COLLECTED WORKS

OF

FRANCIS SIBSON, M.D.

VOL. IV.

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OF

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THE PHYSICAL SIGNS OF RHEUMATIC PERICARDITIS.

In every case of rheumatic pericarditis there is an increase in the amount of fluid in the pericardium, and a layer of ridged, roughened, or honeycombed lymph is spread over the opposing surfaces of the heart and the pericardial sac. The amount of the fluid poured into the sac is made known by the extent of dulness on percussion, the prominence of the sternum and costal cartilages, and the widening of the intercostal spaces over the region of the pericardium, and by the position of the impulse ; while the presence of lymph covering the heart and lining the sac is told by a friction-sound.

Effusion of Fluid into the Pericardium in Rheumatic Pericarditis.—Although in the prescribed order, the examination of the chest by the eye and the application of the hand rightly precede that by percussion, I shall here reverse this order, and begin with percussion, for by it we really judge of the extent of the fluid in the sac.

The pericardium of an adult man with a healthy heart is capable of holding from fourteen to twenty-two ounces of

fluid, and that of a boy of from 6 to 9 years old, about six ounces, when the sac is distended to the full by injecting water

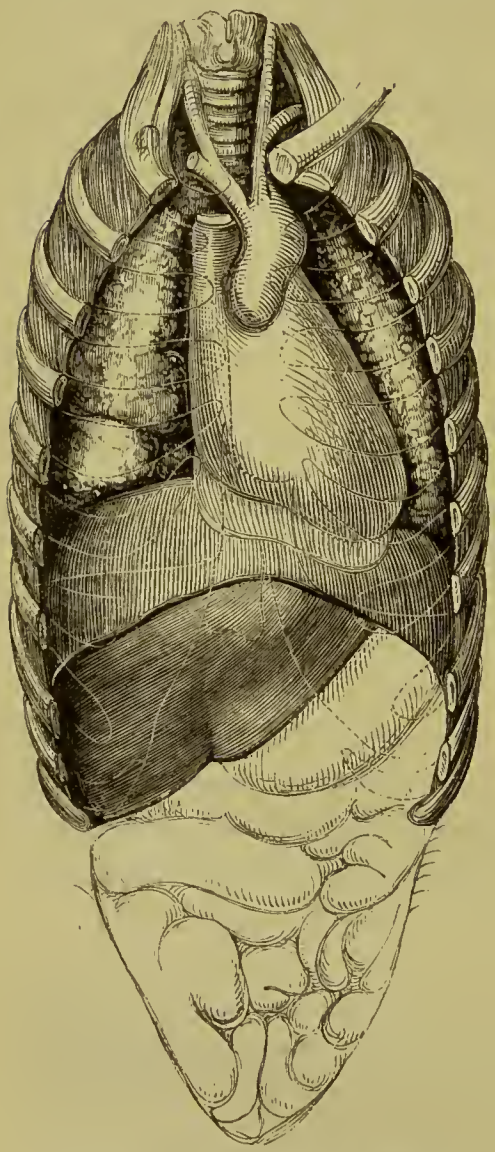


FIG. 1.—Pericardium not distended.

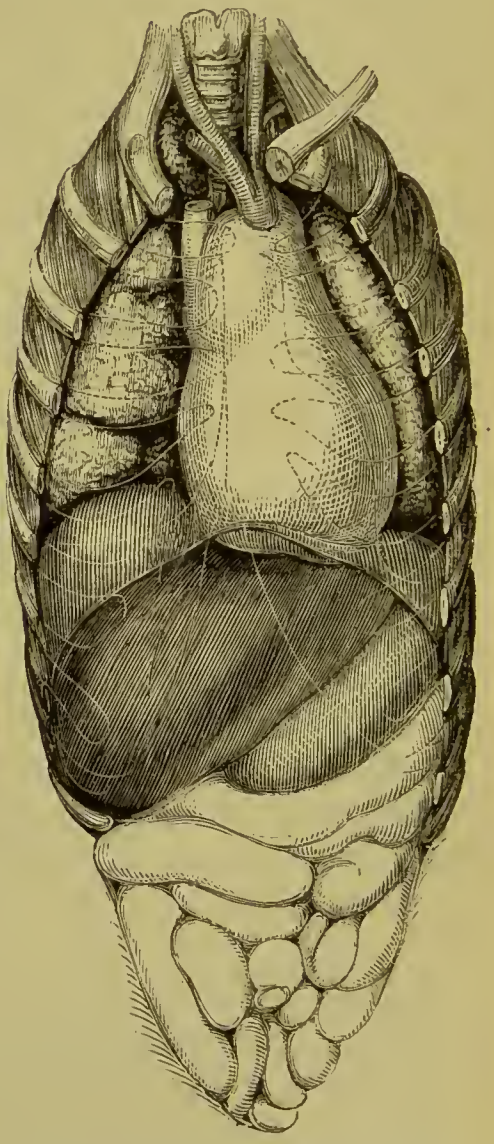


FIG. 2.—Pericardium artificially distended with fifteen ounces of fluid.

into it by a syringe, through an opening made in the anterior wall of the pericardium.

The effect of this artificial distension of the pericardium on the size, form, and position of the sac and on the situation of the surrounding parts is shown in the accompanying figures (1, 2). The pericardium, thus distended, is pyramidal or pear-shaped. It is formed, so to speak, of a larger and a smaller sphere, the smaller one resting on the top of the larger. The larger and lower sphere contains the heart, the ascending vena cava, and the pulmonary veins; and the smaller sphere holds the great vessels. The distended sac occupies the whole centre of the chest, filling up the space between the sternum in front and the spinal column behind; and extending across the chest from a little within the right nipple to a little beyond the left nipple. The whole sac is lengthened; its smaller end reaches upwards almost to the top of the sternum; and its floor, being formed by the central tendon of the diaphragm, presents a large spherical prominence that bulges downwards into the abdomen, occupies the epigastrium, and reaches as low as the tip of the ensiform cartilage and the lower edge of the sixth costal cartilage. The enlarged and swollen sac displaces all the organs and parts surrounding it. In front it separates the two lungs from each other, so as to uncover the pericardium in front of the heart and great arteries. It pushes forwards the two lower thirds of the sternum, the ensiform cartilage, and the adjoining costal cartilages, especially the left, from the third to the sixth; and by counter-pressure backwards it compresses the œsophagus, the descending aorta, the bifurcation of the trachea, and the left bronchus between itself and the bodies of the vertebræ upon which those parts rest. It displaces the lungs to either side and backwards; and the central tendon of the diaphragm where it forms the floor of the pericardium, the stomach, and the left lobe of the liver downwards.

The artificial distension of the pericardium closely corresponds in general form with its natural distension from pericarditis, when the amount of the effusion has reached its acme. I have already sketched [at page 282, vol. iii.] what I believe to be the usual course of the increase of the effusion from the beginning of an attack of pericarditis to the period of its acme. When, however, the inflammation of the pericardium has existed for some time, the walls of the sac, so thin, tough, and firm in health, become comparatively thick, soft, and yielding; and as the sac cannot expand to a material degree either upwards towards the neck, or downwards towards the abdomen, it yields sideways and backwards, and widens to the right and especially to the left, so as to encroach on both lungs, but more seriously on the left lung; as may be seen in the accompanying figure, which is taken from a case of chronic pericarditis of long standing, in which the sac contained three pounds and a quarter of fluid (fig. 3). When thus distended, the sac seems to occupy the whole front of the chest; and it completely conceals the left lung, which is pushed backwards and compressed by it so that comparatively little air is admitted into that lung at its lower and posterior part; this effect being increased by the compression of the left bronchus.

There is another effect of this distension of the pericardium to which I have already alluded, its inferred effect, namely, upon the heart itself. The muscular walls of the ventricles are so thick, and their action is so powerful, that the direct effect of the fluid pressure upon them cannot be very great. But the pressure of the fluid tells inwards upon the weak and unresisting walls of the auricles, the vena cava descendens within the pericardium, and the pulmonary veins, so as to compress and lessen those vessels and the auricles, and to resist and impede the currents of blood, on the one hand from

the system along the cava, and on the other from the lungs along the pulmonary veins. This partial blocking of the double stream from the system and the lungs to the heart lessens the contents of the organ, and tends to diminish the size of its cavities. At the same time the supply of blood to

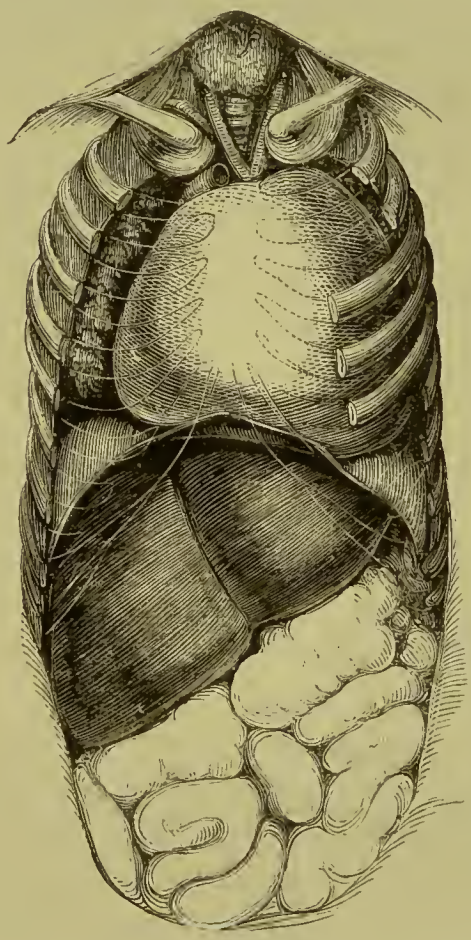


FIG. 3.—Case of pericarditis in which one sac contained 3¼ lbs. of fluid. The patient was under the care of Sir James Alderson.

the aorta is lessened, and the ascending aorta is therefore also compressed by the fluid. The pulmonary artery, however, owing to the obstacle to the flow of blood through the lungs, tends to resist the pressure of the fluid in the swollen sac, and to remain distended.

While, however, this influence on the part of the fluid pressure of the distended pericardium is at work compressing the auricles and veins, a second influence is at work, also set up by the inflammation, to counteract the first influence, and to shield to some extent the weaker parts of the heart. The auricular appendices shrink at an early stage, and the walls of the auricles and veins are thickened and somewhat protected from the pressure of the effused fluid by a leathery coat of mail in the shape of the roughened and honeycombed coating of lymph that clothes and strengthens the feeble natural walls of those parts. Thus the double march of the inflammation supplies at the same time a compressing fluid and a sustaining covering of lymph.

The distension of the pericardium with fluid produces two other effects on the heart. 1. The heart is heavier than the fluid in which it plays, and its ventricles consequently tend to sink backwards, so that the left ventricle rests upon the posterior wall of the pericardium, just as the liver sinks backwards when the abdomen is distended with fluid in cases of ascites. 2. The other effect of pericardial distension on the heart is the lifting or tilting upwards of the organ within the sac. The heart is attached by its great vessels to the posterior and upper parts of the sac, and the whole organ, therefore, tends to shrink upwards and gravitate backwards towards its points of attachment. At the same time the accumulating fluid which occupies in volume the space between the lower surface of the heart and the central tendon of the diaphragm, displaces the organ upwards into the higher part of the pericardium.

The natural effect of this gravitation, shrinking, and upward displacement of the heart, owing to the great accumulation of fluid in the sac, would be, I conceive, if not modified by other agencies, to cause a layer of fluid to be interposed

between the front of the heart and the anterior walls of the chest. Practically, however, we find that this is not usually the case over the mass of the ventricles; for with one or two rare exceptions we can always feel the impulse of the heart beating, sometimes with force, sometimes with a thrill, in the second and third, or third and fourth left spaces, extending from the edge of the sternum to above and beyond the nipple. A layer of fluid is, however, evidently interposed between the lower portion of the front of the heart and the anterior walls of the chest.

The reasons for the presence and pulsation of the heart in the upper intercostal spaces when the pericardium is distended, I believe to be, firstly, the distension of the pulmonary artery, and, to a less extent, of the right ventricle, owing to the difficulty with which the blood flows through the lungs; and secondly, the raised position of the heart, which having left the broader space of the chest below, where it enjoyed free play, occupies its narrower space above, where the heart and pericardium are as it were grasped between the walls of the chest in front and the bodies of the vertebræ behind. The result is that under the combined influence of the elevation of the heart; the distension of the pericardium; and the contracted area of the upper part of the chest in which the heart is lodged, the left lung is displaced from before the organ and the right and left ventricles, and the apex and the great arteries beat against the higher intercostal spaces with which they come into direct contact. In consequence of the withdrawal of the lung from before the heart, and the narrowing compass of the portion of the chest in which the organ is situated, its impulse, besides being raised, is also widened outwards, so that the apex beats against the third or fourth space, at or above the level of the left nipple, where it extends beyond the nipple line.

Although the upper portion of the right ventricle is in immediate contact with the walls of the chest, I am satisfied that a portion of the fluid effused into the sac is interposed between those walls and the lower portion of the right ventricle over its anterior surface.

We shall afterwards see that the impulse is raised in position when the fluid in the pericardium increases, and is lowered in position when that fluid diminishes, so that under these circumstances the varying amount of the fluid is told by the varying position of the impulse.

Cases, included in the following tables, that form the subject of this inquiry into the Physical Signs of Pericarditis.—I possess notes of 44 of my 63 cases of rheumatic pericarditis, of the increase, acme, and diminution of the quantity of fluid in the pericardium, as shown by the enlarging and lessening area of the dulness on percussion over that region ; the progressive changes in the position of the impulse ; and the variations in the tone, intensity, and area of the friction-sound ; all of which signs are at once the effects and the witnesses of the advance and decline of the inflammation. I have arranged these 44 cases in columns in the accompanying tables (see pages 17—32), so that day by day each of those parallel effects of the disease may be seen either singly or in comparison with each other; and have combined with them the co-existing endocardial murmurs, the presence of pain over the region of the heart and elsewhere in the chest, and the affection of the joints ; and I shall now briefly analyse, point by point, these parallel effects in those cases.

PERCUSSION.

The enlarged Area of Dulness on Percussion over the Pericardium, caused by the Increase of Fluid in the Sac.—In 22 of the 44 cases under examination the increased amount of fluid in the pericardium, as indicated by the extended area of dulness over that region, had already at the time of its first observation reached its acme, and from that time, the amount of fluid with its area of dulness steadily declined. One of these cases had a relapse and proved fatal on the 14th day. In the remaining 22 cases the period of the greatest distension of the sac was preceded by a gradual increase, and was followed by a more gradual decrease, in the amount of the fluid; the periods of increase, acme, and decrease of the amount of fluid, being shown by the corresponding gradual enlargement, greatest area, and lessening of the region of dulness on percussion over the pericardium. In 11 of these 22 cases there was a single rise and fall of the tide of the effusion; but in the 11 remaining cases there was a relapse, and the amount of effusion, after lessening considerably, again increased and attained to a second acme. In five of those cases, indeed, there was a second relapse, so that the fluid in the pericardium presented a third, and in one of them even a fourth wave of increase.

The duration of the whole period of increase of dulness on percussion over the region of the pericardium varied much in different patients. Of the 22 cases in which the region of dulness had attained to its greatest area at the time of the first observation, the average duration of the increased dulness from the effusion into the pericardium was eight days, the extreme duration varying from three days on the one hand, to

seventeen on the other. The average duration of the period of increased dulness in the 11 cases in which there was a gradual increase, single acme, and a decrease in the amount of fluid effused into the pericardium, amounted to fully eight days, the extreme variation ranging from four to thirteen days. The average duration of the whole period of increased pericardial dulness was more than twice as long in the 11 cases of relapse as in the cases with a single acme, since in them it amounted to eighteen days, the extremes varying from fourteen to twenty-four days.

The increase of fluid in the early stage was usually rapid. In one-half of the cases in which this increase was watched, the area of dulness had reached its maximum on the second or third day after the first observation (11 in 22), and in all but two or perhaps three of the remainder, on the fourth or fifth day. The early advance of the dulness was, as a rule, more slow in those patients who suffered from a relapse than in those who did not do so.

The time during which the effusion into the pericardium remained at its height was, as a rule, very short. In 39 of the 44 cases the region of dulness extended over its greatest area for about a single day. It may have lasted longer, but on the next examination, made usually on the following day, but sometimes later, the tide had turned and the extent of dulness had lessened. The acme of the pericardial dulness lasted two days in three of the remaining cases, and three days in two of them.

The period of the decrease of the effusion in the pericardium was much longer than that of its increase, its average duration having been, as we have already seen, eight days in the 22 cases in which the effusion was at its acme on the first examination.

We thus see that the period of the advance of the effusion,

dating from the time of its first observation in the early stage, usually lasted about three days ; that the period of the acme of the effusion was usually observed during only one day ; and that the period of the decline of the effusion generally lasted about eight days.

The fluid in the pericardium begins to increase on the first day of the inflammation ; but, as it necessarily gravitates backwards during the early stages, the effusion does not appear in front until it has accumulated so as to occupy the natural hollow at the back of the sac, and the space between the lower surface of the heart and the floor of the pericardium. Dulness on percussion over the region of the pericardium therefore does not declare itself until the inflammation has lasted for a day or two. I have no exact indications telling how soon the fluid advances to the front of the heart in sufficient quantity to push aside the lungs. That it must, however, have been rapid in certain cases is I think shown by the following instances :—

The effusion had reached its acme in one patient three days after the beginning of the attack of acute rheumatism ; and the increased cardiac dulness was observed for the first time on the fifth or from that to the seventh day after the beginning of the illness in nine cases. Pain attacked the heart in three cases the day before, and in one three days before the first appearance of increased dulness over the pericardium ; and from one to four days before the effusion had reached its acme in eight other cases.

Friction-sound, like increased pericardial dulness, is not present at the first blush of pericarditis, and in my cases the two signs usually appeared at the same time. Thus they did so in 16 of the 22 cases in which the dulness on percussion was detected in the early stage ; while in only one of those cases did the first brush of the friction-sound precede, and in

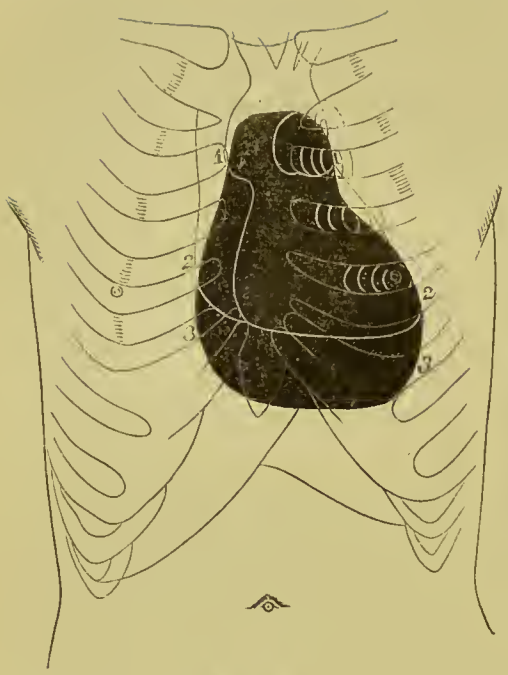


FIG. 4.

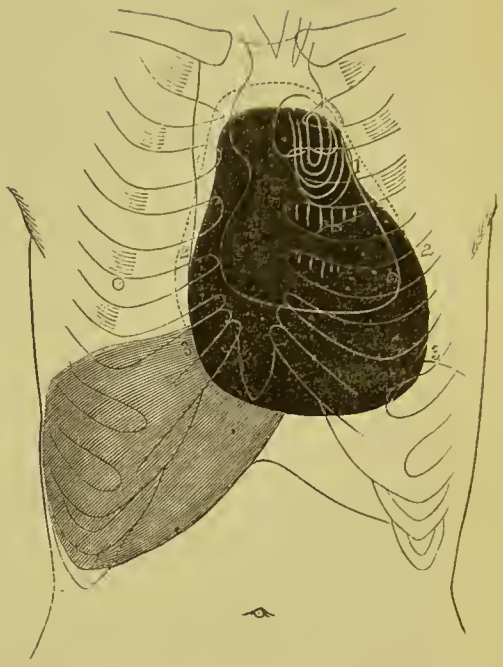


FIG. 5.

Figure 4, from a youth aged 17, affected with rheumatic pericarditis, who recovered in nine days from the time of his admission.

Period of the rapid increase of the effusion into the pericardium, just before the occurrence of its acme. The effusion completely distended the sac.

Day of admission.

The pericardial effusion distends, lengthens, and widens the sac, to the same extent and with the same effect as when the healthy pericardial sac is artificially distended with fluid (see figs. 1, 2, p. 2). The swollen pericardium is pyramidal or shaped like a pear, as in figure 2. Its smaller and higher portion (1, 1,) contains the great arteries; and its larger portion is occupied above (2, 2,) by the heart, and below (3, 3,) by the great volume of fluid which accumulates between the under surface of the heart and the floor of the pericardium. The distended pericardium displaces the lungs upwards, and to each side; and the diaphragm, liver, and stomach downwards: and the fluid in the sac compresses the auricles; and that in the lower portion of the sac, between the under surface of the heart and the floor of the pericardium, elevates the heart. Owing to the displacement of the lungs from before the pericardium, the whole of the anterior surface of the heart and great arteries is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the ascending aorta within the pericardium and the pulmonary artery; and, owing to the elevation of the heart by the fluid, that organ presses and rubs with increased force against the walls of the chest in front of it; the anterior surface of the

the remaining five it followed the onset of the increased pericardial dulness.

The upper boundary of the pericardial dulness when first

heart at its lower portion is, however, separated from the sternum and cartilages by a thin layer of interposed fluid.

This explanation, and that which follows, given once for all, will apply to figures 5, p. 12; 8, p. 44; 10, p. 48; 13, p. 64; and 16, p. 110, which represent, each of them the single, or first or second acme of the pericardial effusion.

There is *prominence over the region of the pericardium*, the left costal cartilages and ribs from the third to the eighth being raised and moved outwards.

The region of dulness on percussion over the distended pericardium, ("pericardial dulness," see the black space,) indicates the extent of the pericardial effusion; has the pyramidal or pear-shaped form of the distended sac; and extends from a little above the lower end of the manubrium, where it displaces the lungs, down almost to the tip of the ensiform cartilage, where it intrudes on the epigastrium. The lower and larger portion of the region of pericardial dulness over the heart and the great body of the effusion is more than twice the width of its upper and smaller portion over the arteries. This narrow upper portion forms therefore a peak which gives to the region of pericardial dulness its pear-shaped form, and which rises high behind the sternum, and occupies the lower portion of the manubrium. The wider portion of the region of pericardial dulness bears chiefly to the left; and its upper border, starting from the foot of its narrower portion, is on a level with one of the higher left costal cartilages or spaces. The upper and left boundary of the region of pericardial dulness is therefore indented; and its upper border is much higher behind the manubrium than behind the adjoining left costal cartilage or space that may form its higher limit. The higher and narrower region of pericardial dulness (1, 1,) over the ascending aorta and plumonary artery, is about two inches in width, and is situated behind the sternum, on a level with the first and second spaces, and for about half an inch to the left of it in those spaces. The lower, larger and wider region of pericardial dulness that extends over the heart itself (2, 2,) and over the accumulated fluid that occupies the depending portion of the sac below the heart, and that bulges downwards into the epigastric space (3, 3,), extends from the upper edge of the third left costal cartilage, and the corresponding portion of the sternum, down to the lower edge of the sixth left cartilage, and almost to the tip of the ensiform cartilage; and from about an inch to the right of the lower half of the sternum, to half an inch or more to the left of the nipple. The lower border of the fifth cartilage, and a line running thence across the sternum to the fourth right space, *probably* forms the lower boundary of the heart (2, 2,) and the upper boundary of the depending space (3, 3,) occupied by the volume of the fluid distending the pericardial sac.

The *impulse of the heart* occupies the third and fourth left spaces, (see the curved lines in those spaces,) and extends in the latter space to just beyond the nipple; and the pulsation of the pulmonary artery is felt in the first and second spaces

observed, was limited by the space between the third and fourth left cartilages in 11 out of 22 cases, by the fourth cartilage in three cases, and by the third cartilage in seven cases. In one patient only did the dulness on its first observation reach as high as the second space.

to the left of the sternum; where the first impulse is followed by a sharp second stroke which is synchronous with the loud second sound of the pulmonary artery, and which gives the effect of a double impulse, one systolic and gradual, the other diastolic and sharp.

Figure 5, from the same patient as figure 4.

Period of the acme of pericardial effusion.

Third and fourth days after admission.

The explanation of pericardial effusion and dulness given with figure 4, applies also to this figure.

The pericardial effusion, which distended the sac on the day of admission (see fig. 4,) has steadily increased in quantity since then, so that the whole pericardium has become enlarged, and has yielded sideways, and especially to the left; but it has not lengthened from above downwards. In this patient, therefore, the *region of pericardial dulness* (see the black space) during the acme is unusually wide, and especially along its left border; this increased width being fully as great above over the great vessels (1, 1,) as lower down over and below the heart (2, 2, 3, 3). The left boundary of the region of pericardial dulness over the great arteries (1, 1,) extends about an inch to the left of the sternum, in the first and second spaces; while the left boundary of the large region of pericardial dulness over and below the heart, extends fully half an inch to the left of the mammary line (2, 2, 3, 3). In all other respects, except the increase of the dulness to the left, the region of pericardial dulness corresponds with figure 4, taken on the day of admission. The apex of the left ventricle seems in this case to be behind the fourth left rib or space, and the lower boundary of the heart *probably* extends along the upper edge of the fifth left cartilage, and across the corresponding portion of the sternum; the heart having been much elevated by the increase of the fluid, which interposes itself between the anterior surface of the heart at its lower border and the walls of the chest.

The prominence over the region of the pericardium has increased.

The impulse is peculiar; it is felt beating (4th day) from the first to the third left costal cartilages, while the third and fourth spaces are retracted during the systole (see the curved and straight lines in those spaces). These movements give to the impulse the appearance of an undulation. The interposition of the fluid between the apex and lower border of the front of the heart and the walls of the chest has combined with the elevation of the organ to raise the impulse.

For later views of this case see figures 6 and 7, p. 40.

The increase of the region of dulness over the pericardium was sometimes gradual, sometimes rapid. In rare instances the gradual ascent was slow and irregular. As a rule, however, the ascent was rapid.

The contour of the area of dulness on percussion over the pericardium when swollen with fluid in acute rheumatism corresponds very closely with the outline of the sac when distended with water after death. (See figures 1, 2, p. 2.) In a paper in the *Provincial Medical Transactions* I gave illustrations of the area of pericardial dulness in which the boundary lines of the effusion were ascertained with care, and I here give figures of those cases (figs. 4, 5, p. 12; 6, 7, p. 40; 8, 9, p. 44; 10, 11, p. 48); and elsewhere, views taken from a case of pericarditis in St. Mary's Hospital, which show the same point during various stages of the affection. (See figures 12, 13, p. 64; 14, 15, p. 108; 16, p. 110.)

The form of the region of pericardial dulness changes as its area increases, its upper boundary being then on a higher level over the sternum than over the costal cartilages, instead of being, as in health, on the same level. The pericardial dulness, at the same time, extends further downwards in the manner shown in the figures just referred to, so as to intrude on the abdomen, and to replace the liver and stomach to a degree proportionate to the amount of the effused fluid.

When the increase of fluid in the pericardium reaches its height, and the sac is completely distended, the area of dulness over the affected region is pyramidal, or, more exactly, pear-shaped, and it extends over and beyond the heart, and in front of the great vessels. The inner borders of the right and left lungs are pushed to each side by the distended sac, so as to expose the whole of the heart and the great vessels.

The region of dulness over the great vessels extends upwards from the level of the third cartilages, sometimes as high as across the middle of the manubrium, or within an inch of the top of the sternum, but more usually to a little above the junction of the manubrium with the long bone of the sternum, or about two inches below the upper end of the bone. This space of dulness over the aorta and pulmonary artery extends across the whole width of the sternum and reaches some distance to the left of it in the first and second spaces.

The area of the region of dulness over the heart itself and the lower portion of the distended pericardium may extend across the chest from an inch or more to the right of the lower portion of the sternum to an inch beyond the left nipple; and from above downwards from the second cartilage to the lower edge of the sixth cartilage. The extreme measurement from side to side of the whole region of pericardial dulness may vary from four and a half to six inches, and somewhat diagonally from above downwards, from five and a half to seven inches.

The lower portion of the region of dulness, from side to side, for the extent of about two inches from above downwards, is situated below the lower boundary of the heart; and is entirely occupied by the effused fluid, which here, as I have before shown, displaces the heart upwards, and the diaphragm, stomach, and liver downwards, to an extent corresponding to the amount of the effusion.

The width of the region of pericardial dulness in front of the great arteries is usually about two inches, and this region usually ascends above the upper boundary of the heart to an extent varying from one inch to an inch and a half.

This upper region of pericardial dulness, over the great arteries, which is two inches wide, is much narrower than the great region of dulness over the heart itself, which at its

CASES OF RHEUMATIC PERICARDITIS.

EXPLANATION.

1.— ——— means the upper boundary of the region of pericardial dulness, and is placed on a line with the space or cartilage that formed the upper limit of the dulness.

2.— ∧∧∧ means the upper or lower boundary, according to its position, of the region of pericardial friction sound over the sternum, and is placed on a level with the part of the sternum that forms the limit of the region of friction sound. The whole space between these upper and lower boundaries is occupied by the friction sound.

3.— ∧∧∧ means the upper or lower boundary, according to its position, of the region of pericardial friction sound over the costal cartilages and their spaces, and is on a line with the space or cartilage forming the limit of the friction sound. The whole space between these upper and lower boundaries is occupied by the friction sound.

4.— ∪ means the position of the heart's impulse, and is on a level with the space where the impulse was felt.

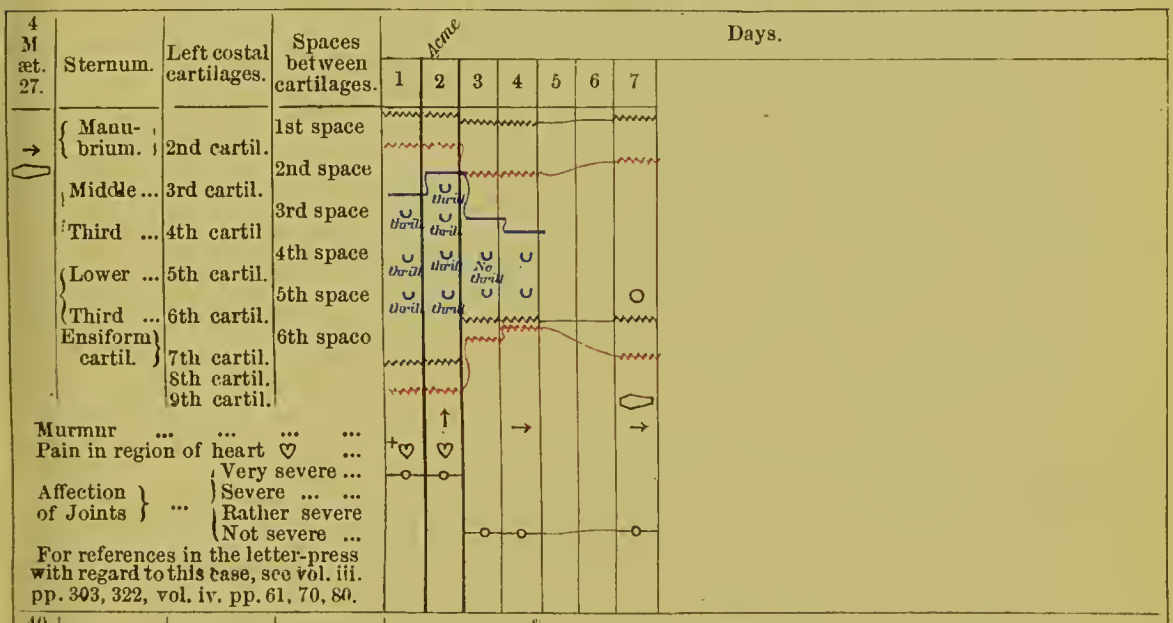
5.—Murmur →, means mitral murmur; ←, tricuspid murmur; ↓, aortic regurgitant murmur; ↑, aortic systolic murmur; ↖, pulmonic murmur; o→ means absence of such murmur.

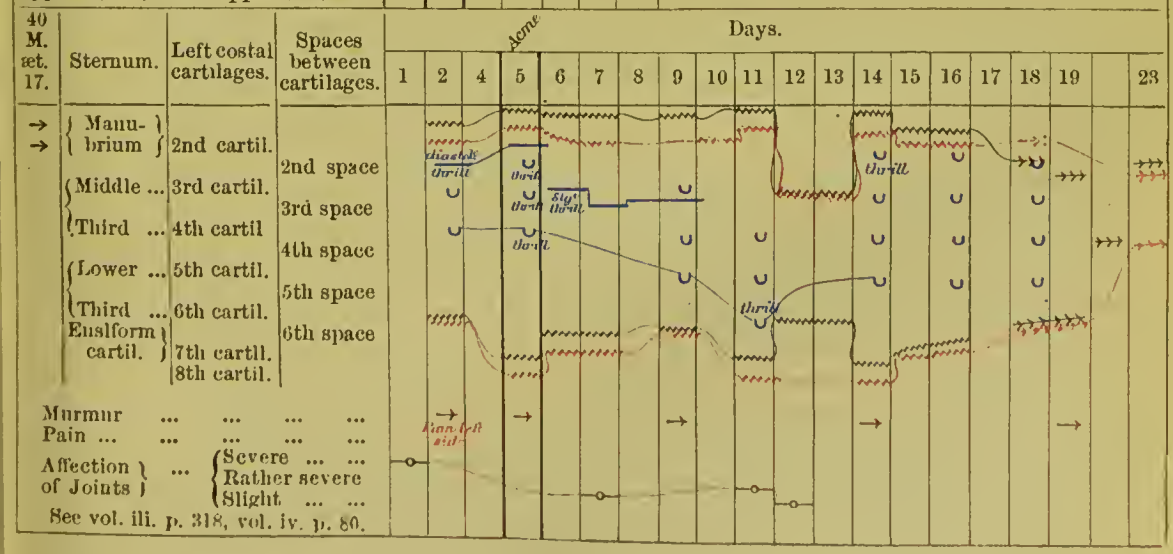
The thin lines merely connect the successive observations with each other, and show that no examination as to the point in question was made on that day.

M., Male: F., Female.

I.—CASES WITH A THRILL OVER THE REGION OF THE HEART OR GREAT VESSELS DURING THE ACME OF PERICARDIAL EFFUSION. (See pp. 70, 72.)

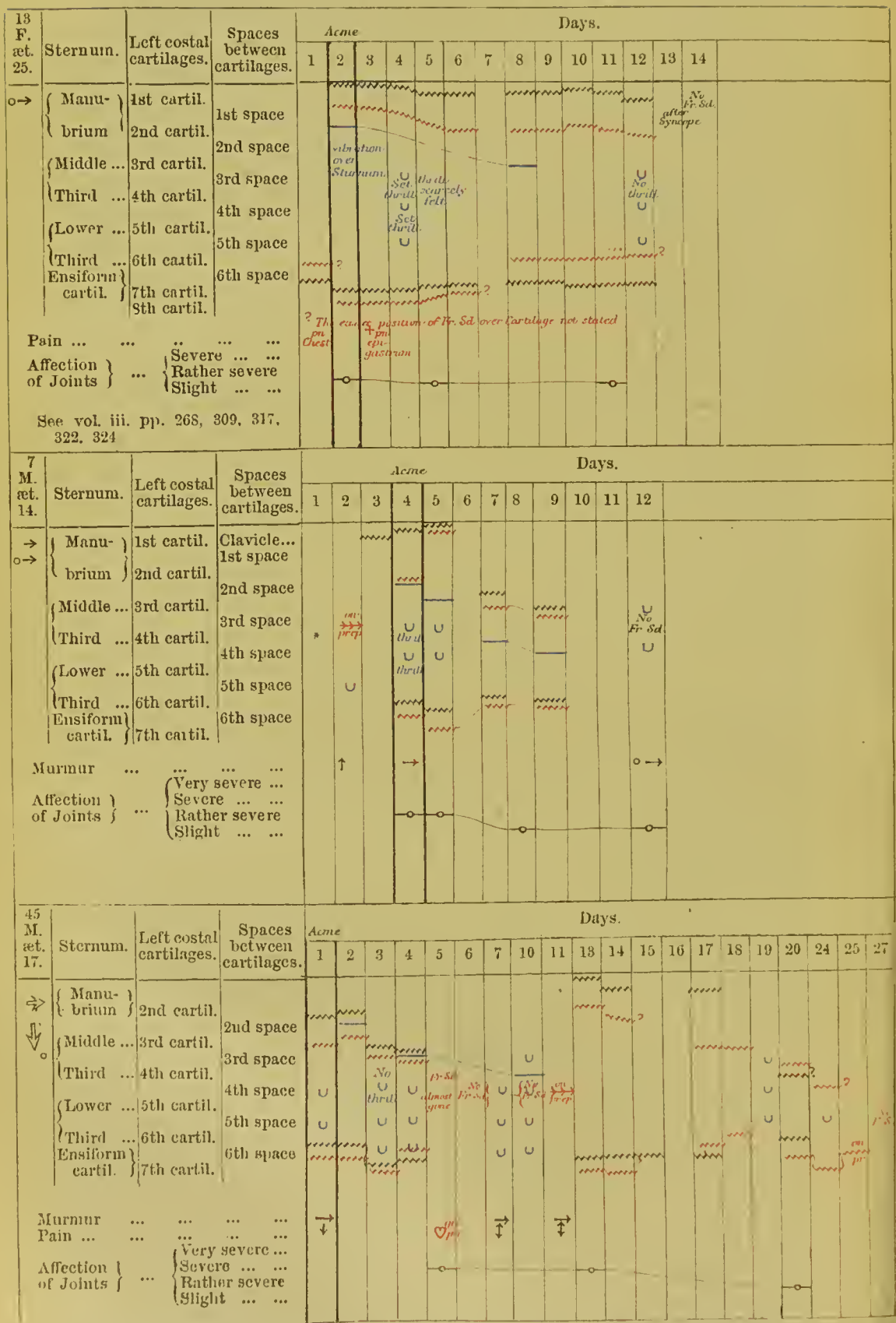
(1.)—*Cases with a creaking friction sound during the acme.* (*See p.* 71.)





For Explanation see p. 17.

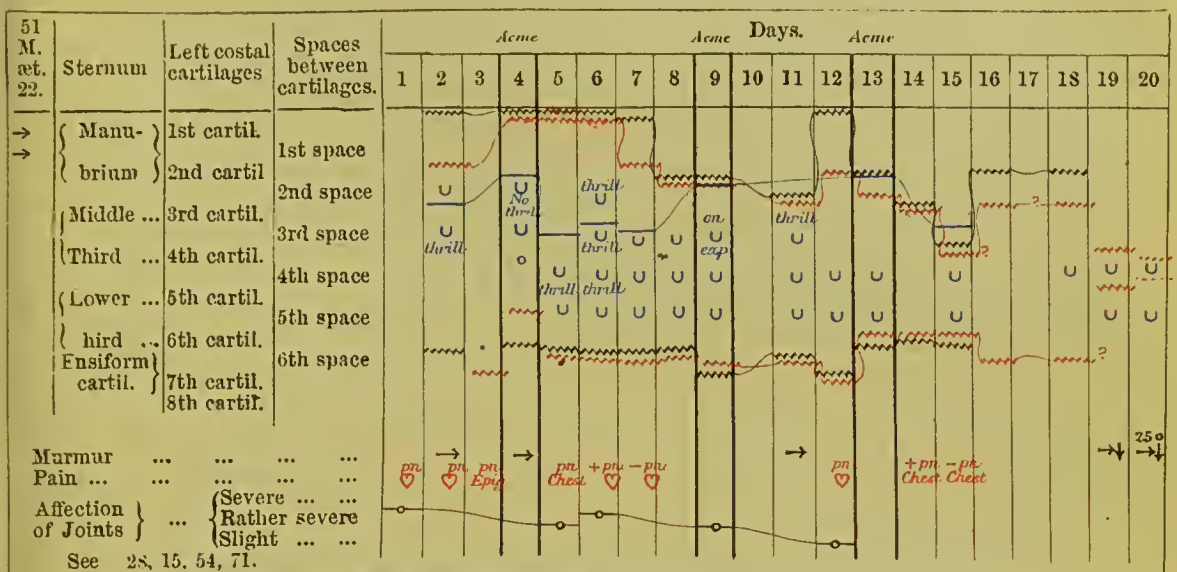
Cases with a thrill.—Cases with a creaking friction sound during the acme (continued).



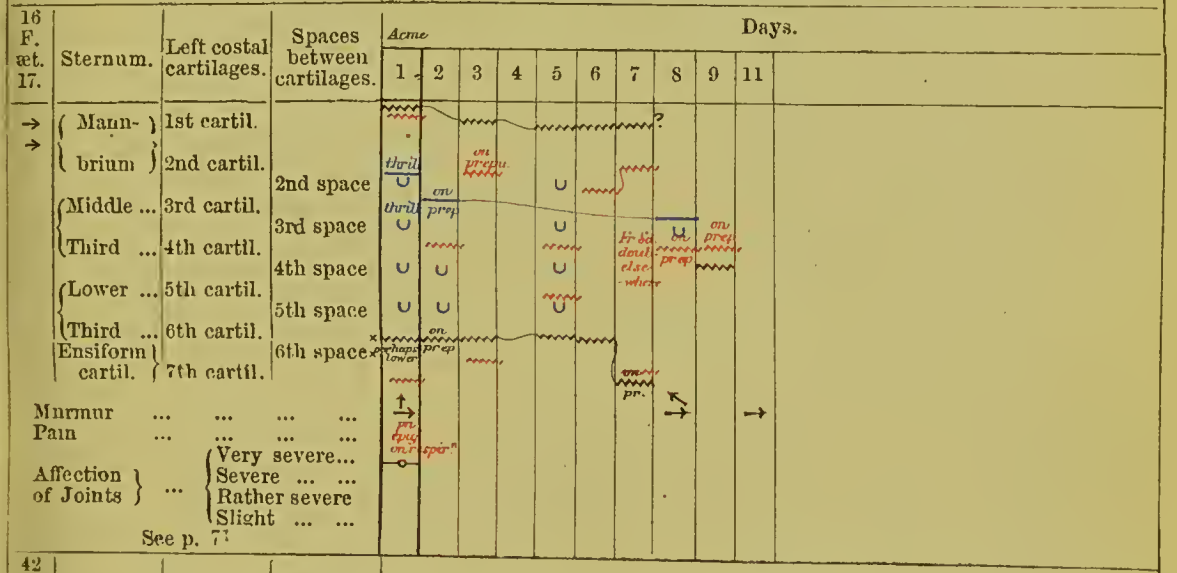
* Here and elsewhere in these tables the word "prep" occurs; sometimes the word "prepura." It is so printed everywhere in the original, and is therefore retained. But on internal evidence it appears to be a misprint for "pressure" variously shortened. This at least "makes sense"; while no interpretation that I can suggest will make sense out of "prep" or "prepura."—Ed

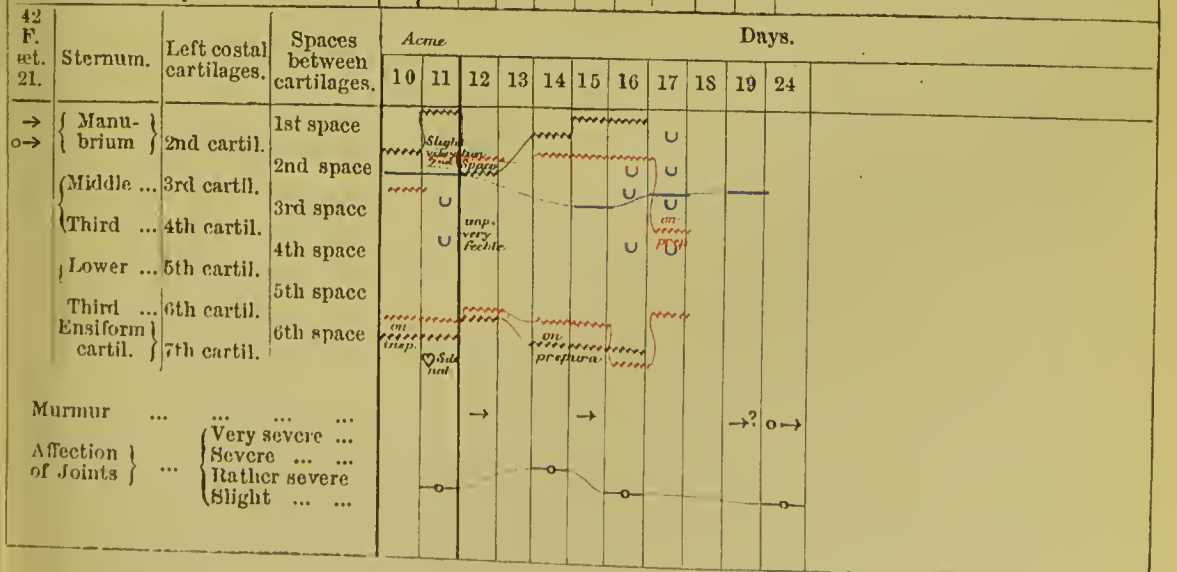
For Explanation see p. 17.

Cases with a thrill (continued). (2).—*Case with a grating friction sound during the acme.* (*See p. 71.*)
(*Case with a treble acme.*)



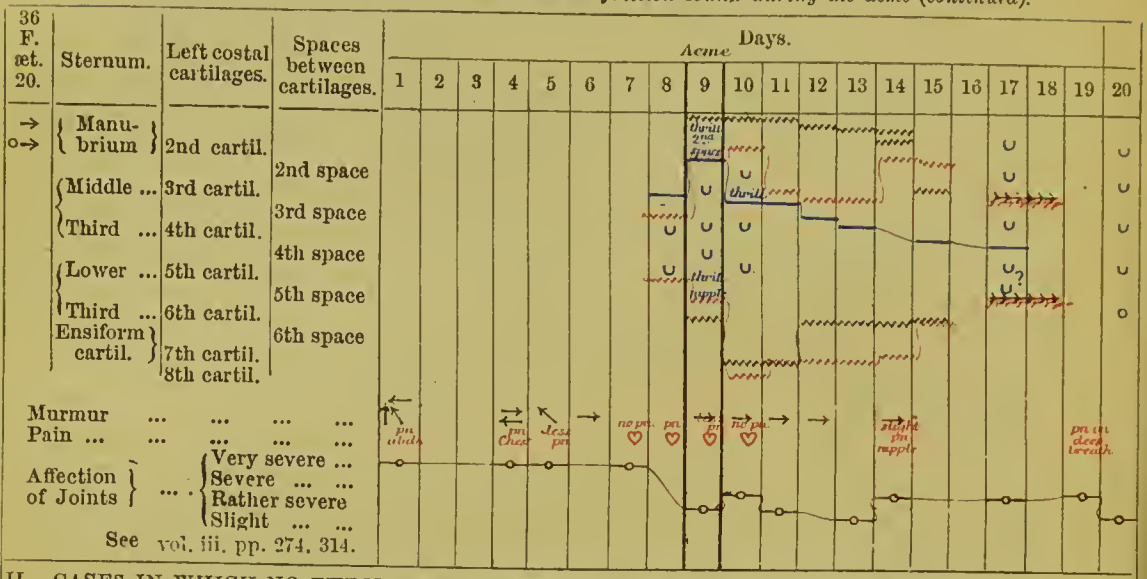
(3.)—*Cases with a harsh double friction sound during the acme.* (*See pp. 71, 72.*)





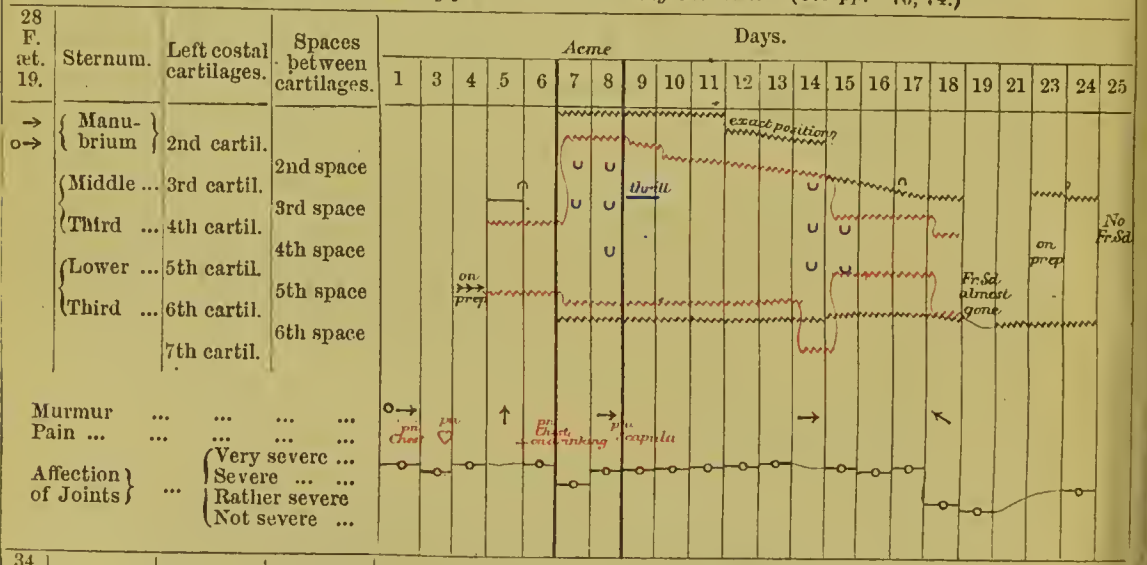
For Explanation see p. 17.

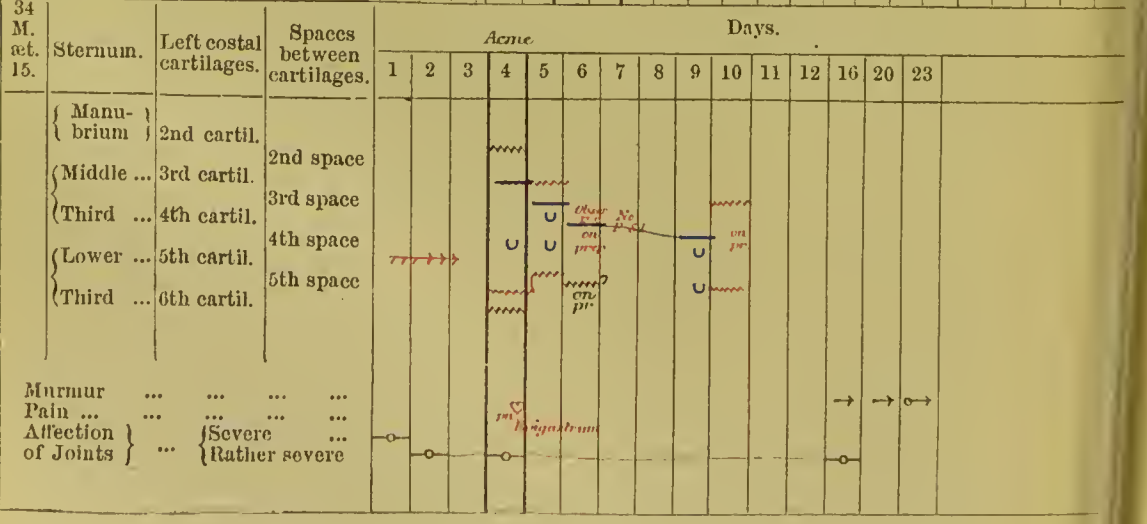
Cases with a thrill.—Cases with a harsh double friction sound during the acme (continued).



II.—CASES IN WHICH NO THRILL WAS OBSERVED OVER THE REGION OF THE HEART DURING THE ACME OF PERICARDIAL EFFUSION. (See pp. 73–81.)

(1.)—*Cases with a creaking friction sound during the acme.* (*See pp.* 73, 74.)





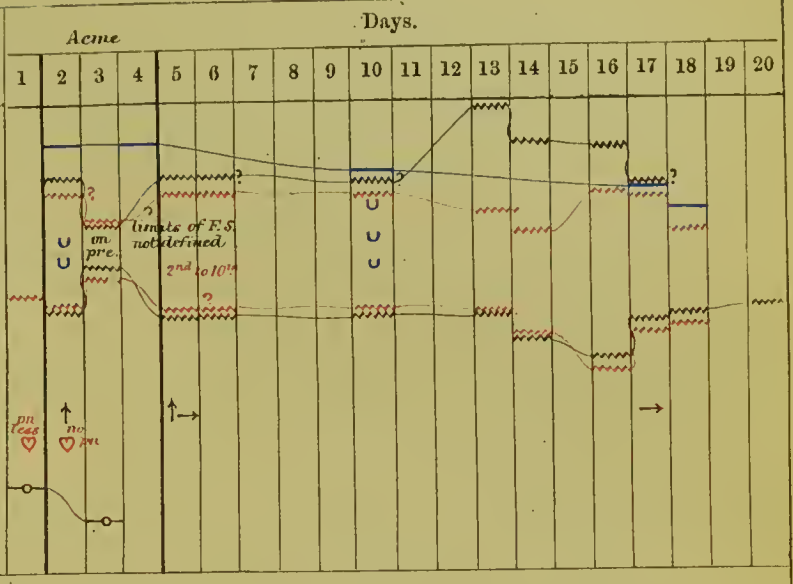
For Explanation see p. 17.

12 F. æt 26.

Sternum.	Left costal cartilages.	Spaces between cartilages.
Manubrium	2nd cartil.	2nd space
Middle ...	3rd cartil.	3rd space
Third ...	4th cartil.	4th space
Lower ...	5th cartil.	5th space
Third ...	6th cartil.	6th space
Ensiform cartil.	7th cartil.	
	8th cartil.	

Murmur ... ...
Pain ... ...
Affection of Joints ... Very severe ... / Severe ... ... / Rather severe / Not severe ...

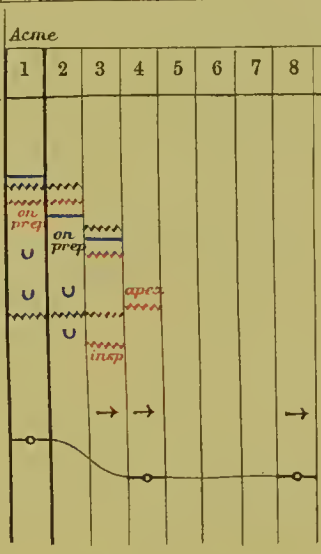
See vol. iii. p. 286, vol. iv. p. 90.



43 M. æt. 18.

Sternum.	Left costal cartilages.	Spaces between cartilages.
Manubrium	2nd cartil.	2nd space
Middle ...	3rd cartil.	3rd space
Third ...	4th cartil.	4th space
Lower ...	5th cartil	5th space
Third ...	6th cartil.	6th space
	7th cartil.	

Murmur ... ...
Affection of Joints ... Severe ... / Rather severe / Slight ...



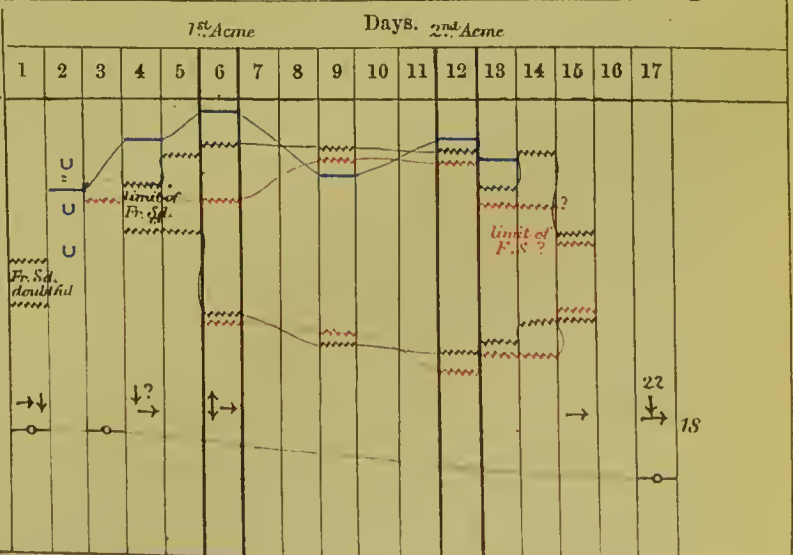
(Case with a double acme.)

25 M. æt. 17.

Sternum.	Left costal cartilages.	Spaces between cartilages
Manubrium	2nd cartil.	1st space
Middle ...	3rd cartil.	2nd space
Third ...	4th cartil.	3rd space
Lower ...	5th cartil.	4th space
Third ...	6th cartil.	5th space
Ensiform cartil.	7th cartil.	6th space
	8th cartil.	

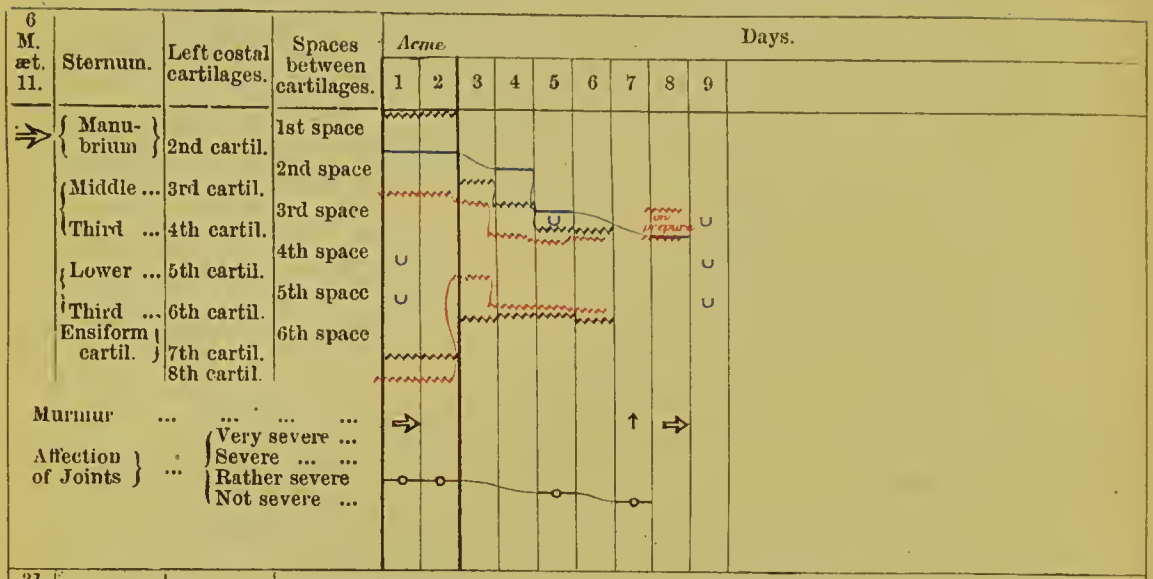
Murmur ... ...
Affection of Joints ... Severe ... ... / Not severe ... / Slight.

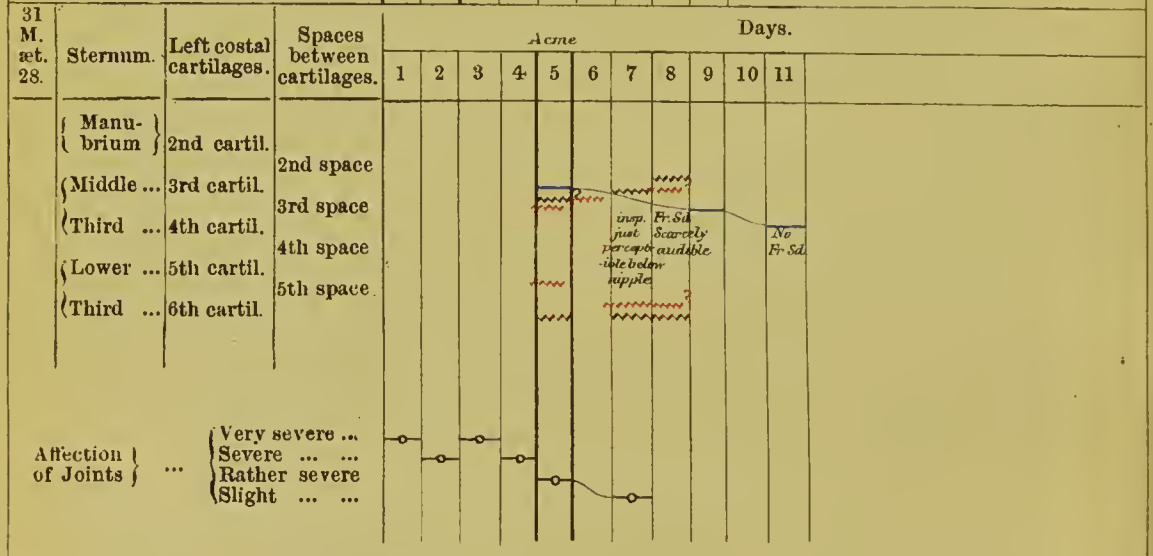
See pp. 15, 92.



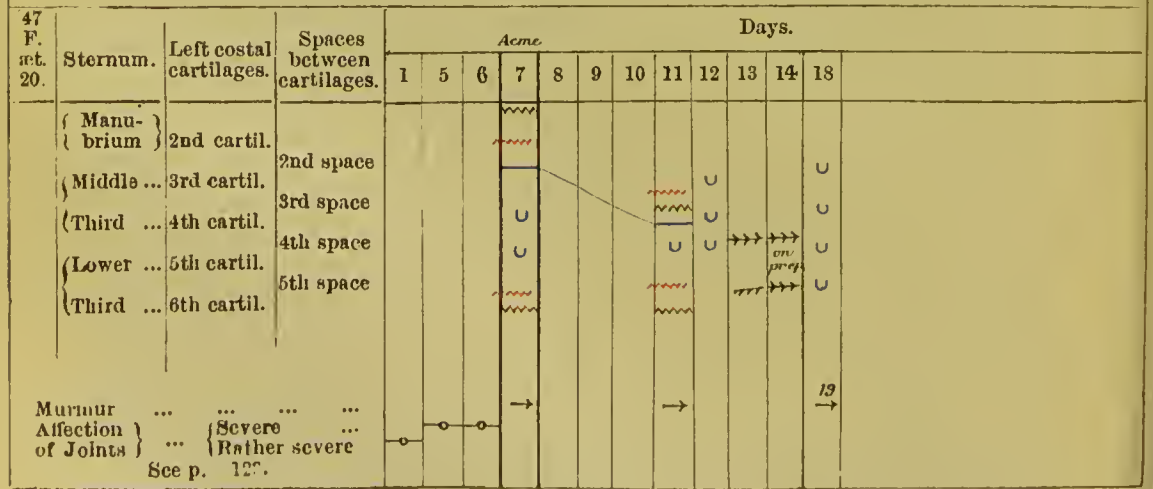
For Explanation see p. 17.

Cases in which the friction sound was almost creaking during the acme.



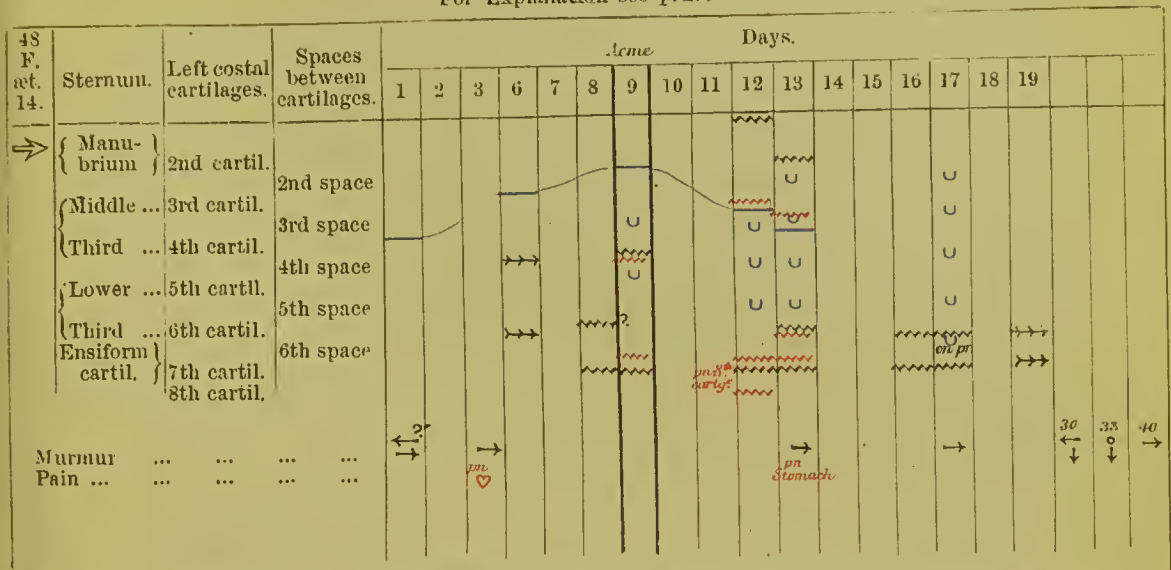


(2.)—*Cases with a grating friction sound during the acme.* (*See p. 74.*)



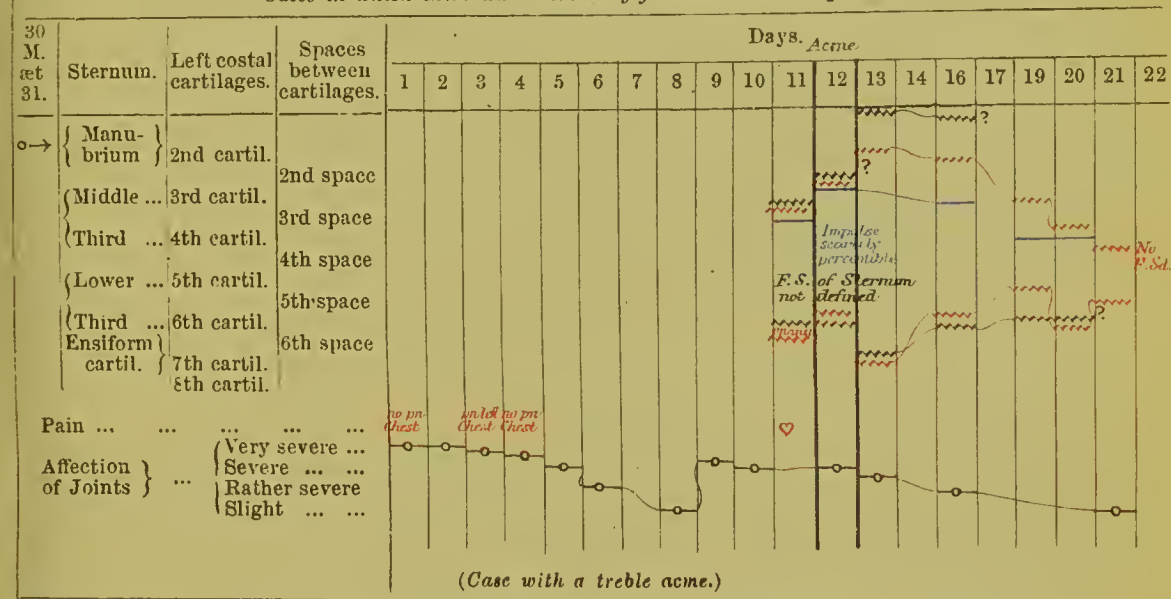
See p. 128.

For Explanation see p. 17.



(3.)—*Cases in which there was a definite double friction sound, usually harsh, during the acme.* (*See pp.* 75–79.)

Cases in which there was a creaking friction sound on pressure.



(*Case with a treble acme.*)

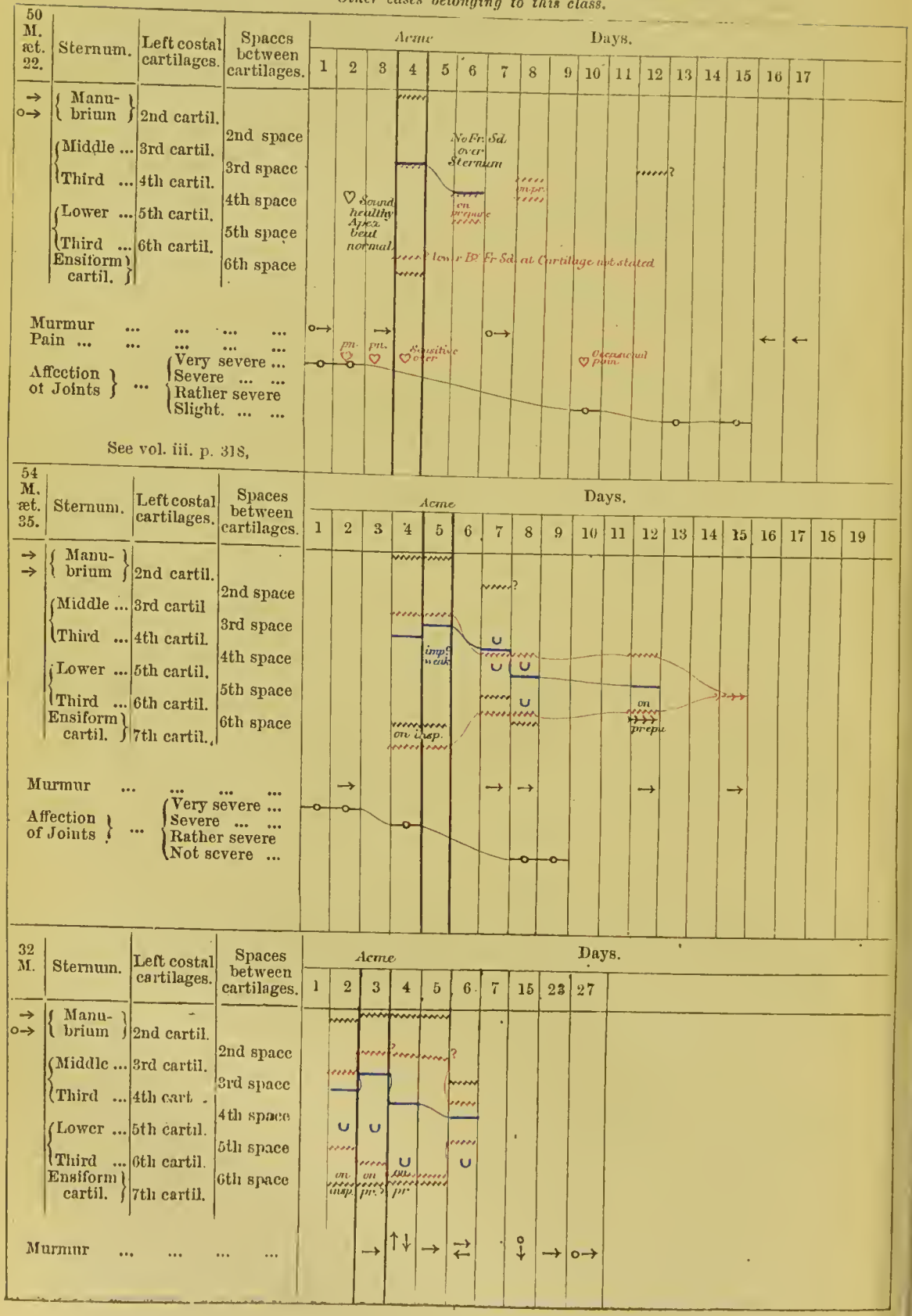
See pp. 15, 62, 98, 108 -110.

*See Figs. 12, 13, p. 80 ; figs. 14, 15, p. 108 ; and fig 16, p. 110

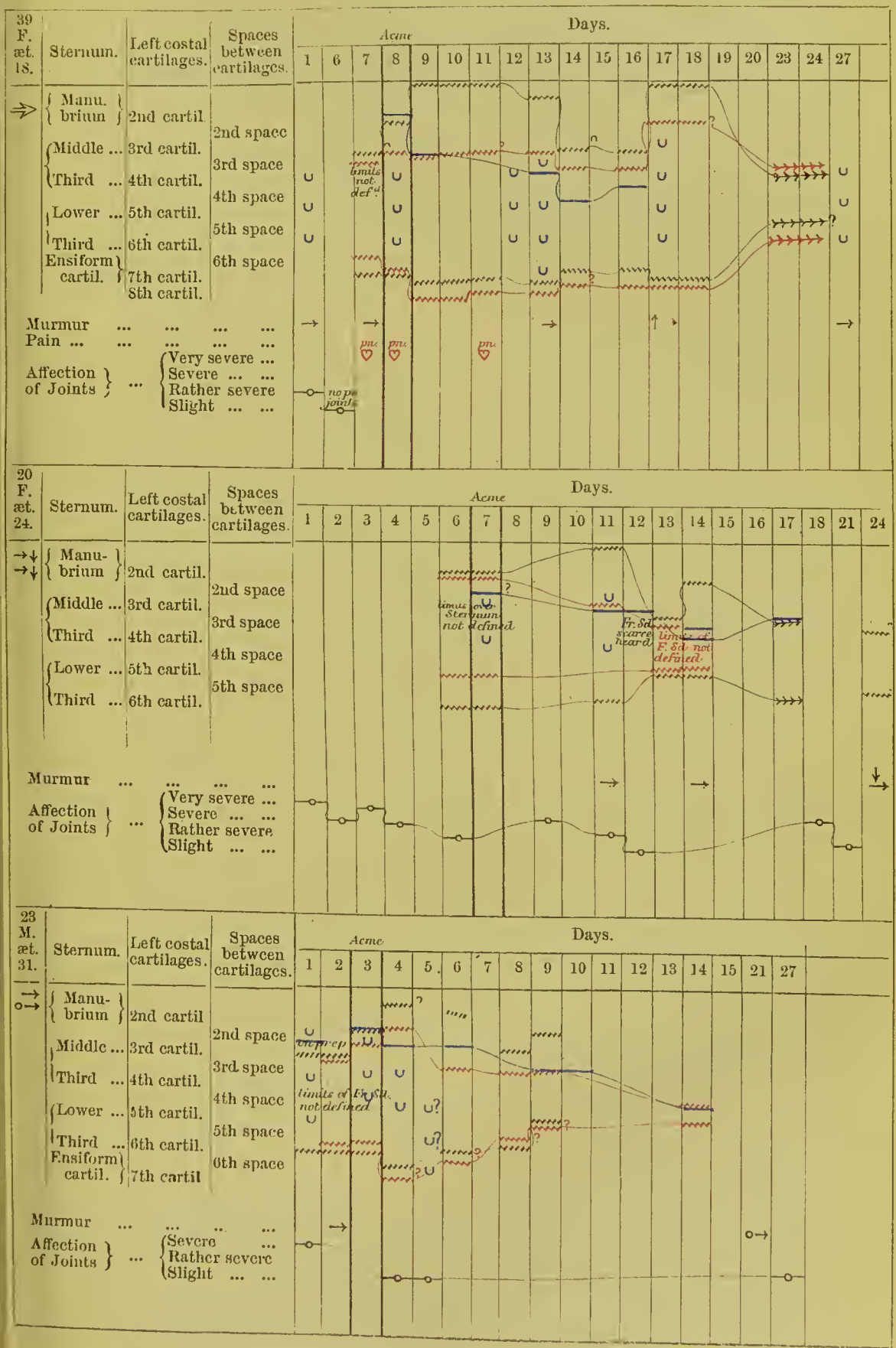
For Explanation see p. 17

Cases in which there was a definite double friction sound, usually harsh (continued).

Other cases belonging to this class.

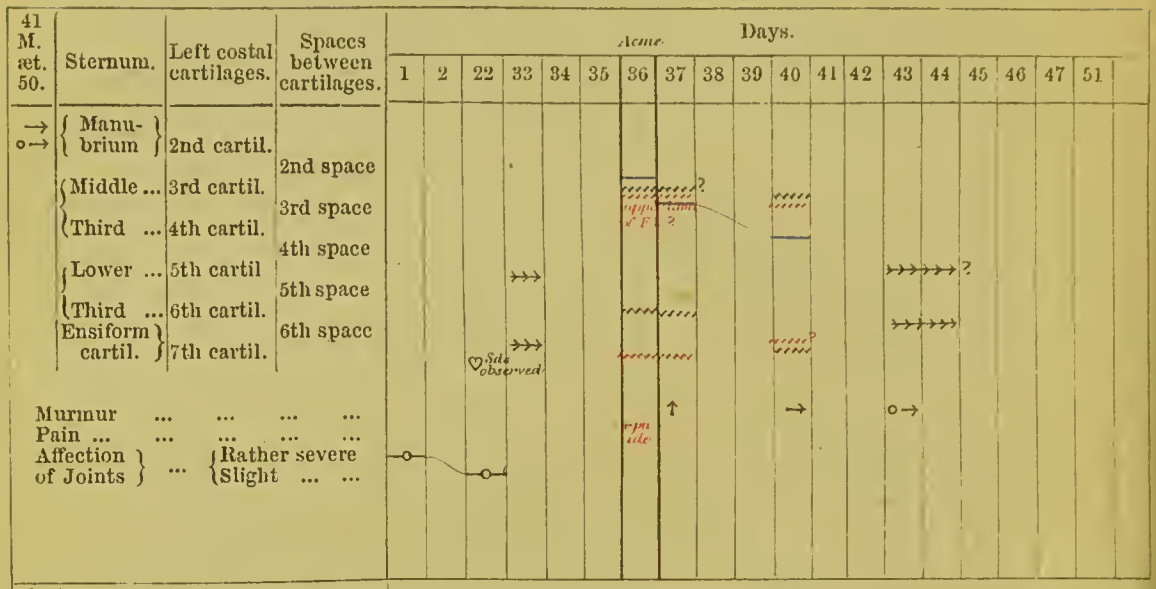


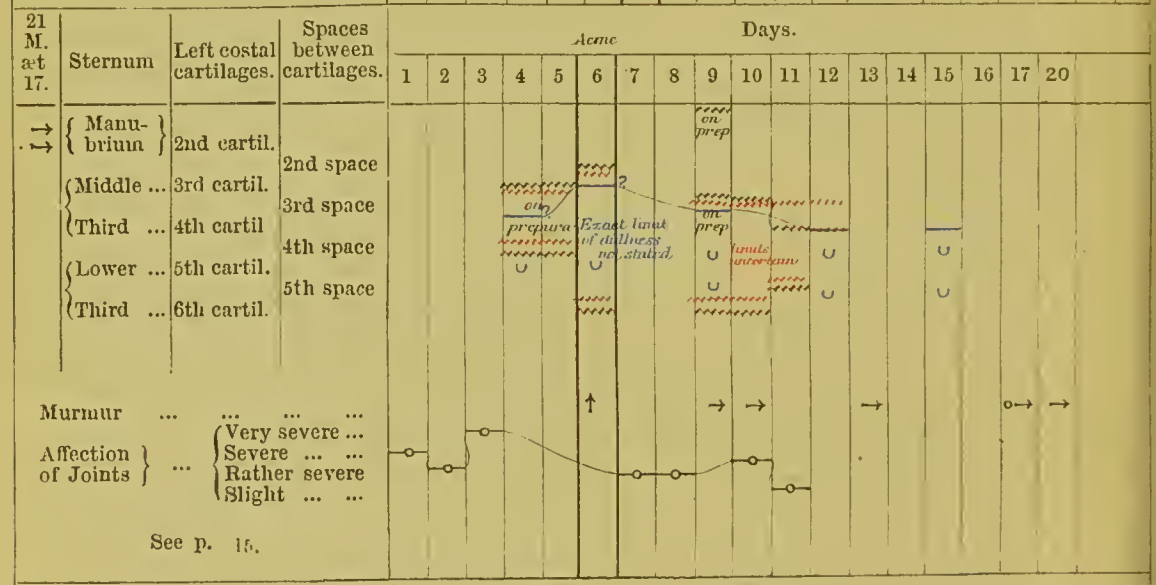
For Explanation see p. 17.



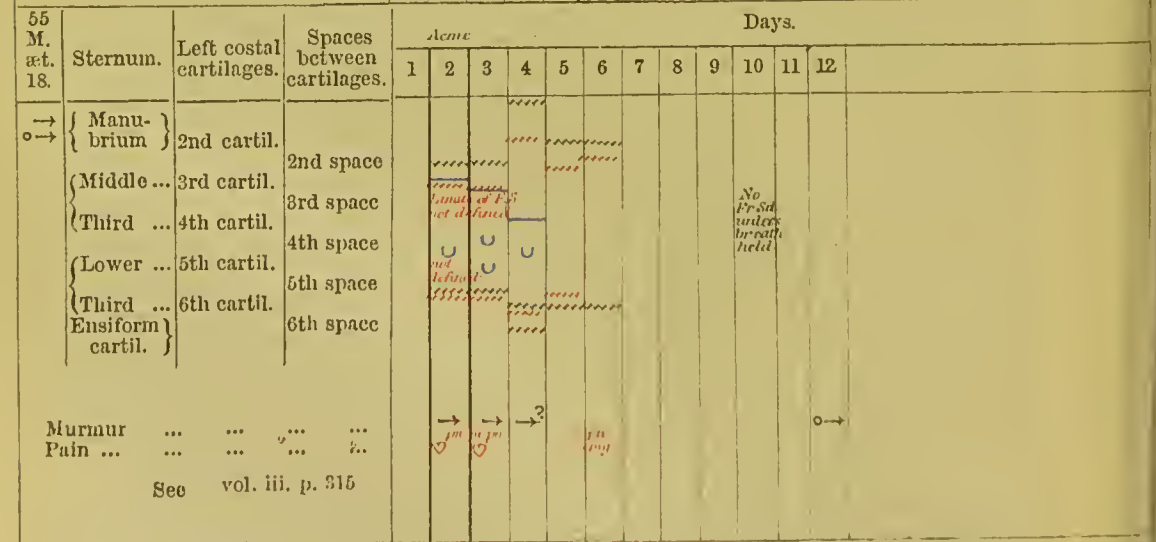
For Explanation see p. 17.

Cases in which there was a definite double friction sound, usually harsh, during the acme (continued).





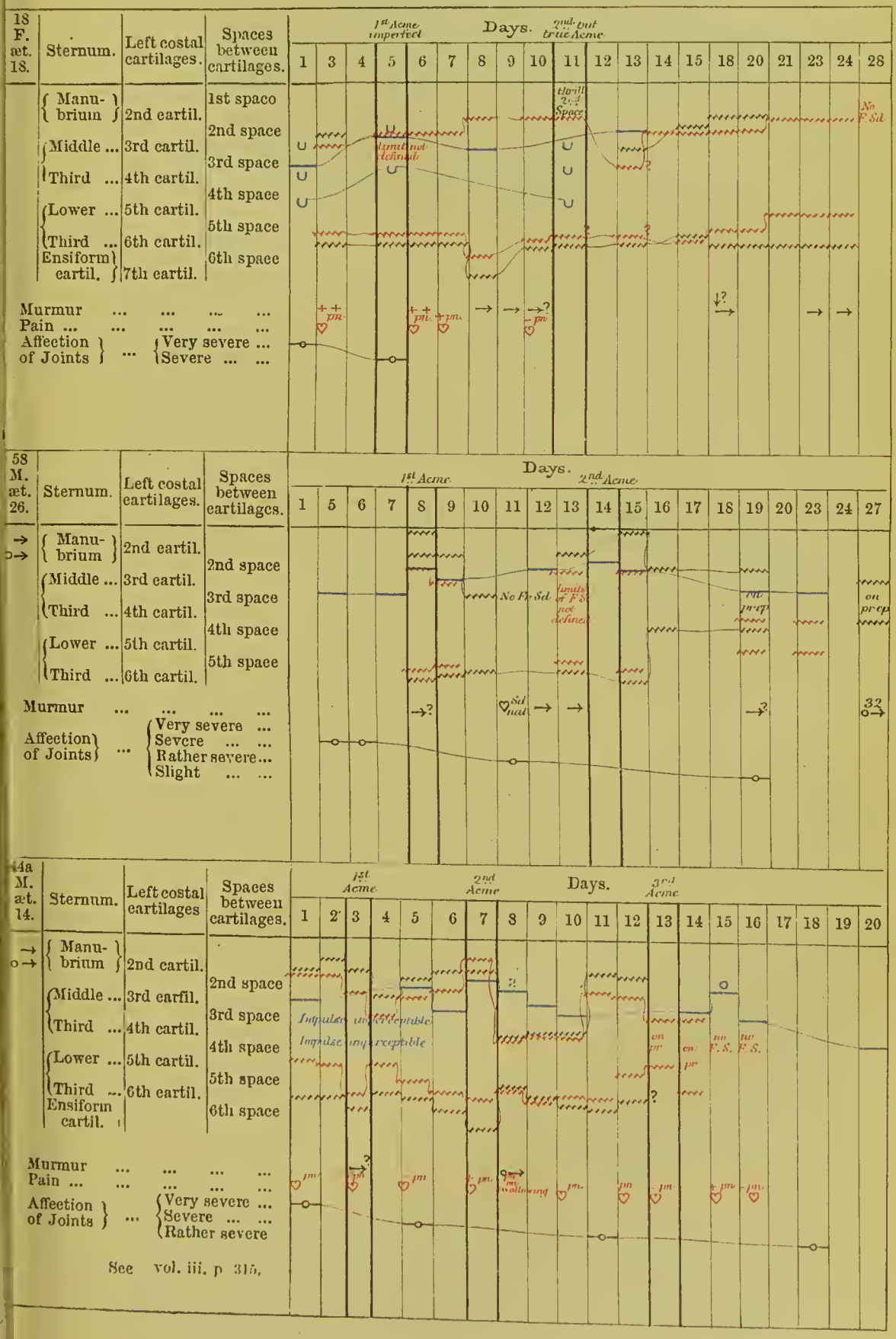
See p. 15.



See vol. iii. p. 315

For Explanation see p. 17.

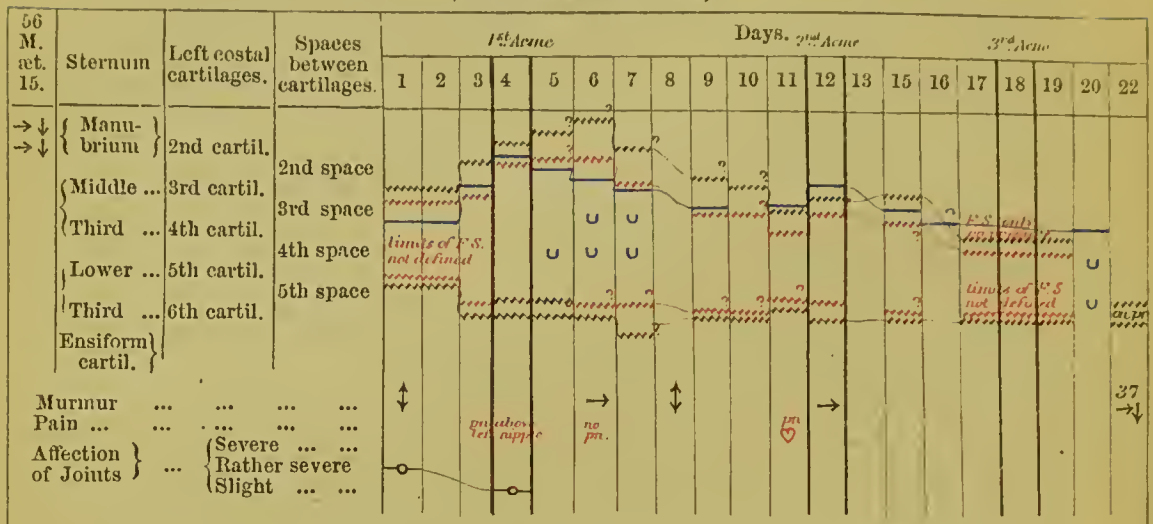
(Cases with a double acme.)



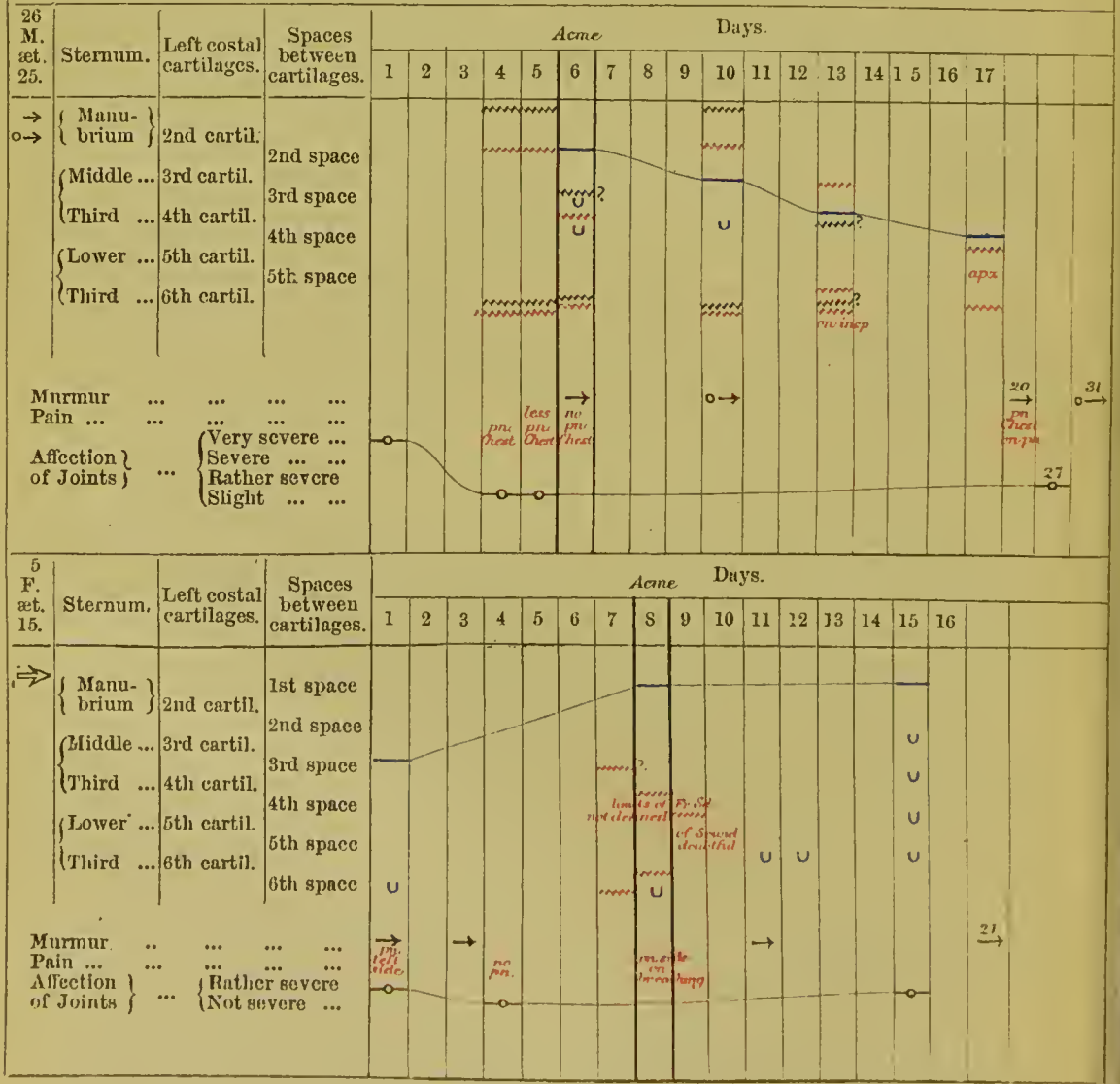
See vol. iii. p 315.

For Explanation see p. 17.

Cases in which there was a definite double friction sound, usually harsh, during the acme (continued).
(Case with a treble acme.)

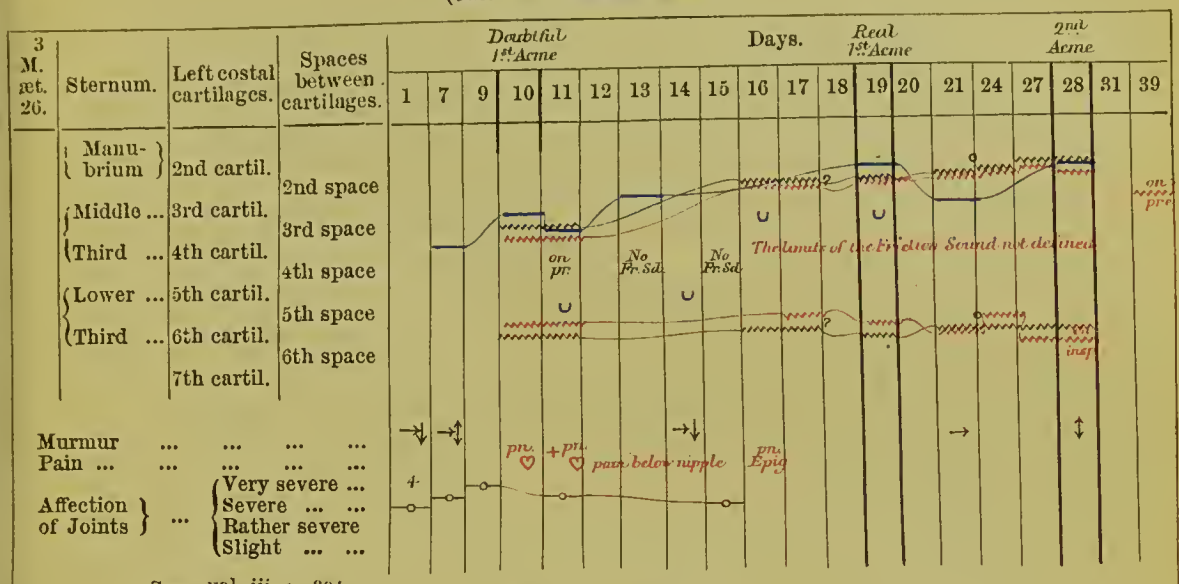


(4).—*Cases in which the friction sound was soft, and was rendered harsh by pressure, during the acme.*
(See p. 79.)



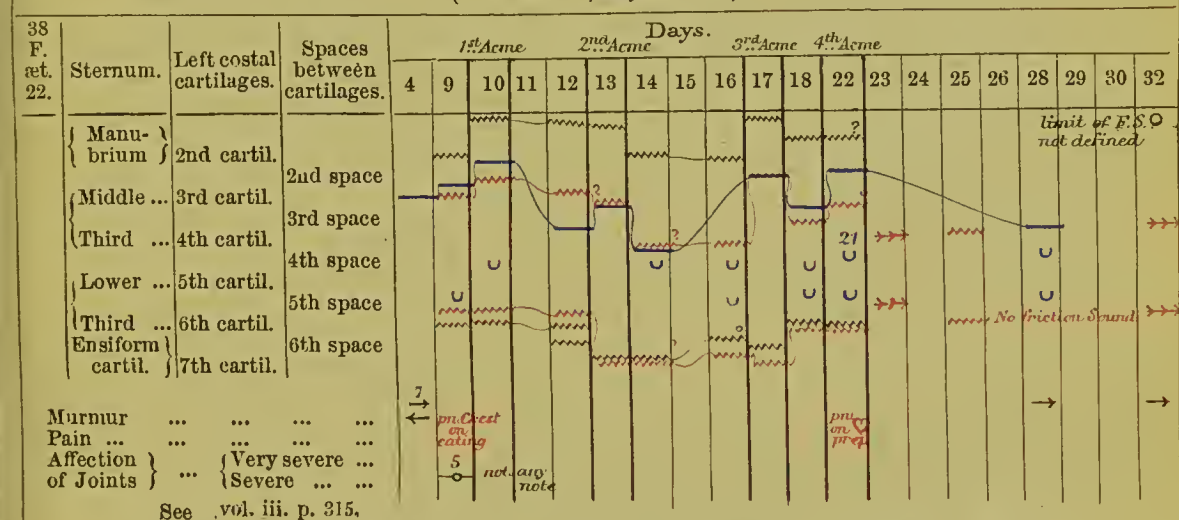
For Explanation see p. 17.

(Case with a double acme.)



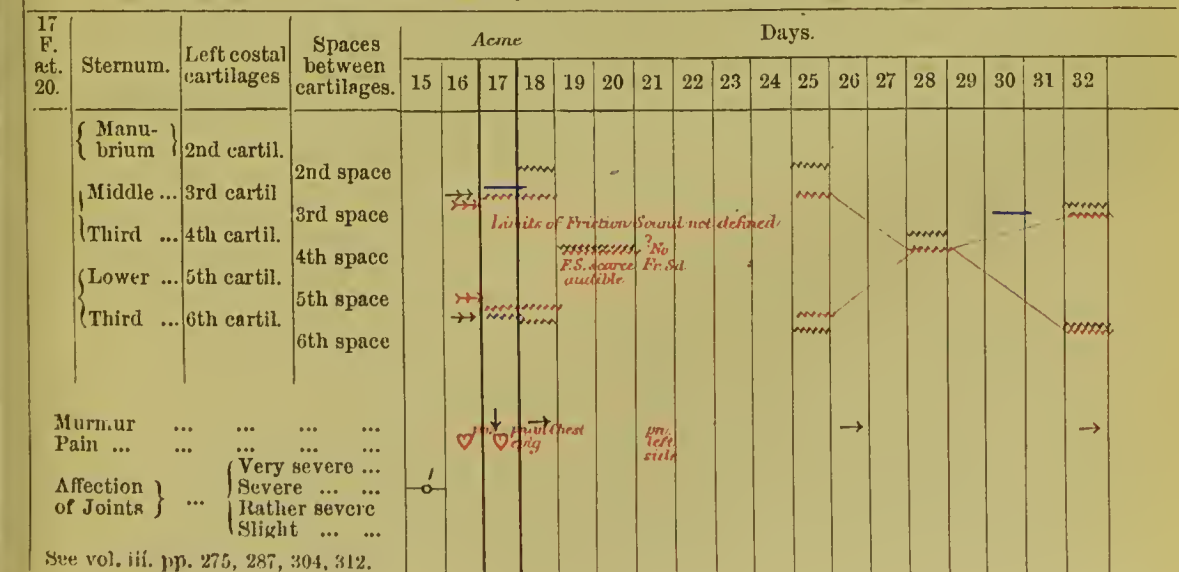
See vol. iii. p. 304.

(Case with a fourfold acme.)



See vol. iii. p. 315.

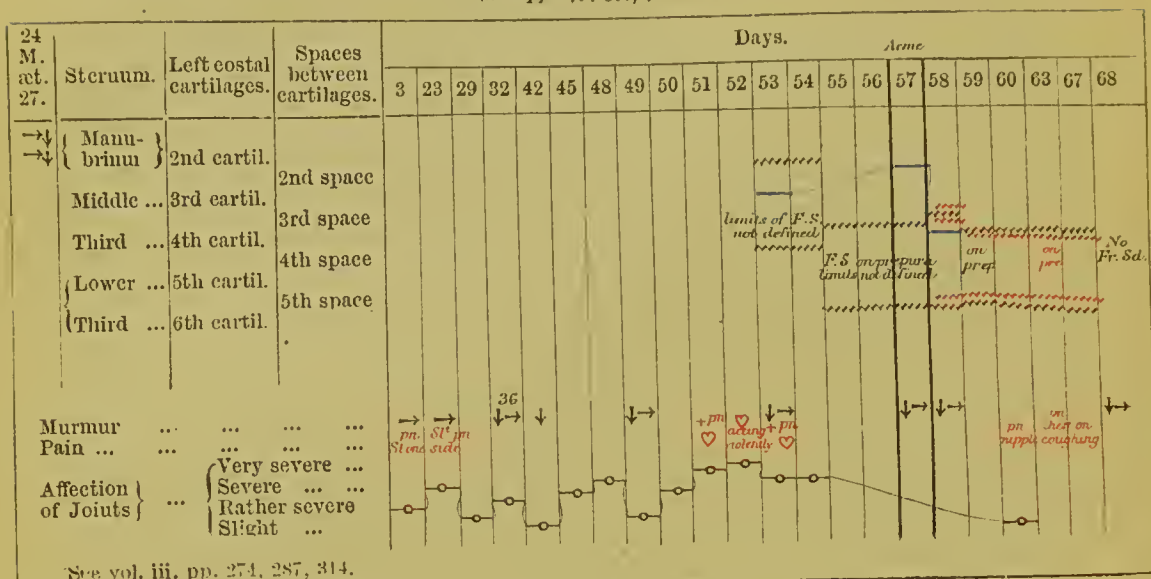
Case in which the friction sound was soft and the pressure test was not employed. *See p. 79.*



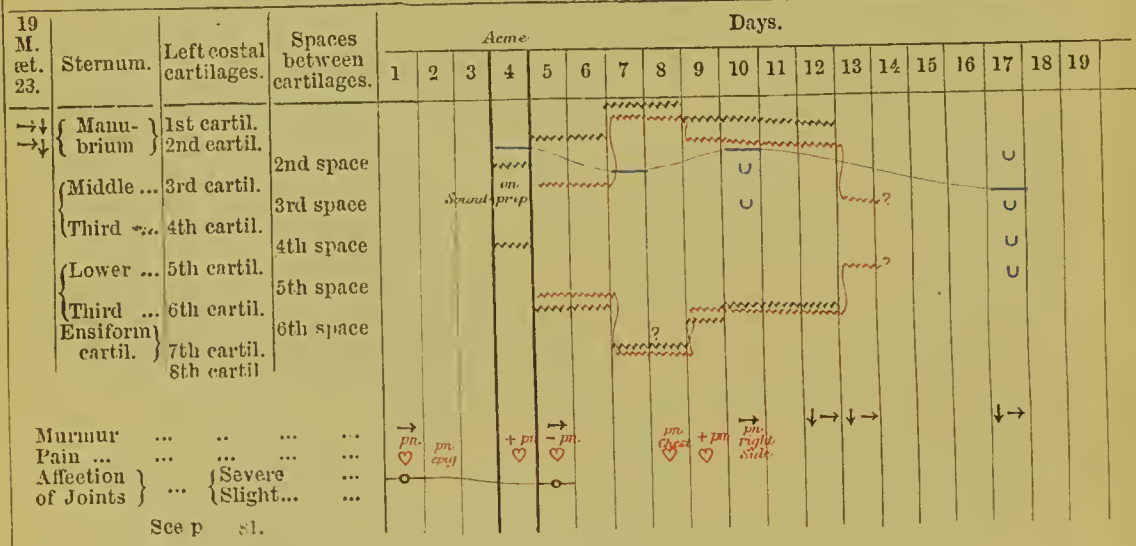
See vol. iii. pp. 275, 287, 304, 312.

For Explanation see p. 17.

(5.)—Cases in which pressure brought out a friction sound that was not otherwise present during the acme. *(See pp. 79, 80.)*

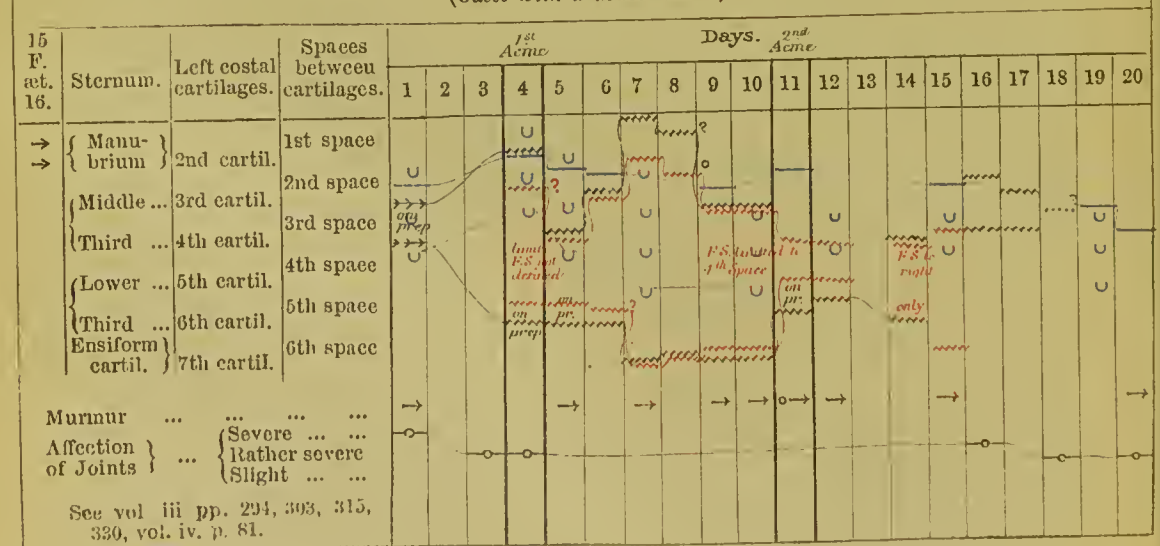


See vol. iii. pp. 274, 287, 314.



See p 81.

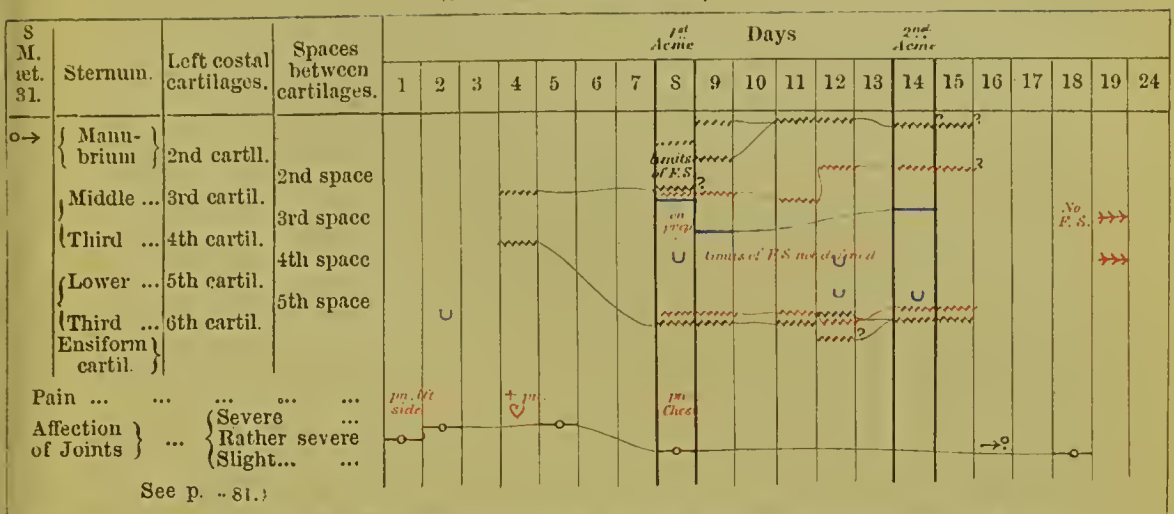
(Cases with a double acme.)



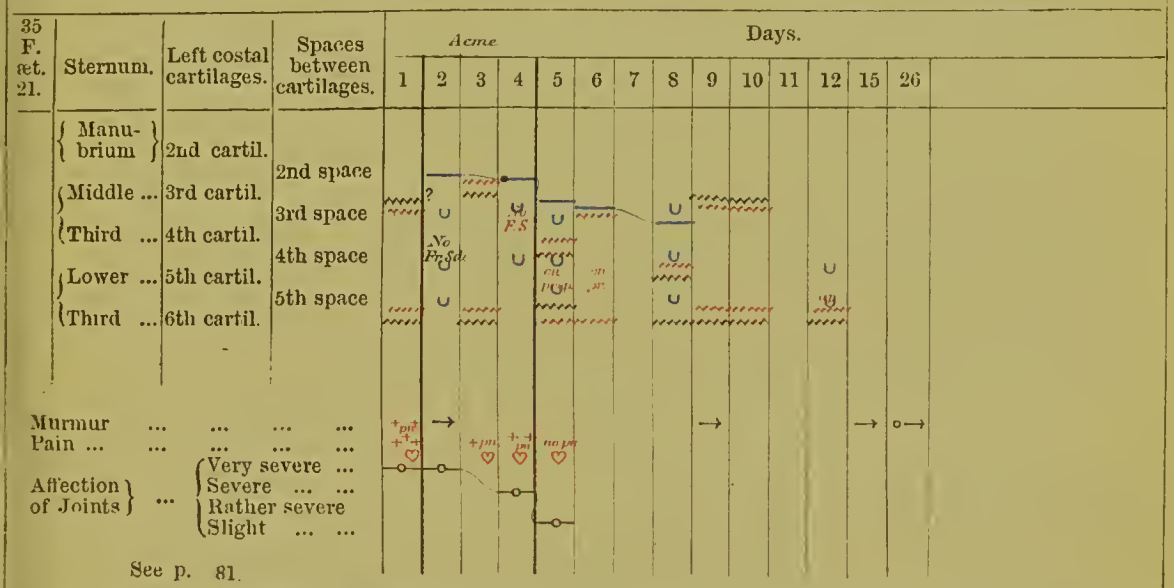
See vol iii pp. 294, 303, 315, 330, vol. iv. p. 81.

For Explanation see p. 17.

(Case with a double acme.)

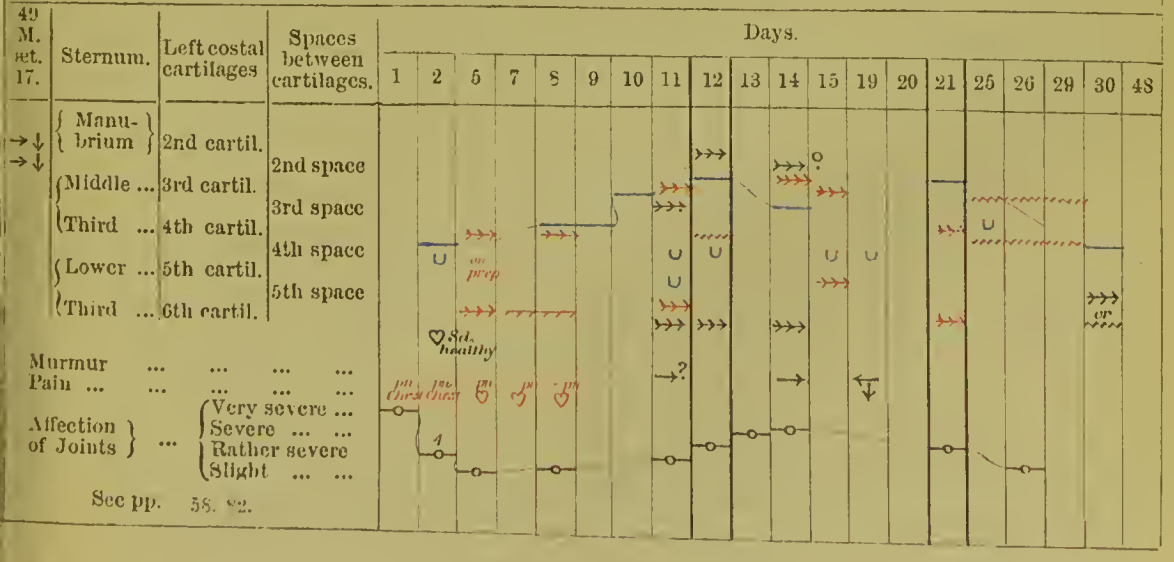


(6.)—*Case in which friction sound was absent during two of the three days that the acme lasted.* (*See pp.* 80, 81.)



(7.)—*Case in which a double friction murmur was present during the acme.* (*See p.* 83.)

(Case with a double acme.)



INFLUENCE OF PRESSURE IN (I.) EXCITING AND (II.) INTENSIFYING A FRICTION SOUND WHEN APPLIED WITH THE STETHOSCOPE OVER THE REGION OF THE HEART IN CASES OF RHEUMATIC PERICARDITIS.

I.—Influence of Pressure over the Region of the Heart in Exciting a Friction Sound when not previously audible.

(For the explanation of this part of the Table, see page 133.)

The figures refer to the cases in the preceding Tables, see pp. 17–31. The repetition of the figure shows that the sign was again audible.	During advance of effusion	During acme of effusion.	During decline of effusion.
Friction murmur excited by pressure—			
Systolic friction murmur	·15, 28	—	16, 40, 47, 50, 50, 50.
Double friction murmur	49	—	34, 35, 47, 47, 49.
Time or rhythm of friction murmur not specified	·7, 24	—	28, 30, 30, 43.
Smooth or feeble friction sound—			
Systolic friction sound	8	—	16, 49 (faint friction sound).
Double friction sound	21	—	·6, 12, 58.
Time or rhythm of friction sound not specified	24	19	·6
Friction sound—			
Systolic friction sound	·23	—	·28, ·30, ·30, 43.
Double friction, or to and fro sound	·8, ·21	8. 15, 32	3, ·32, ·34, 44, 45, 48.
Time or rhythm of friction sound not specified	24, ·24	12, 24; ·15 (2nd *acme*).	8, ·26, 35, 36, 44.
Creaking or grating sound—			
Systolic creaking friction sound	·21	—	·30, ·31 : 42 (grating).

II.—Influence of Pressure over the Region of the Heart in Intensifying a Friction Sound already present.

(For the explanation of this part of the Table, see page 133.)

The large figures indicate the cases given in the preceding tables (see pp. 17–27). The small figures show the days on which the observation was made.

Instances in which it was doubtful whether the sound modified by pressure was a friction sound

Loud ringing sound, increased by pressure, **25** 2nd day after admission.
Murmur, more distinct on pressure, **20**, 18th day.
Soft double sound (? friction), rendered louder by pressure, **20**, 35th day.

Instances in which an endocardial murmur was replaced, on pressure, by a friction murmur or sound,

Feeble murmur, replaced, on pressure, by double friction murmur, **54**, 12th day.
Mitral murmur to right of, or above, nipple (⟶); replaced, on pressure, by a friction sound, **36**, 8th day; **17**, 35th (⟷).
Double endocardial murmur, beginning with accent: on pressure, a double friction murmur, not beginning with accent, **39**, 22nd day.

Friction murmur modified by pressure

Systolic friction murmur; intensified by pressure, **4**, 7th day; **6**, 5th; **30**, 20th.
Systolic friction murmur: on pressure, a double friction murmur, **6**, 7th.
Systolic friction murmur; replaced, on pressure, by a to-and-fro friction sound **28**, 21st; **54**, 8th.
Double friction murmur; intensified by pressure, **24**, 59th; **33**, 21st; **36**, 17th; **39**, 18th; **49**, 11th–29th.
Double friction murmur; converted by pressure into a double friction sound, **26**, 13th; **28**, 14th: **45**, 3rd; **51**, 19th.

Friction sound resembling a murmur, changed by pressure to definite friction sound

Friction sound, like a bellows murmur; much harsher on pressure, **33**, 4th day.
Friction sound, of murmuring character: rendered grazing by pressure, **48** 13th.
Friction sound, almost a murmur; changed, by pressure, to a harsh double friction sound, **26**, 13th.

Systolic friction sound modified by pressure

Friction sound, scarcely audible; on pressure, a harsh double friction sound, **26**, 17th
Systolic friction sound; intensified by pressure, **6**, 1st; **21**, 9th; **40**, 7th; **40**, 13th.
Systolic friction sound; followed, on pressure, by a diastolic murmur, **31**, 5th.
Systolic friction sound; changed, by pressure, to a double friction sound, **15**, 5th **39**, 13th; **42**, 15th; **45**, 20th.

Friction sound intensified and altered in character by pressure

Smooth, soft, or grazing double friction sound; intensified by pressure, **4**, 3rd day; **4**, 17th; **25**, 4th; **25**, 5th; **28**, 5th; **32**, 4th; **38**, 9th; **38**, 25th; **39**, 17th; **40**, 14th; **45**, 4th; **47**, 11th; **54**, 4th; **58**, 21st.
Smooth or soft "friction sound" probably double, intensified by pressure, **4**, 3rd **25**, 1st; **26**, 12th; **39**, 14th.
Double friction sound; intensified or altered in character by pressure, **3**, 21st day; **6**, 1st; **7**, 4th; **8**, 9th; **8**, 14th; **8**, 15th; **12**, 1st; **15**, 5th; **15**, 6th; **19**, 3rd; **20**, 11th; **28**, 18th; **33**, 3rd; **34**, 4th; **38**, 22nd; **39**, 7th; **41**, 4th; **19**, 6th; **50**, 4th; **51**, 1st; **51**, 2nd; **56**, 12th; **58**, 9th; **58**, 16th.
"Friction sound," probably double; intensified or altered in character by pressure, **7**, 9th day; **20**, 6th; **24**, 58th; **32**, 5th; **39**, 9th; **39**, 18th; **44**, 10th; **44**, 12th; **45**, 14th; **45**, 17th; **45**, 18th; **54**, 7th; **55**, 2nd: **56**, 3rd.
Harsh or rough double friction sound; harsher on pressure, **7**, 5th; **12**, 5th; **18**, 7th; **18**, 8th; **28**, 14th; **32**, 4th; **36**, 9th; **38**, 10th; **39**, 8th; **40**, 5th; **42**, 10th; **42**, 11th; **44**, 3rd; **56**, 5th; **58**, 8th.
Harsh or rough "friction sound," probably double, intensified by pressure, **19**, 10th; **21**, 6th; **33**, 12th.

Grating friction sound excited or intensified by pressure.

Friction sound scarcely audible, grating on pressure, **48**, 9th.
Double friction sound; rendered almost grating by pressure, **42**, 13th.
Double friction sound; rendered grating by pressure, **26**, 6th; **44**, 1st; **44**, 2nd **51**, 4th.
Grating friction sound; increased or rendered harsher by pressure, **36**, 8th **47**, 7th.

N B.—The last section of this table, for which there is no space here, is given at p. 137.

upper portion is above four inches wide, the greater width of the cardiac portion of the region of dulness being gained chiefly to the left. This sudden widening of the area of pericardial dulness from distension of the sac gives that area a peaked form above, and an indented outline along its left upper border, that distinguish it from the equally high and extensive area of cardiac dulness due to adherent pericardium and valvular disease, when the heart is enlarged in all directions and especially upwards and to the left, and when the upper left border of the region of cardiac dulness presents a very gradual inclination downwards and to the left without a break. (Compare figure 10 with figure 11, p. 48.) This pear-shaped outline of the region of dulness over the pericardium is quite characteristic, and indicates with certainty the presence of extensive effusion into the sac.

Among the forty-four cases, the upper boundary of the region of dulness when the effusion had reached its acme was over the first space or second cartilage in ten cases, over the second space in twenty-two, and over the third cartilage in twelve. In those cases that suffered a relapse, the first acme was as a rule higher, and the second, and still more the third acme, were lower than the single acme in cases that had no relapse.

If the position of the upper boundary of the pericardial dulness over the cartilages and their spaces is known, the whole area of the region of dulness over the pericardium may be inferred with considerable accuracy; since the whole outline of that area shrinks when its upper boundary is lowered, and widens when it is raised. In this respect, with certain definite reservations, the upper border of the region of pericardial dulness over the cartilages and spaces to the left of the upper half of the sternum serves to measure the whole area of dulness and to define its complete outline; just

as the ebb and flow of the tide, or the rise and fall of a flood indicated on a measuring post, will tell any one accurately acquainted with the coast, or the contour lines of the country, the exact area over which the land is covered by water.

If the upper boundary of pericardial dulness reach to the second space, the contour line defining the dulness extends—to within an inch of the top of the sternum; an inch beyond the right edge of the lower half of that bone; and more than an inch below its lower end, where it may descend as far as the tip of the ensiform cartilage; to the lower edge of the left sixth cartilage; and about an inch beyond the left nipple. (See figures 10, p. 48; 13, p. 64.) If the upper margin of dulness be limited by the third space, the boundary line extends—across the sternum on a level with the third costal cartilages; to the right edge of that bone; and to fully half an inch below its lower end; to the upper edge of the sixth cartilage; and to the left nipple. (See figures 6, p. 40; 12, p. 64.) The lungs, the diaphragm, the liver, and stomach are all correspondingly displaced, to a greater degree all round when the upper limit of dulness is over the second cartilage; and to a lesser degree all round when that limit is over the third space. The intermediate position of the upper edge of dulness over the other cartilages and spaces gives an intermediate outline of the whole area.

The restrictions to this rule are due to age and sex, to previous affections of other organs, to valvular disease of the heart of old standing, to coinciding affections of the lungs, especially the left lung, to the duration of the attack of pericarditis and the occurrence of relapses, to accompanying endocarditis, to the progress of the disease, and to its terminations, whether in complete restoration to health, the valves being intact, in valvular disease, or in pericardial adhesions. These restrictions are numerous in appearance, but practically

they seldom interfere with the rule just stated of the correspondence of the whole area of dulness with the boundary of a particular part of it.

The rule that the region of pericardial dulness in rheumatic pericarditis enlarges over corresponding areas in different cases, holds good in young persons of both sexes, and in women. In men, however, the bony framework of the chest is larger, the lungs are more ample and cover the heart to a greater extent, and the diaphragm is lower than in boys, youths, or women. The result is, that in men both the upper and lower boundaries of the region of pericardial dulness are lower than in the classes just spoken of. Thus the upper boundary of dulness during the acme was over the third cartilage in 8 out of 14 cases of rheumatic pericarditis in men; while in the whole of those of the female sex so affected, except one, that boundary was above the third cartilage. In nearly one-third, or 3 in 11 of the male youths with rheumatic pericarditis, the upper boundary of the region of dulness during the acme was over the third cartilage. This is due to the fact that in the male sex, the lungs at a comparatively early period are more largely developed than in the female sex.

When rheumatic pericarditis attacks a heart enlarged from previous valvular disease, the pericardial sac, being more ample, is capable of containing a larger amount of fluid, and the region of pericardial dulness is of greater relative width than when the affection attacks the virgin heart.

If the lower lobe of the left lung shrinks, owing to the combined effect of the compression of that lobe and of the left bronchus by the swollen sac, and of pleurisy with or without pulmonary apoplexy, a condition of things by no means unusual, the whole area of pericardial dulness tends towards the left, and its left border comes into direct contact with the ribs at the side.

Changes in the Form of the Outline of Pericardial Dulness caused by variations in the Progress and Termination of the Affection.—If the attack lasts long, the pericardial sac, as I have already stated, becomes softened, it yields sideways, and becomes widened to the left and right, while it is not proportionally lengthened above and below (see figure 3, p. 5). This is especially to be noted when relapses take place, and when the effusion, after lessening in quantity, again increases. (See figure 16, p. 110.)

If the affection passes quickly through its stages, and the recovery is perfect, the heart being restored to health, the changes of the increase, the acme, and the decline of the pericardial effusion and of the area of pericardial dulness, pass through the course I have described. (See figures 4, 5, p. 12 ; 6, 7, p. 40.)

If, however, the heart becomes enlarged owing to the establishment of valvular disease, the lessening and disappearance of the effusion are delayed, and the area of dulness is somewhat widened and lowered, especially towards the left.

If along with valvular disease, adhesions of the heart are established, the whole organ is enlarged, upwards, downwards and sideways. The outline of the area of dulness loses its characteristic pear-shaped form, and its peaked outline over the great vessels gives place to a gradual widening of that area from above downwards, that corresponds with the enlarged outline of the heart itself. (Compare figure 10 with figure 11, p. 48.)

Prominence over the Region of the Pericardium.

Increased dulness on percussion over the region of the pericardium is the only reliable sign of the increase of fluid in the sac. Increased prominence of the costal cartilages

over the heart, with widening of the spaces between them, form, however, a secondary sign of some interest and value.

In my paper before alluded to, I state that the distension of the pericardial sac by fluid, besides displacing the surrounding organs, pushes forward the sternum, elevates the costal cartilages from the second to the seventh, widens the spaces between the cartilages and ribs from the second to the sixth or seventh, pushes outwards the sixth left rib, and causes some degree of prominence over the left side.

This condition was observed with care in one or more of the cases of pericarditis examined by me in the Nottingham Hospital. I find that prominence over the region of the pericardium was noticed by me in 19 of 63 cases of rheumatic pericarditis under my care in St. Mary's Hospital. More than three-fourths of those patients (15 in 19) were males, while only 4 were females. The cardiac prominence is obscured in women by the mamma ; that sign having been observed in only one-seventh of the female cases of rheumatic pericarditis (4 in 27), while it was noticed in nearly one-half of the male cases (15 in 36).

The increased prominence over the region of the heart was usually noticed when the effusion into the pericardium was at its height, and it lessened when the effusion declined. In the greater number of the cases (12 in 19), the prominence over the region of the heart is described in general terms, but in seven its area was specified. In one of these it extended from the second cartilage to the sixth ; in two, from the third to the sixth ; in three, from the third to the fifth ; and in the remaining one, from the fourth cartilage to the sixth.

In these cases the cartilages yielded to the distension of the sac, and were displaced by it forwards and upwards ; with the good effect of somewhat relieving the pressure exerted by the swollen sac on those important structures, the bifurcation of

the trachea, the left bronchus, the œsophagus, and the aorta, that are situated between the back of the pericardium and the bodies of the dorsal vertebræ. The prominence over the cardiac region caused by the forward pressure of the enlarged pericardium points out that a serious counter-pressure backwards is exerted at the same time on the three vital tubes that I have just named, which convey air to the lungs, and especially the left lung, food to the stomach, and blood to the lower half of the frame. Indeed, the true value of this sign is that its presence reveals to us at the surface, the existence of deep and serious pressure on important internal parts, a pressure that is augmented when the superficial prominence increases, and that is relieved when that prominence lessens.

It is to be remarked that at the same time that the sternum and cartilages over the region of the distended pericardium are rendered prominent with the effect of somewhat lessening the pressure of the swollen sac upon the bifurcation of the trachea, the left bronchus, the œsophagus and the aorta—the dorsal portion of the spinal column deepens itself and curves backwards so as to afford increased space for the swollen sac and those important tubes that are compressed by it. At the same time the patient sits up, and even leans forward, so as to allow of the gravitation downwards and forwards of the fluid in the pericardium. By this attitude, and the deepened spinal curvature, indeed, the pressure of the distended sac upon those vital parts is materially lessened, breathing and swallowing are rendered less difficult, and blood is supplied through the descending aorta with greater freedom to the body and lower limbs.

The Position of the Impulse of the Heart in Cases of Pericarditis.

When the amount of fluid in the pericardium has increased so as to enlarge the area of dulness on percussion over the region of the heart, the seat of the impulse is raised and extended outwards.

I gave figures of three cases of pericarditis with great increase of fluid in the sac, in my paper on the position of the internal organs, in which the impulse was present in the third and fourth spaces, instead of occupying its usual position in the fourth and fifth spaces. In that paper, attention was I believe called for the first time to the elevation of the impulse in cases of pericarditis with effusion into the sac.

In thirty-seven of the forty-four cases of rheumatic pericarditis, daily details of which are given in columns in the accompanying tables, the exact position of the impulse during successive visits is stated, in five others the impulse is described, but its situation is not specified, and in the remaining two the impulse was almost or quite imperceptible (see pp. 17—31).

In examining these cases I shall study the position of the impulse from two points of view, (1) the elevation of its lower boundary; (2) its diffusion into the higher intercostal spaces during the period of the increase of fluid in the pericardium.

(1) *The Elevation of the* Lower Boundary *of the Impulse.*— In fourteen cases, the extent of dulness on percussion over the region of the pericardium increased, and the effusion attained to its acme after the first observation; and in twelve of these the impulse occupied a higher position at the time

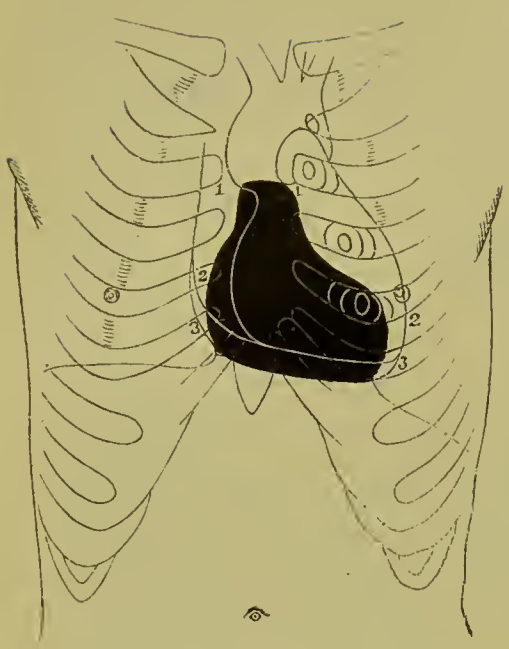


FIG. 6.

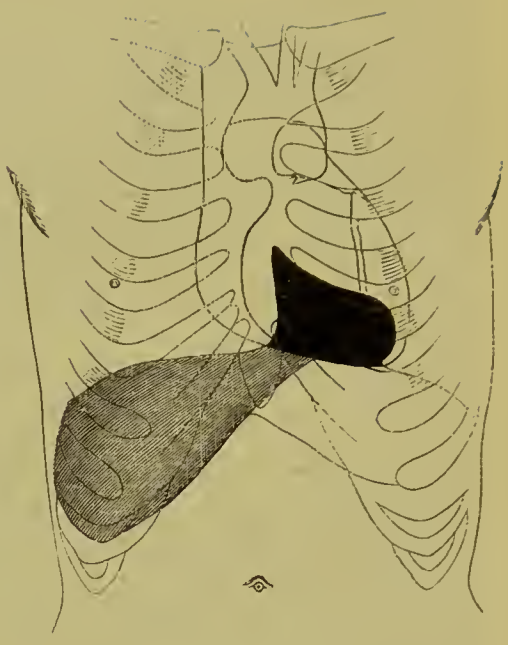


FIG. 7.

For previous views of this case, see figures 4, 5, page 12.

Figure 6, from a youth aged 17, affected with rheumatic pericarditis.

Period of the decline of the pericardial effusion.

Sixth day after the acme of pericardial effusion, eighth day after admission.

The pericardial effusion has diminished to a great extent, and the sac, no longe distended, has contracted, so that it has lost its pear-shaped form, and resumed more nearly that of the heart itself, a little modified and enlarged by undue fulness above. The lower border of the heart is much lower than during the acme, being situated behind the fifth cartilage, and the lower boundary of the pericardium is much higher ; it no longer protrudes into the epigastric space, but has shrunk upwards, being situated behind the upper third of the ensiform cartilage, and behind or above the upper edge of the sixth left cartilage. The right ventricle and the apex of the left ventricle are exposed ; but the upper part of the conus arteriosus and of the front of the left ventricle, the pulmonary artery, and the ascending aorta, are covered with lung.

The prominence over the pericardium has almost disappeared and the left side has nearly resumed its natural shape.

The region of pericardial dulness (see the black space) corresponds with the lessened amount of the pericardial effusion, and instead of being pear-shaped, or longer than it is broad, as it was during the acme, it has now more nearly the contour of the natural region of cardiac dulness, and is broader than it is long. It still, however, presents a peaked form at its upper border behind the sternum, where that border is on a level with the third cartilage, and

of the acme than at that of the first observation, while in two its position was unchanged.

In twenty-two of the patients the amount of fluid in the pericardium was at its greatest height or acme at the time of the first observation; and as the effusion lessened, in eighteen of these the lower boundary of the impulse fell, in three it was stationary, and in one it became higher in position.

We thus see that in thirty of these thirty-seven cases of rheumatic pericarditis, the lower boundary of the impulse was raised in position when the amount of effusion in the pericardium was at its acme.

In one-fifth of the cases (7 in 37) the lower boundary of the impulse was pushed up as high as the third space, and in three-fifths of them it was present in the fourth space (21 in 37). In two patients, one with disease of the aortic and mitral valves, the other with that of the mitral valve alone, of some standing, the impulse was seated in the sixth space,

where it is still much higher than its upper border to the left of the sternum, which is situated at the third left space. Its lower border is situated behind the upper third of the ensiform cartilage; and its right and left borders are respectively behind the right margin of the sternum, and within the left nipple.

The *impulse* is felt in the first, second, third, and fourth left spaces, being feeble in the fourth space. (See the curved and circular lines in those spaces.)

Figure 7, from the same patient as figures 4, 5, 6.

Period of the disappearance of the pericardial effusion and restoration of the heart to its natural position, which is however still rather high.

Eighth day after the acme of pericardial effusion, tenth day after admission.

There is no pericardial effusion, and the chest has resumed its natural shape.

The region of cardiac dulness (see the black space) has regained its natural form and is no longer preternaturally higher behind the sternum than to the left of it. Its upper boundary is situated behind the fourth cartilage, and the adjoining portion of the sternum, its lower boundary, is behind the fifth space and the upper end of the ensiform cartilage; its right margin is a little to the left of the middle line of the sternum, and its left border is fully half an inch within the mammary line.

in three cases it occupied the fifth space, and in three it was felt over the third cartilage.

The existence of previous valvular disease, owing to the increased size of the heart in such cases, exercised a marked influence on the position of the lower boundary of the impulse, and as a rule lessened or prevented its ascent during the acme of the effusion. Thus, of five patients of this class, all of whom had affection of the mitral valve, and one of them of the aortic valve also, in two the lower boundary of the impulse occupied the sixth space, in two the fifth space, and in one it was seated in the fourth space.

If we deduct from the thirty-seven cases these five with valvular disease, which are exceptional both in their nature and as regards the influence of the effusion on the seat of the impulse, we find that in only one of the remaining thirty-two patients was the lower boundary of the impulse as low as the fifth space during the acme of the effusion.

These cases of previous valvular disease are exceptional in another point of view. In three of these five patients the position of the lower boundary of the impulse was not higher during the acme of the effusion than at other times. If we deduct these five cases from the thirty-seven under review, we find that in only three of the remaining thirty-two cases was the position of the lower boundary of the impulse unchanged during the acme of the effusion, while in twenty-nine of them it was definitely higher than in health.

Extent to which the Lower Boundary *of the Impulse was Raised, when the Effusion into the Pericardium was at its Height or Acme.*—In the twelve patients in whom the acme of the effusion was reached after the first observation of increased dulness on percussion, and in whom the lower boundary of the impulse was then elevated, the impulse at its lower boundary ascended two spaces in two instances

(compare figure 12 with 13, p. 64), a space and a half in one, one space in six, and less than a space in three cases; and it descended after the acme two spaces in five instances, one space in five, less than a space in one, and in the remaining case its descent was not observed.

In the eighteen cases in which the effusion had attained to its acme at the time of the first observation, the lower boundary of the impulse subsequently descended two spaces in three patients, one space in thirteen, one rib's breadth in one, and half a space in one case.

If we combine these thirty cases in one group, we find that the lower boundary of the impulse was higher during the acme of the effusion than in the natural state by two spaces in eight cases, by one space in nineteen, and by less than a space in three cases.

Time occupied during the Ascent and the Descent of the Lower Boundary of the Impulse in connection respectively with the Increase, the Acme, and the Decline of the Fluid in the Pericardium.—In the twelve cases in which the impulse at its lower boundary ascended to its highest point after the first observation, and during the period of the increase of the pericardial effusion, the time occupied by its ascent was from one to two days in nine cases, and from four to six days in three cases.

The lower boundary of the impulse fell from its highest position to its natural one in from one to two days in ten cases, in from three to nine days in eighteen, and in sixteen days in two out of a total of thirty cases. The ascent of the lower boundary of the impulse was therefore more rapid than its descent.

Relation between the Extent of the Effusion in the Pericardium, and the Height of the Lower Boundary of the Impulse.—The clinical facts just given show that the lower boundary

FIG. 8.

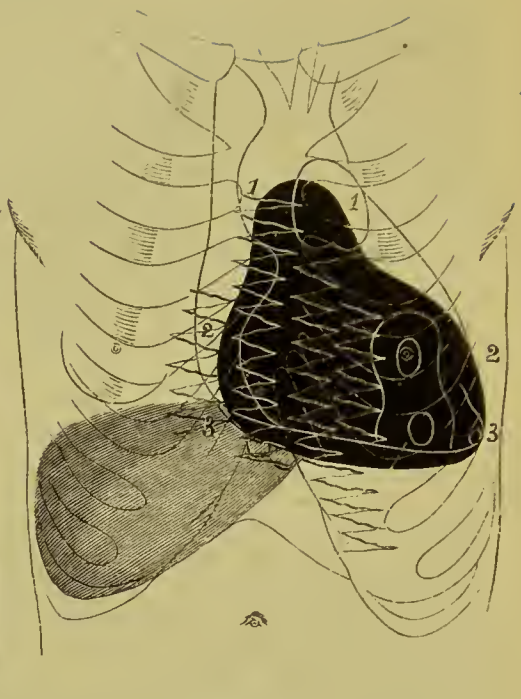


FIG. 9.

Figure 8 from a housemaid aged 17, affected with rheumatic pericarditis.

Period of the first acme of pericardial effusion, fifth day after admission.

The explanation of pericardial effusion and dulness given with figure 4, page 12, applies also to this figure.

The *pericardial effusion* extends less to the left and more to the right than in figure 5, page 12 (acme of pericardial effusion), and is of about equal extent in the two figures from above downwards. The heart, which is enlarged, is elevated by the fluid, but to a less degree than in figure 9. its lower boundary being *probably* situated behind the lower border of the fifth cartilage, and just above the lower end of the sternum.

The whole front of the heart is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the ascending aorta within the pericardium, and the pulmonary artery.

The region of pericardial dulness (see the black space) extends from a little above the lower end of the manubrium and the second left space, down to the tip of the ensiform cartilage, and the middle of the sixth cartilage ; and from a little over an inch to the right of the lower half of the sternum, to a little beyond the left mammary line. The area of dulness includes (1, 1,) the region of the great arteries ; (2, 2,) that of the heart ; and (3, 3,) that of the volume of the effused fluid below the heart, and projecting downwards into the epigastric space.

The impulse is less elevated than in figure 5 (acme), being situated in the second, third, and fourth spaces. (See the curved and circular lines in those spaces.)

of the impulse was raised by the increase of the fluid in the pericardium; and we find, therefore, as a rule, a relation between the extent of the effusion and the height of the impulse in these cases of pericarditis. But this rule is

The *friction-sound* (represented by zigzag lines, the systolic lines being thick, the diastolic thin), is heard, double, over the whole length of the sternum, being audible, with pressure over its upper third (the great arteries), and without pressure over its lower two-thirds; and it is also audible with pressure from the third to the fifth left cartilages (right ventricle); and over, but not beyond the apex of the left ventricle.

A loud mitral murmur → is audible extensively to the left of the heart.

Figure 9 from the same patient as figure 8.

Period of the decrease of the pericardial effusion after the first acme.

Eighth day after admission, third after the acme—for the sounds. Eleventh day after admission, sixth after the acme—for the pericardial effusion and dulness, and impulse.

The pericardial effusion has lessened considerably, but is still present in considerable quantity. The right ventricle and the apex and front of the left ventricle are completely exposed; and the left border of the right auricle, and the lower portions of the ascending aorta and pulmonary artery, are also brought into view. The heart (2, 2,) which is enlarged, has dropped down into its natural place, and even extends beyond that place, at its lower and left boundaries. The amount of effusion between the under surface of the heart and the floor of the pericardium (3, 3,) is very small.

The region of pericardial dulness (see the black space) has lessened considerably in area; it extends from between the second spaces, behind the sternum, down to the lower third of the ensiform cartilage; from the third left space to the upper border of the sixth cartilage; and from the right edge of the sternum to a point an inch beyond the left mammary line. There is reason to believe that adhesions have formed at the apex, so that the latter boundary is not pericardial but cardiac. The region of dulness over the great arteries (1, 1,) is still very marked but has materially lessened; that over the heart (2, 2,) being still extensive; and that over the depending portion of the pericardial effusion between the under surface of the heart and the floor of the pericardium (3, 3,) being very narrow, indeed a mere strip.

The impulse of the apex is felt in the sixth space, considerably to the left of the nipple. The position of the impulse elsewhere is not mentioned in the report, but I have given it in the figure as being present in the fourth and fifth spaces, because three days later, at the time of the second acme, it was felt in those spaces, as well as in the second and third spaces. (See the circles and curved lines in those spaces.)

The friction-sound (see the zigzag lines, systolic thick, diastolic thin) on the seventh day had increased considerably below and to the right, and lessened above and to the left. It was audible over the sternum from below, but not above,

reversed in a small group of exceptional cases, amounting to seven, in which the upper limit of the effusion was as high as the first space or the second cartilage; while the lower boundary of the impulse was present in the sixth space in one, in the fifth space in two, in the fourth space in three, and in the third space in only one of these cases. Three of these patients in whom the impulse was low had valvular disease of old standing, a condition that, as I have already shown, prevents or lessens the ascent of the impulse.

(2) *The Diffusion of the Impulse over the* Higher *Intercostal Spaces during the Acme, and Decline of the Fluid in the Pericardium.*—In three-fifths of the cases (22 in 37) the impulse, at the time of the acme of the effusion, extended upwards above its lower boundary to the extent of one or more of the higher intercostal spaces. In more than one-half of these cases the impulse was felt beating as high as the second space (12 in 22), while in less than one-half of them its upper limit was the third space (10 in 22). The extent to which the impulse was felt in the higher spaces was naturally regulated by the position of its lower boundary. Thus, the impulse was bounded below by the fourth space in ten cases, and in eight of these it extended up to the third space or cartilage, and to the second space in only two; while in eight other patients the impulse, which was bounded below by the third space or cartilage, spread upwards to the second space. According, therefore, to the degree to which the impulse was raised by the increased amount of fluid in

the level of the second spaces, and thence down to the tip of the ensiform cartilage; to the right of the lower half of the sternum; and over the left cartilages, from the third to the seventh, where it extended about two inches below the heart; but it was inaudible over the region of the apex, where there were probable adhesions.

For the later views of this case, see figures 10, 11, p. 48.

the pericardium, it was felt beating in the second and third spaces, or the third and fourth spaces, instead of, as in health, the fourth and fifth spaces.

In these cases there were two agencies at work : one, the increase of fluid in the pericardium, which elevated the heart and its impulse both at their lower and upper boundaries into the contracted space at the higher part of the chest, and caused the heart to beat against the left upper spaces ; the other, the enlargement from distension of the right ventricle and especially of the pulmonary artery, owing to the difficulty with which the blood passes through the lungs from the combined effect of the pressure upon the auricles by the fluid in the swollen sac, and the existence of endocarditis with mitral regurgitation. The enlarged right ventricle and pulmonary artery displace the lungs, and pulsate, the former against the third, the latter usually against the second space ; and in that space the double beat of the artery is then felt, the first being feeble, the second sudden and like a shock, coinciding with a feeble first and intensified second sound heard over the same situation. When the heart is much raised, it is evident that the conus arteriosus must sometimes occupy the second space, the pulmonary artery being elevated into the first space.

After the acme, when the amount of the fluid in the pericardium lessened, the position of the impulse, as we have just seen, as a rule descended at its lower boundary, but it generally retained its place at its upper boundary. Sometimes, indeed, the impulse extended upwards as well as downwards during the period of the lessening of the effusion.

The clinical facts that I have just related as to the extension of the impulse into the upper region during the successive periods of the increase, the acme, and the decrease of the effusion into the pericardium, while its lower boundary

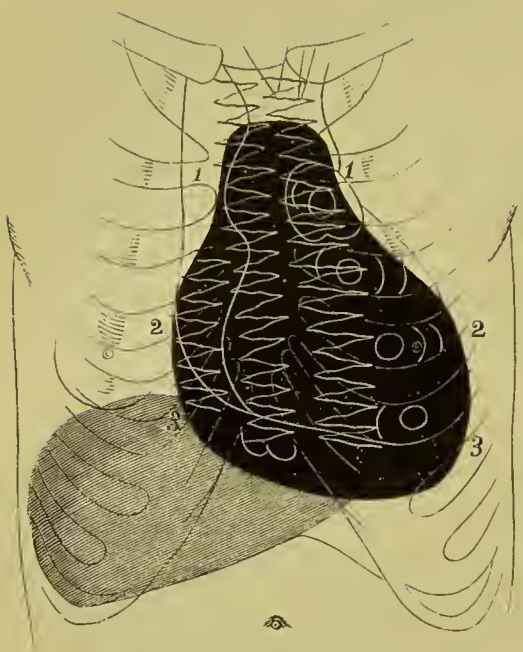


FIG. 10.

FIG. 11.

For previous views of this patient see figures 8, 9, page 44.

Figure 10, from a housemaid aged 17.

Period of the second acme of pericardial effusion owing to a relapse of pericarditis. From the fourteenth to the eighteenth day after admission, from the tenth to the fourteenth day after the first acme (figure 8), and from the third to the seventh day after the period of decrease of the effusion illu trated in figure 9. The period of the acme lasted four days.

Thc explanations of pericardial effusion, prominence and dulness, given with figure 4, at page 12, apply also to this figure.

The pericardial effusion has increased again to a very great extent. The heart is con-iderably enlarged, and is probably adherent at the apex; its lower boundary is therefore much lower than during the first acme, figure 8, and apparently reaches down to the sixth cartilage, and the middle of the ensiform cartilage. The effusion has increased very much, especially upwards, downwards, and to the right ; but owing probably to adhesions at the apex, it has been stationary or has lessened in area at the left side—compared with its amount and area during the period of decrease of the effusion after the first acme shown in figure 9. The effusion extends much higher and more to the right than during the first acme (figure 8), but it is of the same extent at its lower and left boundaries in this as in the first acme. The area of the effusion was much wider in relation to its length, and especially towards the left, in the single acme shown in figure 5, owing to the enlargement of the sac from long-continued distension, than it is in this instance, in which thc expansion of the sac to the left has been apparently stopped by the probablc adhesion of the apex and front of thc left ventriclc.

steadily rose during the increase, and fell during the decrease of the fluid, are to be traced I consider to a succession of causes. I have just considered the two agencies that are at work to extend the impulse into its higher region during the

The whole front of the heart and great arteries is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, and the ascending aorta and pulmonary artery.

The region of pericardial dulness (see the black space), corresponding to the pericardial effusion, extends very high, or to within an inch of the episternal notch; far to the right, or nearly two inches to the right of the sternum; low down, or below the tip of the ensiform cartilage; and, owing probably to adhesions at the apex, proportionally less far to the left, or fully half an inch to the left of the mammary line. The region of dulness over the arteries is unusually high and narrow. Its width on the first day of the acme was little more than one inch; but it had increased to about two inches on the fourth day, when its upper border was not quite so high as on the first day of the acme at its upper part.

The impulse is extensive but not strong, the double pulsation over the pulmonary artery being felt over the second and third spaces; and the impulse of the heart, over the third, fourth, and fifth spaces, where it extends beyond the nipple (see the curved lines and circles in those spaces). The lower boundary of the impulse has therefore been elevated from the sixth space to the fifth since the period of the decrease of the effusion following the first acme, shown in figure 9: it is, however, lower in this second acme than it was in the first acme, when it occupied the fourth space.

The friction-sound (see the zigzag lines, the systolic lines being thick, the diastolic thin) is scarcely audible anywhere without pressure, but with pressure it is heard, double, over the whole region of the pericardial dulness except over the apex and front of the left ventricle, where there are probably adhesions, and where a loud mitral murmur → prevails. The rubbing sounds are louder over the two lower thirds of the sternum and to each side of it, than higher up.

Figure 11, from the same patient as figures 8, 9, 10.

Period of complete adhesion of the pericardium to the heart.

For pericardial dulness—fifty-two days after admission, thirty-nine to forty-three days after the second acme.

For the impulse—eighty-eight days after admission, when the dulness, tested by post-mortem examination, was about the same as on the fifty-third day after admission.

The region of pericardial dulness (see the black space) is very extensive, measuring about seven inches from left to right, with a slight downward inclination, and nearly five inches from above downwards. Its upper boundary was behind the lower border of the manubrium; its lower boundary, behind the lower end of the ensiform cartilage, the sixth left space and the seventh left rib; its

periods of the increase and acme of the effusion ; the increase namely of the pericardial fluid elevating the heart into the contracted space of the chest above ; and the enlargement of the right ventricle and pulmonary artery from obstruction to the flow of blood through the lungs. During the decline of the fluid the first of these influences is reversed, for the heart descends into its natural place, where it beats with comparative freedom ; but the second influence, the enlargement of the right ventricle and pulmonary artery from obstruction through the lungs, often remains in full force to retain the impulse in its higher position ; and this influence is frequently added to by other causes that have a like effect. These additional influences include the thickening and matting of the inflamed pericardium ; the possible adhesion from pleurisy of the left lung to the pericardium at its upper border ; and the deficient or absent expansion of this portion of the lung from adhesion and other causes, such as pulmonary apoplexy, and the imperfect general use of the left lung. These views derive additional confirmation from the fact that in all the cases save one in which the impulse

right boundary was situated midway between the right nipple and the edge of the sternum ; and its left boundary extended to the sixth and seventh ribs at the outer side of the chest.

The impulse on the fifty-third day was present in the fourth, fifth, and sixth spaces from two inches within, to two inches without, the nipple line, and was quite absent from the sternum and the spaces between the cartilages; since that time the patient has been getting gradually worse ; and the impulse has been becoming gradually stronger and more extensive, and is now, on the eighty-ninth day, felt over the whole sternum, the epigastrium, and the cartilages to each side, and on the left side down to the seventh left rib, where it beats against the outer side of the chest (see the curved lines occupying all that region). The impulse heaves up rather slowly during the systole, and immediately after it falls suddenly backward. The impulse in the first and second spaces, over the pulmonary artery, is double, protruding slightly during the systole, and going back with a flapping rapid movement during the diastole, conveying the impression of a sharp impulse or shock, synchronously with the second sound. Ninety-first day : The impulse is still felt over the sternum, but feebler than two days ago, similar in character, but not felt.

extended over the higher spaces during both the acme and the decline of the effusion, there was endocarditis with mitral incompetence, and in several of them, aortic incompetence also.

Position of the Impulse after the Decline of the Pericardial Effusion during the Later Stages of Rheumatic Pericarditis; and after its Cessation.—When the effusion disappears and the heart resumes its natural position, and when the lungs again cover the great vessels and the upper part of the organ in front, the impulse as a rule descends into its natural position, and is again felt in the fourth and fifth spaces.

In those patients in whom the heart becomes again healthy after the attack, the size, position and customary beat of the organ are restored: but in those in whom valvular disease is established, the nature and extent of the disease are made apparent by the force, extent, and position of the impulse. When the resulting mitral disease is severe, the impulse of both the right and left ventricles is extended, and is felt beating from the lower half of the sternum to the left nipple. When, however, the mitral affection is slight, and such as scarcely or not at all to interfere with the function of the organ, then the impulse resumes its natural boundary and strength; and thus the impulse becomes a true measure of the extent of the valvular disease. When both the aortic and mitral valves are affected, the apex-beat and the impulse generally of the left ventricle become more markedly developed, the action of the right ventricle being still unduly strong. In those comparatively rare cases in which the aortic valve is alone affected, the right ventricle is untouched; but the size and force of the left ventricle are increased in exact proportion to the increased labour thrown upon that cavity

by the degree of the crippling of the valve. The apex-beat and general shock of the left ventricle become extended outwards beyond the left nipple, and downwards into the sixth space, when the valvular affection is great; but they are held almost within the natural limits when it is slight. When the heart becomes adherent and there is disease of one or more of its valves, the impulse of the organ becomes extended in every direction—to the right, over and beyond the sternum; to the left, beyond the line of the nipple; downwards, over the ensiform cartilage, and even below it in the epigastrium; and especially upwards, to the second space and to the adjoining portion of the sternum. In some cases the whole impulse bears at first forwards during the systole, and then drags the walls of the chest in a characteristic manner backwards; while in other cases, in which there is complete fibrous attachment of the adherent pericardium to the sternum, that bone and the adjoining costal cartilages are steadily drawn inwards during the systole, and spring forwards with a shock during the diastole. An essential difference is also established between the influence of respiration on the area of the impulse of the adherent and the non-adherent heart. When the heart is not adherent, a deep inspiration, by drawing down the heart and covering it with the expanded lungs, causes a complete transfer of the impulse from the fourth and fifth spaces to the epigastrium and the sixth and seventh cartilages; but when the heart is adherent, the outspread dragging impulse almost retains its position during a deep inspiration, neither materially lessening its area over its upper borders, nor materially increasing it below. There is, in short, no transfer, such as occurs when there are no adhesions, of the impulse during a deep breath from the intercostal spaces to the ensiform cartilage and epigastrium and the adjoining left costal cartilages. Thus in

a patient who has recovered from rheumatic endo-pericarditis we are enabled to judge by the position and force of the impulse, whether the valves, if affected, are seriously or only slightly affected; and, by the extent to which the play of the impulse is influenced by respiration, whether the valvular affection is combined or not with extensive and binding adhesions of the heart.

Vibration or Thrill felt by the Hand over the Region of Pericardial Friction.

A sense of vibration or thrill was felt over the seat of the friction-sound at the region of its greatest intensity in fully one-fifth of the patients with rheumatic pericarditis (13 in 63).

In seven of the cases, or more than one-half of them, the thrill was felt over the whole region of the impulse, extending in two instances over the second and third left spaces; in one, over the spaces from the second to the fifth; in three over those from the third to the fifth; and in one, from the fourth to the sixth spaces.

In two other instances the thrill was confined to the second space, apparently over the pulmonary artery, in three to the region of the apex, and in the remaining case it was present both over the second space and the apex. In all these patients the friction-sound was harsh and grating, vibrating, or creaking in character.

In those cases in which the vibration was felt over the whole region of the impulse, the thrill was present at the time of the acme of the effusion, or in one instance two days after it (see pp. 17-19); and the same may be said, with one exception, of those in which the vibration was felt in the second space.

The duration of the thrill was short. It was observed for only one day in seven cases, for two days in three, for three days in two cases, and for four consecutive days in the remaining one. In two cases (51, see p. 19; 40, see p. 17), the thrill, after being absent from its previous seat over the body of the heart for several days, returned over a limited space when the surfaces were comparatively dry, the effusion having disappeared.

The character of the friction-thrill or vibration is peculiar, and differs from the thrill due to altered blood-currents chiefly in the following points. The blood-thrill presents a succession of equal vibrations, often like those made by a vibrating musical cord; is diffused; has a focus of greatest intensity, from which it lessens and fades away all round; gives the impression to the hand of being deeply seated as well as superficial; begins, when diastolic, after the impulse ends, and often continues, when systolic, for a short period after the cessation of the beat of the ventricle; retains its character, position, focus of intensity, and general outspread, unchanged or with only slight modifications from day to day; and finally, has a long previous history pointing to an affection of the heart, and probably dating from an attack of acute rheumatism. The friction-thrill or vibration, on the other hand, is shallow, giving a sensation as if it were made just under the hand by the rubbing together of two rough surfaces; has often a grating, rasping, or irregularly vibrating character; presents no focus of intensity, but is spread, with varying force, over the region of the impulse; begins and ends rather abruptly, being limited to the period of the impulse and not passing beyond or preceding it; does not end with an abrupt shock; is short-lived and transient, and, if felt on one or two following days, it always changes in extent, and perhaps in position, and alters in character; and

finally has a short previous history of local pain, extended dulness on percussion, increased prominence over the region of the pericardium, and elevated impulse. Sometimes, however, the blood-thrill and the friction-thrill are so much alike that they cannot be distinguished by the hand. The character of the thrill is, however, at once cleared up by the ear; the friction-thrill being accompanied by a friction-sound which is in all cases increased by pressure, and is most vibrating, grating, or creaking and harsh at the very seat of the vibration; while the blood-thrill is accompanied by the murmur, usually musical, that distinguishes the valvular affection.

The thrill of presystolic murmur is distinguished by the position of the thrill over and to the left of the interventricular septum, the peculiar large vibrating character of the murmur; the abrupt shock with which the thrill and murmur terminate; the persistency of the thrill, murmur, and shock from day to day; and the long previous history.

The character of the friction-sound presented in the various cases a close approximation to the character of the thrill or vibration.

The sensation conveyed to the hand when applied over the seat of thrill in the thirteen cases under examination was not always of the same character. Thus, under these circumstances the hand felt a sense of grating or rasping in two, of vibration in four, and of thrill in seven of the cases.

On listening over the region of the thrill or vibration in these cases a loud harsh friction-sound was heard in seven patients, in five of whom the sound was described as being "to and fro;" in five others of them there was a noise resembling the creaking of leather; in three the sound was grating, in one rasping, in two vibrating, in one grazing, and

in one "churning." In several of these cases the friction-sound presented, as we have already seen, different phases at different periods of their progress. In all of them the friction-sound became less harsh and extensive when the vibration or thrill over the region of the pericardium ceased to be perceptible.

It is to be remarked that when the thrill was perceptible in these cases, especially if it extended over the ventricles, and was not limited to the region of the apex or that of the pulmonary artery, the area of the friction-sound was increased as well as the intensity. In one of the cases the rubbing sound was audible over the whole front of the chest, and in several of the patients it spread downwards to the ensiform cartilage and to the left and right seventh and eighth costal cartilage.

The character of the friction-sound, associated with the presence of a thrill over the heart and great vessels, whether creaking or grating, vibrating or rustling, or to and fro, will be considered in the next section.

Auscultation.

Position and Character of the Sounds heard over the Heart and Pericardium during the Early Stages of Pericarditis.—In more than one-half of my cases of rheumatic pericarditis (33 in 63), I observed the character of the sounds of the heart at or soon after the commencement of the attack, and before the effusion into the pericardium had arrived at its height. I was frequently surprised by the rapidity with which the affection attained to its acme. In twenty-three of these patients friction-sound was heard for the first time before the fluid in the pericardium had reached it greatest amount; and

in fifteen of these the rubbing-sound was detected only one day, and in four two days before the time of the acme.

Modification of the Sound of the Heart at the Commencement of Pericarditis, before the Occurrence of Friction-murmur or Friction-sound.—There were five cases in which the sounds of the heart were modified before the occurrence of a friction-sound, or the period of the acme. In one of them the heart-sounds were muffled two days before the occurrence of the friction-sound and the acme; in three of them those sounds were ringing in character from three to four days before the acme; and in one of these the systolic-sound was rough and unduly prolonged four days before that period. All the cases of this group but one presented on pressure either a single or double murmur or a rubbing-sound subsequently to this modification of the heart-sounds, and before the occurrence of the acme.

Position and Character of the Friction-murmur, influenced by Pressure, heard at the Beginning of Pericarditis.—A murmur, which was excited or rendered more intense by pressure, was heard over the region of the heart before the period of the acme of effusion into the pericardium in eight cases.

Pain was felt directly over the seat of the pericardial inflammation in seven of the cases, being excited by pressure on the surface of the chest in three of them. In five of the cases the pain was present at the same time as the appearance of a murmur on pressure, and in two the pain preceded the murmur by a day or two.

In four cases the friction-murmur was single and systolic. In four cases a double murmur, excited or intensified by pressure, preceded the friction-sound and the acme of

pericardial effusion. In the last case of this group (49, see p. 31), a youth aged 17, a fatal case, the friction-murmur prevailed more or less through the whole of the illness until the heart became adherent.

The double friction-murmur, heard during the early period of pericarditis, is thus distinguished from the double murmur caused by aortic incompetence, combined as it usually is with mitral regurgitation. It is accompanied, and often preceded, by pain over the heart, usually increased by pressure; it comes into play suddenly; its area is limited to the middle, or lower half of the sternum, and the adjoining left, and, on rare occasions, right cartilages; it is accompanied by the natural heart-sounds, but is not rhythmical with them, the heart-sounds and the murmur being heard as it were side by side; it does not begin with a double accent or shock, the double accent or shock of the natural heart-sounds, but is of equal intensity throughout; it is invariably rendered more intense by pressure, which often converts it into a true to-and-fro *frottement*, and which always obscures or silences the natural heart-sounds. It is not accompanied by marked visible pulsation of the great arteries in the neck, or by the sudden pulse at the wrist of aortic regurgitation, audible when the arm is raised; it is accompanied by extended dulness on percussion over the region of the pericardium; and as a rule it speedily gives place to a friction-sound, with which, however, it may co-exist, being audible beyond the circumference of the friction-sound especially below, and on either side.

In all these respects the double friction-murmur contrasts notably with the double aortic murmur; which is not usually accompanied by pain over the heart; does not come into play suddenly; is not limited in its area to the middle or lower half of the sternum and the adjoining cartilages—but

extends also to the upper portion of the sternum and to its right; is rhythmical with the natural heart-sounds; commences with a double accent or shock; is not rendered to a material degree more intense by pressure, which never converts it into a friction-sound, and which never abolishes the double accent with which the double murmur begins; is accompanied by marked visible pulsation of the carotid and radial arteries, the pulse of the latter becoming audible as a shock when the arm is raised; is not accompanied by extension of dulness over the region of the pericardium; and does not give place suddenly to friction-sound, but is persistent.

The single systolic friction-murmur is not so easily distinguished from the tricuspid murmur as from other systolic blood-murmurs, but their differences are sufficiently marked. The systolic friction-murmur is accompanied or preceded by pain over the heart, usually increased by pressure; comes into existence suddenly; is limited usually to the base of the right ventricle, being heard over the middle or lower sternum or over the fourth left space; is accompanied by the natural first sound, but is not rhythmical with it, the heart-sound and the murmur being distinctly heard side by side; does not begin with an accent or shock, the accent or shock of the natural first sound, but begins and ends with a single note of equal intensity throughout; extends rarely beyond the period of the systole into that of the diastole; is usually produced, and invariably rendered more intense by pressure, so that it obscures or masks the natural first sounds; is accompanied by extended dulness on percussion over the region of the pericardium; and speedily gives place to a double friction-murmur or a friction-sound.

The several systolic blood-murmurs may be thus distinguished from the single or systolic friction-murmur.

The tricuspid murmur is more likely to be taken for a friction-murmur than any other systolic murmur, for it is situated over the front of the right ventricle—over and to the left of the lower half of the sternum—and, like the friction-murmur, it is a shallow sound, and it may appear and vanish quickly. It differs, however, in these respects: it is rarely accompanied by pain and tenderness over the heart; is never accompanied by the natural first sound over the right ventricle, for that sound is converted into the murmur; always commences with an accent, the accent or shock of the first sound of the right ventricle; may be intensified, but is not changed in character by pressure, which, however, brings the ear more close to the murmur; is not accompanied by extended dulness on percussion over the pericardium; and does not give place to a double friction-murmur or a friction-sound.

The systolic mitral murmur is readily distinguished from the friction-murmur by the intensity with which it is heard to the left of and below the apex; and its great relative feebleness, or silence over the right ventricle—to the left of the lower portion of the sternum; and by its persistence. When the mitral murmur is audible in the situation just spoken of it is feeble, and is accompanied by the natural sounds of the right ventricle. The heart-sounds and the murmur are rhythmical and go well together; and pressure, though it makes the mitral murmur somewhat more clear, does not mask or obliterate the healthy sounds of the right side of the heart.

The direct aortic, and pulmonic systolic murmurs are distinguished at once from the systolic friction-murmur by their situation above the level of the third cartilage; the pulmonic murmur which is often scratching in character, and is therefore apt to be mistaken, when first heard, for a friction-sound,

being limited to the second left space; and the direct aortic murmur being heard over the upper sternum, and to the right of it, and in the neck over the carotid.

The essential features of difference between the friction-murmurs and the blood-murmurs are these:—The friction-murmurs do not begin with an accent, but usually maintain the same tone and pitch throughout; while the blood-murmurs begin with an accent or shock: the friction-murmurs are intensified and altered by pressure, becoming sometimes rubbing in character; while the valve-murmurs are only intensified by pressure: the friction-murmur and the natural heart-sounds are heard at the same time, but they do not play together or in unison, being audible as it were side by side, each having its own rhythm; and on pressure the friction-murmur becomes so loud and even rubbing in character as to mask and extinguish the heart-sounds; while the blood-murmurs are in perfect accord with the heart-sounds: the friction-murmurs come suddenly, with pain and increased pericardial dulness, and are transient; the blood-murmurs come gradually, without pain or increased dulness, and are permanent.

Friction-sound in Pericarditis before the Occurrence of the Acme of the Effusion into the Pericardium.—Friction-sound was heard during the early stage of pericarditis, in every gradation from a sound scarcely to be distinguished from a murmur up to a grating, vibrating, or creaking noise.

In a few of the cases, the early friction-sound was not audible until pressure was made over the heart. In nearly all the cases, the friction-sound was double from the first, but in two, and perhaps three patients the sound was single and systolic when first heard. In a small group of four patients, a smooth or feeble double friction-sound, intensified

by pressure, came into play from one to four days before the occurrence of the acme of the affection, when the friction-sound became louder and more harsh.

In the last great division of cases of pericarditis with friction-sound before the acme, the double friction-sound, as a rule, was loud and harsh, was intensified by pressure, and set in suddenly; and the infusion into the pericardium speedily attained to its acme after the first observation of the friction. This set of cases divides itself into three groups: in the first group (1), the friction-sound became inaudible during the acme; in the second (2), the friction-sound became less loud and harsh during the acme; and in the third group (3), the friction-sound remained during the acme with little or no change.

(1) In two cases, the friction-sound, harsh at the onset, disappeared during the acme. It is difficult to explain the disappearance of the friction-sound at the time of the acme of the effusion in these two remarkable cases on physical grounds, but the following circumstances show that it was mainly due to lowering of the power of the heart. It is natural to expect that when the fluid increases, it should interpose itself between a portion of the right ventricle and the anterior wall of the chest, and so limit the area of the friction-sound, and lessen its intensity. This will not, however, account for the disappearance of the rubbing-sound at the period of the acme, since the impulse was then still preceptible, though higher in position and more forcible.

(2) The second group of this division, in which a loud double friction-sound appeared suddenly before the acme of the effusion, and became less loud during the acme, consists of five patients.

The case of this group (33, see p. 23) that I shall relate, is illustrated by the accompaning figures (12, 13, p. 64);

during its later stages, by figures 14, 15, p. 108; 16, p. 110. A housemaid, aged 20, came in on the fifth day of her illness, the heart-sounds being natural. On the third day there was increased dulness on percussion over the region of the heart; and a to-and-fro friction-sound over the whole of the region of cardiac dulness, to which it was exactly limited. The impulse was present, as before, in the fifth space, but was higher in position. The dulness and the friction-sound extended from the sternum almost to the nipple, and from the third left cartilage to the sixth, but did not pass beyond the sternum to the right, so that the rubbing-sound was limited to the right ventricle. It was stronger over the sternum than the cartilages, and became everywhere much harsher on pressure. On the fourth, the double friction-sound was heard over the greater part of the sternum, and was audible over the manubrium during expiration only. The friction-sound had somewhat the character of a bellows murmur over the fourth space. It was not quite rhythmical with the sounds of the heart, which were also audible. It was harsher and louder during the systole than the diastole, and was rendered more intense by pressure. On the fifth day, the effusion into the pericardium was at its acme—reaching up to the second space and the manubrium. The impulse was raised from the fifth to the third space. The area of the friction-sound was more extensive upwards, but more limited below. It was heard over the whole sternum, being louder over the manubrium on expiration, over the lower portion of that bone on inspiration, and was most harsh and strong over the middle third of the sternum. The rubbing-sound was heard from the second to the fourth cartilage, but not apparently below it, and was harsh in the third space. A bellows murmur was audible over the fourth cartilage on the light application of the stethoscope; but when pressure was

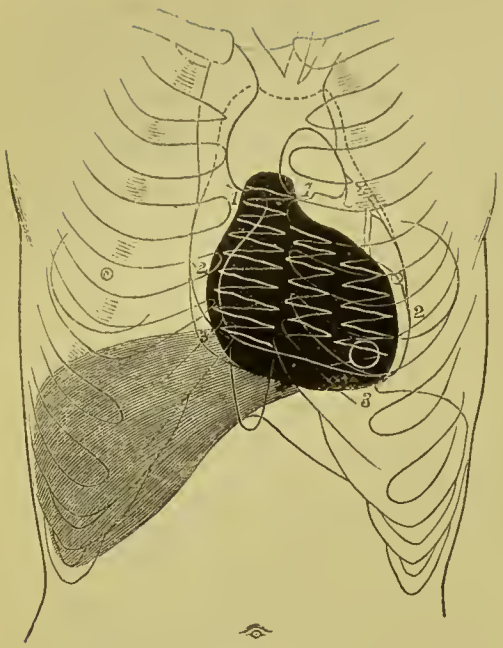


FIG. 12.

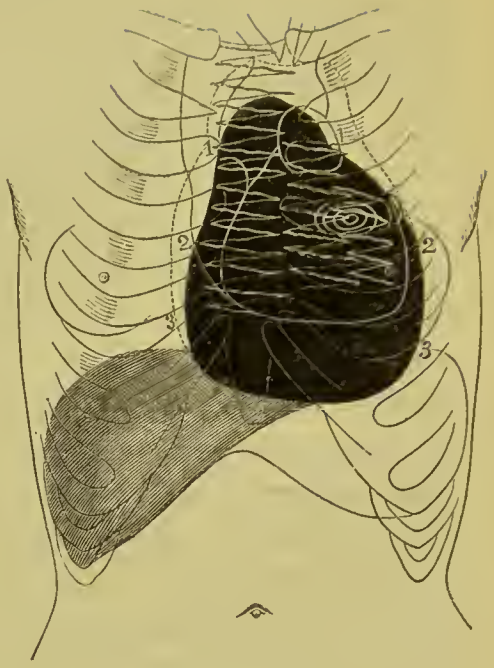


FIG. 13.

Figure 12, from a housemaid aged 20, affected with rheumatic pericarditis (33, see p. 23).

Early period of the increase of the pericardial effusion.

First day of the friction-sound; third day after admission. The sounds of the heart were natural when she was admitted.

The pericardial effusion probably already occupies to some extent the space between the under surface of the heart and the floor of the pericardium, and elevates the heart to a slight degree, and, to a moderate extent, displaces the lungs upwards and to each side; and the centre of the diaphragm, where it forms the base of the pericardium, and the subjacent portions of the liver and stomach downwards. Owing to the displacement of the lungs upwards and to each side from before the heart, the whole of the right ventricle except the upper portion of the conus arteriosus, the inner or left border of the right auricle, and the apex and a portion of the front of the left ventricle are exposed.

Probable region of pericardial dulness on percusssion (see the black space). The outlines of the region of pericardial dulness, which is increased in extent, are not described on this occasion, but the extent of the friction-sound and the position of the impulse are given; and I have assigned to the region of dulness an outline corresponding to the region of friction-sound and the position of the impulse. The region of pericardial dulness has not yet acquired the pyramidal or pear-shaped form that it presents during the acme of the pericardial effusion, but still retains the general form of the healthy

made, a creaking noise was heard there during the systole, and a rubbing sound during the diastole.

I believe that this group and this case represent the natural progress of the friction-sound from the commencement of

region of cardiac dulness, but its outline is considerably enlarged in all directions, and is higher behind the sternum than over the cartilages. It extends across from the right edge of the sternum to the left nipple; its upper boundary probably crosses the sternum on a level with the upper edges of the third costal cartilages, and occupies the third space; and its lower boundary is probably situated a little above the middle of the ensiform cartilage and the upper edge of the sixth cartilage.

Third day. *The impulse of the heart* is felt at the fifth space below the nipple. (See the circle in that space.)

Fourth day. The impulse is feeble, being slightly perceptible below the nipple.

Friction-sound (see the zigzag lines—systolic thick, diastolic thin). Third day. A loud but soft to and-fro friction-sound is heard over the sternum from below the manubrium to its lower end, and up to but not beyond its right border; and over the fourth and fifth cartilages and intermediate spaces, where it extends almost but not quite to the nipple, where it is feebler than it is over the sternum. The friction-sound is rendered much harsher by pressure. Fourth day. The friction-sound is nearly the same in extent, character, and area as it was yesterday, but it is now audible over the manubrium during expiration; it is lower and louder below during inspiration than expiration; and it is louder generally during the systole than the diastole.

Figure 13 from the same patient as figure 14, affected with rheumatic pericarditis.

Period of the first acme of pericardial effusion. Third day of the friction-sound and of the increase of pericardial dulness, fifth day after admission.

The explanations of pericardial effusion and dulness given with figure 4, pag 12, apply also to this figure.

The pericardial effusion completely distends the sac, which is pyramidal or pear-shaped, as in figures 2, p. 2; 4, 5, p. 12; 8, p. 44; 10, p. 48. The extent of the effusion, and of the displacement upwards and to each side of the lungs, and downwards of the diaphragm, liver and stomach, may be inferred from the description given below of the extent of the region of pericardial dulness on percussion. The whole front of the heart is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the pulmonary artery, and the ascending aorta within the pericardium, owing to the extensive displacement of the lungs from before those parts.

The region of pericardial dulness (see the black space) on percussion is pyramidal or pear-shaped, like the distended pericardium. The upper and narrower region of dulness over the great vessels (1, 1,) is situated behind and below the lower half of the manubrium and extends a little way into the adjoining first and second spaces; the larger portion of pericardial dulness, which includes the heart itself and the volume of fluid effused into the space between

pericarditis to its acme when the effusion is at its height. During the first blush of inflammation, the surfaces of the heart and the sac are crowded with vessels, but are as yet scarcely coated with lymph. A single or double friction-murmur, induced or intensified by pressure, may then be the only sound excited by the rubbing of the heart against the pericardium. Speedily their surfaces become coated with a finely honeycombed rugose covering; and the amount of fluid in the sac increases so as to enlarge the area of dulness over the pericardium, and to expose the whole of the right ventricle and the apex, but neither the right auricle nor the great vessels. The heart is slightly raised and the apex beat ascends from the lower to the higher part of the fifth space. A double friction-sound is audible over the whole region of pericardial dulness, to which it is exactly limited, louder and more continuous during the systole than the diastole, and rendered more intense by pressure, which brings into full play both sounds, exciting a to-and-fro rustle or *frou-frou*.

its under surface and the floor of the pericardium (2, 2 ; 3, 3,) extends from the second space down to the lower border of the sixth cartilage, and almost to the end of the ensiform cartilage, and from an inch to the right of the sternum to about half an inch to the left of the nipple. The lower boundary of the heart (2, 2,) is probably situated behind the lower border of the fifth cartilage ; and the heart (2, 2,) extends from this boundary up to the third cartilages : and the volume of effused fluid between the under surface of the heart and the floor of the pericardium extends from the lower boundary of the heart down into the epigastric space, almost to the end of the ensiform cartilage, and the lower edge of the sixth left cartilage.

The impulse has been elevated from the fifth to the third space, and extends outwards to the nipple line. (See the concentric curves in that space.)

The friction-sound (see the zigzag lines—systolic thick, diastolic thin) is double, and extends from the nipple to the lower end of the sternum. It is most harsh about the middle of the sternum, and is louder at the upper end of that bone during expiration, and at its lower end during inspiration ; and is more intense during the systole than the diastole. The frottement is also audible over the left second, third, and fourth cartilages; and is soft without pressure, but with pressure it is creaking over the fourth cartilage.

A mitral murmur —> is audible at the apex.

For the later views of this case see figures 14, 15, 16, pp. 108, 110.

When the effusion has increased to its utmost limits, the heart is elevated, its impulse being raised from the fifth to the fourth or third space; the increased effusion displaces the lungs and so exposes the whole surface of the heart and great vessels; and depresses the central tendon of the diaphragm downwards towards the abdomen, fluid being alone present below the fourth space. The whole region of actual friction is shifted upwards, and with it the whole region of the friction-sound; which is no longer audible below the fourth or fifth cartilage, but spreads outwards over the right auricle and the left ventricle, as well as the right ventricle; and upwards over the great vessels and to the top of the sternum. The friction-sound silenced below is intensified and extended above; so that there is a transfer upwards of the friction-sound; while the dulness on percussion increases in all directions, upwards as well as downwards.

Four cases differed from the rest in this, that while the friction-sound spread upwards at the time of the acme, it also either increased downwards, or, retaining its hold below, increased extensively to the left side.

The comparative relative Area and Intensity of the Friction-sound just before,[1] *and during the Acme of the Effusion into the Pericardium.*—In twenty-nine cases the comparative area and intensity of the friction-sound were observed both before, and at the time when, the effusion into the pericardium was at its height.

Area.—When the effusion into the pericardium increases, the heart is raised, and the lungs are displaced upwards and to the left and right by the increased fulness of the sac and the greater elevation of the heart itself; for the organ is then

[1] At the time of the last observation, made before the effusion had reached its height.

pushed upwards from a wider into a narrower space. It is natural to expect that, under these circumstances, the area of the friction-sound should steadily increase upwards and to each side with the increase of the area of pericardial dulness. This was found to be so in the great majority of instances. Thus the area of friction-sound was greater at the time of the acme than before it in twenty out of the twenty-nine cases; while it was less under the same circumstances in only two of them. In six patients, the area of the friction-sound was equal before and during the acme; and in one case the friction-sound was absent before, but present at, the time of the height of the disease.

These clinical facts show that when the curtain of lung in front of the heart and great vessels is displaced by the distended pericardium and the elevated heart, the friction-sound spreads upwards, and to the right and left; so as to be audible over the whole front of the right ventricle, the great vessels, the right auricle, and the apex.

The lower boundary of the friction-sound, while it retains its place, at the time of the height of the effusion becomes softened in character. The focus of intensity of the rubbing sound is shifted upwards, with the upward shifting of the heart and its impulse; and the intensity of the sound is toned and graduated downwards, from the seat of its focus to that of its inferior limit.

Intensity.—In nearly three-fifths (16 in 29) of the cases, the friction-sound was more intense; and in fully one-third of them (10 in 29), it was less intense, when the infusion into the pericardium was at its height, than just before that time. The tendency, then, is for the friction-sound to increase both in intensity and area, during the acme. The exceptions to this rule are, however, much more frequent as regards intensity than area; for the area lessened at the time of the acme in

only two instances, while the intensity did so at that time in ten instances, out of twenty-nine.

The area of the friction-sound, then, is, as a rule, larger, and its intensity greater at the time of the acme of the pericardial effusion, than at that of the last previous observation, made from one to two days before the acme. The exceptions to this rule are rare as regards the area, but rather frequent as regards the intensity of the friction-sound, which is greater in one-third of the cases on the day before than at the time of the acme. The change, both in area and intensity, is often notably rapid and great ; the character of the friction-sound being sometimes altogether altered, and its area remarkably enlarged in the course of one or two days.

The Character and Area of the Friction-sound at the time of the Acme of the Effusion into the Pericardium.—The friction-sound, audible over the region of the heart and arteries and the pericardium during the acme of the pericardial effusion presented great variety of character, intensity, and area in the forty-four cases under examination. I. In nine of those cases the friction-sound was accompanied during the acme by a thrill over the region of the heart and great vessels ; and II. in thirty-five of them the presence of a thrill was not observed. I. Of the nine cases with a thrill, (1) in five a sound resembling the creaking of a new leather ; (2) in one a grating sound ; and (3) in three a harsh friction-sound was respectively audible over the region of the pericardium. II. Of the thirty-five cases in which a thrill was not observed, (1) in seven a creaking sound was heard ; (2) in two the sound was grating in character ; (3) in fifteen a definite friction-sound, intensified by pressure, which in two instances excited a creaking noise, usually harsh, but sometimes not so, was audible ; (4) in five the friction-sound was soft in character, but was rendered harsh or more intense by pressure, except

in one instance, in which pressure was not employed; (5) in four a friction-sound, previously absent, came into play when pressure was made over the region of the heart; (6) in one friction-sound, present during one, was absent during two of the three days during which the acme lasted; and finally (7), in the remaining case a double friction-murmur, intensified by pressure, was audible over the region of the pericardium during the acme. The cases in the tables at pages 17 to 31 are classified according to this arrangement.

I.—*Cases with Thrill and* (1) *a Creaking*, (2) *Grating, or* (3) *Harsh Friction-sound over the Heart* (see pp. 17-20).—In nine of the forty-three cases under review, a systolic thrill was felt over the heart, and (1) in five of those cases a creaking; (2) in one of them a grating; and (3) in three of them a harsh friction-sound was audible at the seat of thrill at the time of the height or acme of the disease. In six of these cases the thrill was present over the right ventricle, and, in some of those, but not in all, it was probably situated over the left ventricle also; in another of them it was present over the apex and the second space, but not over the right ventricle; in one of the two remaining cases it was felt over the apex; and in the other one over the second space alone.

(1.) *Creaking Friction-sound* (see pp. 17, 18).—In three of the cases with a thrill over the right ventricle, and in one of those with a thrill over the apex alone, a creaking-sound was audible over the seat of thrill.

One of these patients (4, see p. 17), a man aged 27, came in with extensive pericardial dulness; a thrill over the right ventricle extending from the fourth left cartilage to the sixth; a loud systolic creaking friction-sound consisting of five vibrations, the diastolic sound being much smoother than the systolic, over the seat of the thrill; and a double *frottement* extending widely over the front of the chest from the second

cartilage down to the ninth on both sides, and audible at the epigastric space. The pericardial dulness on that day extended upwards to the third space, and on the following day to the third cartilage, when it reached its greatest height. The region of thrill had increased upwards, and extended from the third cartilage to the sixth. A creaking sound was audible apparently over the whole seat of the thrill, but over the fifth cartilage there was a vibrating, grating, systolic friction-sound of churning character, which was creaking towards the end of the systole, the diastolic sound being short and smooth.

(2.) *Grating Friction-sound* (see p. 19).—A grating friction-sound without a creak was present on the presumed day of the acme in one case (51, see p. 19).

(3.) *Harsh Friction-sound* (see pp. 19, 20).—A harsh friction-sound was present with a thrill in three cases. One of these cases (16, see p. 19) a girl, aged 17, came in with an extensive impulse, a double thrill, and a loud, double scraping sound over, but not below, the heart. On the second day there was less dulness, and no note of thrill, and the friction-sound was less harsh and extensive: but, on the third day, there was less effusion, the impulse was lower and more diffused, and the friction-sound was much more intense and extensive.

We may, I think, say, on reviewing these cases, that at the time of the acme of the disease, when a thrill is present over the right ventricle, a creaking noise is audible over the seat of the thrill; and that from this noise, as from a focus, a to-and-fro sound radiates in all directions over the front of the chest, reaching far beyond the limits of the region of actual friction, becoming more feeble towards its outlying margins, and spreading almost up to the clavicles, out to or beyond the nipples, and down to the eighth or ninth cartilages;

and that when the effusion lessens and the thrill disappears, the creak vanishes, and the friction-sound softens and limits its area to the region of actual friction, being bounded below by the sixth cartilage. The reason for the great extension during the acme of the friction-sound upwards, outwards, and downwards beyond the region of actual friction in these cases is obvious. The heart, surrounded by the distended pericardial sac, is displaced upwards into the higher and narrower portion of the cone of the chest. It works in a confined space, and rubs with its roughened surface against the roughened surface of the pericardium; and, the lungs being pushed aside, it presses against the sternum and cartilages, and excites vibrations and a creaking or grating friction-sound over the walls of the chest in front of the heart. The play of the two roughened surfaces of the pericardium upon each other induces vibrations, sensible to the hand, that excite consonant vibrations in the superimposed sternum and cartilages; and these parts, acting as a sounding-board, transmit the sound to a distance over the front of the cage of the chest in all directions, and especially downwards. When the thrill is limited during the acme to the second space, over the pulmonary artery, or to the apex of the heart, or is felt both over the apex and the second space, the creaking or grating noise is limited to the seat of the thrill; and the friction-sound does not extend beyond the region of actual friction, excepting perhaps to a small extent over the circuit of the apex. When in such a case the effusion lessens, the heart descends, and the thrill disappears, the friction-sound may spread downwards, so as to reach the eighth cartilage.

II.—*Cases in which a Thrill was not observed over the Region of the Heart or Great Vessels* (see pp. 20-31).

(1.) *Cases in which a Sound like the Creaking of New Leather was audible at the time of the Acme of the Effusion,*

no Thrill being present (see pp. 20-22).—In seven of the forty-four cases under examination, a creaking noise, usually systolic, was heard without a thrill at the seat of the impulse of the heart at the time of the acme.

In all of these cases, and in several of those in which a thrill over the heart was accompanied by a creaking or grating noise, as soon as the fluid in the pericardium lessened and the heart descended, the creaking noise was replaced by a comparatively smooth friction-sound. This occurred on the day after the acme of the effusion in four of the seven cases. This sudden disappearance of the creaking noise with the diminution of the fluid and the descent of the heart, appears to me to show that the presence or absence of the creaking noise depended more on the position of the heart and on the degree and kind of pressure exerted by it during its contraction, than on the character of the roughened coat of lymph covering the heart and lining the pericardial sac, since that lining cannot have changed materially in one day when the disease was at its height. At the time of the acme of the effusion into the pericardium, the heart is elevated so as to occupy the upper and narrower part of the cone of the chest; and beats with force in its contracted space against the cartilages and sternum which confine its movements. When the heart pulsates thus against the walls of the chest, the movements of the former are resisted by the pressure of the latter. The accumulated force of the heart overcomes the resistance of the walls of the chest, and the accumulated resistance of those walls then overcomes the force of the heart; these two opposite forces by turns arrest and overcome each other and give rise to a series of fine jerks or vibrations that may give birth to a thrill, and a vibrating creaking noise. In one case, this creaking noise consisted of five distinct vibrations; and such a succession of vibrations

forms, indeed, the essential nature of the thrill and its attendant creaking sound.

The creaking sound, and the main varieties of friction-sound, may be imitated by rubbing the forefinger on the thumb with varying degrees of force when the back of the thumb rests upon the ear. When the finger and thumb rub gently or with moderate force upon each other, to and fro, the rubbing sound is smooth or harsh in proportion to the gentleness or force employed. When, however, the pressure exerted by the finger on the thumb is great, the resistance to their onward movement on each other causes them to stop in a succession of jerks, which produce a creaking noise.

When the fluid decreases, the heart descends into the ampler space of the chest ; the organ moves with freedom ; and, as it no longer presses with a resisted force against the walls of the chest, the thrill, vibrations, and creak give place to a moderated friction-sound ; which may be so harsh as to sound like the rubbing of sand-paper; or so soft as to resemble a murmur.

(2.) *Vibrating, Grating Friction-sound* (see pp. 22, 23).— The grating, vibrating friction-sound ranks next to the creaking noise in intensity. It is, in fact, a sister-sound to the creaking noise, with which it is closely allied. Thus, it may be audible when there is a thrill, when it may be heard alone, or associated with a creak ; or it may by pressure be converted into a creak; or it may precede or follow, displace or be displaced, by that sound ; or it may, like it, be produced by pressure. The grating sound, like the creaking sound, is the combined effect of pressure and friction, but the pressure is usually less, while the rubbing surfaces are, I believe, more invariably rough, when the sound is grating than when it is creaking. A grating sound was audible during the acme of effusion in two or three cases in which there was a thrill, and

in two in which there was no thrill; and it was excited by pressure in two. It was, therefore, observed in one-seventh of the cases (6 or 7 in 44).

We have already seen that the creaking sound is usually single but it is the reverse with the grating-sound, which is usually double. The grating friction-sound is a jarring, grating, vibrating noise, rough and to-and-fro in character, made in a succession of jerks, each jerk being separately audible, and varying slightly, and the whole series not combining to form one note like the creaking sound, but, as I have just said, a jarring, grating, vibrating noise. I made out, as I have already stated, that in one case the creak was composed of five vibrations, or at the rate of twenty-two vibrations in a second; but, as I took no special note of it, I do not know what number of vibrations were made in a second by the grating noise. I believe, as I have already hinted, that the grating noise is always associated with the rubbing of the two harsh and roughened surfaces of the heart and pericardium upon each other, but I have no direct proof of this at present.

(3.) *Harsh To-and-fro Friction-sound, intensified by Pressure, at the time of the Acme of the Pericardial Effusion* (see pp. 23-28).—Résumé, *including the whole of the preceding cases, whether with or without a Thrill.*—We have just seen that a creaking noise, usually systolic, was present over the heart at the time of the acme of the disease in one-fourth of the cases in which the dulness was observed at or about the period when the effusion was at its height (12 in 44); while in four other cases it was then excited by pressure, and in two it was heard just before the acme of the effusion. Creaking, therefore, was present as a primary sound in twelve cases; as a secondary sound, or from pressure, in four cases; and in two others it was audible just before the acme. We have also seen that a

grating friction-sound, usually double, was present over the heart when the effusion was at or about its height, as a primary sound in three cases in which there was no creaking, and in one or more in which there was creaking; and as a secondary sound in two in which it was excited by pressure; while in four others it was present just before or after the period of the acme of the disease.

If we combine the two sounds, we find that during the acme the creaking and grating sounds were primary in fifteen cases, and secondary, or excited by pressure, in six; while they were associated with each other in one or more. Besides these fifteen cases, in which creaking or grating sounds were primary, there were nineteen cases in which there was a definite friction-sound, which was usually harsh; in all of these it was double, or to-and-fro in character, being audible both during the systole and the diastole of the ventricle, and in all but two it was intensified by pressure. Three-fourths, therefore, of the patients (34 in 44) in whom the pericardial dulness was observed when at or near its height, presented either a systolic creaking noise, or a double grating, or a definite to-and-fro friction-sound, usually harsh in character.

Besides the nineteen cases in which there was a double *frottement*, usually harsh, at the time of the acme, there were seven cases in which that sound was associated with a creaking noise; and in one it accompanied a grating noise. In these cases the creaking or grating noise was limited to that part of the right ventricle, or the apex, that was pressing with the greatest force upon the costal cartilages or sternum, while the double *frottement* pervaded and often overstepped the rest of the heart and the great vessels.

If we group together the eight cases with harsh double *frottement*, in seven of which the *frottement* was associated with a creaking sound and in one with a grating noise, and

the nineteen cases not so associated, we find that in one-half of those twenty-seven cases the character of the sound is definitely specified (13 in 27); while in twelve it is described as a harsh double friction-sound; and in two as a to-and-fro sound.

Of the thirteen cases in which the character of the double sound was specified, in four it was described as being like that made by rubbing with sand-paper; in seven as being either rasping, or musical, planing, scraping, scratching, grazing or rustling, the latter sound being a genuine *frou-frou;* while in the remaining two the sound resembled that made by sharpening a scythe.

In the whole of the twenty-seven cases except two, pressure with the stethoscope intensified the double *frottement;* it sometimes altered or modified the character of the sound; and in five instances it transformed the double *frottement* into a creaking sound. When the creaking sound was thus brought into birth by pressure, or secondary, it was usually double; but when the creak was always present, or primary, it was, as I have already shown, usually and essentially single or systolic.

In all these cases the double *frottement* was essentially a to-and-fro sound. The character and volume of the sound, and the relative intensity of the to-and-fro, or the systolic and diastolic friction-sounds, varied over the different parts of the heart. As a leading principle, the greater the pressure exerted by the heart, or any portion of it, during its action upon the cartilages or sternum against which it beat, the more intense was the friction-sound.

The friction-sound in the remaining cases of this group was limited to a comparatively small area.

In two of the nineteen cases, in both of which there was a thrill over the right ventricle, the rubbing noise, as I have

already stated, extended over the front of the chest, far beyond the region of actual friction. These two cases, however, stand apart, for in the remaining seventeen the area of the friction-sound was limited to the region of actual friction ; with, however, this slight exception, that in six of the patients the to-and-fro sound spread upwards to the top of the sternum, and in one of them it was diffused outwards as far as the left arm-pit. The upper limit of the distended pericardial sac and of actual friction is rarely higher than the transverse centre of the manubrium, which is about an inch below the top of the sternum ; therefore in the six patients just spoken of, the friction-sound extended itself upwards for from an inch to fully two inches above the actual seat of friction over the great vessels, which, at their higher portion, are partly covered by lung.

The explanation of this extension of the friction-sound upwards beyond the immediate seat of friction is the same as that of the diffusion of the friction-sound over the front of the chest far beyond the region of the distended pericardium and of actual friction, when a thrill and a corresponding creaking noise are present over the heart. The to-and-fro movements of the heart upon the pericardial sac, both being covered with lymph, excite a to-and-fro sound which is audible over the region of those movements. The vibrations that produce the sound are communicated to the sternum, which is played upon by the rubbing surfaces; and the sternum, which acts as a sounding-board, propagates the sound to its own upper end, which is at some distance from the seat of the parent vibrations. The extension of the friction-sound beyond the region of actual friction depends on the loudness and intensity of the rubbing noise, and the force with which the heart, when it is rubbing to and fro, presses against the sternum and cartilages. Of the six cases in which the to-and-

fro sound mounted to the top of the sternum, in three there was a creaking sound over the heart, with a thrill also in two of those; in two others a creaking sound was excited by pressure; and in the remaining one a loud, harsh, double friction-sound was present over the region of the pericardium. Although a creaking friction-sound was audible over the apex in four instances, in only one of them did the to-and-fro sound spread to the left beyond the apex, but in that one the rubbing sound extended outwards into the left arm-pit. In that case there was dulness over the left lower lobe, and bronchial breathing between the left axilla and the spine. It is, therefore, evident that the heart and pericardium were displaced towards the left side owing to the condensation of the left lung, and that this circumstance facilitated the extension of the friction-sound to the left axilla.

With these few exceptions, the region of friction-sound coincided in these cases with the actual region of friction at the time when the effusion into the pericardium was at its height.

(4.) *Cases in which a Soft Friction-sound, audible over the Heart at the time of the Acme of the Effusion into the Pericardium, was converted by Pressure into a Harsh Rubbing Noise* (see pp. 28, 29).—Four cases with a soft friction-sound, in which pressure rendered the sound harsh, come under this heading, and in one of these the friction-sound elicited by pressure resembled the noise made by sharpening a scythe. In a fifth case, with a similar friction-sound, the pressure test was not employed.

In these four cases a comparatively soft double friction-sound was intensified and altered by pressure, becoming converted in one instance into a sound like that made by sharpening a scythe, and in one into a rasping, grating noise. Here pressure expelled any interposed fluid; brought

the opposite roughened surfaces of the pericardium more closely into contact; and aroused counter-pressure on the part of the heart against the cartilages and sternum during its to-and-fro rubbing movements. These effects spoke out not only in a louder and more diffused, but also in an altogether altered sound; so that the soft sounds, sometimes so murmur-like as to be almost doubtful in quality, became instantly transformed into a loud double and broken noise, like that made by sharpening a scythe, or into a rasping, grating, almost creaking sound.

(5.) *Cases in which a Double Friction-sound, not otherwise audible, came into play when Pressure was made over the Heart during the time of the Acme of Pericardial Effusion* (see pp. 30, 31).—In four cases during the acme, on listening without making pressure, the healthy sounds of the heart were alone audible; but on making pressure those sounds were either replaced or accompanied by a double rubbing noise.

In three of these cases, at the time the effusion into the pericardium was at its height, when the stethoscope was applied lightly over the heart, the natural heart-sounds were alone heard, friction-sounds being everywhere inaudible. When, however, pressure was made with the stethoscope, a double friction-sound was immediately brought into play, which could be suspended or renewed at will by withdrawing or replacing the pressure. In one case the friction-sound thus generated was limited to the region of the right ventricle, and in another to the base of that ventricle; but in a third case it was diffused over the whole space occupied by the heart and great vessels. The impulse was feeble in one of these patients, and was felt over the right ventricle in another. It is difficult to say why friction-sound was absent without pressure over the seat of the impulse; but it is self-evident that if we press

the cartilages or sternum inwards upon the walls of the heart moving to and fro, those walls will work with increased counter-pressure against the resisting walls of the chest; and may thus elicit a friction-sound when previously absent, or intensify a friction-sound already existing, owing to the increased friction of the two roughened surfaces. In two of the cases a to-and-fro sound was audible without pressure over the apex, and in one of them over the lower border of the right ventricle also; but it was brought into play by pressure over the whole region of the heart and great vessels.

The subsequent history of these cases illustrates with great clearness the cause of the absence of friction-sound without pressure, and its presence with pressure during the acme of the disease. In three of them (15, 8, 19), as soon as the effusion into the pericardium lessened, the heart descended, and its impulse became stronger and lower; the fluid interposed between the front of the heart at its lower border and the pericardial sac disappeared; and the friction-sound came into spontaneous play where it was before absent without pressure. That sound, indeed, gradually augmented in loudness and intensity, and increased in area upwards, sideways, and especially downwards.

(6.) *Case in which Friction-sound was Absent for Two of the Three Days during which the Acme of Pericardial Effusion lasted* (35, see p. 31).—This patient (35), a woman aged 21, came in with great pain and a double friction-sound all over the region of the heart. The pain was relieved by leeches. Next morning the effusion was at its height, but the friction-sound had vanished and could not be brought back even by pressure. That evening there was a return of pain, and a renewal of the friction-sound, which lasted until next day, but again vanished on the fourth day, when the effusion was still at its acme. She was in great

distress from pain over the heart, but the impulse was faint in the third and fourth spaces. Next day there was less effusion, a lowered impulse, and no distress, and friction-sound was rendered audible by moderate pressure over the right ventricle. Why was the friction-sound absent in this case of pericarditis? When we consider that the impulse was perceptible, it must be allowed that the answer is difficult. The loss of blood on the second day and the great distress on the fourth day may in some measure, however, account for the exit of the friction-sound.

(7.) *Case in which a Friction-murmur was Audible over the Heart at the time of the Acme of the Disease* (see p. 31).—This case (49) of a youth æt. 17, presented a long history, and proved fatal on the forty-eighth day. On examination after death, the heart was found to be universally adherent by means of recent lymph. Throughout the whole period, with rare and doubtful exceptions, the inflammation of the pericardium was made evident, not by the ordinary friction-sound, but by a true friction-murmur.

The Area of the Friction-sound during the Acme of the Effusion.—The area of the friction-sound when the effusion into the pericardium is at its height may, on the one hand, be so extensive as to cover the whole front of the chest, extending from the clavicles down to the ninth right and left costal cartilages; or, on the other, be so limited as to be confined to the middle or lower portion of the sternum. This great diffusion, or narrow limitation of the friction-sound at the period of the acme of disease, is, however, comparatively rare; and, as a rule, the area of the friction-sound corresponds either with the area of actual friction, or with that of dulness on percussion over the pericardium.

The friction-sound was audible over a great extent in all those cases, amounting to nine, in which a thrill was felt over

the heart or great vessels, and especially in those in which it was perceptible over the front of the right ventricle.

In all the cases with thrill the friction-sound was audible over the right auricle and ventricle, the outlying portion and apex of the left ventricle, and the great vessels; in all but one of them, also, it extended to the top of the sternum, beyond the region of the distended pericardium over the great vessels. In these cases, as I have already explained, the friction-sound was most intense over the region of the thrill, and it radiated thence over a wide area, becoming gradually less intense from its focus to its extreme limits, being conducted by the sternum and cartilages acting as a sounding-board.

In six of the twelve cases in which a creaking-sound was heard over the heart, the area of the friction-sound extended down to the seventh, eighth, or ninth costal cartilages; but in five of these the creak accompanied a thrill. In the remaining six cases the *frottement* extended to the sixth cartilage, or occupied an unspecified space to the right and left of the sternum. It is evident, therefore, that the great diffusion of the sound in these cases was due more to the thrill than to the creaking-sound that was audible at the seat of the parent thrill.

I need not here specify the exact limits of the friction-sound in the remaining cases.

These clinical facts show that, when the effusion into the pericardium is at its height, if we put out of view those cases in which a thrill is felt over the right ventricle, the friction-sound is, with a slight exception, practically limited to the region of pericardial dulness, or rather of the heart and great arteries. This exception applies to the presence of the friction-sound over the upper end of the sternum, which is fully an inch higher than the uppermost limit of that

region. This was observed in nineteen cases, and in ten of these no thrill was noticed over the region of the heart or great vessels. In all these cases the friction-sound was conducted to the top of the manubrium, from the actual seat of friction by the sternum itself acting as a sounding-board.

When the lower boundary of the friction-sound reaches to the lower end of the sternum and the sixth cartilage, that limit is still within the lower boundary of the region of pericardial dulness, which is situated, when the pericardium is completely distended, behind the ensiform cartilage and along the lower margin of the sixth cartilage. As I have already shown, however, the lower boundary of the heart, and consequently of the region of actual friction, is, in the great majority of cases, above the lower end of the sternum and the sixth cartilage; for the fluid in the pericardium presses the heart upwards, and interposes itself between the lower border of the heart and the walls of the chest in front of that border. The position of the impulse is a good practical test of the position of the actual seat of friction. In three of the seven cases in which the friction-sound was audible as low as from the seventh to the ninth cartilages, the impulse was felt in the fifth space, and in one of them, a case of established valvular disease with enlarged heart, in the sixth space. But with one single exception, in which the beat of the heart was felt in the fifth space, in all the rest of the cases the impulse was not present below the fourth space, and in nine instances its lowest position was in the third space. In the nature of things, the seat of the actual friction behind the sternum, except at its upper portion, corresponded, as a rule, pretty closely with its seat at the intercostal spaces.

In all the cases save one the friction-sound was audible down to the lower end of the sternum at the time of the

height of the effusion, and in twelve of them it was heard over the sixth cartilage. In all these cases, therefore, it is evident that the friction-sound was audible below the actual seat of friction. The sternum is an excellent sounding-board, and the conduction of the friction-sound to the lower end of that bone, by its own resonant vibrations, at once explains the presence of the sound at its lower end. The presence of the *frottement* over the sixth cartilage, an inch below the actual seat of friction, appears to me to call for a different explanation. The observed facts are indeed different in these two cases. The sound heard at the lower end of the sternum is like that at its upper end, usually of the same harsh to-and-fro quality, and of about the same intensity as that audible over the two rubbing surfaces at the middle of the bone. But this, as a rule, is not so with regard to the friction-sound audible over the sixth cartilage, for that is softer, smoother, and less loud than the sound over the seat of the impulse, from one to two spaces higher up. The presence of the soft muscular space cuts off the direct connection between the fifth cartilage and the sixth. The sixth cartilage is, however, directly attached to the sternum, and that bone, acting as a sounding-board, doubtless conveys some of its own resonant vibrations to the cartilage. But it is to be noted that the sound over the fifth space, though softer and feebler than that over the fourth space, is harsher and louder than that over the sixth cartilage. It is self-evident that the sound over the space can scarcely be conducted from the sternum; and I think, therefore, that we must look to the fluid within the pericardium, and to the inner surface and structure of the roughened and thickened pericardium itself, as the principal media by which the sound is conducted in these cases to the sixth cartilage.

If we except those cases in which a thrill is felt over the

right ventricle or at the apex, we find that when the sac is filled with fluid the friction-sound stops quite suddenly along the left and right margins of the region of dulness over the pericardium. This sudden arrest of the rubbing-sound at its outer border is less marked along the right than the left margin. This is, I consider, explained, firstly, by the softer, smoother, and more equal character of the to-and-fro sound over the right auricle than over the right and left ventricles; and, secondly, by the presence of fluid between the compressed right auricle and the walls of the chest in front of it, along its outer border. If, on the other hand, we look at the left border of the distended pericardium, we find that there the solid ventricles by their own pressure and action against the ribs and spaces, displace the fluid and completely occupy the ground. Here we pass suddenly from the loud double *frottement* made by the two rubbing solid surfaces of the ventricle and the rib lined with roughened pericardium, to the silent, soft, non-conducting surface of the lung.

We may, I think, conclude, with the qualifications just stated, that when the effusion is at its height, as well as when it is increasing in quantity, the friction-sound is limited to the region of pericardial dulness; and, though with less rigour, to the region of actual friction; and that the law originally stated by Dr. Stokes, that the area of the friction-sound is usually limited to the region of the heart, is correct in the great majority of cases, during the period of the acme of the effusion.

Before concluding what I have to state with regard to the area of the friction-sound, I would here estimate, as nearly as I can, the extent to which the sound was heard over the various chambers of the heart and the great vessels during the acme of the pericardial effusion in the forty-four cases now under examination.

In one-half of the cases (21 in 44) the friction-sound was audible over the whole front of the heart, including the right auricle and ventricle, the apex and a portion of the left ventricle, and the great vessels. In seven or eight other cases it was heard over the right auricle and ventricle, in four or five of which it was also present over the apex, and in one over the great vessels. In fifteen other cases the *frottement* was audible over the right ventricle, in nine of which it was also heard over the apex, and in four or five over the great vessels. In six of these cases the friction-sound was limited to the right ventricle. If, upon this estimate, we take each portion of the heart separately, we find that the friction-sound was present during the acme over the right ventricle in the whole of the forty-four cases under notice; over the apex of the left ventricle in thirty-four or perhaps thirty-five of those cases; over the right auricle in twenty-eight or twenty-nine of them; and over the great vessels in twenty-six or twenty-seven of them.

Intensity and Character of the Friction-sound over the different parts of the Heart and Great Vessels during the Acme of the Effusion.—When inquiring into the relative intensity and character of the friction-sound over the different cavities of the heart, except the right ventricle, and the great vessels at the time of the acme of the effusion, I shall take into account the forty-four cases now under examination; but as regards the right ventricle I shall limit myself to the twelve cases with primary creaking-sound, the two with grating friction-sound, and the nineteen cases in which there was a harsh friction-sound intensified by pressure, which form a total of thirty-three cases. Although the left ventricle forms the pivot of the heart's action, and does its work with threefold more power than the right ventricle, I shall first examine the friction-sound as it presented itself over the right ventricle,

because it forms the front of the heart; covers the left ventricle except at its left border and apex; and is the main seat of actual friction.

Right Ventricle.—As the right ventricle forms the front of the heart, it is always in contact to a greater or less degree with the anterior walls of the chest. Owing to the distension of the pericardium during the acme, and the elevation of the heart into the contracted space at the upper part of the chest, the heart and great vessels are stripped of the lung that covered them, and press directly forward upon the middle and upper part of the sternum and the higher costal cartilages and intercostal spaces, from the second to the fifth.

The to-and-fro movements of the right ventricle, by rubbing against the opposed surface of the sac, give birth to the to-and-fro friction-sound audible in front of the ventricle. Those movements play from right to left during the contraction of the ventricle, and from left to right during its dilatation (see Figs. 16, 17, p. 112, vol. iii.). The sweep of the walls is very extensive behind the sternum, at the junction of the auricle to the ventricle; thence it gradually lessens, and comes to a standstill near the septum. The friction movements are therefore greater, and the friction-sounds are louder, at the sternal than the costal halves of the cartilages. As the position of the ventricle is raised from the fourth and fifth spaces to the third and fourth spaces, the *frottement* is usually louder over the sternal portions of those spaces, and the adjoining portion of the sternum, than elsewhere.

As the movements made during the emptying of the ventricles are active, and those made during the filling of the ventricle are passive, the increased pressure made by it upon the cartilage and sternum during the systole often intensifies the *frottement*, and, as I have already shown, may even transform it into a creaking noise. Thus, of the thirty-

three cases under examination, in six there was a systolic creak over the right ventricle; in thirteen the systolic friction-sound was louder than the diastolic; in two the systolic and diastolic sounds were equal; and in twelve it is not stated whether there was any difference between the two sounds.

From these clinical facts it is evident that the active friction-sound made during the contraction of the ventricle is, as a rule, louder than the passive friction-sound made during its dilatation. In a small minority of cases, however, the two sounds are equal, and a true to-and-fro sound is produced, the diastolic portion of which speaks with the same intensity, length, and continuousness as the systolic portion. In these cases I believe that the impulse is feeble, and that the systolic friction-sound, like the diastolic, is, so to speak, passive, and is not intensified by the greater pressure from within of the anterior wall of the ventricle upon the walls of the chest.

The *conus arteriosus* of the right ventricle calls for special notice. It is situated behind the third space and the two adjoining cartilages, and as it enjoys extensive play during the systole, when its movements are twofold, from above downwards, and from left to right, the friction-sound is often notably harsh, loud, and to-and-fro in that situation. Sometimes it is there creaking or grating, when it may be accompanied by a thrill. It sometimes resembles the sound made by rubbing together two opposite surfaces of emery paper, of stuff or of silk; or it is rasping, or scratching, or rustling when it may present a true *frou-frou*; or it may, though less frequently, be soft in character. A friction-murmur is, however, rarely or never present in this situation. Pressure readily intensifies and alters the friction-sound over the conus arteriosus, and sometimes converts it into a creaking-sound. As the conus arteriosus is covered in health by a thin layer of lung, it is not usually the early seat of friction-sound; but as the lung, when

once displaced from before it, does not readily replace itself, the rubbing-sound is often heard in this position up to a late period in the history of the case. The friction-sound is notably double or to-and-fro over the conus arteriosus, and this may be accounted for by the ready completeness with which the right ventricle spontaneously fills itself during the ventricular diastole.

The Apex and Outlying Portion of the Left Ventricle.—The apex and outlying portion of the left ventricle are in health covered by the lung. The extent to which the lung thus affords a protection for the apex depends upon the vigour of the individual, the size of the chest, and the amplitude of the lungs. The portion of left lung immediately covering the apex is a thin tongue, the lowermost protruding angle of its upper lobe, which laps round the apex of the organ, and interposes itself between that part and the ribs. During the diastole, when the ventricle is inactive, the covering of lung is complete; but when the ventricle contracts, owing to the combined muscular rigidity of the organ, and the outward pressure of the blood that is compressed by the contracting cavity, it pushes aside the tongue of lung in front of it, so that the apex sweeps against the ribs and their interspaces. It is thus in young persons and those who are not robust; but in strong adults, inured to exercise, the average size of the lung is increased, and the apex is so embedded in the lung, that its proper beat cannot be felt, except perhaps at the end of a forcible expiration, or when they lie on the left side. In one instance (12, see p. 21) and in one only an obscure friction-sound was heard over and limited to the apex before it was audible elsewhere. This was on the day of admission, but on the following day it had left the apex, and transferred itself to the whole right ventricle and right auricle. I can offer no explanation of this exceptional sign.

As a rule, the friction-sound was, as I have said, limited at first to the right ventricle; but as the disease advanced, the increased fluid in the pericardium displaced the left lung and laid bare the apex, so that the friction-sound spread itself from the right ventricle to the left.

When the effusion was at its height the heart was raised, and the apex-beat was felt in the fourth, or even the third space, at or just above and beyond the nipple. Friction-sound was probably audible over the apex during the acme in thirty-four of the forty-four cases now under notice; it was absent from that point in nine; and its presence there was doubtful in one case.

The movement of the apex is, in its nature, the reverse of that of the right ventricle at its junction with the right auricle; for while, during the systole, that part moves from right to left, the apex moves from left to right, and from below upwards. As the active sweep of the apex takes place during the contraction of the ventricles, it is natural to expect that the friction-sound at the apex should be mainly systolic, and the examination of my cases shows that this is so. Of the thirty-four cases in which a friction-sound was audible over the apex, in six it was heard during the systole only; in ten the *frottement* was double, but was more intense and prolonged during the systole than the diastole; and in none was it stated that the two sounds were of equal intensity during the two periods. In six cases there was a creaking friction-sound, usually systolic, at the apex.

When the lower lobe of the left lung shrinks under the double effect of pulmonary apoplexy within the lung, and pleurisy on its exterior, on the one hand, and of compression of that portion of the lung and of the left bronchus, by the great distension of the pericardium, on the other, the apex becomes completely exposed, and extends far to the left. In

one such case (25, see p. 21), a youth, aged 17, a systolic creaking-sound was audible over and beyond the apex, and the friction-sound extended far to the left, ceasing suddenly in the axilla.

Right Auricle.—The right auricle is in health completely screened from the anterior wall of the chest by the middle lobe of the right lung, which separates it from the middle of the sternum and the costal cartilages to the right of the lower half of that bone. Friction-sound is therefore never audible over the right auricle until the portion of the lung that is interposed between it and the right cartilages is pushed aside by the advancing tide of effusion, so as to lay bare the auricle. When the effusion into the pericardium was at its height, a friction-sound was audible over the right auricle in three-fifths of the cases (28 or 29 in 44).

The expansion of the right auricle is quite passive, and its contraction is made with so little exercise of force, that its movement to the right during its period of filling, and its movement to the left during its period of emptying, are made so quietly, that it exerts no pressure on the cartilages during its to-and-fro movements. It is natural to expect that the to-and-fro *frottement*, the *frou-frou* produced by the passive double friction of the right auricle, should be made up of two equal sounds, and as a rule those two sounds were equal over that cavity.

In twelve of the twenty-nine cases in which friction-sound was audible over the right auricle, the systolic and diastolic sounds were equal; in eleven the *frottement* was double, but the relative intensity of the two sounds was not described; and in one a systolic-sound, almost creaking in character, was audible over the right auricle. In this last exceptional case a similar almost creaking noise was heard over the base of the right ventricle at the lower portion of the sternum, and

that was evidently the source of the rubbing-sound over the auricle.

The two sounds made respectively over the right auricle during the two alternate movements of its dilatation, with contraction of the ventricle, and its contraction with dilatation of the ventricle, are not only equal in character, intensity, and continuousness, but they are also more soft and smooth in tone than they are over the ventricle ; this contrast being most remarkable in some of those cases that present a thrill and a creaking-sound over the right ventricle, and the diffusion of a harsh double friction-sound over the whole front of the chest extending downwards even to the eighth or ninth right and left cartilages.

The question here arises whether under these circumstances the soft double friction-sound audible over the cartilages to the right of the lower sternum is due to the immediate friction of the subjacent right auricle ; or to that of the right ventricle, transmitted through the fluid and softened in its transmission ? I think that we must infer that the latter is the usual source of this sound, when we consider that the yielding right auricle is compressed by the fluid in the pericardium at the time of the acme. Why, under these circumstances, the two sounds are usually equal, I cannot say.

The Ascending Aorta and Pulmonary Artery.—In health the two great arteries lie behind the upper half of the sternum and the spaces to the left of it, above the level of the third cartilages. They not only have the bony protection thus afforded them, but they are additionally sheltered by a thin covering of lung that is interposed between them and the bony shield in front, and is made up of the inner adjoining margins of both lungs. The aorta is guarded by the strongest portion of the sternum, and the pulmonary artery lies behind the second space and cartilage, and the adjoining margin of

the sternum. In the early stages, therefore, of pericarditis, friction-sound is never audible over the great vessels. When the fluid increases, the distended pericardium and the elevated heart and great vessels push the double curtain of lungs to each side, so as to bring the great arteries into contact with the sternum and the first and second spaces and cartilages. The heart and great vessels then, as I have already said, occupy the narrower space in the upper portion of the cone of the chest, and there is now no longer room both for them and for the portion of lung superficial to them in health, which is therefore displaced.

In considering the character of the friction-sound over the two great arteries, we must distinguish the aorta from the pulmonary artery. The roots of those arteries, including under that term their apertures, valves, and sinuses, descend and ascend fully half an inch during the successive periods of the systole and diastole of the ventricles; the movement of the systole being more active than that of the diastole.

The root of the pulmonary artery is situated at the front of the heart, and when the lung is displaced from before it, the artery lies immediately behind the second, and sometimes also the first, left intercostal space, the second costal cartilage, and the adjoining border of the sternum. The movement of the pulmonary artery, like that of the conus arteriosus from which it springs, is downwards and from left to right during the systole, and the reverse during the diastole. The friction-sound over the pulmonary artery, is not, therefore, so far as I know, to be distinguished from that over the conus arteriosus. The to-and-fro sound caused by those two adjoining and connected parts must resemble and blend with each other; but while that of the pulmonary artery is situated over and above the second space and the adjoining border of the sternum, that of the conus arteriosus extends downwards

from that point to the fourth cartilage, but widening to the right, so as to occupy the whole breadth of the centre of the sternum.

A peculiar systolic scratching noise, that somewhat resembles a friction-sound, is sometimes audible over the pulmonary artery during the course of acute rheumatism, and is generally associated with endocarditis. This sound is evidently caused by the vibration of the blood advancing during the systole along the artery when not in a state of tension; and is to be distinguished from friction-sound by its limited area, the sound being confined to the second space, and not accompanied by friction-sound elsewhere over the heart; its restriction to the period of the systole and its consequent total want of a to-and-fro character; its freedom from change when pressure is made over it; its unaltering character on successive days; and the absence of pain over the heart or other symptoms or signs of pericarditis.

The root of the aorta instead of being exposed in front, like that of the pulmonary artery, is buried deeply in the centre of the heart, being covered by that artery, the conus arteriosus, and the left border of the right auricle. The root of the aorta cannot therefore cause a friction-sound. The ascending aorta, where it comes into view above the right auricle and behind the lower half of the manubrium, is in health deep in situation, being covered by the adjoining margins of the opposite lungs. When, however, the heart and great vessels are lifted upwards by the advancing invasion of the fluid in the pericardium, the lungs are displaced from before the ascending aorta, which may possibly be pressed against the back of the manubrium. Even then, however, it can only excite a partial friction-sound, for its movements, which are downwards and upwards, are very slight.

Friction-sound was audible at the manubrium over the

ascending aorta and the adjoining portion of the pulmonary artery at the time of the acme of the effusion into the pericardium, and especially during expiration, in twenty-six or perhaps twenty-seven of the forty-four cases under review; but this friction-sound was evidently not generated by the double movement of those vessels, but was conducted upwards by the sternum, acting as a sounding-board, from the harsh double friction-sound over the right ventricle. This was shown by that sound reaching with full intensity to the top of the sternum, which is a little above the transverse aorta, in twenty-six or perhaps twenty-seven of the forty-four cases; and by the close correspondence between the character of the double *frottement* over and above the great vessels at the upper half of the sternum, and that over the right ventricle, at the lower half of the sternum.

At the time of the acme of the effusion into the pericardium the whole heart is raised, and the lungs are separated from each other in front, so that the pulmonary artery, the conus arteriosus and the rest of the right ventricle, the apex and outlying portion of the left ventricle, and the right auricle, are uncovered, and brought into immediate contact with the walls of the chest in front of them.

The whole front of the right ventricle bears upon the sternum and left cartilages with varying force. Sometimes it produces a thrill during its contraction, which may extend over its surface from the third to the sixth cartilages, and is often accompanied by a systolic creaking-sound. At other times, sometimes with, but generally without, a thrill, a double grating-sound or a harsh friction-sound of various tones, the systolic-sound being usually louder than the diastolic, springs up over the whole right ventricle. In rare instances the two sounds are equal. More rarely a soft friction-sound, rendered

harsh by pressure, or a to-and-fro sound, excited by pressure but absent without it, is present over the right ventricle.

A friction-sound is heard over the apex during the acme in about three-fourths of the cases. The apex may, like the right ventricle, present a thrill and a creaking-sound during the systole ; or a loud, prolonged systolic friction-sound, and a short, feeble diastolic one. In no instance are the systolic and diastolic friction-sounds equal over the apex.

During the acme the right auricle in two-thirds of the cases presents over its surface, to the right of the lower half of the sternum, a double, smooth, to-and-fro murmur or friction-sound, equally loud during its dilatation and contraction. This double smooth *frottement* over the right auricle is probably transmitted, softened in its transit, through the fluid, from the noisy and active right ventricle.

The friction-sound, if any, that may be made during the acme by the ascending aorta and the adjoining portion of the pulmonary artery behind the manubrium, is almost always masked by the friction-sound of the right ventricle, which is conducted by the sternum acting as a sounding-board, the sound being thus conducted in more than half of the cases to the upper end of the bone.

The double *frottement* proper to the pulmonary artery when covered with lymph is undoubtedly audible during the acme over the second space, where it must resemble and blend with the double *frottement* proper to the conus arteriosus.

In every instance pressure intensifies the two friction-sounds ; and it sometimes transforms an ordinary *frottement* into a creaking or grating-sound ; or a soft friction-sound into a harsh rubbing noise ; or it excites a friction-sound when one was previously absent.

Second Acme.—*Renewed Increase of effusion into the Pericardium owing to Relapse.*—In eleven cases the effusion into the pericardium, after it had reached its height and commenced to decline, again increased in quantity, and attained to a second acme. Another case that had a relapse and a second acme, that was admitted during the period of the first acme, has not been included in the inquiry that is about to follow. In five of those eleven patients under consideration the fluid, after declining for a second time, again increased so as to present a third acme of pericardial effusion, and in one of the five there was a fourth wave of increase.

I shall examine in these cases with relapse and renewed acme, the comparative height of the pericardial effusion; the extent of the heart's impulse; the area and intensity of the friction-sound; the severity of the general illness; and the intensity of the accompanying endocarditis, and its permanent effect on the functions of the valves of the heart during the period of the later acme.

Extent of the Effusion into the Pericardium.—In five of the cases the effusion into the pericardium was equal in extent during the first and the second acme; while in five it was greater, and in one it was less, during the first than the second acme. In six of the cases, from two to five days, and in five of them from six to eight days elapsed between the end of the first period and the beginning of the second period of the height of the effusion.

Position of the Impulse.—In six of the cases, and probably in a seventh, the impulse at its inferior boundary occupied a lower position by from one to two intercostal spaces during the second acme of the effusion than the first (compare Figs. 16, p. 110; and 10, p. 48 respectively with Figs. 13, p. 64; and 8, p. 44); in two cases the impulse occupied the same position during the two periods; in one instance it

was imperceptible throughout; and in one it was very feeble.

We thus find that in the great majority of the cases the impulse of the heart was lower during the second acme of effusion, or the period of relapse, than during the first acme. The reason is, I think, evident. When the fluid in the pericardium begins to increase during the early period of pericarditis, the heart, which is then yielding in structure and usually of the natural size, is steadily floated upwards by the increasing tide of effusion into the pericardium, which may indeed compress the auricle and lessen the size of the heart. The heart, under the double influence of the inflammation on its exterior, and the resulting thick coating of lymph, on the one hand; and the inflammation on its interior, and the resulting crippling of valves, enlargement of cavities, and the thickening of walls, on the other, becomes increased in size. The whole organ is, in fact, enlarged, and it is often unyielding in its position owing to its tough new covering, and perhaps to partial adhesions that may have already connected the double surfaces of the thickened pericardium and the heart, especially along and near the septum; and although the renewed increase of fluid elevates the heart to a certain extent, this second elevation of the impulse is not usually so great as the first elevation.

Thrill.—A thrill was felt over the heart for the first time during the second acme in three of the cases. In two of them the thrill was present over the apex, and this was the natural effect of the lowered position, greater prominence and increased force of that portion of the heart during the second acme than the first. In the other case the thrill was present over the second left space, but in this patient the second acme was the true one, for the effusion was considerably higher during the second than the first acme. A thrill is, in fact,

more frequently present over the second space during the first acme than the second, and over the apex during the second acme than the first, for the reasons that I have just stated.

Area and Intensity of the Friction-Sound during the Second Acme of increased Effusion into the Pericardium.—During the second acme of the pericardial effusion a creaking friction-sound was audible over the heart in four cases, and a grating noise in one ; a to-and-fro sound in five patients, and a double friction-murmur increased by pressure in one.

In five of the cases the area of the friction-sound was greater, and in four it was less during the second than the first acme of the effusion into the pericardium, and in two it was of equal extent during both periods. In five of them the friction-sound was audible over a lower position during the second acme than the first, and in none of them was the friction-sound lower during the first acme than the second.

In like manner, the friction-sound was more intense in six cases, and less intense in four, during the second acme than the first ; and in one it was of equal intensity during both periods. In four of the patients both the area and the intensity of the *frottement* were greater, and in three they were both less during the second than the first acme.

The following agencies, on the one hand, tend to increase the area and intensity of the friction-sound during the second acme as compared with the first :—The greater size of the heart ; the increased thickness and force of its walls ; the lowered position of the organ and its impulse ; and the greater roughness and toughness of the lymph covering the heart and lining the pericardium.

The following, on the other hand, tend to lessen the area and intensity of the friction-sound during the second acme as compared with the first :—The greater extent to which the

lungs sometimes cover the heart; the restraint placed on the movements of the heart, and especially of the right auricle, by the thickness and toughness of its envelope of lymph; and the adhesions that have often already taken place between the pericardium and the heart; and especially along the septum, between the ventricles, and at the apex. Vital influences blend with and counteract these physical influences in producing the result.

My analysis of the cases does not enable me to assign to each of those causes its proper share in the production of these effects.

In the one fatal case the heart was universally adherent, and in that patient the friction-sound was less in extent and intensity during the second acme than the first, owing, I consider, to adhesions that had already begun to form between the heart and the pericardium.

The friction-sound, as we have seen, was lower in extent during the second acme than the first in one-half of the cases (5 in 11), owing to the lower position of the heart and its impulse during that period.

In five of the cases the friction-sound maintained the same character during the second acme as during the first, but in six others it was altered. Thus, one that had a friction-sound on pressure, one that had a smooth friction-sound harsher on pressure, and one that had a harsh friction-sound creaking on pressure during the first acme, presented, all of them, a creaking friction-sound during the second acme; while one with a to-and-fro sound during the first, gave a grating noise during the second acme.

From what I have just said, it is evident that the influences tending to increase the area and intensity of the friction-sound during the second acme were in greater force than the influences tending to lessen them; and that the friction-sound

was usually more intense and more extensive, especially downwards, during the second acme than the first.

Comparative Area and Intensity of the Friction-Sound just before, during, and soon after the Second Acme of Effusion into the Pericardium.—The friction-sound is, as a rule, louder and more extensive during the second acme of pericardial effusion than either just before or soon after that period. At this stage, therefore, the *frottement* increases with the advance, and decreases with the decline of the fluid.

Degree of the General Illness during the Second Period of Increased Pericardial Effusion.—In five of the cases the illness was extreme, in three it was severe, and in three it was slight or probably so during the second acme.

Of the five cases in which the illness was extreme, the face was anxious in four; there were choreal movements and rigidity, chiefly of the left arm, in one; breathing was laborious in one and quick in four, the respirations ranging from 32 to 48; pain was present over the heart in one, and in another pain was felt, apparently in the side, on a deep breath; swallowing was difficult in two; one had diphtheria; and one raised phlegm tinted with blood.

The patients who were thus affected with relapse of the inflammation of the pericardium suffered more frequently with symptoms of great severity during the first than the second period of the increase of the effusion into the sac. Thus during the first acme in seven patients the illness was extreme, including the five in which it was so during the second acme, and in three it was severe. In one case the symptoms were not described.

Of the seven cases in which the illness was extreme during the first acme, perspiration was very profuse in three; the face was anxious, pallid, livid, or of a leaden hue, in four; there were slight choreal movements in one; breathing was

quick in five, the respirations ranging from 40 to 48 ; pain was present over the heart in four of those seven patients and in two others in whom the symptoms were less severe; and swallowing was difficult or painful in three.

We thus see that pain attacked the region of the heart in six cases during the first acme, and in only one case during the second acme. The breathing also was more urgently affected during the first acme, when they numbered from 40 to 48 in the minute, than during the second acme, when they ranged from 32 to 48.

On the other hand, depression and anxiety were more marked during the second acme than the first.

The general illness was much more often extremely severe during the first acme in those cases that suffered from a relapse, than during the single acme in those that had no relapse. Thus of those patients in whom there was a renewal of the acme, the illness was extreme during the first acme of the effusion in two-thirds (7 in 10 or 11), and severe in one-third (3 in 10 or 11) ; while of those who had no renewal of attack, the illness was extreme in only one-third of the cases (10 in 30 or 32), severe in one-half (14 in 30 or 32), and not severe in one-fifth (6 in 30 or 32). In one case of the series with a relapse, and in two cases of the series without a relapse, the symptoms were not recorded.

Intensity of the Endocarditis accompanying the Pericarditis during the Second Acme of the Effusion ; and Permanent Effect of the Endocarditis on the Valves.—All the cases that had a relapse of pericarditis were affected with endocarditis in an intense degree. One of the patients had old-standing disease of the mitral and aortic valves ; and in seven of them valvular disease was established when they left the hospital, the mitral valve being affected in all of them, and the aortic valve also in three. In three cases the mitral valve, which

was incompetent during the attack, owing to inflammation of the valve, completely regained its function.

The proportion of cases in which the valves were permanently crippled among those who were affected with relapse of the pericarditis was much greater than among those who were not so affected. Thus the valvular incompetence became permanent in two-thirds of the patients with relapse of the affection (7 in 10), three of them being affected with both aortic and mitral disease; and in only about one-fourth or one-third of those who had no relapse (11 in 52 and 7 others who left with lessening murmur).

These clinical facts tend to make it probable that when there is a relapse of the inflammation of the exterior of the heart, there is a relapse also of the inflammation of the interior of the heart and its valves; and that the inflammation when thus prolonged tends to cripple the valves for life.

Second Relapse of Pericarditis with a Third Acme *of Pericardial Effusion.*—In five of the eleven cases with relapse and a renewed increase of effusion into the pericardium, after the fluid began to decline, there was a second relapse, and the fluid increased in quantity for a third time. In one of those cases there was indeed a third relapse followed by a fourth acme of pericardial effusion.

In one of the cases the effusion into the pericardium was equal in amount during the first acme, the second, and the third, the wave of increase rising on each occasion to the same height. In two of them the fluid was equal in quantity during the first acme and the third, but was less during the intermediate period of renewed increase; and in the two remaining cases the wave of increased effusion lessened on each repetition, the effusion being less during the third acme than the second, and less during the second acme than the first.

In those five cases from three to five days elapsed between the second acme and the third.

The impulse, at its inferior boundary, was lower during the third acme than the first in three of the cases; and it was lower in one case and higher in another during the third acme than the second; while its position was unchanged during those two periods in a third. In one of the cases the impulse was imperceptible throughout, and in another it became so at the period of the third acme.

The presence of a thrill was not observed in any of the cases during the third acme.

The friction-sound is in a declining state during the third acme. The *frottement* was of a definite rubbing to-and-fro character, intensified by pressure, in only one of the four cases during the final acme. In one of those patients the friction-sound was double and smooth in character; in another it was single and systolic; in a third it was almost like a bellows murmur; and in the remaining case it was absent with light pressure, but firm pressure brought a to-and-fro sound into existence.

Four of the five patients belonging to this group were affected with great general illness during the final acme; their breathing was distressed and rapid, numbering respectively from 36 to 60 in the minute; while two of them had pain in the chest, and the remaining two pain in the region of the heart.

The Area and Intensity of the Friction-Sound during the Decline *of the Pericardial Effusion.*—In forty-three cases the comparative area and intensity of the friction-sound were observed both when the effusion into the pericardium was at its height, and during the period of its decline.

(1) The friction-sound spread downwards to a greater

extent during the decline than the acme of the effusion into the pericardium, in three-fifths of the cases (26 in 43.) (2) In less than one-fourth of the cases (10 in 43) the reverse took place, the friction-sound being more extensive, and especially downwards, during the acme of the effusion than when the fluid diminished. (3) The area of the friction-sound extended downwards to an equal extent during the acme and the decline of the effusion in a still smaller proportion of the cases (7 in 43).

(1) *Cases in which the Friction-Sound spread downwards to a greater extent during the Decline than the Height of the Effusion into the Pericardium.*—I shall consider (1) the time of occurrence; and (2) the duration of the downward extension of the friction-sound in these cases; (3) the area; and (4) the character of the sound; and the position of the heart and its impulse and thrill; and (5) the degree of the general illness during the period in question.

1. *Time of the Occurrence of the Downward Extension of the Friction-Sound.*—The friction-sound spread rapidly downwards soon after the fluid in the pericardium began to decline in all but a very small proportion of these cases. Thus the rubbing-sound had already extended downwards to its lowest position in four-fifths of the patients (21 in 26) during the first three days after the acme. During the three following days the descent of the rubbing-sound appeared in four more of the cases; but in the last of these this condition came into play quite suddenly on the twelfth, and still more on the fourteenth day after the fluid began to lessen.

2. *Duration of the Extreme Downward Extension of the Friction-Sound.*—The downward extension of the friction-sound lasted in these cases for a very short period. In two-thirds of them (17 in 26) it was observed during only one day, and in but two cases, or rather one, did it last over

three days. This extension downwards of the friction-sound during the decline of the fluid was therefore short and transitory.

3. *Area of the Downward Extension of the Friction-Sound.* —When the fluid in the pericardium, after having reached its height, lessens in quantity, the heart descends, its impulse is lowered by from one to two intercostal spaces, and the friction-sound extends downwards. The area of the rubbing noise is, as a rule, by no means limited to the area occupied by the heart itself; but spreads downwards to an extent varying from one to four inches below the lower boundary of the heart. The friction-sound does not, in these cases, diffuse itself downwards over the whole breadth of the region below the heart; for it is usually silent over the front of the abdomen in the epigastric space; while it is present along the right and left seventh and even eighth costal cartilages that bound that space to the right and left; and over the ensiform cartilage that dips downwards from the lower end of the sternum at the centre of that space.

The rubbing noise is usually heard with equal intensity over the right and the left seventh and eighth cartilages. Sometimes indeed the sound was louder and more extensive over the right seventh and eighth cartilages than the left; and it appeared as if in those cases the cartilages that rested on the liver conducted the sound better than the cartilages that covered the stomach. The contrast between the harsh rubbing noise heard in some cases over those cartilages, and the complete silence present over the intervening epigastric space was very remarkable.

The friction-sound, besides travelling downwards, extended also upwards in one half of these cases (14 in 26) when the fluid in the pericardium lessened, and the heart and its impulse descended. In one-third of the patients (8 in 26)

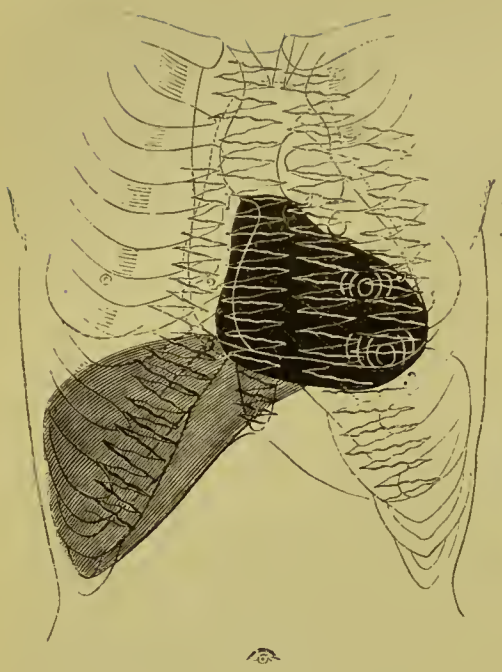


FIG. 14.

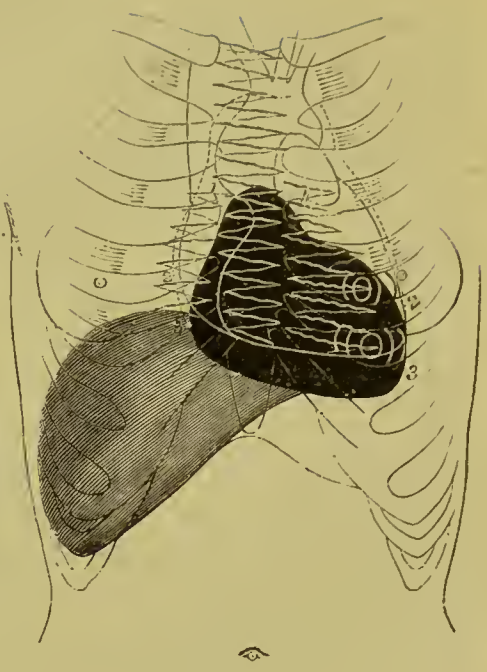


FIG. 15.

For previous views of this case, see Figs. 12, 13, page 64.

Fig. 14, from a housemaid, aged 20, affected with rheumatic pericarditis (33, see p. 23).

Period of the decline of the pericardial effusion after the first acme.

Fifth day after the acme of pericardial effusion, seventh day after the first observation of the friction-sound and increased pericardial dulness, and ninth day after admission.

The pericardial effusion has lessened much since the period of the acme, its upper boundary (1, 1) having descended from the middle of the manubrium to the middle of the sternum; and its lower boundary having probably ascended from a little above the end to about the middle of the ensiform cartilage. The heart (2, 2) is lower, and the amount of fluid between the under surface of the heart and the floor of the pericardium (3, 3), though still considerable, has evidently lessened by at least one-half. The right ventricle, the inner or left half of the right auricle, and the apex and front of the left ventricle are exposed; but the great arteries are covered with lung.

The region of pericardial dulness (see the black space) probably extends from the middle of the sternum between the third cartilages and from the fourth left cartilage, down to the middle of the ensiform cartilage, and the lower third of the sixth cartilage; and from a little to the right of the lower half of the sternum to the nipple line. The lower boundary of the heart is behind the upper edge of the sixth rib, and the top of the ensiform cartilage.

The impulse has descended from the third space during the acme, to the fourth and fifth spaces. (See the circular and curved lines in those spaces.)

the area of the friction-sound was equally high over the front of the chest during the period of the acme of the effusion into the pericardium, and that of its decline.

A double friction-sound (see the zigzag lines—systolic lines thick, diastolic thin), which is more harsh on making pressure, is heard over the whole length of the sternum : which is most intense at the middle of the bone, and is louder at its lower end during inspiration, and over the manubrium during expiration ; a creaking-sound is audible during systole over the third, fourth, and fifth left spaces ; and a friction-sound is heard to the right of the lower portion of the sternum. (The nipple is too far to the left in this figure.)

Fig. 15, from the same patient (33) as Figs. 12, 13 (page 64), 14, and 16.

Period of the decline of the pericardial effusion ; second view, taken the day after a slight and transient second acme, when the fluid was again declining.

Remarkable extension of the friction-sound over the greater part of the front of the chest, and especially downwards.

Tenth day after the first acme of pericardial effusion ; twelfth day after the first observation of the friction-sound and of pericardial dulness ; fourteenth day after admission ; and four days before the occurrence of a second acme.

The pericardial effusion has diminished. There is therefore less fluid between the under surface of the heart and the floor of the pericardium (3, 3) ; the roughened front of the heart is more dry, and is closer to the corresponding roughened surface of the pericardial sac ; the heart (2, 2), which is somewhat enlarged, is lower in position ; and the upper boundary of pericardial effusion (1, 1) is lower, and its inferior boundary is somewhat higher than when Fig. 14 was taken five days previously. The whole right ventricle, the left border of the right auricle, and the apex and front of the left ventricle are exposed ; while the great arteries and part of the conus arteriosus are covered with lung.

The region of pericardial dulness (see the black space), which is bounded above by the fourth cartilage, and below by the sixth cartilage, is probably rather less extensive above, below, and to the right than in Fig. 14, taken on the ninth day after admission.

The impulse is lower, stronger, and more extensive than in Fig. 14, and is present from the third to the fifth spaces, and up to or beyond the mammary line (see the circular and curved lines in those spaces) ; and gives therefore direct evidence that the heart is lower in position, and that the effusion has lessened.

The friction-sound (see the zigzag lines—systolic lines thick, diastolic thin) has gained a very great extension, being audible over a great part of the front of the chest, from the first costal cartilage to the seventh left and the eighth right cartilages ; and from the top of the sternum to the bottom of the ensiform cartilage. This extension of the friction-sound is especially marked downwards, where it extends for about four inches below the heart and is lower on the right than on the left side, reaching over the right eighth cartilage in front

In four instances the whole area of the friction-sound shifted bodily downwards when the pericardial effusion lessened, and the heart and its impulse descended; so that the

of the liver, and the seventh left cartilage in front of the sternum. This is the reverse above, when the rubbing-sound extends four inches to the left, and only about two inches to the right of the sternum.

The friction-sound is harsher than it was yesterday; over the midsternum it is louder during the systole than the diastole; and it is intensified by pressure; over the manubrium, the to-and-fro sounds are equal; over the ensiform cartilage, friction-sound is present during inspiration; it is creaking during systole over the second and first spaces; and it becomes louder below the mamma during inspiration.

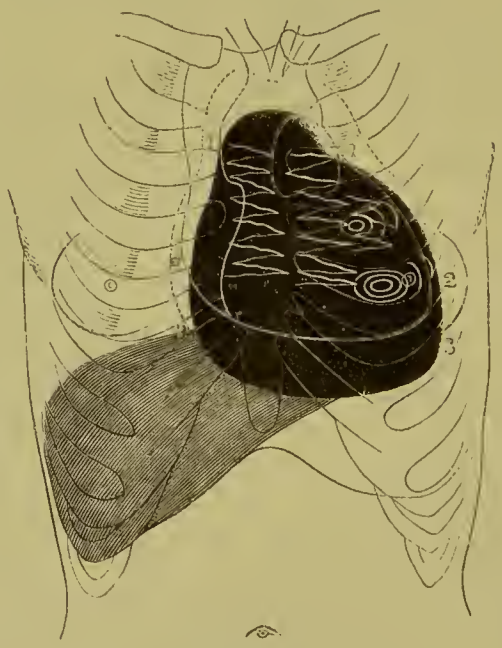


FIG. 16.

For previous views of this case, see Figs. 12, 13, page 64, and 14, 15, page 108.

Fig. 16, from a housemaid affected with rheumatic pericarditis (33, see p. 23.)

Third acme of pericardial effusion (the second acme was very slight and transitory). Thirteenth day after the first acme; eighteenth day after admission.

The pericardial effusion is greatly increased, but its extent and limits are not definitely described. If we compare this third acme with the first acme (Fig. 14, page 108), we find that the distended pericardium, though it contains less fluid, is wider in relation to its length; that the heart is larger; and that the

upper and lower borders of that area were then simultaneously lowered. In these four cases while the lower boundary of

lower boundary of the heart is lower, in this the later and renewed, than in that of the earlier and original acme. In the first acme the heart was not yet enlarged, or being compressed by the fluid in the distended sac, was possibly lessened in size; and the walls of the pericardium were still unyielding, so that the swollen sac took the form that it would naturally take if artificially distended with fluid (see Figs. 1, 2, page 2). In this, the third acme, the heart has become enlarged both by pericarditis and by mitral endocarditis; the lower boundary of the heart, although elevated by the accumulated fluid, is lower than in the first acme; and the walls of the pericardium have become thicker, softer, and more yielding than in health, so that the distended sac yields to the right and left, where it meets with no resistance, to a greater extent than it does upwards and downwards, where it meets with much resistance; and is therefore wider in relation to its length than it was during the first acme, when its form was more purely pear-shaped. The whole front of the heart and great vessels is exposed, including the right auricle and ventricle, the apex and front of the left ventricle, the pulmonary artery, and the ascending aorta within the pericardium. The fluid has evidently interposed itself to a greater extent between the surface of the lower portion of the front of the heart and the walls of the chest during this, the third acme, than during the first acme.

The region of pericardial dulness (see the black space), the limits of which are not described, corresponds in general form and outline to the pericardial effusion and evidently extends more to the right and left, and less upwards and downwards than it did during the first acme.

The impulse has again been elevated at its lower boundary, and this time from the fifth space, as in Fig. 15, page 108, to the fourth space, where it is feeble; and it is felt over the third space during expiration. (See the concentric circles in those spaces.) The lower boundary of the impulse is therefore lower by one space than it was during the first acme, when it occupied the third space (see Fig. 13, page 64).

The friction-sound (see the zigzag lines—systolic lines thick, diastolic thin) is softened, and is limited in area, being heard over the middle region of the sternum, where it is double, and although frictional in character is almost like a bellows murmur; and is audible over the second and third spaces during the systole.

Later progress of this case.—On the following day, the nineteenth after admission, the friction sound was almost creaking, or like the sound made by rubbing with sand-paper, over the second and third left spaces. On the twenty-first day, or the fourth after the third acme, the extent of dulness over the pericardial region had lessened; and a double friction murmur, which was not rhythmical with the sounds of the heart, was audible over the base of the right ventricle, and became harsh on pressure. The friction-murmur was still heard on the following day, and after this it was scarcely audible.

the region of friction-sound descended from the sixth to the seventh cartilages, its upper boundary descended in two of them from the second left cartilage to the third, and in the other two from the third cartilage to the fifth.

In two-thirds of those cases (16 in 26) in which the friction-sound extended much downwards after the acme, it was also audible up to the top of the sternum. In three of those cases the friction-sound so covered the front of the chest as to be audible up to the clavicles, while in one of them it reached the first cartilage. In the whole of these cases the friction-sound extended from two to nearly four inches above the actual seat of friction. The region of pericardial dulness was limited above in all but three of the patients by the third intercostal space or the fourth cartilage; and the space between this limit and the top of the sternum nearly measures the extent to which the *frottement* extended above the seat of the friction.

When the fluid in the sac declines, the roughened heart rubs against the roughened pericardium, and in doing so bears directly upon the lower half of the sternum with which it is almost in contact, owing to the removal of the anterior layer of the fluid and the descent of the heart and its impulse. The sonorous vibrations excited by the movements of the heart are directly conveyed to the sternum, and that bone and the costal cartilages attached to it act as a sounding-board and transmit the rubbing noise in all directions.

In three of the cases the sound was audible over the whole front of the chest. Usually, however, it extended only slightly to the right, and over a greater extent to the left of the lower half of that bone. As a rule, therefore, the rubbing noise extended in a straight line from the top to the bottom of the sternum, and there it divided into two diverging lines, one along the right, the other along the left seventh

cartilage, where they form the boundaries of the intervening epigastric space. The area of friction-sound thus extending along the sternum and the right and left seventh cartilages closely resembles in shape the inverted letter ⅄. Since however the friction-sound also extends downwards over the ensiform cartilage, its area is somewhat like a trident with a short central prong.

In one-fifth of the cases (5 in 26) the area of the friction sound dwindled during a short period after the time of the acme, and then suddenly expanded, and especially downwards, at a later date during the decline of the effusion.

In one case the friction-sound alternately lessened and increased in area and intensity during the ten days that intervened between the termination of the acme, and the time at which the *frottement* had a remarkable downward development.

4. *Character and Intensity of the Friction-Sound; and Position of the Heart and of its Impulse and Thrill.*—At the time that the friction-sound spread downwards when the effusion lessened, the sound was intense, loud, and of a marked character in all but three or four of the twenty-six cases that belong to the group under consideration.

In nine of those cases the friction-sound was creaking (6), or grating (3); in thirteen it was harsh and loud; and in four its intensity was not specified.

The friction-sound in the twenty-six cases under review, as a rule, gained in intensity as it gained in area; and lost in intensity as it lost in area. Thus in all but six of the cases, the rubbing noise became more harsh when it increased in extent; and in all but two of them it became softer when it lessened in extent.

When the effusion lessened, the impulse, while it descended at its lower boundary, was still felt beating in the higher

spaces into which it had been forced during the acme in fully one-fourth of the cases (5 in 19): while, curiously, the impulse ascended to a higher space than it had occupied during the acme in six other patients.

A thrill was felt over the heart in five of these twenty-six cases during the acme of the effusion. In four of these the thrill disappeared when the effusion lessened, and in one it remained, though with lessened intensity. In three other patients a fresh thrill came into play during the decline of the fluid; in two of them over the apex, and in the other case at the second space.

In these twenty-six cases, when the effusion into the pericardium lessened, the heart, relieved from the pressure of the fluid, descended into its natural space, and even below and beyond it. The heart thus relieved, beat with increased force; its right cavities were enlarged, owing to the increased supply of blood from the system, and the continued resistance offered to the flow of blood through the compressed lung and the incompetent mitral valves; and, as the general result, its anterior walls played with greater power and noise upon the sternum and cartilages, and the friction-sound was heard over a largely increased area.

5. *Degree of the General Illness.*—At the time that the area of the friction-sound was most extensive, especially downwards, when the fluid in the pericardium lessened, twenty of these twenty-six cases were less ill or in better health, three of them were probably better, and three were worse in health than they were during the acme.

In a large proportion of the cases under review, when the fluid in the pericardium lessened, the heart descended and gained freedom of movement and power, and the general health improved; and as a natural result the friction-sound increased in extent, and especially downwards. The compar-

atively dry roughened surface of the heart rasped to and fro upon the roughened surface of the pericardium. These influences combined to cause the increased harshness and extension of the friction-sound; which, starting from its focus of greatest intensity over the right ventricle, radiated in all directions over and beyond the region of the heart and the great vessels, outwards to the right and left, upwards to the summit of the sternum, and especially downwards over the ensiform cartilage and the diverging right and left seventh and eighth cartilages.

(2) *Cases in which the Friction-Sound was audible downwards to a greater extent during than after the Acme of the Effusion.*—In ten cases the friction-sound was audible to a greater extent downwards when the effusion was at its height than during its decline.

Two series of influences are at work in these cases, acting at different times, to enlarge the area of friction-sound during the acme, and to lessen it during the decline of the effusion.

1. When, during the acme, the friction-sound is creaking or grating, being sometimes associated with a thrill, over the right ventricle, and when it radiates thence in all directions, softened in character, beyond the region of actual friction, the heart, raised by the increased effusion into the narrower space at the upper part of the cone of the chest, beats with increased force directly against the sternum, the higher cartilages, and their spaces, and so excites an intense and widely diffused friction-sound.

When the fluid lessens the heart descends and is again partially covered with lung; and as it beats over a smaller extent, and with less pressure against the sternum and cartilages, the friction-sound lessens in intensity and area.

2. When the friction-sound is of moderate intensity and

extent during the acme, it sometimes lessens during the decline of the effusion. In these cases the impulse at its inferior boundary is not notably lowered, while it disappears from the upper spaces. In some of these cases the action of the heart is throughout feeble; and probably in others of them slight adhesions take place at the apex and septum which restrain and lessen the descent of the heart, the rubbing movements of the right ventricle, and the area and intensity of the friction-sound over the higher intercostal spaces.

(3) *Cases in which the Friction-Sound extended Downwards to an equal extent during and after the Acme of the Effusion.*—In seven cases the friction-sound was of equal extent during the two periods, when the fluid in the pericardium was at its height and was declining.

Character and Intensity of the Friction-Sound during the Decline of the Effusion, and the Relation of the Intensity to the Area of the Friction-Sound.—I shall examine these conditions during three periods in the order of time of the decline of the effusion, (1) the beginning of the decline of the effusion; (2) the gradual and the interrupted progress of the decline of the effusion; and (3) the final dying away of the friction-sound; and (4) shall then inquire into cases in which the ordinary friction-sound gave place to a friction-murmur towards the end of the attack.

1. *Character and Intensity of the Friction-Sound at the Beginning of the Decline of the Effusion.*—When the amount of fluid in the pericardium began to lessen, if the friction-sound increased or diminished in intensity, it usually increased or diminished also in area.

As a rule, the friction-sound increased in intensity and area in those cases in which the *frottement* extended further

downwards after than during the acme; while it lessened in intensity and area in those in which the friction-sound spread more downwards during the acme than after it.

When the friction-sound spread downwards during the decline of the effusion, the sound gained in area in nearly every case (25 cases in 26), and in intensity in two-thirds of the cases (18 in 26). We thus see that while an increase in the intensity of the *frottement* almost invariably leads to an extension of its area—for I find only one exception to this rule—and while a diminution of its intensity likewise generally causes a diminution of its area; yet, in certain cases, the friction-sound gains in extent, though it lessens (4 cases in 43) or remains unchanged (3 cases in 43) in intensity. This is explained by the lowering of the heart, and the consequent descent of its impulse during the decline of the effusion in all the cases—the surface of the roughened organ being thus brought into more extensive contact with the sternum at its lower half, and with the corresponding costal cartilages: while in the small number of cases in which, although the friction-sound gains in area, it is lessened or not increased in intensity, the heart, released from its confinement in the contracted space of the chest above, where it rubbed with force and noise against the sternum and cartilages in front of it, finds itself moving with ease in its proper place in the lower and wider part of the chest, and so presses with less force and less noise than before on the sternum and cartilages in front of it. The causes of the increased intensity as well as area during the decline of the effusion, which, as we have just seen, occur in the great majority of the cases under examination, have been already considered at page 115.

2. *The Gradual and Interrupted Progress of the Decline of the Effusion.*—In thirty-one of the forty-three cases now being examined, the effusion in the pericardium steadily

and gradually declined, and, as we have already seen, in twelve of them, owing to relapse, the effusion, after beginning to decline, again increased in quantity generally once, sometimes twice, and on one occasion even a third time.

The progress of the friction-sound during the decline of the effusion was rarely uniform. It was in several of the cases silenced and suspended for a time (6 in 43); it more frequently, however, when in full play, became feebler during a short period, and then again louder (13 in 43). In a larger number of the cases the *frottement*, after attaining to its greatest intensity, more or less steadily lessened in loudness and extent until it finally disappeared (23 in 43).

In one case the friction-sound suddenly and permanently disappeared after an attack of syncope. In this patient, a girl, the friction-sound vanished when the action of the heart became enfeebled ; and she died in a second attack of syncope a few hours after the first attack.

Cases in which the Friction-Sound vanished and reappeared during the Decline of the Effusion.—In six of the forty-three cases under review and in one other patient the friction-sound disappeared and reappeared during the decline of the effusion. In five of these cases the *frottement* was absent for from two to three days, and in one of them for about seven days.

In three of the patients the friction-sound, as in the case just referred to, vanished for a time after the application of leeches for the relief of pain.

If we view these cases as a whole, and take into the survey the case of the female servant who died from a second attack of syncope, the first attack having permanently quenched a loud and pervading friction-sound, we shall, I think, see that when the force of the heart's action and the volume of the blood in circulation are lessened—either by immediate

syncope, by loss of blood from leeching, by diarrhœa, sickness, or other exhausting influences, by pain in or over the organ, by extreme distress in breathing, or more often by a combination of several of these lowering agencies—then the rubbing-sound, when in full play, may gradually or suddenly vanish, and may suddenly rekindle into full volume after a longer or shorter period of silence.

Cases in which the Friction-Sound lessened and then increased in Area and Intensity during the Decline of the Effusion.—In thirteen of the forty-three cases under examination, and in three other cases, the friction-sound, when in full play, lessened in extent and intensity, and after a longer or shorter interval again resumed more or less nearly its full sway.

In one of these sixteen cases the diminution of the *frottement* was associated with sudden faintness; in two with loss of blood from leeching; in eight with increase of the general illness—in seven of which as the health improved the friction-sound resumed its extent and intensity—in two with an amelioration of the symptoms; in two with irregularity and intermission of the pulse and the action of the heart; and in two the state of the health is not described.

In eight cases the diminution of the friction-sound corresponded with an increase of the general illness, which showed itself generally by an anxious expression, accelerated and difficult breathing, and pain over the heart; sometimes with cough and rusty phlegm; and sometimes with abundant perspiration. With the renewed increase of the rubbing-sound there was in all these cases, save perhaps one, a marked improvement in the health; manifested usually by a comparatively cheerful expression, more easy respiration, lessening or absent pain over the heart, and assuaging of cough with diminution of phlegm.

(1) *Duration and* (2) *Progress of the Friction-Sound during the Decline of the Effusion.*—(1.) *Duration.*—The friction-sound lasted for a very variable period during the decline of the disease.

In the group of thirty-one cases that had no relapse and no return of the effusion into the pericardium, the friction-sound lasted from three to nineteen days, its average duration being ten days.

In the group of twelve cases that suffered from relapse with return of the effusion into the pericardium, the friction-sound lasted from eleven to twenty-two days, its average duration being fifteen days.

(2) *Progress.—Cases in which the Maximum Development of the Friction-Sound took place during the Decline of the Effusion.—Period between the Maximum Development and the Cessation of the Friction-Sound.*—In thirteen of the nineteen cases under examination the area of the friction-sound steadily lessened from the day of its maximum extension to that of its final disappearance. It contracted gradually from right to left and from left to right, from above downwards and from below upwards, towards the centre or focus of actual friction. It thus died away from beyond and over the great vessels, the right auricle, and the apex, and from the region that it had previously occupied below the lower boundary of the heart. Towards and over the region of actual friction it step by step concentrated itself, and after lingering over the right ventricle with softening tones for a shorter or longer period, it quietly died away. In about one-half of the cases (6 in 13) this subdued sound outlived the period of its greatest intensity and extent for from one to two days; in the remainder, for from three to seven days; and in only one did it exist for nine days.

The front of the right ventricle was, as I have just said, the

last home of the friction-sound, as it had been indeed the seat of its birthplace. As the position of that ventricle varied in different patients accordingly as the heart was larger or smaller in size, higher or lower in situation, the final seat of the softened friction-sound varied in different cases, from the left third and fourth cartilages to the fifth or sixth; and from the middle third of the sternum to the ensiform cartilage.

There was a general but by no means invariable correspondence between the area of the friction-sound on its last observation, and the position of the impulse.

In only three of the nineteen cases now under review did the impulse occupy the same position when the friction-sound was heard for the last time, as when it was most extensive. In four cases it had descended at its lower boundary from the fourth space to the fifth; and in four cases it had disappeared from the upper space at the time of the last observation of the friction-sound, when compared with the time at which it was predominant. There was therefore in these patients a tendency for the heart and its impulse to take up a lower position, and to be covered to a greater extent with lung as the friction-sound was about to disappear, and the case advanced towards its termination. On the other hand, in two other cases the impulse gained ground above, and appeared in the second space for the first time when the *frottement* was heard for the last time.

The descent of the impulse both above and below when the case advances to recovery and the friction-sound is dying out, appears to me to be the natural bias in these cases when the heart is not adherent, and descends into its natural situation; when the right ventricle and pulmonary artery are not greatly enlarged; and when the upper lobe of the left lung expands in front so as to cover the pulmonary artery and the upper portion of the right ventricle.

When, however, the heart becomes more or less adherent; when the pulmonary artery and right ventricle become enlarged owing to mitral regurgitation; when mitral incompetence is combined with adherent pericardium; when the walls of the pericardium are thickened; or when the left lung does not expand in front of the upper border of the heart so as to cover the pulmonary artery and the conus arteriosus; and notably when two or more of these conditions combine their influence, then the impulse tends to remain in or attain to the higher intercostal spaces, and especially the second space.

In one remarkable case belonging to the group of nineteen now under review, the friction-sound was lost on the fifth day after the acme, and reappeared on the twelfth day with greater intensity and over a larger area than at any previous time. In three other cases the friction-sound, after gradually diminishing in intensity and area, became suddenly reinforced; and in two others a similar diminution and increase of the *frottement* took place, but to a comparatively slight degree.

3. *The final dying away of the Friction-Sound.*—The friction-sound offered greater variety in different cases just before the time of its extinction than at any other period of its existence.

(1) In a very small number of the cases (4 in 43) the friction-sound, when in full play, suddenly disappeared; (2) in two-fifths of them (16 in 43) the *frottement*, after being more or less loud up to a certain date, rapidly declined, and vanished in one or two days; (3) in a fifth of them (8 in 43) the decline of the friction-sound was gradual; (4) and in two-fifths of them (16 in 43) the ordinary rubbing-sound gave place towards the end of the case to a friction-murmur sometimes double, and increased by pressure (8), sometimes double and

excited by pressure (5), sometimes single and systolic and intensified by pressure (2), and in one case a single friction-murmur was excited by pressure.

4. *Cases in which the ordinary Friction-Sound gave place to a Friction-Murmur towards the end of the attack.*—In fifteen patients, and possibly in a sixteenth, a friction-murmur was audible in lieu of the ordinary friction-sound towards the end of the attack of pericarditis.

We have already seen that in a certain number of cases, at the beginning of the attack, the ordinary friction-sound was preceded by a friction-murmur: and that in one remarkable case a friction-murmur prevailed throughout the whole course of the disease to the exclusion of the usual rubbing-sound. I would here refer to what has already been said as to the friction-murmur as it was observed during the beginning of the attack, at pages 57-61.

In one case a systolic friction-murmur audible on making pressure, and in another case a systolic friction-murmur increased by pressure, was respectively the final sign of pericarditis.

In six cases a double friction-murmur was audible on pressure towards the close of the affection. One of the cases of this group (47, see p. 22), a servant girl aged 20, presented on the seventh day, when the effusion was at its height, an extension of the *frottement*, when there was a double grating friction-sound. On the eleventh, when the effusion was declining, there was a feeble murmur-like friction-sound over the right auricle, to the right of the lower sternum; and later in the day the heart-sounds were natural over the lower sternum, but pressure brought out a double friction-murmur not quite rhythmical with the sounds of the heart. A systolic friction-sound was audible over the left fifth cartilage. On the fourteenth day a faint double murmur was still excited by pressure

over the lower sternum. This was the last day of undoubted pericardial friction-sound, but on the eighteenth day a double grating friction-sound burst out on pressure at the end of a deep breath, that was probably pleuritic.

In several of these cases a friction-murmur either prevailed over the right ventricle during the early stages, or was limited to certain favourite spots, such as the right auricle, when the friction-sound was at its height. Later, the friction-murmur gradually again developed itself as the harsher friction-sounds became softened, and at length spread itself over the heart. Soon, however, this disappeared as a constant sound, but for one or two final days of the disease it could be again awakened by making pressure over the right ventricle. Several of these cases ended with a double friction-murmur that was intensified by pressure.

In addition to these cases in which the friction-murmur prevailed exclusively towards the termination of the disease, there were others in which, while the friction-sound was harsh, and even creaking or grating over the focus of its greatest intensity, it was yet so toned down towards the lower margins of the area of rubbing-sound, especially at and below the ensiform cartilage, that a double friction-murmur was audible there, when a loud double grating noise was heard over the right ventricle. In some of the cases also, when a creaking, or grating, or rasping-sound prevailed with a thrill over the right ventricle, a double friction-murmur was audible over the right auricle. Here the stormy noises prevailed over the forcible ventricles, and the soft murmuring sounds over the passive auricle.

The occurrence of a creaking, grating, or harsh friction-sound depends on the force with which the heart contracts and presses against the cartilages and sternum, and on the roughness of the lymph-covered rubbing surfaces; the

creaking-sound being mainly excited by pressure, the grating noise by the roughness of the two surfaces when the one rubs actively upon the other. The friction-murmur, on the other hand, is due to the gentle or restrained movements of the heart, and the comparative smoothness of the rubbing surfaces all over the heart, that occur towards the end of the attack. It may also be present in its softest and most murmur-like tones over the comparatively smooth and feeble right auricle, and below the heart over the epigastrium, when the attack is at its height, and is speaking with the greatest harshness and noise over the more vigorous parts of the organ; and when the harsh friction-sound is evidently softened and rendered murmur-like during its transmission through the fluid intervening between the seat of active friction, and the comparatively distant surface of the chest over the right auricle or the epigastrium.

I have already given the distinctions between the friction-murmur and the valvular murmur when inquiring into the occurrence of the former during the first blush of the affection. The rules that apply to the distinction of the friction-murmur during the early period of the attack apply also to its distinction during the later period. These rules have been already given at pages 58-61, but the following is a *résumé* of the more important distinctions between the friction-murmur and the valvular murmur :—

The friction-murmur is not rhythmical with the natural heart-sounds, but the two sounds are heard side by side; the valve-murmur is rhythmical with the natural heart-sounds, and the two sounds are in perfect unison. The friction-murmur does not begin with an accent or shock, but is of equal tone throughout; the valvular murmur begins with an accent or shock, the accent or shock of the corresponding first or second sound which serves as the starting-point for the murmur.

The friction-murmur is greatly intensified, and is often altered in tone on pressure ; the valvular murmur is brought nearer to the ear by pressure, it is not altered in tone.

There are certain differences between the early and the late friction-murmur, although their characters in the main correspond.

In situation the early and late friction-murmurs for the most part correspond, being generally seated over the base or body of the right ventricle. The early friction-murmur was situated to the left of the sternum in six cases (6 in 8), in four of which it was also heard over the sternum ; and it was present over the sternum alone in two cases (2 in 8). The late friction-murmur was audible over the sternum alone in four cases ; over that bone and to the left of it in five ; to the left of the sternum alone in four ; and to the right of the sternum in three cases, including one case in which it was also audible to the left of the sternum. From these figures it would appear that the early friction-murmur is always situated over the right ventricle ; but that while the late friction-murmur is present over the right ventricle in seven-eighths of the cases, it is audible over the right auricle in one-fifth of the cases.

The late friction-murmur is smoother and more equal in tone ; more prolonged ; less rustling and more murmur-like ; more alike in tone and intensity during the systole and the diastole ; varies less from day to day ; and lasts much longer than the early friction-murmur. Pressure intensifies both of them and often modifies their tone, but I think that the early friction-murmur is more frequently converted by pressure into a true rubbing sound than the late friction-murmur.

The complication of a co-existing aortic murmur with the friction-murmur is more frequent during the late than the early period of the affection.

The Character and Tests of Pericardial Friction-Sound.

I shall, before concluding the subject of pericardial friction-sound, briefly consider the characteristic nature and tests of that sound, including its character and rhythm; its position and extent; the influence exercised over it by respiration; its variation from day to day in character, intensity, rhythm, position and extent; and finally, the effect upon it of external pressure over the region of the pericardium during pericarditis, or the *pressure test* of friction-sound.

Character of the Pericardial Friction-Sound.—The friction-sound when in full play, and of its usual to-and-fro character, speaks for itself. I have already illustrated, in the preceding pages, the clinical history of the forms and variations, the growth, ripening, and decline of the friction-sound. When the friction-sound is smooth and soft, almost resembling a murmur, or when a friction-murmur is present, the sound no longer declares itself, from its very nature, to be of a rubbing quality, and requires for its distinction that other points shall be considered besides the tone, nature, and to-and-fro quality of the sound. The clinical history and distinguishing characters of the friction-murmur during the early advance and the late decline of the attack of pericarditis have been given respectively at pages 57 and 125.

The Rhythm of the Friction-Sound.—In a large proportion of my cases it was noticed that when the friction-sound was not of its completely developed to-and-fro and rubbing character, that is, during both the advance and the decline of the pericarditis, the healthy sounds of the heart were heard along with the double or single friction-sound. The natural

sounds of the heart and the friction-sounds were never welded or incorporated together, but were each of them heard separately, and, so to speak, side by side. They did not seem to begin or end together ; and although they were both sounding at the same time, they yet appeared to be completely separate and apart. They were not therefore rhythmical with each other. That the natural heart-sounds are in play within the period of the to-and-fro friction-sound is evident, for when that sound becomes sufficiently loud and continuous, whether by the natural advance of the disease or by pressure made from without, the sounds of the heart are overwhelmed, being masked by the predominant rubbing noises.

When the to-and-fro friction-sound is loud, harsh, and in full play, the systolic and diastolic sounds being equal in duration—though rarely in loudness, the systolic sound being the louder—each sound seems almost to fill up its respective space, leaving two very short intervals of silence between the two sounds. These two friction-sounds never begin with an accent or shock, but they commence, continue, and end as a rule with the same tone throughout. In these respects they differ from the natural heart-sounds. The first sound always ends in a shock, followed by a short but definite space between itself and the second sound ; and the second sound consists in a short shock, followed by a prolonged space between itself and the first sound. The mitral murmur always begins with a shock or accent, the shock of the first sound, and the murmur fills up the space more or less completely between that shock and the second sound. The diastolic aortic murmur also commences with a shock or accent, the shock of the second sound, and it usually fills up the space, but not always completely, between that shock and the first sound. The absence of a commencing shock or accent from the friction-sound or friction-murmur and the presence of a commencing shock or

accent with the valve-murmurs distinguish those two classes of sounds from each other.

The first contraction of the ventricles precedes by an appreciable period the flow of blood from them into the great arteries; and after that flow has ceased, the exterior of the heart is still in motion. The play of the surface of the heart against that of the pericardium therefore precedes, accompanies, and follows the natural first sound of the heart, and precedes and accompanies the coinciding valvular murmur if present. The closure of the aortic valve precedes the second sound by the tenth of a revolution of the heart's action. The diastolic *frottement* therefore both precedes and follows the second sound; and accompanies a diastolic murmur, if present, throughout its whole period. The friction-sound being made by the moving exterior of the heart, is in relation to the healthy heart-sounds and the valvular murmur, which spring from the interior of the heart, as if it were made, so to speak, by an instrument playing outside the room, while they are made as if by an instrument playing inside the room. The friction-sound is therefore a surface noise, working apart from, and often overriding the healthy heart-sounds and the valvular murmurs. The healthy heart-sounds and the valvular murmurs are, on the other hand, internal noises made simultaneously and by the same parts, and playing together inseparably and in unison.

When listening to the two sounds, the frictional and the natural heart-sounds, playing together but not in concert or unison, I have found it very difficult to say whether the systolic friction-sound commenced before the first sound of the heart or not. For the reasons just given, however, and that a considerable space of time intervenes between the beginning of the systole and its final shock, amounting to about two-fifths of the healthy revolution of the heart's action,

it is evident that the commencement of the systolic friction-sound must precede the final shock of the first sound. In one case I heard a short brush at the beginning of the systole, and this no doubt represents the natural beginning of the prolonged systolic friction-sound. As a rule the systolic friction-sound is of equal tone throughout, whether it is creaking, grating, rubbing, or rustling ; but in one instance that sound became suddenly less loud about the middle of its course, and remained so to the end of the systole, the second half of the sound being weaker than its first half.

In one instance a systolic brush, excited by pressure, occupied the latter two-thirds of the systole ; in another a systolic whiff, excited by pressure, extended into the diastolic period ; and in a third, a double brush was excited by pressure, the systolic being the longer, and each brush occupied a part of the systole and a part of the diastole. I state these signs as I heard them, but cannot account for them.

The diastolic friction-sound presents much greater variety in character and rhythm than the systolic friction-sound. While the systolic sound is usually continuous through the whole of its proper period, the diastolic friction-sound is often of short duration ; when it is, I believe, usually present about the beginning of the diastole, and when it accompanies but is separate from the natural second sound : in one instance, however, the natural second sound was followed by a diastolic graze. Sometimes there was a double graze or rub during the diastole ; when the entire friction-sound resembled the noise made by sharpening a scythe, having one forward or systolic, and two backward or diastolic strokes. When the friction-sound was to-and-fro, the second sound appeared generally to be equal in duration, but not in loudness, to the first. When a creaking-sound was present it was mostly

limited to the systole: this was not so, however, with the grating noise, which was usually a double sound.

The diastolic sound was usually equal in intensity and length to the systolic over the right auricle, both sounds being in all but one instance soft in character. This double soft to-and-fro sound over the right auricle was evidently transmitted, softened during its transit, from the loud-speaking right ventricle, through the fluid, to the cartilages in front of the right auricle.

The diastolic friction-sound was often absent, and, relatively to the systolic friction-sound, was always short and feeble over the apex. In more than one instance, in adults, the diastolic friction-sound at the apex appeared to have in it a peculiar twist.

Respiration exercised in many of my cases a definite and speaking influence upon the area, and in a few of them upon the intensity of the friction-sound. The friction-sound became more loud or harsh in three cases during expiration, and in four during inspiration ; and in one the *frottement* disappeared at the end of a deep breath.

The area of the friction-sound increased below during inspiration in a large number of cases, or thirty-one, while in a much smaller number of instances, or eight, it increased above during expiration.

The Friction-sound varied in character, intensity, rhythm, and position from day to day. The clinical history contained in the previous pages of the friction-sounds during pericarditis is pervaded throughout with instances of the great daily variability of the friction-sound in all its relations. This changing condition of the friction-sound during the successive phases of the disease is one of the important characteristic features of that sound. This feature has been already abundantly illustrated.

Position and Extent of the Pericardial Friction-sound.—Dr. Stokes[1] in 1834 stated that the friction-sounds in pericarditis are audible generally only over the region of the heart. I stated independently, in 1843, that I had never heard the friction-sounds beyond the region of the heart.[2] We have seen in the previous pages that during the advance of the effusion, and usually during its acme, the friction-sound is limited to the region of the heart, but that in certain cases with a thrill, the friction-sounds spread during the acme from the seat of the thrill as from a focus, in all directions, over the front of the chest, and especially downwards.

During the period of the decline of the effusion, the friction-sound, as we have seen, also often extends beyond the region of the heart, over the front of the chest, and especially downwards to the seventh and eighth, and even the ninth cartilages (see pp. 107, 113). The various changes in the area of the friction-sound are given in the previous pages, and to those I refer for the more extended study of this subject.

The position, limitation, and extension of the pericardial friction-sound supply characteristic differences between pericardial friction-sounds and endocardial murmurs.

The Effect of Pressure with the Stethoscope *over the region of the Pericardium during Pericarditis on the Friction-sound; or the* Pressure Test *of Pericarditis.*—I called attention in 1843, in my paper on the situation of the internal organs,[3] to the effect of pressure made with the stethoscope over the region of the pericardium in rheumatic pericarditis, in intensifying or even bringing into play a pericardial friction-sound. Since then Dr. Walshe—who, in the *British and*

[1] *Dublin Journal*, iv. 60.

[2] Situation of the Internal Organs. *Prov. Med. Trans.* xii. 52.

[3] *Prov. Med. Trans.* xii. 540.

Foreign Medical Review, very kindly reviewed my paper just referred to, soon after its publication, and Dr. Stokes, independently, observed this sign. This effect of pressure is thus spoken of by Friedrich. "Sehr brauchbar is das von Sibson, Walshe, und Stokes, angegebene Zeichen, das nämlich Reibungs geräusche bei Druck mit dem stethoskop stärker werden, was allerdings Endocardiale Geräusche nicht thun."[1]

The pressure test shows itself in two ways, (I.), when pressure over the region of the heart elicits a friction-sound that was previously absent; and the other, (II.), when pressure made over the seat of a friction-sound intensifies, changes, or modifies that sound.

I. *Influence of Pressure over the region of the Heart in exciting a Friction-sound not previously audible* (see Table I. at p. 32).—Pressure made with the stethoscope over the region of the heart elicited a friction-sound not otherwise audible in twenty-nine of the forty-four cases that are included in the tables of cases of pericarditis given at pages 17—31, and in the special table at page 32, in all of which cases the acme of the pericardial effusion was observed. As might be expected, it was usually (1) during the period of the commencement of the attack or (2) that of its decline that this sign was observed; and a friction-sound otherwise latent was also thus brought into play by pressure (3) at the time of the acme of the effusion in four patients whose cases have already been touched upon at page 80, and in one case during a second acme of the effusion.

1. *Friction-sound excited by Pressure during the Onset and early period of the attack of Pericarditis.*—In eight cases, as has just been stated, the attack of pericarditis first declared itself by a friction-sound induced by pressure over the region of the heart. As a rule this sound, so awakened, was smooth

[1] Friedrich, *Die Krankheiten des Herzens*, p. 229.

in character. In three instances it appeared as a single or double friction-murmur, in one as a whiff, and in one as a soft to-and-fro sound. In the other three cases, however, the rubbing-sound was more marked, being harsh and systolic in one, of a winnowing character in another, and creaking, in the third of those cases. In three of these eight cases, pressure was required to bring out the friction-sound over the right ventricle during the advance of the effusion. The friction-sound was excited by pressure made, in six cases over the sternum, in one over the fourth cartilage, and in one over the heart. As a rule the spontaneous friction-sound partook somewhat of the character of the friction-sound previously generated by pressure. Thus it was creaking in the case in which it was originally creaking; harsh in one of those (23) in which it was harsh; to-and-fro in that in which it was to-and-fro (56), rather smooth in the patient (28) with a systolic friction-murmur; and a double friction-murmur prevailed through the long history of the fatal case (49), in which a double friction-murmur was originally aroused by pressure.

The acme of the pericardial effusion usually occurred in these cases very soon after the first appearance of the excited friction-sound, or from the first to the third day, in six of the eight cases.

2. The four cases in which a friction-sound, otherwise absent, was elicited by pressure during the acme of the disease have been already considered under their proper heading at page 80.

3. *Friction-sound excited by Pressure during the Decline of the Effusion into the Pericardium; and during the dying away of the attack.*—In the great majority of the cases in which pressure was required to elicit the friction-sound during the period of the decline of the pericardial effusion, this sign was a prelude to the dying away of the friction-sound. Thus in

nineteen of the twenty-four cases that belong to this class the *frottement* never again appeared as an independent sound ; and the attack of pericarditis was coming to an end. In three of the cases the friction-sound, after being for a time only audible when excited by pressure, reappeared for from five to ten days as an independent to-and-fro sound. There was a complete suspension of the friction-sound in connection with extreme general illness in two of these cases (45), and the return of the spontaneous friction-sound was in both of them associated with improvement of health, and was preceded by the appearance of a pressure friction-sound.

The friction-sound became inaudible except on pressure in nearly one-half of the cases under examination during the first four days after the acme of pericardial effusion (11 in 24) ; and in more than one-half of them this sign came into play from five to twenty-one days after the occurrence of the acme (13 in 24).

The character of the spontaneous friction-sound last observed before the pressure friction-sound was called forth was, with few exceptions, decidedly of a subdued tone.

The lower two-thirds of the sternum was the favourite seat of the pressure friction-sound which was heard in eleven of the cases over that bone, including two in which it was heard over the ensiform cartilage. In seven of the cases the rubbing sound was excited by pressure over the cartilages from the third to the fifth, in one other instance over the second space, and in one over the fourth space. Besides these cases the pressure friction-sound was heard over the heart in one case, the right ventricle in three, and the apex in three.

II. *Influence of Pressure over the Region of the Heart in intensifying a Friction-sound already present* (see Table II. page 32).—Pressure exercised a marked influence on the friction-sound in all but one of the forty-four cases under inquiry, and in that single exception there is no mention of the

employment of pressure over the region of the heart during the attack of pericarditis. Pressure, therefore, as a means of diagnosis, and of illustrating the clinical conditions of the friction-sound in pericarditis, is essentially interwoven into every part of what has gone before in relation to friction-sound in that affection; and one part has been devoted to the study of cases in which a soft friction-sound audible over the heart at the time of the acme of the effusion into the pericardium was converted by pressure into a harsh rubbing noise (see page 80). It is not, therefore, needful to give here again in a detached form what has already appeared distributed naturally through the preceding pages.

In four instances or observations, an endocardial murmur was masked on pressure by the occurrence of a friction-murmur or friction-sound. A friction-murmur was modified by pressure in fifteen instances: a systolic murmur being intensified (in 3), rendered double (in 1), or transformed into a double friction-sound (in 1), by the employment of pressure; and by the same means a double friction-murmur was intensified in five and converted into a double friction-sound in four instances. In a few instances (3) a friction-sound resembling a murmur acquired its complete frictional character by pressure; and in a greater number a systolic friction-sound was thus intensified (in 4), or rendered double (in 5). An ordinary friction-sound usually double, sometimes soft or grazing (in 18), sometimes of the usual to-and-fro character (in 38), sometimes harsh (in 18), was intensified, or altered in tone, or rendered more harsh in seventy-four instances or observations. As a rule a succession of observations was made upon each case, and the same patient often reappears again and again under the varying phases of the friction-sound and of the influence of pressure upon that sound.

I have not, as a rule, illustrated in this summary the various

transformations that the friction-sound may undergo under the touch of pressure ; but those two remarkable noises, the grating and the creaking friction noises, have been separately analysed, and all the instances in which either of those sounds replaced another character of friction-sound, or was strengthened by pressure, are given in the summary at page 32, and in this place.

INFLUENCE OF PRESSURE IN INTENSIFYING A PERICARDIAL FRICTION-SOUND.—*Continued from* p. 32.

Friction-sound rendered Creaking by Pressure.

Friction-sound rendered almost creaking by pressure.	Systolic friction-sound rendered almost creaking by pressure, 31, acme, 3rd space. Soft double friction-sound (bellows murmur) almost creaking on pressure, 26, acme. Double friction-sound rendered almost creaking by pressure, 6, acme, 3rd space, 40, acme, 39, after acme, 123, acme ?
Friction-sound rendered creaking by pressure.	Systolic friction-murmur rendered creaking by pressure (diastolic rub), 33, acme, 4th cart. 33, 3 days later. Musical friction-sound, changed to creak on pressure, 30, acme, 3rd space, (30, 2 days later, systolic creak produced by pressure). Friction-sound like sand-paper rubbing, creaking on pressure, 30, after acme, 22, acme ? double friction-sound. Grating sound changed to creaking by pressure, 45, after acme. Harsh double brush, replaced by double creak on pressure, 30, before acme. Friction-sound rendered creaking by pressure, 33, after 2nd acme, 33, ditto, 44, second acme.
Creaking friction-sound increased by pressure.	To-and-fro sound partly creaking, increased by pressure, 40, after acme. Creaking systolic friction-sound, increased by pressure, 34, acme, 43, acme, 42, after acme. Systolic creaking friction-sound, double creak on pressure, 7, acme, 4th space, 122, acme ?

A friction-sound of indefinite quality was rendered grating by pressure in six instances, and in two a grating friction-sound was intensified or rendered more harsh by pressure. A creaking friction-sound was in an especial manner the offspring of pressure when applied over the seat of an ordinary friction-sound, since in six instances a friction-sound, double in all but one, was rendered almost creaking by pressure, and in twelve instances, various kinds of friction noise, grating, harsh, smooth, and murmuring, were transformed by pressure into a creaking-sound ; while in two others, pressure converted a systolic creaking-sound into a double creaking-sound. These eighteen instances occurred in fourteen different cases. In each of two of these patients a creaking-sound was excited

by pressure four different times in the course of the clinical history of the case; showing a strong tendency to the repeated recurrence of this sign when it has been once excited. In six cases a creaking friction-sound was rendered more intense by pressure, and only one of these cases appears also among those just spoken of in which an ordinary friction-sound was converted by pressure into a creaking-sound.

Although I have only noticed in the summary those two more striking noises, the grating and the creaking, as being excited by pressure, yet there are many other friction-sounds of a definitely individual character that are thus brought into existence. These sounds differ in no essential respect from those that are spontaneously excited from within by the simple rubbing of the heart against the pericardium, when their opposing surfaces are covered with roughened lymph. Pressure over the heart affected with pericarditis excited—either originally or by transformation, among my various cases—a single and a double friction-murmur; a whiff; a single, and more often a double brush; rustling, grazing, scraping, scratching, and sawing friction-sounds; a double sound like that made by rubbing with sand-paper; and a peculiar double sound, broken during the diastole, that brings to my ear a noise like that made by sharpening a scythe. A to-and-fro sound was not unfrequently excited by pressure. I again and again noticed that under the influence of pressure the two friction-sounds, and especially the diastolic one, became more continuous.

Owing to the increased intensity and continuousness of the friction-sound caused by pressure over the heart in pericarditis, the natural sounds of the heart which were previously audible side by side with the friction-sound, but were not strictly rhythmical with it, were frequently silenced under the influence of pressure.

The Movements of Respiration in Pericarditis.

In the Cases included in the following Table the movements of respiration were observed with the aid of the chest measurer.

TABLE SHOWING THE MOVEMENTS OF RESPIRATION IN PERICARDITIS.

I.—CASES IN WHICH THE RESPIRATORY MOVEMENTS OF BOTH THE CHEST AND THE ABDOMEN WERE OBSERVED.

* Explanation.—These figures indicate the movements of respiration in hundredths of an inch. For explanation of Symbols see page 17.

15 (See p. 30). Female, æt. 16. ⇉

1st day. Friction-whiff on pressure, pain left side.

Rib.	*Rt.*	*Lft.*
2nd	15*	8.*
5th	5	2.
9th	9	7.
abdom.	5	9.
abd. below ens. cartil.		—10.

4th day. Acme of pericardial effusion.

Rib.	*Rt.*	*Lft.*
2nd	25*	15.*
6th	6	5.
9th	14	6.
abdom.	6	5.
abd. below ens. cartil.		—8.

5th day. Pain in epigastrium.

Rib.	*Rt.*	*Lft.*
2nd	9*	12.*
6th	3	3.
9th	5	5.
abdm.	3	3.
abd. below ens. cartil.		—6.

7th day. Better, pericardial effusion less.

Rib.	*Rt.*	*Lft.*
2nd	15	12.
5th	6	5.
9th	9	7.
abdom.	—10	3.
abd. below ens. cartil.		—3.

19th day. No friction-sound; better.

Rib.	*Rt.*	*Lft.*
2nd	9	7.
6th	3	2.
abd. below ens. cartil.		—4.
abdomen at navel.		—7.

26th day. abd. below ens. cartil. —10. abd. at navel 15.

36 (See p. 20). Female, æt. 20. ⇉

9th day. Acme of pericardial effusion, less pain heart, resp. 52.

Rib.	*Rt.*	*Lft.*
2nd	5	4.
5th	2	2.
6th	3	3.
9th	7	5.
abdom.	6	0.
abd. below ens. cartil.		—3.

10th day. Resp. 48.

Rib.	*Rt.*	*Lft.*
2nd	8	4.
6th	1	1.
7th	7	3.
9th	7	4.
abdm.	4	4.
bel. ens. car.	—6.	
abd. at navel	7.	

12th day. Feels better, Resp. 50.

Rib.	*Rt.*	*Lft.*
2nd	18	12.
6th	5	3.
abdm.	3	2.
abd. below ens. cartil.		—0.
abd. at navel	0.	

15th day. Friction-sound more limited; feels better.

Rib.	*Rt.*	*Lft.*
2nd	15	15.
6th	4	3.
9th	12	8.
abd. below ens. cartil.		—0.
abd. at navel	5.	

18th day. Lying on right side, friction-sound on pressure.

Rib.	*Rt.*	*Lft.*
2nd	14	12.
6th	7	4.
9th	10	7.
abdm.	3	5.
abd. below ens. cartil.		0.
abd. at navel	4.	

21st day. Better, aspect good, resp. 30, no friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	12-20	10-15
6th	5	?
9th	10	,,
abdm.	8	10.
abd. below ens. cartil.		4.
do. deep breath	20	
abd. at navel	5.	

22nd day. Weak. below ens. cartil. 5. ditto, deep breath 25.

33rd day.—Below ensiform cartilage, deep breath, 50.

37th day.—Below ensiform cartilage, deep breath, 90.

5 (See p. 28). Female, æt. 15. ⇒

1st day. Pn. lft. side ill a week. No friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	20	15-20.
6th	6	3.
9th	4	7.
abd.	6	6.

8th day. Acme, resp. 54, pain side.

Rib.	*Rt.*	*Lft.*
6th	3	3.
9th	9	4.
abd. below ens. cartil.		—1.

12th day. Better, but resp 55, no friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	18	15.
6th	6	2.
9th	9	2.
abd.	1	3.
abd. b. ens. c.	—2.	

4 (See p. 17). Male, æt. 27. ⇉

2nd day. Acme, very ill. resp. 36.

Rib.	*Rt.*	*Lft.*
2nd	7-9	7-10.
6th	4.	4.
9th	9.	5.
abd.	7.	12.

3rd day. Better.

Rib.	*Rt.*	*Lft.*
2nd	6	7.
6th	6	3.
9th	,,	9.
abd. below ens. cartil.		—12.

7th day.

Rib.	*Rt.*	*Lft.*
2nd	6	7.
6th	4	2.
9th	4	7.
abd. at navel.	20.	

18 (See p. 27). Female, æt. 18.

5th day Acme, pain over heart.

Rib	*Rt.*	*Lft.*
2nd	30	20.
6th	7	6.
9th	10	10.
abd. below ens. cartil.		—2.

24th day.–Slight friction-sound

8th day. Pain in chest, friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	30	20.
6th	6	5.
9th	15	7.
abdm. —3		—2.
abd. below ens. cartil.		—4.
abd. at navel		0.

11th day. Acme.

Rib.	*Rt.*	*Lft.*
2nd	30	20.
6th	6	3.
9th	10	7.
abd.	6	3.
abd. below ens. cartil.		—4.
abd. at navel		0.

19 (See p. 30). Male, æt. 23.

7th day. 3days after acme, very extensive friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	20	20.
6th	6	5.
9th	5	7.

10th day. Improving; less friction-sound; left pleurisy.

Rib.	*Rt.*	*Lft.*
2nd	25	20.
6th	9	10.
9th	13	10.
abdm. 12		5.
abd. below ens. cartil.		—1.

13th day. Better, very slight friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	15	12.
6th	6	4.
9th	13	12.
abdm.	6	10.
ab. b. ens. c.		2.
abd. at navel		6.

26 (See p. 28). Male æt. 25.

6th day. Acme, lessening pain over heart.

Rib.	*Rt.*	*Lft.*
2nd	20	20.
4th	10	10.
7th	9	10.
9th	9	10.

10th day. Resp. 22, better, friction-sound.

Rib.	*Rt.*	*Lft.*
2nd	8	7.
6th	16	8.
10th	9	9.
abdm.	25	20.
abd. below ens. cartil.		—20.
abd. at navel		40.

13th day. Better, sits up in bed, friction-sound on pressure.

Rib.	*Rt.*	*Lft.*
2nd	15	15.
6th	10	10.
abdm.	30	35
abd. below ens. cartil.		—30.
abd. at navel		40.

49 (See p. 31). Male, æt, 17.

8th day. Before acme pain in heart.

Rib.	*Rt.*	*Lft.*
7th	10	4.
abdm.	12	7.

12th day. Acme. }—abdomen below ensiform cartilage,—7.

II.—CASES IN WHICH THE RESPIRATORY MOVEMENTS OF THE CENTRE OF THE ABDOMEN WERE OBSERVED.

A.—*Cases observed*—(1) *below the Ensiform Cartilage, and* (2) *at the Navel.*

25 (See p. 31).	Male, æt. 17		12th day, second acme...	Below ens. cartil.,	—3,	at navel, 4.
			21st day, no friction-sound	,, ,,	*0 ,,	*12.
			85th day	,, ,,	18 ,,	20.
34 (See p. 20).	Male, æt. 15		4th day, acme, pain epigast.	,, ,,	6 ,,	10.
			5th day, after acme ...	,, ,,	5 ,,	
			6th day, ,,	,, ,,	4 ,,	
16 (See p. 19).	Fem., æt. 17		2nd day, acme?	,, ,,	0 ,,	10.
40 (See p. 17).	Male, æt. 17		1st day, acme	,, ,,	1 ,,	12.
			7th day, after acme ...	,, ,,	5 ,,	
			15th day, friction-sound...	,, ,,	3 ,,	12.

B.—*Cases observed below Ensiform Cartilage.*

51 (See p. 19).	Male, æt. 22		4th day, acme..	...Movement below ens. cartil.,		–2.
			7th day, decline of fld. ...	,, ,,	,,	6.
			9th day, second acme ...	,, ,,	,,	9.
			29th day, well. Deep breath	,, ,,	,,	110-170.
35 (See p. 31).	Fem., æt. 21		5th day, after acme ...	,, ,,	,,	–2.
			11th day, improving ...	,, ,,	,,	3.
			32nd day, clothes on. Deep breath	,, ,,		50.
56 (See p. 28).	Male, æt. 15		1st day, before acme ...	,, ,,	,,	16.
			3rd day, acme	,, ,,	,,	6.
			11th day, well	,, ,,	,,	20.
38 (See p. 29).	Fem., æt. 22		13th day, second acme ...	,, ,,	,,	–2.
			22nd day, third acme ...	,, ,,	,,	0.
28 (See p. 20).	Fem., æt. 19		9th day, after acme ...	,, ,,	,,	1
			11th day	,, ,,	,,	–7
20 (See p. 25).	Fem., æt. 24		17th day, acme	,, ,,	,,	–3.
			25th day	,, ,,	,,	17
44*a* (See p. 27).	Male, æt. 14		6th day, after first acme	,,	,,	3.
13 (See p. 18).	Fem., æt. 25		4th day, acme, or after ...	Mvt. bel. ens. cartil. or lower,		4.
58 (See p. 27).	Male, æt. 26		5th day, before friction-sound.	Mvt. below ens. cartil.,		5.
54 (See p. 24).	Male, æt. 35		8th day, no friction-sound.	,, ,,	,,	20.

The movements of respiration were affected in pericarditis in three different relations ; (1) those of the ribs ; (2) those of the abdomen on each side, just below the eighth cartilage ; and (3) those of the centre of the abdomen.

(1.) The respiratory play of the upper ribs was more than doubled in extent in three-fourths of the cases observed (5 in 7), so that respiration was as a rule high. This was due to the arrest or restraint of the action of the diaphragm caused by the extensive inflammation of the central tendon of the diaphragm, where it forms the floor of the pericardium.

In one (4) of the two exceptional cases, the movements of the second ribs were not at all or only slightly augmented throughout the whole period of the illness; but in the other case, in which the respiration was greatly accelerated, the action of those ribs, which was slight during the acme of the affection, was much increased during the decline of the effusion.

The respiratory movement of the ribs on the left side of the chest was less than that of those on its right side, as might naturally be expected, in more than one-half of the cases (5 in 8) ; but in the remaining three patients the action of the two sides was nearly equal both during the acme and the decline of the pericarditis. The difference in the movement of the two sides of the chest was not, as a rule, limited to the ribs adjoining the pericardium, but extended along their whole range, from the second to the ninth. The study of the Table will show, however, that there were some exceptions to the rule that the play of the ribs was restrained throughout on the left side ; since in two of the three cases in which the two sides of the chest moved with equal freedom, the ninth left rib was greatly restrained in its movements.

(2.) The lateral movements of the abdomen below the eighth cartilages were greatly restrained in three-fourths of the

cases (6 in 8); and the respiratory play of the left side of the abdomen was much less than that of its right side in the same proportion of cases (6 in 8).

(3.) The inspiratory movement of the abdomen below the ensiform cartilage was either reversed (in 12), arrested (in 1), or restrained (in 6) in every case of pericarditis in which that sign was observed. This is at once accounted for by the inflammation, in that disease, of the central tendon of the diaphragm where it forms the floor of the pericardium, which leads to the virtual paralysis of the central portion of the diaphragm. This fact, that the anterior wall of the epigastric space, instead of advancing, recedes during inspiration, gives us a physical sign of great value in the diagnosis of pericarditis, and of the advance and decline of that disease. Thus in the first case in the Table (15), a girl, aged 16, the anterior wall of the abdomen below the ensiform cartilage fell backwards during inspiration for the tenth of an inch during the three early days, when the disease was at its acme; then, as the tide turned and the effusion diminished, the abdomen receded less and less up to the seventh day, when it did so for only the fiftieth of an inch; after this it regained its natural forward movement, and on the twenty-sixth day the abdomen at the epigastric space advanced as much (the tenth of an inch) as it had receded on the day of admission. In the other case (56), the front of the abdomen advanced the sixth of an inch on the day of admission, when the pericarditis had scarcely pronounced itself; the sixteenth of an inch on the third day, when it had reached its acme; and the fifth of an inch on the eleventh day, when it had declined and disappeared. In my paper on the movements of respiration I showed that in health the abdomen at the navel advanced during inspiration a quarter of an inch or a little more, but I did not ascertain the respiratory movement at the epigastric

space. A short time ago I observed, with Mr. Rossiter, the respiratory movements of the abdomen in eleven patients in St. Thomas's Hospital, several of whom were convalescent, and one had pericarditis ; when we found that the inspiratory advance at the epigastric space varied from the sixth to the fifth of an inch. The latter was also the extent of the advance in two healthy men. I consider that this forward movement fairly represents the healthy respiratory play of the part in question ; that in pericarditis, as a rule, the whole of this advance is lost ; and that in addition the play is reversed to the extent of from the fiftieth to the tenth of an inch. It is worth noting, in conclusion, that in the case of pericarditis observed by Mr. Rossiter and myself in St. Thomas's Hospital, a boy, aged 12, in whom the disease was at its height, the wall of the abdomen receded during inspiration at the epigastric space from the sixteenth to the twentieth of and inch, and at the navel from the thirty-fifth to the fiftieth of an inch.

CASES OF BRIGHT'S DISEASE EXAMINED AFTER DEATH

With Especial Reference to the Occur-

CONDITION OF KIDNEY.	CONDITION OF HEART.	Total Number.	Percentage to total number of cases in this class.	Age.—Below 16 years.	Age.—16 yrs. to 30 yrs.	Age.—Above 30 yrs.	Male Patients.	Female Patients.	Pleurisy.	Pneumonia.
Acute Bright's Disease following Scarlet Fever	Heart of natural size	2	40%	2	...	...	2	...	1	...
	Ditto with partial Pericarditis	1	50	1	...	...	1	...	1	...
	Heart rather large	1	20	1	...	...	...	1	1	...
	Heart large or very large	2	40	1	1	...	...	2	1	...
	Size of heart not described	1	16·6	...	1	...	...	1	...	...
Ditto. TOTAL		6	2·1%	4	2	...	2	4	3	...
Ditto with partial Pericarditis		1	16·6	66·6%	33·3%	...	23·3%	66·6%	50%	...
				av. age 10·5	av. age 23					
Acute Bright's Disease	Heart of natural size	2	14·3%	1	1	...	1	1	...	...
	Heart rather large	4	28·6	...	2	2	1	3	...	...
	Heart large or very large, hyptrd.	8	57·1	...	4	4	7	1	1	2
	Ditto with general Pericarditis	2	25	...	1	1	2	...	1	2
	Size of heart not described	1	6·6	...	...	1	1	...	...	...
TOTAL with Acute Bright's Disease		15	5·3%	1	7	7	10	5	1	2
Ditto with general Pericarditis		2	13·3	6·6%	46·6%	46·6%	66·6%	33·3%	6·6%	13·3%
				av. age 15	av. age 20·6	av. age 51				
Fatty Kidney	Heart small	14	25%	...	4	7	5	8	2	2
	Ditto with partial Pericarditis	1	7·2	...	...	1	...	1	...	1
	Heart of natural size	18	32	1	4	11	15	3	6	7
	Ditto with partial Pericarditis	1	6	...	...	1	1	1	...	...
	Heart rather large	10	18	...	...	9	7	3	5	2
	Ditto with partial Pericarditis	1	10	...	...	1	1	3	...	...
	Heart very large, hypertrophd.	14	25	1	3	9	14	...	2	3
	Size of heart not described	6	9 6	...	1	3	3	3	1	2
	Ditto with general Pericarditis	1	16·6	...	...	1	1	...	...	1
	Ditto with partial Pericarditis	2	33·3	...	1	...	...	2	1	1
TOTAL		62	21·7%	2	12	39	44	17	16	16
Ditto with general Pericarditis		1	1·6	3·7%	22·6%	73·6%	72%	28%	26%	26%
Ditto with partial Pericarditis		5	8·0	...	...	...	...	...	...	...
					av. age 25	av. age 45				
Granular Kidney. Kidney lessened in size	Heart small	4	3·5%	...	...	3	2	2	4	...
	Heart of natural size	24	20·5	...	5	18	10	13	5	2
	Ditto with general Pericarditis	5	20·8	...	2	3	3	2	1	1
	Ditto with partial Pericarditis	2	8	...	...	1	...	1	1	...
	Heart rather large	20	17	...	4	16	10	10	2	1
	Ditto with partial Pericarditis	1	5	...	...	1	...	1	...	...
	Heart very large, hypertrophd.	69	59	...	8	55	48	21	16	9
	Ditto with general Pericarditis	5	7·2	...	1	2	4	1	3	1
	Ditto with partial Pericarditis	3	4·3	...	...	4	1	3	1	...
	Size of heart not described	11	8·6	...	2	7	7	3	3	1
	Ditto with general Pericarditis	3	27·2	...	2	...	1	2	2	1
	Ditto with partial Pericarditis	1	9	...	...	1	1	...	...	...
TOTAL		128	45%	...	19	99	77	49	30	13
Ditto with general Pericarditis		13	10·1	...	16%	84%	61%	39%	23·4%	10%
Ditto with partial Pericarditis		7	5·5	...	...	...	...	...	...	...
					av. age 26	av. age 51				
Granular Kidney. Kidney of natural size, and larger than natural	Heart of natural size	5	16·6%	...	...	3	3	2	1	...
	Heart rather large, hypertrop.	4	13·3	...	...	3	3	2	2	...
	Ditto with general Pericarditis	1	25	...	...	1	...	1	...	...
	Heart very large	21	70	...	3	16	6	3	2	2
	Ditto with general Pericarditis	1	4 8	...	...	1	1	...	...	...
	Size of heart not described	4	11·7	...	1	3	2	2	...	...
	Ditto with general Pericarditis	1	...	...	...	...	1	...	...	...
TOTAL		34	12%	...	4	25	24	9	5	2
Ditto with general Pericarditis		3	8·8	...	14%	86%	72·7%	27·3%	14·7%	6%
					av. age 23	av. age 46·3				
Granular Kidney—GRAND TOTAL		162	57%	...	23	124	101	58	35	15
					16%	84%	63·5%	36·5%	21·6%	9·3%

AT ST. MARY'S HOSPITAL DURING THE YEARS 1851–69,

ence of Pericarditis in that Disease.

Pleuro-pneumonia.	Pulmonary Apoplexy.	Pleuro-pulmonary Apoplexy.	Purulent deposit, or abscess in lung.	Total attacks of inflam of lungs (including Pulm. Apoplexy & Pleurisy).	Total cases with inflam. of lungs or pleura.	Phthisis.	General Pericarditis. (Pus in Pericardium includ. in General Pericarditis	Partial Pericarditis.	Adherent Pericardium.	General Peritonitis.	Total cases of Disease of Valve with incompetence.	Total cases of Thickening of Valve, the Valve being competent.	Atheroma or Dilatation of Aorta.	Disease of Coronary Artery.	Total cases with Affect. of Valves, with and without valvular incompetence, or with adherent pericardium.	Cases with emaciation great weakness, or exhausting disease.[1]	Cases in good bodily condition.
...	...	...	...	1	1	...	...	1	...	1	...	...	...	...	...	1	...
...	...	...	...	1	1	...	...	...	...	1	...	...	...	...	...	...	...
...	...	...	...	1	1	...	...	...	...	1	...	...	...	...	...	...	...
...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	3	3	...	...	1	...	2	...	...	...	...	...	1	...
...	...	...	...	50%	50%	...	...	16·66%	...	33·3%	...	...	...	...	...	...	...
...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1	...
...	1	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	3
1	1	...	...	4	3	...	2	...	3	...	5	1	...	...	6	1	5
1	...	...	...	3	2	...	...	...	...	...	1	...	1	...	1	1	2
1	2	...	...	5	4	1	2	...	...	...	5	2	1	...	7	3	8
6·6%	13·3%	...	...	33·3%	26·6%	6·6%	13·3%	...	20%	...	33·3%	13·3%	6·6%	...	46·6%	27·2%	72·7%
...	...	...	...	4	4	7	...	1	...	1	...	3	...	...	3	12	1
...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...
4	1	...	2	16	12	1	...	1	...	...	1	1	...	...	4	9	2
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	1	1	1	9	7	2	...	1	...	...	2	4	2	...	6	2	4
...	...	...	1	2	1	...	...	...	...	...	...	...	...	...	...	1	...
2	3	...	...	8	6	...	...	...	5	1	6	3	2	...	12	1	4
...	...	...	1	4	3	...	1	2	1	...	2	1	1	...	3	1	...
...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	2	2	...	...	...	...	...	...	...	...	...	...	...	...
8	5	1	4	41	32	10	1	8%	6	2	11	12	8%	...	28	25	11
13%	8%	1·6%	6·5%	66%	51·6%	16%	1·6%	...	9·6%	3·4%	17·7%	19·4%	...	...	45%	70%	30%
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	4	4	3	...	...	...	...	...	1	...	...	1	4	...
2	2	1	...	9	6	4	5	2	...	3	2	3	3	...	8	11	2
1	...	...	...	2	1	...	...	...	...	1	...	...	...	...	...	2	1
...	...	...	...	1	1	...	...	...	...	1	...	...	...	...	...	2	...
...	1	...	...	4	3	1	...	1	...	1	3	4	1	...	7	7	5
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...
5	10	6	...	35	24	1	5	3	3	...	27	9	13	...	46	21	22
1	...	...	...	4	3	...	...	...	...	...	...	1	...	...	1	2 or 1	3
...	2	1	...	3	2		...	...	...	...	1	1	1	...	3	1	2
1	1	...	...	5	4	1	3	1	...	2	2	1	...	...	4	6	3
1	...	...	...	3	2	...	...	...	...	...	...	...	...	...	...	...	3
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
8	14	7	...	57	41	10	13	7	3	6	34	18	17	...	66	49	32
12%	11%	5·5%	...	44·5%	32·6%	7·8%	10%	5·5%	2·3%	4·7%	26·5%	14%	13·2%	...	51·5%	60%	40%
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	1	...	1	2	2	1	...	...	...	...	...	...	...	...	...	3	1
...	...	...	...	3	3	...	1	...	...	2	1	2	1	1	3	2	...
...	...	...	...	...	...	...	...	...	...	...	1	...	1	...	1	...	...
...	4	1	...	9	8	...	1	...	2	...	11	4	6	...	16	2	9
...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	1	...	...
...	...	...	...	...	...	...	1	...	1	...	2	1	...	...	3	...	...
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	5	1	1	14	13	1	3	...	3	2	14	7	7	1	22	7	10
...	14·7%	3%	3%	41%	38·2%	3%	9%	...	9%	6%	41%	20·6%	20·6%	3%	64·7%	40%	60%
8	19	8	1	71	54	11	16	7	6	8	48	25	24	1	88	56	42
5%	11·7%	5%	6%	42·2%	32·6%	6·7%	9·9%	4·2%	3·7%	5%	29·6%	15·4%	14·8%	·6%	54·3%	57%	43%

CONDITION OF KIDNEY.	CONDITION OF HEART.	Total number.	Percentage to total number of cases in this class.	Age.	Age.—Below 16 years.	Age.—16 years to 30 years.	Age.—Above 30 years.	Male Patients.	Female Patients.	Pleurisy.	Pneumonia.
Cases of actual and probable Lardaceous Disease.	Heart small	5	25%	...	...	4	1	5	...	...	...
	Heart of natural size	8	40	...	1	2	4	8	...	2	2
	Heart rather large	3	15	...	...	1	2	3	...	...	1
	Heart large, or very large	4	20	...	...	...	3	3	1	...	...
	Ditto with general Pericarditis	1	25	...	...	...	1	1	...	...	...
	Size of heart not described	2	18·2	...	...	...	2	2	...	1	1
	Ditto with general Pericarditis	1	50	...	...	...	1	1	...	1	1
TOTAL of actual and probable Lardaceous Disease		22	7·7%	...	1	7	12	21	1	3	4
Ditto with general Pericarditis		2	9%	...	5%	35%	60%	95%	5%	14%	18%
					10	23·5	42				
1 Kidney large, 1 Atrophd	Heart prob. of natural size	1	...	...	...	...	1	1	1	...	...
Embolism of Kidney	Heart very large.	3	...	...	...	1	2	2	1	...	1
Obstr. Congesn. of Kidney	Heart very large	3	...	...	...	...	3	1	2	1	1
Nature of Kidney, disease doubtful.	Heart of natural size	1	10%	...	...	...	1	1	...	...	...
	Ditto with general Pericarditis	1	...	...	...	...	1	1	...	...	...
	Heart rather large	4	40	...	...	...	4	1	3	...	...
	Ditto with general Pericarditis	2	50	...	...	...	2	1	1	...	...
	Ditto with partial Pericarditis	1	25	...	...	...	1	...	1	...	...
	Heart large or very large	5	50	...	...	...	4	3	2	1	2
	Ditto with general Pericarditis	1	20	...	...	...	1	1	...	1	1
	Ditto with partial Pericarditis	1	20	...	...	...	1	...	...	...	...
	Size of heart not described	1	9	...	...	...	1	...	...	...	...
Ditto. TOTAL		11	3·8%	...	...	...	10	5	5	1	2
Ditto with general Pericarditis		4	36·3	...	...	...	100%	45%	45%	99%	18·1%
Ditto with partial Pericarditis		2	18·1	...	...	...					
						av. age	49				
				av. age	below16	16 to 30	above30				
Total cases of Bright'sDisease; arranged according to the size of the heart	Heart small	23	9%	35	...	8	11	12	10	6	2
	Ditto with partial Pericarditis	1	4·5	18	...	...	1	...	1	...	1
	Heart of natural size	61	23·6	37	5	12	37	40	20	15	11
	Ditto with general Pericarditis	6	10	43	...	2	4	4	2	1	1
	Ditto with partial Pericarditis	4	6·6	31	1	...	2	2	1	2	...
	Heart rather large	46	17·4	44	1	7	35	24	21	10	4
	Ditto with general Pericarditis	3	6·6	38	...	...	3	1	2	...	...
	Ditto with partial Pericarditis	3	6·6	56	...	...	3	1	2	1	...
	Heart large or very large, hypd.	123	50	45	2	19	99	94	34	24	19
	Ditto with general Pericarditis	10	8	41	...	2	6	7	2	5	4
	Ditto with partial Pericarditis	4	3·1	54	...	...	4	1	3	1	...
	Size of heart not described	26	10	47	...	5	18	15	11	5	4
	Ditto with general Pericarditis	6	23	58	...	2	2	4	2	3	3
	Ditto with partial Pericarditis	3	11·5	52	...	1	1	2	2	2	1
TOTAL		285	...	43	8	51	199	185	96	60	40
				av. age	3%	19%	78%	66%	34%	22%	14%
Ditto with general Pericarditis.		25	8·8%	...	...	...	...	16	8	9	8
Ditto with partial Pericarditis.		15	5·3	...	...	...	...	6	9	6	2
Calculus in Kidney, &c.; or dilated pelvis; Hydronephrosis.[1]	Heart small	1	10%	...	...	1	...	...	1	...	...
	Heart of natural size	3	30	...		2	...	3	...	1	1
	Heart rather large	4	40	...	...	1	2	2	2	1	1
	Heart large	2	20	...	...	...	2	1	1	...	...
	Size of heart not described	2	16 6	...	...	...	2	1	1	...	...
Ditto. TOTAL		12	...	...	...	4	6	7	5	2	2
						40%	66%	60%	40%	16 6%	16 6
						av. age	av. age				
						28	54				
Suppurative nephritis from stricture, &c.[2]	Heart small	4	30·7%	...	...	1	2	4	...	...	...
	Heart of natural size	7	54	...	...	3	1	7	...	2	1
	Ditto with general Pericarditis	1	14·8	...	...	...	1	1	...	...	1
	Heart rather large	2	15·3	...	...	...	2	2	...	...	...
Ditto. TOTAL		13	...	...	...	4	5	13	...	2	2
Ditto with general Pericarditis		1	7·7%	...	...	44·4%	66·4%	100%	...	15·4·0	15·4
						av. age	av. age				
TOTAL NUMBER OF CASES		310				20·5	50·8				

[1] Calculus in kidney, pelvis, or ureter (4); affection of bladder (3); both affections (1).

ST. MARY'S HOSPITAL DURING THE YEARS, 1851-69—(*continued*).

Pleuro-pneumonia.	Pulmonary Apoplexy.	Pleuro-pulmonary Apoplexy.	Purulent deposit or abscess in lung.	Attacks of inflam. of lungs (inc. Pulm. Apopl. & Pleurisy.)	Cases with inflam. of lungs or pleura.	Phthisis.	Gen. Pericarditis. (Pus in pericar. incl. in Gen. Pericard.)	Partial Pericarditis.	Adher. Pericardium	General Peritonitis.	Disease of Valve, with incompetence.	Thickening of Valve; competent Valve.	Atheroma or Dilatation of Aorta.	Dis. of Coron. Artery	Affect. of Valves, with and without valvular incomp. or with adherent pericardium.	Dis. Heart & Aorta.	With emaciation, great weakness, for exhausting disease.	Cases in good bodily condition.
...	...	...	...	...	...	...	...	...	...	2	...	1	...	...	1	...	5	...
1	...	...	1	5	4	3	...	...	...	...	1	...	...	...	1	...	6	...
...	...	...	1	2	2	...	...	...	1	1	1	1	...	...	1	...	2	...
...	...	...	...	2	...	...	1	...	1	1	3	...	...	...	4	...	...	2
1	...	...	...	...	...	...	1	...	...	1	...	...	...	...	...	...	...	...
...	...	...	...	2	1	...	...	...	...	...	...	...	...	...	...	...	...	...
1	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...
2	...	...	2	9	7	3	2	...	2	4	5	2	...	...	7	...	13	
9%			9%	41%	32%	14%	9%		9%	18·2%	23%	9%			32%		87%	13%[2]
...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...
1	...	...	...	3	2	...	...	...	2	...	3	...	...	...	3	...	2	...
1	...	...	...	2	2	...	...	...	...	...	...	...	...	2	...	...	...	...
...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	1	...	...	1	1	...	2	1	...	1	4	...	...	...	4	...	2	...
...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	2	...	...	...
...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	1	...	...	...
1	1	...	...	4	3	...	1	1	1	...	1	1	...	...	2	...	2	2
1	...	...	...	2	1	...	...	...	...	...	...	...	...	...	...	...	1	0
...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...
2	2	...	...	5	4	...	4	2	1	1	5	1	...	...	6	...	5	
18%	18%			45·4%	36·3%	...	36·3%	1·8%	1·9%	1·9%	45%	9%			54·5%		72%	28%[2]
...	...	...	...	8	8	10	...	1	...	3	...	5	...	...	5	...	21	1
...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...
7	3	1	5	33	25	10	6	4	...	5	4	4	3	3	13	...	31	5
1	...	..	...	2	1	...	...	...	...	1	...	...	...	...	...	...	2	1
...	...	...	2	2	2	...	...	...	...	2	...	...	...	...	...	...	2	...
2	5	...	1	21	18	3	3	3	1	5	11	10	4	...	21	...	14	12
...	1	...	...	1	1	...	...	...	...	...	1	...	1	...	2	...	1	...
...	...	...	1	1	1	...	...	...	...	...	1	...	...	...	1	...	...	...
10	21	8	...	63	46	1	10	4	17	3	56	19	21	4	92	...	27	43
3	2	...	...	11	6	...	...	...	1	...	...	1	...	...	2	...	1 or 2	3
...	...	1	...	2	2	...	...	...	...	...	2	1	1	...	3	...	1	2
2	1	...	1	11	8	1	6	3	2	2	7	3	2	...	11	...	7	24
2	...	...	...	6	4	...	...	...	...	1	...	...	...	...	...	...	1	3
1	...	...	...	3	2	...	...	...	...	...	1	...	...	...	...	...	...	...
21	30	9	7	136	105	25	25	15	20	19	78	41	30	7	142	...	100	65
7·4%	10·5%	3·1%	2·4%	48%	37%	9%	9%	5·2%	7%	6·7%	28%	14·4%	10·5%	2·4%	50%		60%	40%[2]
6	3	...	...	20	12	...	...	...	1	2	2	...	...	...	4	...	5 or 6	7
1	...	1	3	9	8	...	...	...	...	2	4	...	...	...	4	...	3	2
...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	1	...
1	...	...	...	2	1	...	...	...	...	...	...	...	...	...	...	...	1	...
1	...	...	...	2	1	1	...	...	...	...	1	1	1	...	2	...	1	2
...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	1	...	...	...
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	...	...	...	4	2	2	...	...	...	...	1	2	1	...	3	...	3	
16·6%				33·3%	16·6%	16·6%					8·3%	16·6%	8·3%		25%		60%	40%[2]
...	...	...	...	...	...	2	1	...	...	...	...	1	1	...	1	...	4	...
...	...	...	...	3	3	2	...	...	...	...	...	...	...	...	...	...	7	...
...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...
...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	2	...
...	...	...	...	4	4	4	1	...	...	...	...	1	1	...	1	...	13	...
...	...	...	...	31%	31%	31%	7·7%	...	...	...	...	7·7%	7·7%	...	7·7%	...	100%	...

[2] Stricture, or affection of prostate or bladder (11); pyæmia (1); calculus in ureter (1).

FREQUENCY OF PERICARDITIS, PLEURISY, PERITONITIS, AND PNEUMONIA IN CASES OF BRIGHT'S DISEASE OF THE KIDNEYS.

COLLECTED CASES.]	Cases.	Pericarditis.	Pleurisy.	Peritonitis.	Pneumonia.	Pulmon. Apoplx.	Partial Pericard.	Pur. dep. in lung.
CASES OBSERVED IN ENGLAND AND SCOTLAND.								
Dr. Bright (*Guy's Hospital Reports*, I.)	100	7 or 8	17	12	7	3	...	...
Ditto (*Guy's Hospital Reports*, V.)	8	0	1	1	1	...	...	...
Drs. Bright and Barlow (*Guy's Hospital Reports*, second series, I.)	9	2	1	1	0	1	...	...
Dr. Gregory (*Edin. Med. Journ.*, vols. XXXVI., XXXVII.	39	0	4	5	1	...	...	...
Dr. Christison (*On Granular Degeneration of Kidneys*)	15	0	2	0	1?	...	...	...
Dr. Johnson (*Diseases of Kidneys*)	11	1	2	2	0	...	...	...
Dr. Wilks (*Guy's Hosp. Rep.* new ser. VIII.)	76	6	12	7	6	...	...	...
Dr. Basham (*On Dropsy*)	14	0	0	1	0	1	...	...
Dr. Dickinson (*On Albuminuria*)	155	20	20	12	26	...	...	...
Dr Miller (*Kidneys in Scarlet Fever*)	10	1	5	2	5	...	...	...
Dr. Taylor (*Med. Chir. Trans.*, vol. XXIII.)	50	5	11	5	12	...	...	...
Dr. Roberts (*Urinary and Renal Disease*)	8	0	0	0	0	...	...	...
Dr. Grainger Stewart (*Bright's Disease*)	131	9	19	5	13	...	...	...
Dr. Greenfield (Table contributed to Author)	16	3	3	1	4	1	...	...
The Author (*St. Mary's Hosp. Post-Mortem Records*, 1851–69)	285	25 1 in 11·23 8 8%	60 1 in 4·8 21%	19 1 in 15 6·7%	39 1 in 6·4 15·5%	30	14	...
TOTAL	927	80 1 in 11·6; 8·6%	157 1 in 5·9 17%	73 1 in 12·6 7·9%	115 1 in 8 12·4%	...	...	...
Dr. Chambers (*Decennium Pathologicum*).	454	36 or 37 1 in 12·5 8%						
TOTAL	1381	116 1 in 11·9 8·4%						
CASES OBSERVED IN GERMANY.								
Malmsten (*U. Brightsche Nierenkrankheiten*)	33	3	2	0	3	...	...	...
Frerichs (*U. Brightsche Nierenkrankheiten*)	21	1	1	...	3	...	...	...
Bamberger (*Virchow's Archiv.*)	48	8	9	2	11	1	...	...
Traube (various sources)	19	2	4	0	5	...	...	...
Wagner (*Virchow's Archiv*, III.)	8	0	0	0	0	...	...	...
Tüngel	19	2	1	0	0	...	...	...
Treits (*Prager Vierteljahrschrift*, 1854)	5	0	3	1	0?	...	...	1or2
TOTAL	153	16 1 in 9·5 10·4%	20 1 in 7·6 13%	3 1 in 51 2%	22 1 in 7 14·4%	...	...	...
Rosenstein (*Nierenkrankheiten*, p. 105)	114	17 1 in 6·7 14·9%	22 1 in 5·1 19·2%	13 1 in 8·7 11·4%	25 1 in 4·5 21·9%			
Ditto (*ditto* p. 198)	114	8 1 in 14 7%	19 1 in 6 16·6%	10 1 in 11·4 8·7%	20 1 in 5·7 17·5%			
(Second Summary of the same cases with different results)								
Stein (*Myocarditis*)	10	1	...	...	1			
CASES OBSERVED IN FRANCE.								
Martin Solon (*De l'Albuminurie*)	10	0	2	0	1?	...	...	...
Rayer (*Maladies du Rein*)	45	3	9	5	6	3	...	...
Becquérel (*Séméiotiques des Urines*, Adults)	45	1	7	6	8?	...	...	...
Ditto (*ditto* Children)	17	0	3	6	6	...	...	...
Lanceraux (*Encyclop. des Sc. Med.*)	14	0	...	...	...	...	...	...
TOTAL	131	4 1 in 33 3%	21 1 in 6·2 16%	17 1 in 7·7 13%	21 1 in 6·2 6·7%	...	...	...
CASES OBSERVED IN INDIA.								
Dr. Morehead (*On Diseases of India*)	17	0	3	2	4			
GRAND TOTAL	1228	100 1 in 12·3 8·1%	201 1 in 6·1 16·4%	93 1 in 13 7·6%	162 1 in 7·6 13·2%	...	...	...
	*1682	136 1 in 12·3 8·1%						

* With the addition of Dr. Chambers's 454 cases.

FREQUENCY OF PERICARDITIS, PLEURISY, PERITONITIS, AND PNEUMONIA IN THE VARIOUS FORMS OF BRIGHT'S DISEASE.

	Cases.	Pericarditis.	Pleurisy.	Peritonitis.	Pneumonia.	Pulm. Apop.	Part. Peric.	Absc. of lung.
ACUTE BRIGHT'S DISEASE FROM SCARLET FEVER.								
CASES BELOW 16 YEARS OF AGE.								
Dr. Dickinson (*On Albuminuria*, from Tables kindly communicated to the Author)	21	1	4	2	6 or 7	...	...	...
Dr. Greenfield (from ditto)	4	1	0	0	0	...	...	...
The Author (*St. Mary's Hosp. Post Mort. Rec.*)	4	0	2	2	0	...	1	...
TOTAL	29	2 1 in 14 7%	6 1 in 5 20·7%	4 1 in 7 13·8%	6 or 7 1 in 5 21%	...	1	...
CASES ABOVE 15 YEARS OF AGE.								
Dr. Greenfield (*loc. cit.*)	3	0	0	3	2	...	...	...
The Author (*St. Mary's Hospital*)	2	0	1	0	0	...	...	...
TOTAL	5	0	1	0	2	...	...	...
ACUTE BRIGHT'S DISEASE, *NOT* FROM SCARLET FEVER.								
CASES BELOW 16 YEARS OF AGE.								
Dr. Dickinson (*loc. cit.*)	3	0	0	0	1	...	...	...
Dr. Greenfield (*loc. cit.*)	1	0	1	0	0	...	...	...
The Author (*St. Mary's Hospital*)	1	0	0	0	0	...	...	...
TOTAL	5	0	1	0	1	...	...	...
CASES ABOVE 16 YEARS OF AGE.								
Dr. Dickinson (*loc. cit.*)	4	0	1	1	1	...	...	...
Dr. Greenfield (*loc. cit.*)	8	2	2	1	2	1	...	...
The Author (*St. Mary's Hospital*)	14	2 1 in 7 14·3%	1 1 in 14 7%	0	2 1 in 7 14·3%	1	...	...
TOTAL	26	4 1 in 6·5 15·4%	4 1 in 6·5 15·4%	2 1 in 13 7·7%	4 1 in 6·5 15·4%	2	...	...
CASES IN WHICH THE AGE WAS NOT SPECIFIED.								
Bamberger, "first stage," (*loc. cit.*)	8	1	1	1	3	1	...	...
TOTAL with Acute Bright's Disease, *not* from Scarlet Fever. All ages	39	5 1 in 7·8 13%	6 1 in 6·5 15·4%	3 1 in 13 7·7%	8 1 in 4·8 20·5%	...	...	...
Transitional Cases, passing from Acute Bright's Disease to the Fatty or Large White Kidney.								
Dr. Dickinson (*loc. cit.*)	4	1	0	0	0	...	...	...
CASES WITH FATTY OR LARGE WHITE KIDNEY.								
Dr. Dickinson (*loc. cit.*)	6	0	2	1	0	...	...	...
Bamberger, "second stage," (*loc. cit.*)	23	1	4	1	5	...	...	...
Dr. Wilks (*loc. cit.*), "large white," 23; "coarse," 5; "fatty," 17, some with Lardaceous Kidney	45	3	8	5	2	...	...	...
The Author (*St. Mary's Hosp. P.M. Records*)	62	1 1 in 62 1·6%	16 1 in 4 25·8%	2 1 in 31 3·2%	16 1 in 4 25%	4	5	...
TOTAL with Fatty Kidney	136	5 1 in 27 3·7%	30 1 in 4·5 22%	9 1 in 15 6·6%	23 1 in 6 17%	...	...	...
GRANULAR KIDNEY, CONTRACTED.								
Dr. Wilks (*loc. cit.*)	31	3	4	2	1	...	...	...
Dr. Dickinson (*On Albuminuria*)	38	16	7	3	7	...	...	...
Dr. Grainger Stewart (*loc. cit.*)	13	1	2	0	1	...	...	...
Bamberger, "third stage," (*loc. cit.*)	16	5	4	0	3	1	...	...
Author (*St. Mary's Hospital*)	128	13 1 in 10 10%	30 1 in 4·3 23·4%	6 1 in 21·3 4·7%	13 1 in 10 10%	11	7	...
TOTAL with Contracted Granular Kidney	226	38 1 in 6 16·8%	47 1 in 4·8 21%	11 1 in 20·5 5%	25 1 in 9 11%			
LARDACEOUS (AMYLOID) DISEASE OF KIDNEY.								
Dr Dickinson (*loc. cit.*, "depurative")	48	3	5	4	9	...	...	...
Dr. Granger Stewart (*loc. cit*)	50	4	3	3	2	...	...	...
Author (*St. Mary's Hospital*, Lardaceous Disease actual and probable)	22	2 1 in 11 9%	3 1 in 7 14·3%	4 1 in 5·2 19·4%	3 1 in 7 14·3%	...	...	2
TOTAL with Lardaceous Disease of Kidney	120	9 1 in 13·8 7·5%	11 1 in 10·8 9·2%	11 1 in 10·8 9·2%	14 1 in 8·5 11·7%			

Pericarditis in Bright's Disease of the Kidneys.

Dr. Bright, in the first volume of Guy's Hospital Reports, gives 100 cases of albuminuria, seven of which, according to the tables, and eight according to his description, had pericarditis. Subsequently Dr. Gregory and Sir James Christison, in Edinburgh; Martin Solon, Becquerel and Rayer in France; and Malmsten in Germany, gave each of them a series or summary of cases of Bright's disease, in all of which cases, except those communicated by Malmsten, pericarditis was either infrequent or absent.

Dr. Taylor called attention, in 1845, to the large proportion in which cases of pericarditis are affected with Bright's disease, and to the frequency with which pericarditis occurs in cases of Bright's disease. He found that out of thirty-one patients with pericarditis, nine, if not eleven, had Bright's disease; and that of fifty post-mortem inspections of cases with Bright's disease, five, or one in ten, had pericarditis.

Several years later, or in 1851, Frerichs published his important work on Bright's disease, which contains a valuable table, showing various conditions that existed in 292 cases collected by him from various sources, and including 21 observed by himself. He states that in 13 of those collected cases there was pericarditis; that is in only $4\frac{1}{2}$ per cent. or 1 in 22 of the cases. This return, which has been, and still is, much quoted, gives a lower proportion of attacks of pericarditis in Bright's disease than in the cases given or enumerated by Dr. Bright (7 or 8 per cent. or 1 in 14 or 12), Dr. Taylor (10 per cent. or 1 in 10), M. Rayer (5·4 per cent. or 1 in 18), and Dr. Gregory (5 per cent. or 1 in 20); and a higher proportion than in the cases observed by Becquerel (1·6 per cent. or 1 in 62). Frerichs appears to

have overlooked some of the cases of pericarditis in his analysis. To test his figures, I examined as nearly as I could the same cases or tables given by the observers quoted by him, and I find that in a total of 326 cases, 17 or 19 had pericarditis, or about 5·5 per cent. or 1 in 18.[1]

During the nineteen years ending in 1869, 285 cases of Bright's disease were examined after death in St. Mary's Hospital, and of these 25, or 1 in 11·3, or 8·8 per cent. were affected with pericarditis ; which was present therefore somewhat more frequently in those cases than in 1,691 collected cases of Bright's disease, 136 of which, or 1 in 12·3 or 8·17 per cent. had pericarditis.

Besides the twenty-five cases of pericarditis noted in the records of St. Mary's Hospital, there were fifteen of partial or doubtful pericarditis ; but these cases ought not, I think, to be taken into the general account.

If we separate the various forms of Bright's disease occurring in St. Mary's Hospital from each other we shall see the proportion in which each form was affected with pericarditis.

SUMMARY.

Acute Bright's disease, from scarlet fever, total number, 6 ; affected with pericarditis, 0 ; with partial pericarditis, 1.

Acute Bright's disease, *not* from scarlet fever, total number, 15 ; affected with pericarditis, 2, or 1 in 7·5, or 13·3 per cent.; with partial pericarditis, 0.

Fatty or large white kidney, total number, 62 ; affected with pericarditis, 1, or 1 in 62, or 1·6 per cent. ; with partial pericarditis, 5.

Contracted granular kidney, total number, 128 ; affected with pericarditis, 13, or 1 in 10, or 10 per cent. ; with partial pericarditis, 7.

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[1] *Frerichs* : Dr. Bright, 100 cases ; Sir James Christison, 14 ; Dr. Gregory, 37 ; Martin Solon, 8 ; Rayer, 48 ; Becquerel, 45 ; Bright and Barlow, 10 ; Malmsten, 9 ; Frerichs, 21 ; Total 292. *Author* : The same authorities respectively ; 100, 14, 39, 10, 55, 45, 9, 33, 21 ; Total, 326.

Cases of pericarditis in the above, Frerichs, 13 : Author, 17 or 19.

Granular kidney of natural or large size, total number, 34; affected with pericarditis, 3, or 1 in 11·3, or 8·8 per cent.

Granular kidney, grand total number, 162; affected with pericarditis, 16, or 1 in 10, or 10 per cent.; with partial pericarditis, 7.

Lardaceous disease of kidney, actual and probable, total number, 22; affected with pericarditis, 2, or 1 in 11, or 9 per cent.

Nature of kidney disease doubtful, 11; affected with pericarditis, 4, or 1 in 2·7, or 36 per cent.; partial pericarditis, 2.

Total number of cases of Bright's disease, 285; affected with pericarditis, 25, or 1 in 11·3, or 8·8 per cent.; with partial pericarditis, 15.[1]

Calculus in kidney, pelvis, or ureter, or dilated pelvis (hydro-nephrosis), total number, 12; affected with pericarditis, 0.

Suppurative nephritis from stricture, &c., total number, 13; affected with pericarditis, 1, or 1 in 13, or 7·7 per cent.

That I might enlarge the area of observation, I have brought together from various sources, including the returns from St. Mary's Hospital, in the accompanying table, the number of attacks of pericarditis in 1,681 cases of Bright's disease; and the number of attacks of pleurisy, peritonitis, and pneumonia, in 1,228 cases (see p. 148).

I have also given in another and more extended table (see p. 144), the number of cases with pericarditis, pleurisy, and peritonitis, pneumonia, pulmonary apoplexy, and purulent deposit or abscess of the lung; and certain conditions of the heart and aorta in the various forms of Bright's disease among the 285 cases examined at St. Mary's Hospital; distinguishing also those cases in which the heart was small, of natural size, rather large, and large or very large, giving separately those various conditions as they appeared in the cases affected with pericarditis.

Among the cases of Bright's disease collected from various sources, 8·1 per cent., or 1 in 12·3, were attacked with pericarditis.

[1] For details of the cases of partial pericarditis see pages 159, 162.

These cases are arranged in three sections devoted respectively to England, Germany, and France; and the occurrence of pericarditis in Bright's disease is here shown to be most frequent in Germany (1 in 9·5, or 10·4 per cent.), and least frequent in France (1 in 33, or 3 per cent.), while it is of medium or average frequency in England (1 in 11·9, or 8·4 per cent).

Comparative frequency of Pericarditis in the various Forms of Bright's Disease.—I have added to the table of 1,682 cases collected from many sources, a series of secondary tables (see p. 149), showing the relative frequency of pericarditis, pleurisy, peritonitis, and pneumonia in the various forms of Bright's disease, in a certain number of the cases; and I shall here inquire into the frequency of pericarditis in the different forms of that disease.

Pericarditis is not frequent in cases of acute Bright's disease from scarlet fever in the young, since it only occurred in 1 in 14, or 7 per cent. of the patients under 16 years of age. The tendency to pericarditis in children in such cases is slight, as was pointed out to me by Dr. Dickinson, who kindly supplied me with the valuable tables of his cases of that class, amounting to 21. Pericarditis is on the other hand frequent in acute Bright's disease in the adult, since it was present in 1 in 6½, or 15·4 per cent. of those cases. The value of these returns has been greatly added to by the cases of acute Bright's disease kindly communicated to me by Dr. Greenfield.

During the transitional period, when acute Bright's disease slowly gives place to the fatty or large white kidney, pericarditis is probably frequent, since it occurred in one of Dr. Dickinson's four transitional cases.

When, however, acute Bright's disease instead of recovering passes into the second or chronic stage, in the form of large white kidney, the tendency to general pericarditis disappears,

since it only occurred in 1 in 27, or 3·7 per cent., of the collected cases, and 1 in 62, or 1·6 per cent. of the St. Mary's Hospital cases, and the kidney in that single case was in the third or contracted stage of fatty disease. Five, however, of the St. Mary's Hospital cases with fatty kidney had partial pericarditis, showing that this affection, although still inherent, does not tend to develop itself in that form of the disease.

The two great and opposite forms of Bright's disease, the fatty kidney, or the chronic stage of acute Bright's disease, and the contracted granular kidney, show a marked difference in the proportion with which they were respectively affected with pericarditis; which attacked those with contracted granular kidney from six to four times as often (1 in 10[1] and 1 in 6[2]) as those with fatty kidney (1 in 62[1] and 1 in 26·6[2]).

Cases of lardaceous disease of the kidney have pericarditis with a moderate or average frequency (1 in 11, or 9 per cent.,[2] and 1 in 13·3, or 7·5 per cent.[2]).

Inquiry into the Influence respectively of the Fatty Kidney, and the contracted Granular Kidney, in the production of Pericarditis.—When inquiring into the influence of these two forms of Bright's disease in the production of pericarditis it may be well to consider two points which appear to be associated with the production of pericarditis, though for different reasons; (1) the proportion in which cases with fatty and contracted granular kidney were affected respectively with pleurisy, peritonitis, and pneumonia: and (2) the relative proportion in which the heart was enlarged and its left ventricle was hypertrophied in those two forms of disease; and the immediate relation, if any, that the enlarged heart may have had to the production of pericarditis.

[1] In 285 cases examined after death in St. Mary's Hospital.

[2] In the collected cases.

1. Pleurisy attacked 60 of the 285 cases with Bright's disease occurring in St. Mary's Hospital (1 in 4·8, or 21 per cent.[1]; and 1 in 6, or 16·4 per cent.[2]) It will thus be seen that in these cases of Bright's disease pleurisy was twice as frequent as pericarditis (1 in 11·3[1] and one in 12·3[2]). We have here a marked difference between the pericarditis of acute rheumatism and the pericarditis of Bright's disease, since while in the former disease, or acute rheumatism, the inflammation of the pericardium is much more common than that of the pleura; the pleurisy when present, being usually either due (1) to the spreading of the inflammation of the pericardium to the pleura, or (2) to pulmonary apoplexy which is the consecutive effect of the double inflammation of the heart, inside and out; in the latter affection, or Bright's disease, the pleurisy is an independent affection, and is, as we have just seen, twice as frequent as pericarditis in the cases under inquiry.

The same in principle may be said of peritonitis, which is practically unknown in acute rheumatism; while it occurs nearly as often as pericarditis in Bright's disease; the numbers being 93, or 1 in 13,[2] and 19, or 1 in 15[1] of peritonitis against 100, or 1 in 12·3,[2] and 25 or 1 in 11·3[1] of pericarditis.

Two-fifths of the cases of pericarditis were also affected with pleurisy (10 in 25), and three-fifths were free from that affection (15 in 25); while only 2 in 25 of those cases had peritonitis.

The relative frequency of pleurisy and peritonitis on the one hand, and pericarditis on the other, varied much in the different forms of Bright's disease.

In acute Bright's disease from scarlet fever in the young, pleurisy occurs three times (1 in 5) and peritonitis twice

[1] In 285 cases examined after death in St. Mary's Hospital.

[2] In the collected cases.

(1 in 7) as often as pericarditis (1 in 14); but it is otherwise in acute Bright's disease in the adult, not from scarlet fever, since in such cases pericarditis is as frequent as pleurisy (each 1 in 6·5), while it is twice as frequent as peritonitis (1 in 11·5).

Pleurisy attacks many more cases (1 in 4[1] and 1 in 4·5[2]) with fatty kidney than pericarditis (1 in 62[1] and 1 in 27[2]); while in those with contracted granular kidney, pericarditis (1 in 10[1] and 1 in 6[2]) occurs, judging by the collected cases, nearly as often as pleurisy (1 in 4·3[1] and 1 in 4·8[2]). Although pleurisy is rather more frequent, pericarditis, as we have seen, is much less so in cases with fatty than in those with contracted granular kidney; and it is therefore evident that the causes producing the two inflammations have but little in common, and that the one rarely excites the other. Peritonitis occurred twice as often (1 in 31[1] and 1 in 15[2]) as pericarditis in cases with fatty kidney, while pericarditis attacked three times as many as peritonitis (1 in 21) in those with contracted granular kidney.

Pleurisy and peritonitis (each 1 in 10·8[2]) were both of them more frequent than pericarditis (1 in 13·3[2]) in cases of lardaceous disease of the kidney.

Pneumonia, which when it occurs by itself is an occasional cause of pericarditis, while it is less common (1 in 6·4[1] and 1 in 7·6[2]) than pleurisy (1 in 4·8[1] and 1 in 6[2]) is more common than pericarditis in cases of Bright's disease. Those two secondary affections, pneumonia and pleurisy, were of exactly equal frequency in cases of acute Bright's disease, whether from scarlet fever or not; so that what has been said with regard to the latter of those affections applies to the former.

[1] In 285 cases examined after death in St. Mary's Hospital.

[2] In the collected cases.

Pneumonia was common (1 in 4[1] and 1 in 6[2]) and pericarditis was rare (1 in 62[1] and 1 in 27[2]) in cases with fatty kidney. It was almost the reverse in those with contracted granular kidney, in which pneumonia (1 in 10[1] and 1 in 9[2]) scarcely equalled pericarditis in number (1 in 10[1] and 1 in 6[2]). The proportion of pneumonia was, therefore, about twice as great in cases with fatty, as in those with contracted granular kidney, while pericarditis, rare in the former, was frequent in the latter form of the disease, making it evident that there was little in common between the production of pneumonia and that of pericarditis in these cases. Pneumonia was present in only one-third of the cases of Bright's disease that were affected with pericarditis (8 in 25).

2. Enlargement of the heart, usually with hypertrophy of the left ventricle, was present in one-half of the cases of Bright's disease under review (129 in 259) in which the size of the heart was described. The heart was large in more than half of the cases of pericarditis in which the size of the heart was defined (10 in 19[3]); or 10 in 129 of the total number of cases of Bright's disease with enlargement of the heart. Pericarditis occurred in six cases in which the heart was of natural size (or 6 in 61). It would thus appear that 1 in 10·1 of the latter in which the heart was natural in size, and 1 in 12·9 of the former, with hypertrophy of the heart, had pericarditis. This would seem to say that hypertrophy of the heart had no apparent influence in the production of pericarditis in these cases. If, however, we add the cases in which the heart was small (23), none of which had general pericarditis, to those in which it was natural in size (61), we

[1] In 285 cases examined after death in St. Mary's Hospital.

[2] In the collected cases.

[3] The size of the heart was doubtful in six cases with pericarditis.

find that 6 in 84 or 1 in 14 of those combined cases had that affection. If to these we join the cases in which the heart was rather large (45) three of which had pericarditis, the result is that 9 in 129, or 1 in 14·3, were thus attacked. From this analysis, it would appear that enlargement of the heart exercised a definite but not a predominant influence over the production of pericarditis in cases of Bright's disease.

Although hypertrophy of the heart is absent in almost one-half of the cases of Bright's disease with pericarditis, we know that in every form and case of that disease, whether acute or chronic, fatty or granular, the action of the left ventricle is unduly strong; for it has to send the poisoned blood through vessels of great tension that oppose resistance to the onflow of the blood. The result is that in every case of Bright's disease, the left ventricle, whether hypertrophied or not, is beating with undue force; and thus tends, by the pressure of its wall with undue force against the pericardium, to induce pericarditis. The heart is prevented from becoming enlarged in many cases of Bright's disease by the exhausting loss of albumen, the general waste, and the lowering character of the disease. This especially applies to cases of fatty, lardaceous, and suppurative kidney. The left ventricle, notwithstanding the great waste of tissue that goes on in those cases, is actually hypertrophied in a certain proportion of them; and it is so in the greater number of those with acute Bright's disease, in spite of the waste of tissue entailed by the great loss of albumen and blood in such cases. We have already seen that in acute rheumatism, over-action of the heart tends to induce pericarditis. It is, therefore, consistent with analogy, reason, and the clinical facts, that in Bright's disease over-action of the heart should increase the tendency to pericarditis, that tendency being already resident in the disease. May it not be that, on the one hand, the lessened

force of the heart, induced by the weeping of albumen, dropsy, and other secondary wasting diseases in cases with fatty disease of the kidneys, explains to some extent the rarity of general pericarditis (1 in 62[1] and 1 in 27[2]), and the comparative frequency of partial and undeveloped pericarditis (1 in 12·4), in that disease? and that on the other hand, the increased size and action of the heart in cases with granular kidney, which usually lose little albumen, are not dropsical, and are free from exhausting secondary disease, tend to increase the frequency of general pericarditis in that affection (1 in 10,[1] and 1 in 6[2])?

Although the cases of partial pericarditis, which amounted to fifteen, cannot be classed rightly with those of general pericarditis—for the partial variety appears to have a tendency to remain partial, and those cases are not usually included among those with pericarditis,—yet those cases ought to be studied. One of the fifteen cases of partial pericarditis had acute Bright's disease from scarlet fever (1 in 6, or 16·6 per cent.); five of them had fatty kidney (5 in 62, or 1 in 12·4, or 8 per cent.); seven of them had contracted granular kidney (7 in 129, or 1 in 18·3, or 5·5 per cent.); and in two the state of the kidney was not specified.

The proportion in which partial and general pericarditis respectively attacked the different forms of Bright's disease somewhat correspond.

In four of the cases of partial pericarditis the heart was very large (1 in 32·2), and in three it was rather large (1 in 15); while in five of them the heart was of natural size or small, (1 in 16·8), and in three the size of the heart was not described.

[1] The cases of Bright's disease examined after death in St. Mary's Hospital.

[2] The collected cases.

It thus seems that great enlargement of the heart does not favour the persistence of partial pericarditis, but rather tends to develop it into general pericarditis.

Amount of Fluid in the Pericardial Sac in Pericarditis from Bright's Disease.—The amount of fluid in the pericardial sac varied considerably in the twenty-five cases of pericarditis from Bright's disease, the smallest quantity being two drams, and the largest about a pint, in which case the contents of the sac were purulent.

In one-fifth of the cases (5) the contents of the pericardium are not described; and in one-fifth of them (5) there were recent adhesions. The sac contained only a small quantity of serum, or not more than one ounce in one-third (5) of the remaining cases (15); a moderate amount, or a few ounces, in another third of them (6); and much fluid, eight ounces in one instance, a pint in another, in the remaining third (4) of those cases. It is evident that the presence of adhesions, or of a small, a moderate, or an abundant amount of fluid in the pericardium, depends on the stage of the pericarditis at the time of death; and that in the several cases the fluid had either been removed, or was lessening, increasing, or at its height, when the final observation was made. It may, I think, be admitted that in the pericarditis of Bright's disease there is less effusion in the pericardium than in rheumatic pericarditis; but from the evidence here given it would appear that there is no very material difference in the amount of fluid in the sac at the time of death in the two classes of cases.

Character of the Exudation on the Surfaces of the Heart and Pericardial Sac in Pericarditis from Bright's Disease.—In a small proportion of cases the lymph covering the heart and lining the pericardium in case of pericarditis from Bright's disease presents the same pale and rough surface, firm to the

finger, with "cat's-tongue"-like projections, so usual in pericarditis from acute rheumatism. It was thus in two of the twenty-five cases that were examined after death at St Mary's Hospital. In two other cases also, both of acute Bright's disease, a rather firm layer of fibrin easily peeled off from the heart, leaving a finely-injected red surface underneath.

In the majority of cases of pericarditis from Bright's disease the exudation differs from that usual in rheumatic pericarditis. Universal adhesions of the heart, rare in the latter, are common in the former affection; the heart having been completely adherent in three instances, extensively so in one, and doubtfully so in another of those cases. There was pus in the sac in two cases. The lymph—was soft, granular, imperfectly organized, or in patches in six, in two of which the presence of pericarditis was perhaps doubtful; or was bloody or very red on the surface, or mixed with blood, in three of the twenty-five cases of pericarditis from Bright's disease. These conditions, which affected nearly two-thirds of those cases, are rare or unknown in rheumatic pericarditis. The remaining cases were less definite in character, the heart in four of them having been covered by recent lymph, while in two the pericardium was affected with "recent pericarditis."

Appearances in Partial Pericarditis.—The cases of partial or doubtful pericarditis varied much in their features. In four of them flakes of lymph floated in the serum contained in the pericardial sac, the surfaces of the heart not being named. Pericarditis was limited, slight, or in traces or patches in seven other cases, and in two more it was highly vascular or congested. One case presented rough lymph easily detached, leaving an apparently healthy surface; and in the last instance there was a red fluid containing flakes of lymph in the sac, and lymph on the heart, the surface of which was healthy. These two cases, and the four in which flakes of lymph

floated in the serum, were probably free from actual pericarditis.

Physical Signs of Pericarditis occurring in Bright's Disease.—Dr. Taylor gives careful reports of nine cases of Bright's disease with pericarditis, in three of which there was a friction-sound, while in six of them there was no definite sign of the affection. In three of these six cases there were complete recent adhesions, rendering friction-sound impossible. In one of the three cases in which pericarditis was not discovered during life, a layer of soft lymph coated the heart, but there was no lymph on any part of the loose pericardium, and this appears to account for the want of friction-sound. In one of the three cases that presented a friction-sound, a double creaking noise was heard between the apex of the heart and the sternum; and the heart and sac were covered with soft, slightly rough lymph.

In two of the three cases without friction-sound, excluding the three with complete adhesions, and in two of the three with friction-sound, there was no adequate explanation, after death, of the absence of that sound in the two former cases, in which the opposed surfaces of the heart and sac were rough and scabrous; nor of its presence in the two latter cases in one of which there were extensive adhesions of the heart; while in the other the surface of the heart was simply red from fine injection, and there were but a few spots of lymph on the anterior coronary artery.

I possess notes of the symptoms during life, and the appearance after death of nine fatal cases of Bright's disease with pericarditis. I cannot find the notes of a tenth case with regard to which I find two lines of an abstract of symptoms. In seven of the cases immediate signs of pericarditis were observed, and in three of them the signs of pericarditis were not observed.

Cases in which the Signs of Pericarditis were not observed.—In one patient, a man, aged 61, with granular kidneys, the heart, which was very fat, was covered and the sac was lined with recent lymph. On the third day after his admission, on which day he died, the heart's action to the left of the ensiform cartilage was loud; and loud mucous rattles were audible all over his chest. In the second case, a man, aged 47, the opposite surfaces of the pericardium, and the heart, at its base, and along the great vessels were rough with a deposit of fibrin. This patient was in the hospital fifty-two days, but there is only one note of the state of his heart, which was on the fifth day after his admission, when its sounds were rather loud.

I cannot find the notes of the remaining case with Bright's disease and pericarditis; but the following is the brief abstract preceding the notes of the examination after death. "At first, doubling of the first sound, afterwards systolic murmur after epistaxis," so that friction-sound was evidently not observed in this case.

Cases in which the Signs of Pericarditis were observed.—(1) A creaking noise with a thrill was present in three of the seven cases of pericarditis with friction-sound; (2) a creaking sound without a thrill in two of them; and (3) in the remaining two there was a "friction-sound."

(1.) *Cases with Thrill and a Creaking Friction-sound over the Seat of the Impulse, and Frottement extending far beyond and especially below the Region of the Pericardium.*—There were three cases of this class. One of them, a woman, aged 32, who was in the hospital for a week, presented after death some fluid in the pericardium, and a rough deposit of recent lymph of a bright red colour, which covered the heart and lined the sac. On the day after her admission a systolic murmur was audible over the cardiac region. Two days

later, when she complained of pain going across the chest, the upper border of cardiac dulness was situated at the third space; and a rasping, creaking friction-sound, chiefly systolic, was heard all over the front of the chest, and down to the eighth and ninth costal cartilages, its maximum intensity being at the centre of the sternum, and during the middle of the systole. Next day a strong thrill extended over the heart from the right of the sternum to the nipple, and as high as the third cartilage; and the creaking sound was triple, being exactly like that made by the rise and fall and rise in the saddle. On the following day, the fifth, the thrill was less intense, and there was a triple creak at the apex, the friction-sound being still audible over the lower cartilage; and two days later she died.

The second patient, a woman, aged 27, with contracted granular kidney and pericarditis, had several patches of recent lymph on the surfaces of the heart and the free pericardium, and presented a double thrill, a double creak, and an extensive friction-sound, which were all absolutely suspended for one day, under the influence of flooding.

The third case, a man, aged 33, had mitral-aortic incompetence, and highly albuminous urine. The heart and pericardium were greatly increased in size, and the right ventricle was covered with a white fibrinous structure, rough to the finger, like a cat's tongue. On admission he had pain over the heart; and for two days, mitral and double aortic murmurs were audible. He became worse, and on the fourth day the diastolic murmur disappeared. On the ninth day he was drowsy, a strong thrill was felt with each impulse from the third cartilage to the fifth; a loud grating double friction-sound was present over the seat of the thrill, the rubbing noise radiating thence up to the top of the sternum, down to the eighth cartilages, and to the left and right; a leather

creak was audible at the apex; and a sound of a friction character was heard behind, over the dorsal spine. On the next day, when he died, the vibration had increased, and extended from the third to the seventh cartilages; it lessened in extent above, on inspiration; below, on expiration; and was accompanied by a loud creak during systole, and a fainter creak during diastole, the sound spreading from the seat of the vibration over the front of the chest and the upper third of the belly.

(2.) *Cases with a Creaking Friction-sound, no Thrill being observed, over the Seat of the Impulse, and a Frottement extending beyond, and especially below the Region of Pericardial Dulness.*—One of the two cases of this class was a young married woman, with granular disease of the kidney. A firm coating partly in ridges and partly like a cat's tongue covered the heart and lined the sac. On her admission a creaking systolic friction-sound was audible at the apex, in the fifth space. Four days later, when the pericardial dulness was at its acme, reaching up to the third cartilage, her respirations being fifty, the friction-sound was no longer creaking but presented itself as an occasional brush; but three days after this, or on the eighth day, there was a loud leather creak over the whole region of the pericardium. After this the friction-sound almost disappeared; but on the twelfth and preceding days it had again burst into full play as an extensive leather creaking noise, covering the whole pericardium, and extending down to the seventh cartilage; and eight days later she died.

In the second case, a man, aged 30, with small, probably granular, kidneys, recent, bloody, honeycombed lymph lined the pericardium and covered the heart. On the day of his admission the two sounds of the heart were indistinct. Next day the impulse was extensive, and a loud double creaking

sound, more intense during systole, occupied the whole region of the heart, extending downwards to the seventh and eighth cartilages, and into the epigastrium. During the next few days the *frottement* was much smoother and more restricted in area. On the eighth day he was weak and in distress; the friction-sound was audible over the whole pericardium, and beyond it, from the top of the sternum to the lower cartilage; and he could scarcely swallow or speak: and in the evening he died.

(3.) *Cases with "Friction-sound."*—One of the two cases of this class, a man aged 38, with granular kidney of full size, had recent lymph over the whole surface of the heart, and in some places the heart and pericardium were adherent, by cord-like prolongations of lymph. On the fifty-seventh day there were doubling of the second sound, and a murmur over the third cartilage. On the seventy-fifth day, which was eight days before his death, "double friction-sound over the pericardium," was noted for the first time. Three days latter the pericardial friction-sounds, which were scarcely audible without making pressure, were mingled with pleuritic friction-sounds; but after this he was too ill for examination.

The other patient, an old woman, with contracted granular kidney and pericarditis, the whole surfaces of the heart and sac being covered by recent soft granular lymph, complained, on the twenty-first day after her admission, of great pain at the region of the heart. Next day there was pericardial dulness, and friction-sound was present between the sternum and the left nipple; and three days later she died.

Several of these seven cases of Bright's disease and pericarditis presented certain broad features in common. In three of them a thrill or tactile vibration could be felt over the region of the heart's impulse, extending from the third to the fifth, the sixth, and in one instance to the seventh cartilages.

In one of these cases the thrill extended from the right border of the sternum across the chest to the nipple. In these three cases, and in two others in which a thrill was not observed, a loud sound like the creaking of new leather, usually double, but more intense and prolonged with the systole, was audible over the whole seat of the thrill, or when that was absent, over the region of the heart's impulse. The friction-sound was, however, in none of the five instances restricted to the area of the thrill or impulse, or even of the distended pericardium; but extended upwards to the top of the sternum, downwards to the right and left along the seventh and eighth costal cartilages, and over and even below the ensiform cartilage. In these cases the widespread friction-sound became softer in tone, and especially downwards, as it widened away from the focus of its greatest intensity. In two of these five cases with creaking and extended friction-sound the deposit of fibrin or lymph on the surface of the heart was firm and like a cat's tongue, in one of them it was rough, in one it was bloody and honeycombed, and in the fifth, patches of recent lymph were present on the heart.

In three of these cases there was a period of complete or partial suspension of the creaking and extensive friction-sound; which after spreading with great intensity, and over a large area, became silent or feeble and contracted in area for a time, and then suddenly burst forth again with full intensity, and over a wide space. It was evident that under these circumstances, some influences were at work exciting the heart at the time of the creaking and widespread friction-sound, and depressing the heart when that sound ceased or became feeble. In one instance, the suspension of the thrill and creak was traced to the influence of flooding.

In the two other patients the surface of the heart is described as being covered with recent, and in one of them with

soft lymph. In neither of them is it noted that the coating of lymph was rough. In both of these cases it is simply stated that a "friction-sound" was present over the region of the heart.

In all of these patients pressure intensified the friction-sound.

Cases with a Friction-sound that were not Fatal, or not examined after Death.—Besides these seven fatal cases of Bright's disease with pericarditis in which friction-sound was observed during life, I find three other cases in which the signs of pericarditis were observed when the patients were in the wards.

One of these cases, probably fatal, admitted during the recess, very imperfectly recorded, presented a pericardial friction-sound, which was chiefly present at and below the left nipple.

Another patient, a carpenter, aged 35, had Bright's disease and aortic regurgitation of some standing. On the eighty-second day he had great pain in the heart, and four days later a rough double noise resembling a friction-sound was audible over the cardiac region. Four days after this there was dulness over the pericardium from the third space downwards, and pain over the heart, relieved by leeches; and next day a to-and-fro friction-sound was audible over the heart, which continued for six days; after which, when he was in distress from aching over the heart, and sickness, the rubbing noise vanished, being replaced by the most diastolic murmur of aortic regurgitation. This case left the hospital in improved health.

The last case of Bright's disease with friction-sound was one of great interest, a cab-driver, aged 45. His urine was loaded with albumen, and contained coarse granular and fatty casts. There was, on the fourth day, an extensive impulse,

and a remarkable doubling of the first sound heard all over the region of the impulse, which was heard along with, but apart from, a peculiar pericardial friction-sound chiefly systolic, which was audible for two inches below the nipple. This sound which was rasping at first, became creaking two days later, and five days after that was only audible when pressure was made over the same spot, the sound being like that caused by rubbing together two pieces of emery paper. Next day there was great extension of the friction-sound, which required no pressure for its production, over the whole region of the pericardium ; and four days later, the seventeenth after admission, the friction-sound was soft, double and murmur-like, chiefly heard on pressure, and was accompanied by the natural heart sounds, with which it was not rhythmical. I could not make out which sound had the start of the other. For a few days a systolic friction murmur was audible on passing beyond the nipple line, and a double rustle was heard on pressure down to the twenty-eighth day. The extensive doubling of the first sound held its ground throughout, and on the forty-fourth and fifty-third days a little *frottement* was again present, produced by pressure. On the sixty-fifth day he felt lighter over the heart, and a tremor or thrill was perceived, extending over the cardiac region from the right to the left nipple. A loud double new-leather creak extended over the whole of this region, but the rubbing noise spread far and wide, being heard from axilla to axilla, and down the ensiform and seventh and eighth cartilages. The thrill and creak retained their intensity and area for five days, but on the sixth day the thrill was feeble, and the creek was replaced by a to-and-fro sound extending from the third to the sixth cartilage. Doubling of the first sound was mixed up with the friction-sound, but pressure intensified the latter and extinguished the former. On the seventy-third day there

was no thrill, and a systolic friction-sound, double on pressure, was present between the fourth and sixth cartilages. Two days later the rubbing sound was no longer audible without pressure, and was quite lost on the seventy-ninth day. In this remarkable case the friction-sound was present over a limited region near the apex, from the fourth to the twenty-eighth day ; came into play slightly on the forty-fourth and fifty-third days ; and on the sixty-fifth day burst out, with a thrill, with great intensity over the region of the impulse, and radiated thence as from a focus, all over the front of the chest, and down to the eighth costal cartilages, being audible with a lessening area and diminishing intensity to the seventy-fifth day. This long and intermittent duration of pericardial friction-sound appears to me to be peculiar to the pericarditis of Bright's disease, and is certainly never found in rheumatic pericarditis.

These ten cases—which I have given with some detail, as with the exception of Dr. Taylor's cases and two related, in this respect briefly, by Traube, I have found no cases of pericarditis from Bright's disease in which the signs are related—presented features that are common in them, but are comparatively rare in rheumatic pericarditis. A thrill was present, as we have just seen, in four of these cases, or almost one-half (4 in 10) ; and a sound like the creaking of new leather was heard in six of those cases, or more than one-half (6 in 10), over the seat of the thrill or impulse ; and that radiated thence as a softening sound over the front of the chest, beyond the region of the pericardium, and downwards over the ensiform cartilage and the seventh and eighth costal cartilages. These signs were much less frequent in rheumatic pericarditis, since a thrill was present in only one-fifth of those cases, or 13 in 63, and was distributed over the region of the impulse in only seven, was limited to the second space

in two, to the apex in three patients, and to both those regions in one ; and a creaking friction-sound was present at or near the time of the acme of the pericardial effusion in about one-fourth of those cases, or about 18 in 63. The long duration of the friction-sound, and its frequent suspension, observed in several of those cases of pericarditis from Bright's disease, likewise distinguish them from those with rheumatic pericarditis.

Calculus in Kidney, Pelvis, or Ureter ; or Dilated Pelvis :— and Suppurative Nephritis from Stricture, &c.—I have added, in the Table of Pericarditis in Bright's disease, two sections of cases that, without ranking under that affection, float upon its borders; and substantially belong to the same disease in this respect—that the blood is poisoned, owing to the retention within it of the débris of the broken-up tissues of the body, owing to the imperfect action of the diseased kidney. In the first series, the secreting structure of the kidney is often atrophied by the backward compression of the organ, owing to the distension of the pelvis from the presence of calculus in the ureter, pelvis, or kidney. None of these cases, amounting to twelve, had pericarditis. In the second series of cases, numbering thirteen, there was suppurative disease of the pelvis or kidney, owing mainly to stricture or disease of the prostate, or bladder (in 11 cases) ; in one case, to calculus in the ureter, and in another to pyæmia. One of these cases had pericarditis.

I refer to the Table for the general condition of these two sets of cases.

Pericarditis, neither Rheumatic nor from Bright's Disease.

Rheumatic pericarditis, so common in the wards, is rare in the post-mortem room; and pericarditis, as we have seen, occurs in as many as eight or nine per cent. of all fatal cases of Bright's disease. Although uncomplicated pericarditis is a very rare affection, yet its association with other diseases when fatal, and generally as an effect of those diseases, is by no means rare. There is no single malady that is associated with pericarditis nearly so often as the two just mentioned; yet if we combine all the other fatal cases with that affection, except those with Bright's disease and acute rheumatism, we shall see that pericarditis is found on examination after death nearly twice as often in those combined affections as in Bright's disease, and three or four times as often as in fatal cases of acute rheumatism.

The records of the examinations made after death at St. Mary's Hospital during the nineteen years ending 1869-70 contain forty cases of pericarditis that were neither rheumatic nor from Bright's disease. The accompanying summary shows that thirty-nine of these cases of pericarditis were associated with some other disease, general or local, and that in only one case was the affection uncomplicated.

Besides these forty cases of pericarditis, there were sixteen with partial or slight pericarditis.

In addition to these cases I have analysed in one view (1) Dr. Chambers' complete and valuable table of the causes of pericarditis in 136 cases observed after death in St. George's Hospital during ten years; (2) thirty-seven cases with pericarditis published in the *Pathological*

Transactions; and (3) seventy-nine cases collected from various sources.[1]

(A.) Three cases of pericarditis and three of slight pericarditis had pyæmia, one had scarlet fever, and in one the affection was associated with tubercular disease of the supra-renal capsule; (B.) twelve cases of pericarditis were associated with affections of the heart or aorta; (C.) fifteen with affections of the lungs or pleura; (D.) one with ulcer, and one with cancer of the œsophagus; (E.) five with affections of the abdomen; (F.) and besides these cases of secondary or associated pericarditis, there was, as I have just said, one in which the affection appeared to be primary, or uncomplicated.

(A.) *General Diseases.*—One of the three cases of pyæmia was a schoolboy whose leg was doubled up under him five days before his admission. He came in with hurried breathing, blue lips, and tenderness over the chest and abdomen; on placing the hand over the heart a sense of friction was felt, and a loud pericardial friction-sound was heard all over the cardiac region. He had delirium, and died during the night. The surfaces of the heart and sac were covered with recent lymph in ridges, and connected by threads; and the muscular substance of the heart was firm, and contained numerous minute purulent dots scattered through the fibres of the left ventricle. Dr. Trotter observed this patient.

This case is typical of a frequent method in which pyæmia induces pericarditis. In such cases the inflammation does not at once attack the surface of the heart, but spreads to it from

[1] Corvisart (6); Bertin (5); Andral (9); Bouillaud (16); Dr. Stokes (13 including 4 from Testa); Dr. Law (2); Sir Thomas Watson (3); Tringel (13); Dr. Graves (5); Dr. Mayne (3); Dr. Green (1); Dr. Beattie (2); and Dr. Thwaites (1): Total, 79 cases.

(A.) PERICARDITIS AND PERICARDIAL ADHESIONS—

I. NOT ASSOCIATED WITH ACUTE RHEUMATISM OR BRIGHT'S DISEASE.
II. ASSOCIATED WITH BRIGHT'S DISEASE.

CASES.	I. Not associated with Bright's Disease.					II. Associated with Bright's Disease.				
				Pericardial adhesion.			Pericarditis.		Pericardial adhesion.	
	Total	With Pericarditis.	With slight Pericarditis.	General.	Partial.	Total.	General.	Partial.	General.	Partial.
A—Pericarditis, &c., associated with general or Constitutional Disease :—										
Secondary inflam. pyæmia. phlebit. erysip.	71*	3	3	2	...	12	...	1	...	...
With scarlet fever and mitral endocarditis .	1	1	...	...	...	...	...	...	...	...
With disease of the supra-renal capsules .	1	1	...	...	...	...	...	...	...	...
With cancer, not includng. ? 10 cancer of heart	63	...	1?	...	...	...	...	...	...	...
A—Total	136	5	4	2	...	12	...	1	...	...
B—Pericarditis, &c., associated with affections of the Heart :—										
Wound of the Heart	2	1	...	...	...	...	...	...	...	...
Tubercular pericarditis (both had phthisis)	2	2	...	...	...	...	...	...	...	...
Aneurism of the heart	1	1	...	...	...	...	...	...	...	...
Aneurism of the ascending aorta	25	...	1	3	...	...	...	...	...	...
Cancer of the heart (total cases of cancer 73)	10	1	...	1	...	...	...	...	...	...
Heart enlarged without valvular incompetence or any other disease—										
a. valves healthy in structure	9	1	...	6	...	54	3	3	4	...
b. with some thickening of mitral valve .	2	...	...	1	...	19	...	...	...	...
Mitral regurgitation	33	5	1	13‡	...	29	2	2	4	1
Mitral obstruction	21	...	2	1	2	9	1	...	1	...
Aortic regurgitation—*a*. ascending aorta, not stated to be atheromatous . . .	13	...	1	...	1	15	...	...	3	1
b. aorta atheromatous, valve diseased. .	14	...	...	1	...	5	1	...	...	...
c. aorta dilated, valve incompetent, flaps healthy	5	...	...	1	...	1	...	...	...	...
Mitral-aortic valve-disease	31	1	...	11	2	20	1	...	...	1
Tricuspid valve-disease	1	...	...	1	...	...	...	...	...	...
Fatty disease of heart	11	...	...	1	...	...	...	...	...	...
B—Total	180	12	5	40	5	152	8	5	13	3
Total with valvular disease . .	118	6	4	28	5	79	5	2	8	3
Apoplexy	17	...	...	1	...	...	...	...	...	...
C—Pericarditis, &c., associated with affections of the Lungs and Pleura :—										
Pneumonia	49	8	3	2	1	40	7	1	2	...
Pleurisy, not with empyema or pneumonia .	22	3	2	...	...	39	3	4	1	...
Empyema, not including pneumonia . . .	14	2	...	2	...	...	...	...	...	...
Pneumothorax	8	...	...	...	...	...	...	...	...	...
Phthisis, not including pneumothorax, 5; empyema, 2 ; tubercular pericarditis, 2. .	126	2†	1	3	...	25	...	...	...	...
Bronchitis, emphysema	18	...	...	...	...	...	...	...	...	...
C—Total	237	15	6	7	1	104	10	5	3	...
D—Pericarditis, &c., asso iated with ulcer, 1 ; cancer, 1, of the œsophagus	2	2	...	...	...	...	...	...	...	...
E—Pericarditis asso. with affec. of the abdo. :—										
Abscess of liver, 3a ; (of diaphragm, 1) . .	4	2a	...	...	...	...	...	...	...	...
Diaphrag. hernia, 1 ; tumr. con. with stom. 1	2	2	...	...	...	...	...	...	...	...
Peritonitis, including 6 tubercular peritonitis, not including 5 phthisis. Total cases .	71	1	1	1	...	19	2	1	1	...
E—Total	77	5	1	1	...	19	2	1	1	...
F—Pericarditis appar. not asso. with other affec.	1	1	...	...	...	9	5	4	...	...
Pericardial adhesion ; heart healthy, nat. in size	1	...	...	1	...	...	...	...	...	...
Total	651	40	16	52	6	285§	25	16	17	3

* Had also endocarditis.

† One with tubercular pericarditis.

‡ In four of these cases the mitral-valve was thickened, but it was doubtful whether there was regurgitation through the mitral orifice.

§ In this column, the same case often reappears under different headings.

Cases with douhtful indications of Bright's disease, but without albuminuria or general dropsy :— Pyæmia, 6 ; mitral regurgitation, 1 ; mitral contraction, valve disease of aorta, 6 ; mitral aortic valve-disease, 5 ; pneumonia, 5 ; pleurisy, 4 ; phthisis, 10 ; peritonitis, 4.

(B.) SUPPLEMENTARY TABLE, *showing the Size of the Heart in the cases enumerated in the preceding Table,* (1) *in their total number;* (2) *in those with Pericarditis complete and partial; and* (3) *with Pericardial Adhesions.*

CASES.	Total Number.	Heart enlarged.				Heart rather large				Heart of natural size, or "healthy."				Heart small.				Size of heart not described or known.			
		Total.	Pericarditis. General.	Pericarditis. Partial.	Pericardial Adhesions.	Total.	Pericarditis. General.	Pericarditis. Partial.	Pericardial Adhesions.	Total.	Pericarditis. General.	Pericarditis. Partial.	Pericardial Adhesions.	Total.	Pericarditis. General.	Pericarditis. Partial.	Pericardial Adhesions.	Total.	Pericarditis. General.	Pericarditis. Partial.	Pericardial Adhesions.
A—*General Disease.*																					
Pyæmia	71	3	...	...	1	9	...	...	...	44	...	3	...	4	...	...	...	11	3	...	1
Scarlet fever	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	..	...
Addison's disease . .	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Cancer (not including 10 with cancer of heart)	63	1	...	...	...	3	...	...	...	48	...	1	...	3	...	...	...	8	...	...	...
A—TOTAL . .	136	4	2	...	1	12	...	...	..	92	...	4	...	7	...	...	...	19	3	...	1
B—*Diseases of Heart and Aorta.*																					
Wound of heart . . .	2	...	...	...	...	...	...	...	...	2	1	...	...	...	...	...	...	...	...	...	...
Tubercular pericarditis	2	1	1	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Aneurism of heart . .	1	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Aneurism of asc. aorta	25	7or8	...	...	2	8or9	...	1	...	2	...	...	...	...	...	...	...	7	...	...	1
Cancer of heart . . .	10	3	1	...	1	...	...	...	..	2	...	...	...	2	...	...	...	3	...	...	...
Uncomplicated enlargement of heart, except with adhe. peri. . .	11	11	1	...	7	...	...	...	..	...	...	...	...	...	...	...	...	...	...	...	...
Mitral regurgitation .	33	20	3	1	10	5	1	...	...	2	...	...	...	...	...	...	...	6	1	...	3
Mitral obstruction . .	21	8	...	2	1	6	...	...	1	5	...	...	...	..	...	...	...	2	...	...	1
Aortic regurgitation .	32	25	...	1	2	2	...	...	...	1	...	...	...	1	...	...	...	3	...	...	1
Mitral-aortic valve dis.	31	26	1	..	11	1	...	...	...	...	...	...	...	...	...	...	...	4	...	...	2
Tricuspid valve disease	1	1	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Fatty degeneration of the heart	11	...	...	...	...	4	...	...	1	5	...	...	...	...	...	...	...	2	...	...	...
Total affection of heart and pericardium . .	180	104	8	4	35	27	2	1	2	19	1	...	...		...	...	...	27	1	...	8
Total with valvular disease	118	78	4	4	25	14	1	...	1	8	...	...	...	1	...	...	...	15	1	...	7
Apoplexy	17	7	...	...	1	...	...	...	...	7	...	...	...	1	...	...	...	2	...	...	...
C—*Diseases of Lungs and Pleura.*																					
Pneumonia	49	5	1	...	1	17	...	...	2	17	1	2	...	...	...	...	...	10	6	1	...
Pleurisy	22	1	...	..	...	3	...	...	...	13	...	1	...	...	...	...	...	5	3	1	...
Empyema	14	...	...	...	...	4	1	...	...	5	...	...	...	1	...	...	...	4	1	...	2
Pneumothorax . . .	8	...	...	...	...	...	...	...	...	6	...	...	...	...	...	...	...	2	...	...	...
Phthisis	126	6	...	...	...	32	1	...	1	63	...	1	1	10	...	...	...	15	1	...	1
Bronchitis	8	1	...	...	...	6	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...
Emphysema	10	7	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...
C—TOTAL . .	237	20	1	...	1	63	2	...	3	105	1	4	1	11	...	...	...	38	11	2	3
D—*Ulcers, Cancers of Œsophagus.*	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	2	...	...
E—*Diseases of Abdomen.*																					
Peritonitis includ. 6 of tubercular peritonitis, not inclu. 5 of phthisis	71	3	1	...	1	5	...	...	...	45	...	...	...	6	...	...	...	12	...	1	...
Abscess of liver, 3; of diaphragm, 1; diaphr. hernia, 1; tumour connected with stomach, 1	6	...	...	...	...	2	1	...	...	2	1	...	...	...	...	...	...	2	2	...	...
E—TOTAL . .	77	3	1	...	1	7	1	...	...	47	1	...	...	6	...	...	...	14	2	1	...
Uncomplic. pericarditis	1	...	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...
Uncomplicated pericardial adhesions . .	1	...	...	...	...	...	...	...	...	1	...	...	1	...	...	...	...	...	...	...	...
GRAND TOTAL .	651	138	12	4	39	109	5	1	5	272	4	8	2	28	...	...	...	104	19	3	12
BRIGHT'S DISEASE.	285	129	10	4	17	46	3	3	1	61	6	4	...	23	...	1	...	26	6	3	2

(C.) PERICARDITIS IN ITS ASSOCIATION WITH OTHER DISEASES.

Cases collected from all sources.

CASES.	A. Dr. Chambers' (Decennium Pathologicum) Pericarditis.	B. Post-Mortem Record, St. Mary's Hospital Pericarditis. General.	B. Partial.	C. Pathological Transactions. Pericarditis. General.	C. Partial.	D. Various Authors. Pericarditis.
Pericarditis associated with Acute Rheumatism	19	?	...	8	1	13
With Bright's disease	36	25	15	3	1	...
Dropsy	...	...	...	...	...	1
A—With general or Constitutional Diseases:—						
Pyæmia, secondary inflammation	18 or 17	3	3	5	1	3
Erysipelas (included with Pyæmia, St. Mary's Hospital)	4	...	...	...	...	1
Small-pox	...	...	...	...	...	1
Fever	4	...	...	...	...	1
Scarlet Fever	...	1	...	...	...	...
Cutaneous eruption	...	...	...	...	...	1
Tubercular disease, supra-renal capsule	...	1	...	...	...	...
Tubercle	...	...	...	1	...	...
Cancer	...	...	1?	1	...	...
Syphilis	...	...	...	...	...	1
A—Total	26 or 27	5	4	7	1	8
B—With Affections of the Heart and Aorta:—						
Wound of the heart	...	1	...	...	...	...
Blow over the heart (1), fracture of the sternum (1)	1	...	...	...	...	1
Tubercular pericarditis	...	2	...	1	...	1
Cancer of heart, pericardium or neighbourhood	3 or 4	1	...	1	...	1
Neighbouring abscess (2 in heart)	2	...	...	...	...	2
Fibroid disease of walls of heart	...	...	...	2	...	...
Aneurism of heart	...	1	...	...	...	...
Aneurism of ascending aorta	2	...	1	3	...	2
Enlargement of heart without assigned complications.	...	1	...	...	...	...
"Diseased heart" and "dropsy"	18	...	...	...	...	...
Valvular disease of heart	...	6	4	3	1	8
Cyanosis, malformation	...	...	...	1	...	...
B—Total	26	12	5	11	1	15
C—With Affections of the Lungs and Pleura:—						
Pneumonia (generally with pleurisy)	10	8	3	...	...	11
Pleurisy (including empyema)	5	5	2	1	...	12
Phthisis	8	2	1	...	...	3
Communication between pericardium and abscess of lung	1	...	...	...	...	1
Indefinite affection of the chest	...	...	...	...	...	1
C—Total	24	15	6	1	...	28
D—With wound (1); slough (1); ulcer (1); and cancer (1) of œsophagus	1	2	...	1	...	...
E—With Affections of the Abdomen, including the Diaphragm:—						
Diaphragmatic hernia (1); tumour connected with stomach (1)	...	2	...	...	...	...
Abscess of liver (3); one communicated with pericardium	...	2	...	...	...	1
Peritonitis	3	1	1	...	...	...
E—Total	3	5	1	...	...	1
Pericarditis not associated with other affections	2?	1	...	1	1	13
Grand Total	136	40*	16*	32	5	79

* Not including those from Bright's disease

the points of suppurative inflammation minutely scattered through the muscular walls of the organ, just as pleurisy is caused by the masses of suppurative inflammation spread through the lungs. Dr. Moxon[1] has seen several cases of pyæmic abscesses of the heart, mostly in youths with suppurative periostitis, or acute necrosis of the long bones, in which pericarditis was often caused by the bursting of small abscesses into the pericardium. This is not however the invariable mode in which pericarditis is caused by pyæmic abscesses of the heart, since in my case, just given, and in Mr. Stanley's,[2] there was evidently no rupture of the minute collections of pus in the walls of the heart. Dr. Moxon finds that in cases with pyæmic inflammation of the lung near its surface the pleura becomes involved, and thus every diseased portion of tissue is covered with a layer of lymph ; and that when general pleurisy takes place, the abscess has generally burst into the pleura, and so caused the serous inflammation (p. 32). This well represents the parallel conditions in cases of pericarditis caused by pyæmic abscesses in the heart.

Another case may be named, a man, who had rigors on the day after being operated upon for perineal fistula, and was seized on the following day with violent pain in the region of the heart, the sounds of which were natural. Next day there was a distinct pericardial friction-sound, which was feeble in the evening, and was not again distinctly audible. He died on the twelfth day after the operation, and the pericardium was found to be adherent to the heart by a thick layer of recent lymph. In this case, unlike that related above, the pyæmic inflammation evidently struck directly at the pericardium, since violent pain seized the heart the day

[1] *Lectures on Pathological Anatomy*, by Dr. Wilks and Dr. Moxon, p. 122.
[2] *Medico-Chirurgical Transactions*, vii. 323.

after the operation, and next day there was a pericardial friction-sound. These two cases show the rapidity with which the processes of inflammation pass through their stages in pyæmia.

Pyæmia, including with it erysipelas, was a much more frequent cause of pericarditis in Dr. Chambers' cases observed in St. George's Hospital (22 or 23 in 81, or 1 in 3·8 of the cases of pericarditis that had neither acute rheumatism nor Bright's disease) than in those recorded in St. Mary's Hospital (3 in 46, or 1 in 13·6 ; or including partial pericarditis 6 in 56, or 1 in 9·5).

Fever, in which the serous inflammations are rare, was only associated with pericarditis in six instances among those from every source. This does not include one of small-pox, properly pyæmic, nor one of scarlet fever.

Those constitutional diseases, tubercle, cancer, and syphilis, were very rarely complicated with pericarditis, or in only one each among the whole of the combined cases, not including however tubercular pericarditis or cancer of the heart, in which the action of the disease was strictly local.

One single instance of cholera, which is so closely connected with acute rheumatism, had pericarditis. This occurred among the collected cases.

The case of pericarditis associated with disease of the supra-renal capsules is figured at page 5. This man could not lie down, his chest was universally dull on percussion in front and at the left side, and the sounds and impulse of his heart were absent. Upon these grounds Sir James Alderson, under whose care he was, correctly inferred that he had pericarditis.

(B.) *Affections of the Heart and Aorta.*—In one case, a man, pericarditis was caused by a wound of the heart. The right ventricle was penetrated by a wound about half an inch long,

and the surface of the heart and that of the pericardial sac were covered with recent lymph, stained red in many places. He survived the injury nearly five days. The left ventricle was penetrated by a wound half an inch long. In another patient who survived nearly two days, fibrinous coagula were found on either side of the wound, but there was no definite note of pericarditis. Pericarditis was caused by an injury inflicted over the region of the heart in two of the collected cases.

Local affections of the pericardium itself, and of the immediately adjoining structures, whether bearing upon it from within, and occupying the walls of the heart or ascending aorta, or from without, and seated in the neighbouring tissues, all tend to produce pericarditis. Tubercular pericarditis occurred in two instances ; and as tubercular disease of the pericardium is rare, it is evident that this affection has a strong tendency to inflame the surface of the heart.

Among the affections of the structure of the heart that excited pericarditis by bearing outward upon the pericardial surface of the heart, there were four cases with cancer of the heart ; two with fibroid disease of the heart, in which the disease extended to the surface of the organ ; and two of abscess of the heart, in one at least of which, described by Dr. Graves, there was no pyæmia, and in which instance the abscess contained two ounces of pus, and did not therefore cause pericarditis by bursting into the sac. These cases are derived from all sources.

Aneurism of the heart was the cause of pericarditis in another patient, a well-formed woman, aged 53. The pericardium was distended with about eight ounces of fluid, and was adherent in front to the right ventricle, and behind to the left ventricle by quite recent attachments. The mitral valve was thickened and incompetent. An aneurism was

discovered, on examination, in front of the left ventricle about the size of a small orange. The walls of the left ventricle were thickened, but in the position of the sac there was not a trace left of muscular tissue, and the wall was only formed by the parietal layer.

In all these cases, whether of cancer, fibroid disease, abscess, or aneurism of the heart with pericarditis, the inflammation of the surface of the heart is excited in the same manner. The new mass projecting into the pericardium, and bearing upon it during the active contraction of the organ with a rude and unaccustomed force, excites inflammation in the opposite surfaces of the heart and the pericardial sac, and so establishes pericarditis.

Aneurism of the ascending aorta excited pericarditis in eight of the cases derived from all sources; and three of the twenty-six cases of that affection observed in St. Mary's Hospital presented evidence of previo us pericarditis in the form of pericardial adhesion. In these cases the pericarditis is excited by the constantly enlarging aneurism bearing upon the pericardium, in the same manner that it is excited by cancer, abscess, fibroid disease, and aneurism of the heart.

Cases with valvular disease of the heart, including all its varieties, without Bright's disease, were attacked with pericarditis in definite, but by no means frequent members, since that affection appeared in only six of the 117 fatal cases in which the valves of the heart were incompetent (1 in 20). These proportions are increased if we strike out the thirty cases of the class under examination in which there were complete adhesions of the heart, and in which pericarditis was therefore forbidden. Thus corrected, the attacks of pericarditis number 6 in 87 (or 1 in 14·5). It will be interesting to ascertain whether valvular incompetence with Bright's

disease was more frequently visited with pericarditis than when it existed free from that affection. In 78 cases of Bright's disease with imperfection of the valves, five had pericarditis (1 in 15·6), or, deducting nine in which the heart was completely adherent, the numbers stand 5 in 69 or 1 in 14. From these comparative results it would seem that Bright's disease scarcely increases the tendency to pericarditis in valvular disease of the heart, for the proportion is almost identical in the two sets of cases. Partial pericarditis was present in four of the 117 cases with valvular insufficiency that were free from Bright's disease; and in three of the 78 cases of that class in which the kidneys were affected.

The six cases of pericarditis have been just distributed over the whole series of cases with valvular disease, the varieties of the affection being merged under one common title. If, however, we distinguish the different affections of the valves from each other, we find a remarkable difference in the proportion in which they were respectively attacked with pericarditis. The cases of mitral incompetence included all but one of those attacks of pericarditis, or 5 in 32; or, deducting 12 with complete adhesions of the heart, 5 in 20 or 1 in 4 of those cases were thus affected. The remaining instance of pericarditis appeared in one of the thirty-one cases of mitral-aortic insufficiency, or, deducting fourteen with complete pericardial adhesions, 1 in 17 of those cases. Not one of thirty-two cases with aotic valve-disease, or of twenty cases with mitral obstruction, had pericarditis.

Pericarditis in case of valvular disease had a strong but not exclusive preference for mitral incompetence among the collected cases, including those in the *Pathological Transactions*, for among eleven cases in which the affection of the valve was specified, eight had mitral insufficiency, while two had mitral-aortic, and one had aortic valve-disease. May not the

comparative frequency of pericarditis in mitral valve-disease be due to the resistance to the flow of blood through the lungs, and the consequent distension of the right ventricle with blood; the powerful action of that ventricle, which presses so strongly upon the walls of the chest in front; and the fulness of the coronary veins—which occur in the final stage of that affection?

The cases of pericarditis in Bright's disease, with valvular insufficiency, were equally distributed over the whole series; two with mitral incompetence, one with mitral contraction, one with aortic and one with mitral-aortic valve-disease being thus affected.

Pericarditis attacked one case in which there was hypertrophy of the heart without valvular disease, or any other complication except pericardial adhesion. There were altogether eleven cases of hypertrophy of the heart thus circumstanced, and as in six of them the heart was adherent, rendering pericarditis impossible, that affection attacked one in five case of this class.

It will be well to inquire as to the proportion in which pericarditis attacked cases with and without hypertrophy of the heart. The heart was enlarged in 130 out of 655 cases of all the kinds enumerated in the supplementary table at page 175, that were free from Bright's disease, and among these 130 cases, 12, or 1 in 11, had pericarditis. The heart was diseased in 86 of those cases in which the organ was enlarged, excluding eleven without other complications except adhesion; and including those cases with adherent pericardium, the heart was not diseased in 45 instances. Of the cases just referred to, 26 of the 86, and 9 of the 45, had pericardial adhesions, and could not therefore have pericarditis. After deducting the cases with adhesions, 7 in 60 (or 3 in 8·6), with disease of the heart, and 5 of the 36 (or 1 in 7), without other

affection than hypertrophy of that organ, had pericarditis. Without going into detail it may be briefly stated that of the rest of the cases, after deducting those with adherent pericardium, 6 in 104 (or 1 in 18) of those in which the heart was rather large, 4 in 267 (or 1 in 66) of those in which that organ was natural in size, and 1 of the 26, in which it was small, had pericarditis.

These returns make it evident that enlargement, or hypertrophy of the heart exercises a powerful influence on the production of pericarditis. Besides the cases enumerated, there were 107 (or 1 in 6) in which the size of the heart was not described, and of these sixteen (or 1 in 6·7) had pericardial adhesions, and nineteen (or 1 in 4, excluding those with adhesions) had pericarditis. It thus appears that the size of the heart was not described in nearly one-half of the cases with pericarditis, owing evidently to the mind of the reporter being preoccupied by the morbid anatomy of the inflamed organ. One of the cases in which the size of the heart is not noted had mitral incompetence, and may therefore be ranked with those in which the organ was enlarged; and ten of them had pneumonia (in 6), pleurisy (in 3), or empyema (in 1). In these ten cases the labour of the right ventricle must have been increased and prolonged, with the effect of enlarging the right side of the heart. This would tell more on the cases with pleuro-pneumonia than in those with simple pleurisy or empyema, but in such cases, with much effusion into one side of the chest, the obstacle to the stream of blood through the lungs is often great. This was well evidenced in a case already alluded to at p. 227, vol. iii., of extensive effusion into the right side of the chest which I saw through the kindness of Dr. Wane. Mr. James Lane drew off a large quantity of fluid from the affected side. Before its removal there was a mitral murmur and doubling of the second sound. The

doubling disappeared when the fluid was being extracted, and after a time the murmur vanished. In these cases, therefore, the prime effect of the spreading of inflammation from the pleura to the pericardium was heightened by the added secondary influence of the increased size and labour of the right ventricle.

(C.) Eight patients with pneumonia (8 in 46), three with pleurisy (3 in 26), and two with empyema (2 in 17) had pericarditis. In all these cases (13 in 89), whether the primary affection was pneumonia or pleurisy, it was the pleurisy affecting the outer surface of the pericardium, and spreading thence to its inner surface, that immediately kindled the pericarditis.

Three of the eight cases with pneumonia and pericarditis were under my care, but in none of them did I detect a friction-sound.

Two of the three cases with pleurisy and pericarditis were my patients, and in both of them friction-sound was heard. One of these was a little girl, who had been attacked a fortnight before with pain in the left side and over the heart, and was brought to the hospital in the mother's arms, in distress, pale, and breathing hurriedly. There was extensive pleurisy of the left side, and next day there was dulness on percussion, and a double, rather smooth friction-sound over the whole pericardium. Chorea soon appeared, and on the seventh day, when there was a mitral murmur, the effusion had reached its acme. Two days later, when the friction-sound was limited to the lower sternum, she died. The other case was a man who had been ill six months with pleurisy of the left side. On the eleventh day after admission double pericardial friction-sound came into play, and continued to the nineteenth day. After two days it vanished from over the heart, and was only audible at the apex; it was thus ten days

later, and on the following day he died. The heart was almost universally adherent by yellow lymph.

Although in these thirteen cases the pleurisy excited inflammation of the exterior of the pericardial sac, which travelled through its fibrous structure to its interior, and then attacked the surface of the heart; yet in many of the seventy-six other cases with pleuro-pneumonia or pleurisy the exterior of the pericardium was inflamed, and yet the sac proved to be a barrier to the inflammation, which did not extend inwards so as to excite pericarditis. We have seen that in rheumatic pericarditis the inflammation habitually travels through the fibrous walls of the sac, and attacks its exterior, or pleural surface, exciting pleurisy; so that pericarditis tends to pass from within outwards much more than pleurisy of the pericardium does so from without inwards.

A case of pleurisy with pericarditis, under my care, that recovered presented a peculiar pericardial friction-sound on pressure, to the left of the lower sternum, that lasted about three weeks.

I have just alluded to the important secondary influence which the increased size and force of the right ventricle exercises in reinforcing the primary influence of the extension of the inflammation from the pleura to the pericardium in cases of pneumonia and pleurisy.

Pericarditis attacked two cases of phthisis out of a total number affected with that disease amounting to 12. This does not include the two cases of tubercular pericarditis with phthisis already spoken of. Dr. Stokes gives an important case communicated to him by Dr. McDowell in which pneumo-pericarditis was caused by a fistulous communication between the pericardium and a small cavity at the summit of the right lung; the apices of both lungs were healthy, but the

bases of both lungs were solidified from a deposit of miliary tubercle and from pneumonia.[1]

(D.) Two cases were attacked with pericarditis owing to disease of the œsophagus where it passes behind the pericardium. In one of these patients, who was under the care of Dr. Chambers, the œsophagus was ulcerated from the bifurcation of the trachea to half an inch above the diaphragm. The ulcer gave way into the pericardium, which was filled with fluid from the stomach, and the interior of the sac was lined, and the heart was covered with recent fibrin.

The other patient, with cancer of the œsophagus behind the pericardium, a woman, aged 47, a cook, under my care, complained of slight difficulty in swallowing, referred to the fauces. A to-and-fro friction-sound, louder with the diastole than the systole, was audible over the cardiac region, being most intense over the sixth cartilage, and heard from thence to the ninth cartilage. Pleural friction was also present. This patient died on the fifth day after admission.

(E.) There was a small and remarkable group of cases, in which pericarditis was caused by affections involving the diaphragm. One of them had diaphragmatic hernia; two others had abscess of the liver involving the diaphragm; and another had a tumour connected with the pericardium, and communicating with the stomach.

In the case of diaphragmatic hernia which was under the care of Sir James Alderson, the stomach, omentum, spleen, and transverse colon were forced through an opening into the left side of the chest, which contained six pints of liquid, partly digested blood, partly food. The heart was displaced to the right of the sternum, and there was pericarditis.

In one of two other cases an abscess, with thickened walls,

[1] Dr. Stokes, *On Diseases of the Heart and Aorta*, p. 25.

containing several ounces of greenish pus, was situated between the pericardium and the liver, involving the diaphragm, and communicating with a small abscess in the liver. The pericardium contained many ounces of puriform fluid, and its lining membrane and the surface of the heart were "hyperæmic," the latter being very red and velvety. In the other case, the diaphragm was pushed up by the liver in a conical projection, which was formed by an abscess occupying the interior portion of the left lobe of the liver, and the contiguous part of its right lobe. The pericardium contained two or three ounces of turbid fluid, and the surface of the heart was roughened by a recent deposit of lymph. Dr. Graves gives an important case in which pneumo-pericarditis was caused by a hepatic abscess which communicated with the pericardium and the stomach.

In the fourth case the pericardium was full of thick yellow fluid, and there were some nodules on the aorta; a dense white tumour which was interposed between the pericardium and the diaphragm was softened in the middle, and formed a cavity which communicated with the stomach and spleen, and resembled an ulcer.

One case of peritonitis out of a total of 64 had general, and another had partial pericarditis.

(F.) There remains one fatal case of pericarditis in which there was no evidence that the affection was secondary to, or associated with any other disease.

In this patient, a woman, aged 44, the pericardium was nearly the eighth of an inch thick, and its sac contained a large quantity of sero-purulent fluid. The surfaces of the heart and the sac were covered with recent layers of plastic deposit, which was arranged at the base in a honeycomb shape, and was lengthened out at the apex into bands. The heart was small, hard, and contracted; the lungs were congested behind;

and there was a quarter of a pint of brown fluid in each lateral cavity of the chest.

Two cases of pericarditis, under my care in St. Mary's Hospital, presented no other definite affection. One of these, a schoolboy, aged 12, was attacked, eighteen days before his admission, with pain in both sides of the chest, worse in the left. On admission the impulse of the heart was in the fifth space, there was fulness over the pericardium, dulness from the second cartilage to the sixth, and a loud to-and-fro sound, which was intensified by pressure, over the same region and up to the top of the sternum. Next day the dulness had lessened, but the friction-sound was strong and grating, and extended beyond the region of dulness. For several days it was more feeble and limited ; on the fourteenth, and two days later, it was again louder, but on the nineteenth day it had vanished. The other patient, a pregnant woman, took cold six weeks before admission. The heart's action was tumultuous, and on the third day the impulse extended from the sternum to two inches and a half beyond the left nipple, a to-and-fro sound appeared over and below the region of the heart, and a mitral murmur at the apex. Next day an impulse of a grating character, almost a thrill, extended over the region of the friction-sound. These signs continued with variations, but lessening, and on the fourteenth day the impulse had shrunk inwards for two inches and a half, being bounded by the nipple line. Three days later a systolic murmur was converted by pressure into a friction-sound, which disappeared on the eighteenth day.

The Treatment of Pericarditis.

Pericarditis, as we have just seen, is so rarely met with except as a combination of, or associated with, some other disease, that in the treatment of such cases we have to consider mainly the primary affection, and along with this the local management of the secondary inflammatiom of the pericardium. I shall of course here practically limit myself to this latter and local point. It will be important, however, to touch upon the measures, in the treatment of the main disease, that may tend to prevent the occurrence of pericarditis. I shall briefly consider (1) the preventive treatment of acute rheumatism, in relation to the possible occurrence of pericarditis, and (2) the local treatment that the presence of pericarditis may render desirable in those diseases which are more or less frequently complicated with that affection.

1. The chief objects to be kept in view in the treatment of *acute rheumatism* are, (1) the mitigation of the endocarditis that is the usual and natural effect of that disease, and (2) the prevention of pericarditis, which, though the frequent, is not the customary complication of that disease. Fortunately the measures that tend to palliate the inflammation of the interior of the heart tend also to prevent the inflammation of the exterior of that organ. The Address in Medicine, given at the meeting of the British Medicine Association in Newcastle-on-Tyne, was devoted to the treatment of acute rheumatism by rest and the relief of local pain, with a view to prevent pericarditis and lessen the severity and permanent ill effects of endocarditis. The absolute rest of every limb and joint; and the soothing application of the belladonna

and chloroform liniment, sprinkled on cotton-wool, to the affected joints, supported by flannel, applied over the seat of pain with uniform and comfortable pressure, are the most important measures in the treatment of acute rheumatism for the prevention of pericarditis. The rest and support of the affected joints should be strictly maintained for several days after the disappearance of the local inflammation ; for the too early use of an affected joint or limb, after the relief of pain and swelling, often leads to a relapse, first attacking the joints of the over-used limb, extending to other joints, and often producing endocarditis and pericarditis. I have given at pp. 275, 276, vol. iii., brief notes of six cases in which a relapse of the joint affection, usually thus occasioned, induced endocarditis and pericarditis.

2. The employment of a few leeches, and the application of cotton-wool or a poultice, sprinkled with the belladonna and chloroform liniment, over the region of the heart during the early and painful period of an attack of pericarditis, are the means that I have for a long time employed in the treatment of that affection.

I have before me the collected notes of 36 cases of pericarditis, in which several leeches were applied over the region of the heart. In 29 of these cases there was pain over the region of the inflamed pericardium, and in seven of them there was no note of the presence of pain. In 24 of the cases suffering from pain, marked relief, sometimes complete, followed upon the application of the leeches ; and this relief in a fair proportion of the cases so speedily followed the local bleeding that the relief must be attributed to the leeching. Brief notes of cases in which the application of leeches relieved the pain over the region of the inflamed pericardium will be found in the preceding (pp. 276, 287, 303, 314, vol. iii.). The local bleeding, besides assuaging the local pain, lessened

the oppression in the chest and the difficulty of respiration in many cases.

In one instance leeches were applied over the seat of pain five times; although on each occasion relief seemed to follow, yet the pain soon again increased.

In five cases leeches gave little or no relief. Although in these cases pain was not materially lessened by the local bleeding, yet in every instance but one, its action on the patients' state seemed to be favourable. In that patient (17), whose case has been already referred to at pp. 287, 304, 309, 312, vol. iii., there was pain over the heart, the action of which was very tumultuous at the time of admission. Leeches were applied with great relief, but unfortunately the bleeding from one of them could not be stopped, and she lost much blood. After this the action of the heart was irregular and intermittent, and she was evidently weakened by the hæmorrhage. She finally died after a long and severe illness, which was closed by an attack of small-pox.

The employment of leeches produced a definite but very variable effect on the friction-sound, and tended to lessen the force and extent of the impulse. Sometimes the friction-sound was lessened in intensity (in 8), but as often it became more intense (in 8) after the local bleeding. In one patient (35, pp. 35, 81) its effect was to suspend the rubbing sound, which had been previously extensive and rough, for one day; but in the evening pain returned, and with it the *frottement* over the region of the heart. Another patient (16) on admission had excessive pain across the heart, where there was a double thrill, and a double harsh scraping friction-sound; four leeches were applied; and next morning there was scarcely any pain, no friction-sound, and no note of thrill. The friction-sound returned on pressure that afternoon, and was again present on the following day. In one instance—

I speak from memory—I examined a patient with pericarditis immediately after the withdrawal of leeches, and found that the friction-sound that had been previously audible was entirely abolished. This disappearance of the friction-sound in such a case is evidently not due to any change in the character of the lymph on the surfaces of the heart and sac, although their vascularity may be lessened, but to the diminished force of the action of the organ. In direct confirmation of this, we have already seen that in several cases friction-sound was abolished, suspended, or softened, by the weakening of the action of the heart (see p. 309, vol. iii., p. 81, vol. iv.).

The effect of leeching the region of the heart on the amount of effusion in the pericardium in cases of pericarditis was not very marked. The leeches were applied at the time of the acme of the effusion in ten cases, and in all of them but two the amount of effusion had lessened on the following day, and in the remaining two on the third day after the local bleeding, which lessened local pain in eight of these cases. To balance these instances, in eight others the effusion increased after the application of the leeches, and attained its acme in a day or two; at the same time, however, the pain over the region of the heart was relieved in six of those cases, but was not so in two of them.

Blisters applied over the heart are frequently employed in the treatment of pericarditis. I resorted to them occasionally up to the year 1856. I cannot, however, find any instance in which they appeared to be of service, and they were certainly, in some cases, a source of discomfort. It is evident that a blister over the region of the heart adds a second and outward inflammation to the primary and inward inflammation, and it therefore, unless there is a counterbalancing gain, increases the evil. Blisters were the definite cause of mischief in a case that I shall have occasion to quote when I speak of the

removal of the fluid from the distended pericardium. In that instance they were applied seven times in succession over the præcordial region. A blister cannot alter the lymph covering the heart and lining the sac; and cannot directly lessen the amount of fluid in the pericardium, which, as we have again and again seen, tends of itself to diminish rapidly when it has reached its acme. It appears to me that a blister over the distended pericardium would rather increase than lessen the morbid supply of blood to those inflamed parts to which it is so contiguous. Blisters, besides inflicting local injury, taint the blood by increasing its fibrin, and are apt to lead to a secondary and low kind of inflammation in distant parts, and perhaps even to degrade the character of the pericardial inflammation itself, and to prolong its existence.

It may be said that exciting pain at the surface of the chest in these cases lessens the severity of the internal pain. This is true, but this effect may be induced innocuously, by the application of chloroform over the seat of suffering, combined with belladonna liniment, sprinkled on cotton-wool, and covered with oiled silk.

Paracentesis of the Pericardium.—We have seen again and again that when the fluid in the pericardium has reached its acme, it very soon begins to diminish. It is therefore evident that puncture of the pericardium is very seldom called for. In some rare instances, however, the quantity of serum in the sac is so great as to interfere seriously with the action of the heart, breathing, swallowing, and speech; owing to the compression of the auricles and venæ cavæ, the trachea and left bronchus, the œsophagus and the descending aorta; and the inflammation of the recurrent nerve. Generally the fluid of itself lessens so quickly that these threatening symptoms pass by without real danger to life. In some rare instances, however, life is in danger owing to the distension of the

pericardium, and then paracentesis of the pericardium may become urgently called for.

Riolan,[1] in 1649, proposed that in dropsy of the pericardium, the sac might be opened by trephining the sternum an inch from the ensiform cartilage. Senac,[2] and Laennec,[3] at long intervals, both gave the same advice, the point selected by Laennec being immediately above the ensiform cartilage. Desault[4] attempted to open the pericardium between the sixth and seventh ribs, and Larrey,[5] between the fifth and sixth ribs; but they both evidently failed to enter the pericardium. Romero[5] opened the pericardial sac in three cases of "hydro-pericardium," twice with success, through an incision made in the fifth space, near the junction of the cartilages to the ribs, this wound being made, partly to explore, partly to open the pericardium or the pleura. The first circumstantial account of tapping the pericardium was in a patient of Skoda's, with pericarditis from cancer of the heart, operated upon by Schuh in 1840,[4] who first inserted a trochar by a perpendicular puncture through the third space close to the sternum over the great arteries, and failing to get fluid, penetrated the sac through the fourth space and obtained a certain amount of reddish serum. This patient lived for nearly six months, and died with extensive cancer of the chest.[6] In

[1] *Encheiridium Anatomicum et pathologicum*, p. 213.

[2] Senac, *De la Structure du Cœur*, ii. 369.

[3] Laennec, *Traité de l'Auscultation Médiate.*

[4] Trousseau et Laségue, *Arch. Gén. de Méd.*, Nov. 1854.

[5] *Dict. des Sc. Médicales*, v. xl. p. 370. These cases are given imperfectly.

[6] Trousseau and Laségue publish this case at length in the *Archives*, but in his *Clinique Médicale* Trousseau states that Schuh penetrated in his first puncture a mass of cancer, altogether of a thickness of six inches, which had invaded the sternum. It was not, however, until more than a month after the operation that this tumour showed itself. *Arch. G. de Méd.*, 1854, p. 520.

1841 Heger performed paracentesis of the pericardium in another patient of Skoda's, with pericarditis. He entered the pericardium through the fifth space, two inches from the left border of the sternum. Altogether 1,500 grammes (about 48 ounces) of a brownish serum, finely flocculent, escaped, and nineteen days later, the fluid having re-accumulated, he again punctured the pericardium at the same place, and 500 grammes (about 16 ounces) of a reddish troubled fluid escaped in the course of four hours. This patient died fifty-one days after the second operation. The pericardium was in great part adherent, and there were nine and five pints respectively in the two sides of the chest, and a tubercular cavity of the left lung. These two patients died from the primary diseases, cancer and tubercle; but both operations were successful.

Behier thought that he punctured the pericardium through the sixth left space in a case related by him in 1854; the patient died twenty-six days afterwards, but there was no pericarditis, and no mark of puncture in the walls of the sac. Jobert,[1] in 1854, after cutting the skin, punctured the pericardium with a trochar, in a case of pericarditis, a patient of M. Trousseau's, through the fifth left space, 1·2 inch from the edge of the sternum. The cannula was agitated by the beating of the heart—the fluid came at first in drops and then very slowly, and altogether 400 grammes (about 13 ounces) of liquid flowed in the course of an hour and a half. The patient left the hospital eleven weeks after the operation, suffering from phthisis. Trousseau,[2] in 1856, operated on another case, and opened the chest with a bistoury below the nipple through the nearest intercostal space, and penetrated into the pericardium, from which flowed nearly 100 grammes

[1] Trousseau et Lasègue, *Arch. G. de Méd.*, 1854.

[2] Trousseau, *Clinical Medicine. New Syd. Soc.*, iii. 365.

(about three ounces) of a red serosity; and twice as much yellow serum came from the pleura. The patient died five days after the operation. The last of the French operators that I shall name was M. Aran,[1] who in 1855, after cutting through the skin, penetrated the pericardium with a trochar through the fifth space, about an inch from the extreme limit of pericardial dulness, and withdrew about 350 grammes (fully 11 ounces) of reddish transparent fluid, and then injected a solution of iodine. Twelve days later he tapped a second time and withdrew 1,350 grammes (about 40 ounces) of albuminous liquid. This patient recovered from the operation, but three months later presented signs of phthisis.

I have now to speak of two important cases of pericarditis with symptoms threatening life, in which Dr. Clifford Allbutt resolved with his colleagues on the performance of paracentesis of the pericardium. One of these cases was operated upon by Mr. Wheelhouse, who vividly describes the condition of the patient and the steps of the operation. He found the patient sitting up in bed, his head resting on his hands, his elbows on his knees, struggling for breath. I quote the following from his description, and refer to his paper for the full details of the operation; and the precautions adopted during its performance:—"I chose for my purpose a small trochar. This I placed on the upper margin of the fifth rib, half an inch to the left of the sternum; and inclining it upwards and inwards, thrust it steadily forwards through the intercostal space towards what I believed to be the centre of the ventricle. I pushed it onwards until I could distinctly feel the movements of the heart with the instrument; and then, sheathing the point, I advanced the cannula well up to the heart, until I could feel and see, and demonstrate to those

[1] *Bulletin de l'Académie Royale de Médecine*, xxi. 142.

around, the impulse of the heart as communicated to the instrument. The trochar was then withdrawn, and the fluid allowed to escape. This it did at first in a steady stream, which soon subsided into a saltatory flow coincident with the heart's contractions. The fluid consisted of a pale pink coagulable serum, and, upon the whole, about three ounces escaped. During the operation the patient gradually obtained relief; and after the cannula was withdrawn, the bed-rest was removed, and he was able to lie down."[1] This patient completely recovered, and was in perfect health the other day when Mr. Wheelhouse, in reply to my inquiries, kindly informed me as to the state of the patient. In the second of Dr. Clifford Allbutt's patients Mr. Teale drew off, as Mr. Wheelhouse had done, through a fine cannula five ounces of fluid which gave the patient great relief. The re-accumulation of the fluid called for a second operation, which was performed with considerable relief. Finally, however, this patient, a girl, died of bronchitis.[2]

The operation has been performed within the last three years on three occasions, and I owe the references to these cases to the kindness of Mr. Holmes. M. Villeneuve, in 1873, operated by means of the aspirator, on a child with arching and fluctuation over the præcordial region. He punctured the tumour at its most prominent part, and removed two syringefuls of serum. On withdrawing the cannula, a jet of liquid spirted out of the wound, which remained open owing to the internal wall of the cavity having been very much thinned by the repeated application of blisters, seven of them having been placed one after another, without any improvement, on the same place. A pericardial fistula, yielding pus, was established and did not

[1] See *British Medical Journal*, Oct. 10, 1868, p. 385.

[2] See Dr. Clifford Allbutt's important paper, *Lancet*, 1869, i. 807.

heal up until the sixth month after the operation.[1] In the other case, a man in whom paracentesis of the chest and abdomen had already been performed, Dr. Valtosta, in 1874, opened the pericardium by making an incision over the fifth space, commencing about half an inch from the sternum. The layers of muscles were then carefully divided and an elastic dilatation was felt. A puncture was made in this, the point of a small trochar was introduced, and about ten ounces of fluid was removed with immediate relief. This patient died four weeks after the performance of the operation.[2] M. Chairon contributed a third case in 1875, in which more than 1,000 grammes (about 33 ounces) of liquid were removed from the pericardium. The result is not given. With reference to the method of operation, he says the spot to be preferred is the fifth intercostal space, at an intermediate point between the nipple and the sternum, rather nearer to the former, always being guided by the apex of the heart. The aspiratory method should, he considers, be preferred.[3]

Proposed Operation for Paracentesis of the Pericardium.— This operation cannot well be called for unless the amount of effusion into the pericardium be so great as to compress the venæ cavæ and the auricles, the œsophagus, trachea, and left bronchus, and the descending aorta, so as to interfere with the action of the heart, swallowing, breathing, and the supply of blood to the abdomen and lower limbs. Under these circumstances the pericardial sac is greatly distended downwards towards the abdomen, and the heart itself is elevated. The result is that the mass of the fluid occupies a large space below the heart, measuring between one and two inches from above downwards, between the lower surface

[1] *London Medical Record*, iii. p. 532.

[2] *Ibid.* pp. 275, 532.

[3] *Ibid.* p. 694.

of the ventricles and the floor of the pericardium, where it is formed by the central tendon of the diaphragm; which is depressed downwards almost or quite to the level of the upper border of the sixth space, in the manner represented in the figures at pages 11, 44, 48, and 110, and also, in principle, in Pirogoff's important work.

When it is considered that in these serious cases the lower border of the heart is above, while the mass of the fluid is below the level of the lower edge of the fifth cartilage, I advise that the fine trochar, such as that used by M. Aran, Mr. Wheelhouse, Mr. Teale, and M. Chairon, should be inserted into the distended pericardium at a point just above the upper edge of the sixth cartilage at the lowest part of its curve, more than an inch within the mammary line; and that the instrument should penetrate gently inwards with a direction slightly downwards, so that it may advance into the collection of fluid below the level of the heart; and that the liquid should be slowly and gently extracted by the use of a syringe or the aspirator. By this proceeding the collected fluid will be alone penetrated and the heart will be quite untouched. Extensive incisions and the injection of irritating fluids should of course be avoided.

In every case in which the heart has been previously healthy and is of the natural size, its lower border is elevated above the level of the fifth space when the effusion into the pericardium is at its height, so that in such cases the procedure I have advised, which has the sanction of Aran's and Chairon's operations, can be performed with ease and safety.

When, however, the heart is enlarged owing to the existence of valvular disease of some standing, the heart is sometimes, as in the cases spoken of at page 42, to be felt beating in the fifth or even the sixth space at the time of the acme of the effusion, when the urgent distress and danger of the patient

may demand paracentesis of the pericardium. Under such circumstances, which can be readily discovered by ascertaining the position of the impulse—which should always be some distance above the point of penetration, for a thin layer of fluid interposes itself between the surface of the heart above its lower border and the front of the chest—another point than that just indicated in the fifth space must be chosen for the operation. This point should then be selected at the space between the left edge of the ensiform cartilage and the right border of the seventh cartilage in the epigastric region; or, if needful, owing to its margin being covered by the seventh costal cartilage, the ensiform cartilage at its left border may itself be perforated, first with the point of a bistoury, and then with the fine trochar. Trousseau states that Larrey advised that the puncture of the pericardium should be made through this space; but in the operation which he performed with a view—erroneous in this instance—to enter the pericardial sac, that great surgeon, as we have seen, entered the cavity of the chest between the fifth and sixth ribs. The lower border of the fully distended pericardium is usually a little above, and sometimes even below, the lower end of the ensiform cartilage, as in figure 10, page 48; which is from a case, exactly in point, with mitral regurgitation and enlargement of the heart; and the pericardium may therefore be safely punctured through a point corresponding to the middle or the lower portion of that cartilage. The presence or absence of the impulse of the right ventricle in the epigastric space, and the position of the lower border of the pericardial dulness in that space, must be previously ascertained. Those two important points of diagnosis, which can be readily made, will prove a safe guide to the surgeon as to the place which he should select for the operation, which he will rightly fix sufficiently below

the seat of the impulse, so as to avoid the heart ; and sufficiently above the lower border of pericardial dulness, so as to prevent the cannula being tilted upwards when the floor of the pericardium elevates itself as the sac is being emptied. When he pushes the trochar onwards, he must use all the precautions so clearly described by Mr. Wheelhouse, so that if the point of the instrument comes upon the front of the heart, he may withdraw the trochar at the same time that he gently presses the cannula forwards and downwards.

In the great majority of cases the fluid, after it has reached its acme, soon begins to lessen, and continues to do so steadily from day to day. Under these circumstances I do not advise the use either of aperients, which tend to disturb and lower the patient, or of diuretics. If, however, the quantity of the fluid is stationary, or lessens very slowly, then diuretics may sometimes be of use.

XVII.

ADHERENT PERICARDIUM.

THE discovery of adherent pericardium during life is in some cases impossible, and in some, doubtful or difficult; but in others, and these are amongst the most important cases, its existence may be ascertained during life on reasonable and well-ascertained grounds.

When the adhesions are partial, or when the heart, though completely adherent, is small, is not bound by external adhesions to the anterior walls of the chest, and is covered to the natural extent by the lungs, their expansion being free and unconstrained, then the varying relation of the heart and lungs to the chest is quite natural, and the diagnosis of the adhesions is impossible. If the adherent heart be enlarged, and is not attached to the lower half of the sternum and the cardiac cartilages by combined pericardial and pleural adhesions, so that the active or automatic and the passive or respiratory movements of the heart are scarcely or but little interfered with, the inspiratory expansion of the lungs is freely permitted, and the diagnosis of the adherent pericardium may be difficult, obscure, or even impossible.

When, however, the heart is, as usual, enlarged, being often affected with valvular disease, the adhesions may be short, fibrous, and binding; and the front of the organ may be fixed

to the two lower thirds of the sternum and the adjoining cartilages by pleuro-pericardial adhesions, so that the automatic and respiratory movements of the heart, and the inspiratory expansion of the lungs, are restrained; thus the discovery of the adhesions during life may generally in such cases be made by a careful study of the physical signs; its diagnosis being the more certain and easy in proportion as the heart is more enlarged, and more firmly fixed to the anterior walls of the chest.

Anatomical Description of Adherent Pericardium.

Partial Adhesions.—Pericardial adhesions vary greatly in firmness of tissue and length of fibre, and when they are partial, they are usually longer than when they are general.

Four conditions seem to regulate the position, extent, and firmness of partial adhesions of the heart. (1) The amount of movement of the various parts of the heart and arteries; for it is evident that the more limited the movement of any part, the greater must be its tendency to adhesion: the relation of the surrounding sac (2) to the heart; and (3) to the outer borders of the pericardium, which are close to the heart, and are therefore more often adherent: (4) the gravitation of the heart in the fluid, since the posterior or depending parts of the heart, when the patient lies on the back, attach themselves readily to the parts on which they rest.

Partial adhesions take place most frequently near the apex and along the line of the ventricular septum; at the outer border of the left ventricle and the outer side of the right auricle, where the movements of those cavities are most limited, and to which parts the outer borders of the sac cling;

the posterior surfaces of the left auricle and of the ventricles which rest upon the sac; and the great arteries at their higher parts, where the extent of their movement is least, and where they are most contiguous to the pericardium. The visible commencement of the ascending aorta is often free from adhesions, owing to the hollow, containing liquid, formed in front of that part of the vessel, between the appendix of the right auricle and the origin of the pulmonary artery. In several instances a patch of the right ventricle, to the right of the septum, and midway between the pulmonary artery and the lower border of the ventricle, was adherent when the rest of the ventricle was free; and it is to be remarked that this patch is the part of least movement, or stable equilibrium, of the walls of the right ventricle (see fig. 16, p. 112, vol. iii.). A frequent seat of partial adhesions is a point a little above and to the left of the apex of the heart. These adhesions near the apex frequently become stretched and attenuated, and at length give way. Several pendulous, filamentous fibrous bands often hang from this point, near the apex, on the surface of hearts that are free from internal disease; but which display white fibrous patches on their surface; the filaments and the patches being evidently alike the result of a previous attack of pericarditis.

The parts of the surface of the heart and arteries that are usually not adherent when other parts are so are, the front of the right ventricle, especially in the neighbourhood of the right auricle and pulmonary artery, and above its own lower border; the appendix and ventricular border of the right auricle; and the parts of the aorta and pulmonary artery nearest to the heart, those being the parts that have respectively the greatest extent of movement during the action of the heart, as may be seen in the figures at p. 112, vol. iii.

General Adhesions.—The adhesions are formed of fibrous

threads of variable and often of considerable length, and they usually allow of a fair amount of movement of the heart. Long and loose adhesions interfere but little with the free play of the heart; but short, close, and firm attachments embarrass the action of the organ. The length of the fibres of adhesion varies over the different parts of the heart; their length usually corresponding to the amount of movement and the power exercised by the respective parts during the action of the organ. The adhesions are generally longer at the apex than elsewhere: those over the left ventricle are longer than those over the right ventricle; those over the auricular portion of the right ventricle are longer than those over its body and near the septum, and I believe that the same applies to the left ventricle also. The adhesions over the right auricle are much shorter than those over the right ventricle; and the auricular appendix is contracted in size by the fibrous covering. The attachments of the left auricle, the aorta, and the pulmonary artery are generally closer than those of the right auricle.

When the adhesions are long and loose, and the heart is free from valvular disease, and from any other influence tending to cause enlargement of the organ, the size of the heart is usually natural. It was thus in two of the cases examined after death at St. Mary's Hospital, in four cases that I observed at Nottingham, in many of those referred to by Dr. Stokes, in ten briefly described by Dr. Gairdner, and in 34 out of 90 cases collected by Dr. Kennedy.

When pericardial adhesions are associated with valvular disease, the heart is always enlarged. It was so in 25 out of 26 cases, and in the remaining instance, a case with mitral contraction, the heart was rather large. I have compared a double series of cases of valvular disease side by side, in one series with and the other without adherent pericardium, and,

not going here into details, I may say that the cases with adhesions were on an average five and a half ounces heavier than those in which there were no adhesions, an increase that was to a considerable extent accounted for in many instances by the augmented thickness and weight of the pericardial sac. The increased size of the heart would seem, therefore, in such cases, judging by this analysis, to be traceable more to the affection of the valves than to the adherent pericardium. We find, however, that in two-thirds of the cases without valvular disease in which the pericardium was adherent, the heart was enlarged (12 in 19); and in one-fifth of them it was rather large (5 in 19); while in only one-tenth of them the organ was of natural size (2 in 19). These proportions are borne out by Dr. Kennedy's important analysis of collected cases of adherent pericardium, who found that in fifty instances the heart was enlarged, in thirty-four it was of natural size, while in five it was atrophied. We may therefore conclude that in cases with the double affection of valvular disease and adherent pericardium, the valvular disease is the essential cause of the enlargement of the heart; yet that the adhesions, by giving an additional spur to the action of the organ, add to the more important enlarging effect of the valvular disease of the organ.

It is the natural effect of pericarditis for the inflammation to spread from the pericardial to the pleural surface of the fibrous sac. When, therefore, the pericardium becomes adherent to the heart in those cases, it becomes adherent also to the walls of the chest in front of the pericardium. These pleural adhesions often occupy an extensive space in front of the chest, and may extend from the second left cartilage to the sixth; from the manubrium to the upper half of the ensiform cartilage; and from the right border of the sternum to the apex of the heart, to the left of the nipple line, as in

the cases referred to in former pages, and there described. Though these are extreme instances, yet they are typical of many cases with pleuro-pericardial adhesions.

When the adhesions are short and powerful, and when, being pleuro-pericardial, they bind the walls of the heart extensively to the walls of the chest in front of them, a great and constant strain is put upon the ventricles ; for they cannot contract upon themselves to expel their contents until they have dragged the sternum and cartilages powerfully inwards. The ventricles thus expend their force in two directions, one towards the interior to expel their contents, resisted in doing so by valvular incompetence ; the other from the exterior, to compel the front of the chest, which is united to them like a solid buckler, to share in their contraction. Under these influences the ventricles tend to undergo a change in form, and to become flattened out, the one in front of the other. Two cases observed by me in Nottingham were thus influenced. The enlarged and thickened right ventricle, instead of sweeping half round the left ventricle, usually cone-shaped, lay directly in front of it ; and the septum between the ventricles, instead of bulging forwards into the right cavity, became flattened.

When the adhesions, being extensive and pleuro-pericardial, are not short and close, but of moderate length, and do not, therefore, bind the sternum and cartilages to the heart like a buckler, they do not seriously embarrass the commencing action of the ventricles ; but during their contraction the ventricles at length begin to draw upon the walls of the chest ; and in the course of the systole they drag those walls inwards.

When the adhesions are, as usual, longer and less solid, the ventricles contract more after their wont, and retain more or less perfectly their power. The right ventricle is usually

enlarged as well as the left, but not always, for the size of the ventricles is necessarily influenced by the valvular affection. When that affection is mitral or mitral-aortic, the right ventricle shares the labour and the enlargement with the left ventricle; when the aortic valve is alone affected, the left ventricle is often alone enlarged; and when there is mitral obstruction, the enlargement may mainly affect the two auricles, that of the ventricles being somewhat moderate.

The ventricles, when the pericardium is adherent, tend to enlarge outwards in every direction, and especially upwards to the manubrium, as well as downwards, into the epigastric space, to the right, and to the left. The great arteries are lifted up on the top of the ventricles into an unusually high position, and are crowded into the narrowed space at the top of the chest, almost as high as the root of the neck.

When the adhesions are dense, strong, and contracted, they sheathe the whole heart in a tight, tough envelope, which grasps the auricles and ventricles, prevents their free expansion, and forcibly lessens the organ.

Physical Signs of Adherent Pericardium.

Clinical History.—(A) *From a Successsion of Observers.*—Dr. Burns, in 1809, gave cases to show that when the pericardium is adherent, pulsation is felt in the epigastrium—a sign that had been previously observed by Korner—caused, he says, by the repercussions of the heart affecting the liver, which is the immediate seat of the pulsation.[1] He gives a case of adherent pericardium in which Dr. Rutherford found a strong pulsation of the heart, accompanied by a jarring motion, most remarkable at the contraction of the ventricles. Heim,

[1] Burns, *On the Diseases of the Heart*, p. 62.

according to Kreysig,[1] observed that a hollow appeared under the ribs during each systole when the pericardium was adherent. Sander[2] found, in a case of adherent pericardium with great enlargement of the heart, deepening of the space on the left side of the ensiform cartilage, followed quickly by

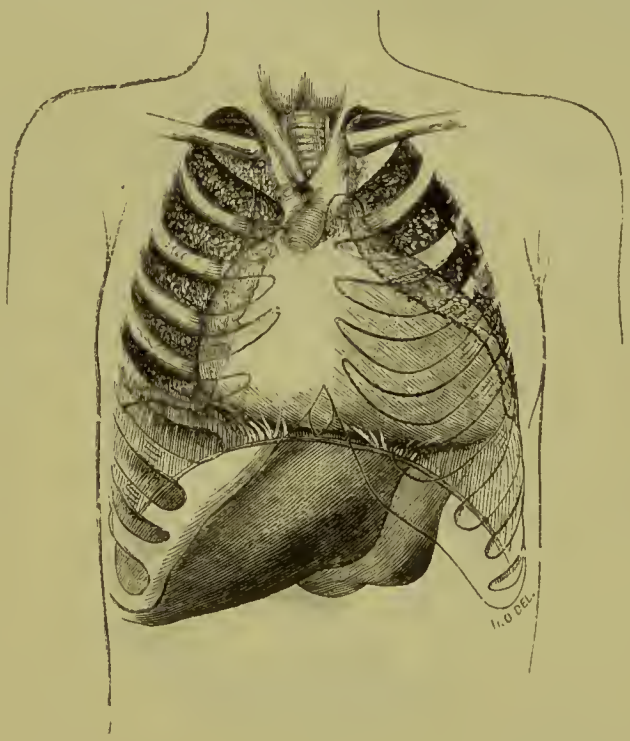


FIG. 1.

a shock, perceptible to the hand ; fulness over the cardiac cartilages ; and extensive impulse over the front of the chest.

Corvisart[3] noticed that in these cases respiration is high, and this he connects with the trouble of the whole heart

[1] Kreysig, *Die Krankheiten des Herzens*, ii. 625.
[2] *Hufeluuaud Bibliothek d. p. Heilkunde*, Bd. 51, 120.
[3] Corvisart, *Sur les Maladies du Cœur*, p. 35.

caused by the laborious action of the diaphragm, to which it is attached by the adhesions.

Dr. Hope,[1] in 1839, observed that pericardial adhesions sometimes caused a prominence of the cardiac cartilages, sometimes an abrupt jogging motion of the heart, corresponding with the systole and the diastole, that with the

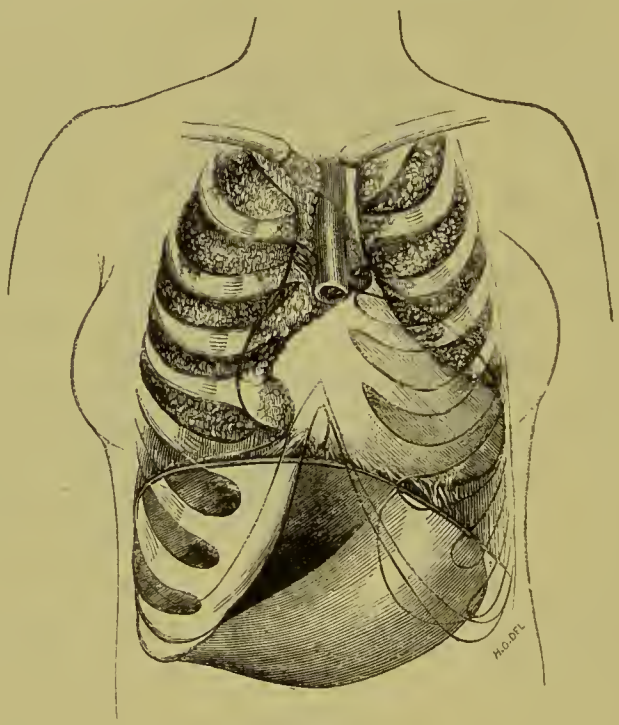


FIG. 2.

diastole having the character of a receding motion suddenly arrested. In the recital of four of his cases, to which his general account does little justice, he states that they presented a second or diastolic shock or back-stroke.

Dr. Williams,[2] in 1840, remarked that when the pericardium adheres both to the heart when enlarged, and to the walls of

[1] Dr. Hope, on the *Diseases of the Heart*, p. 194

[2] Dr. Williams, on the *Diseases of the Chest*, p. 24.

the chest, the heart pulsates in close contact with those walls; so that the pulsations are felt very widely, extending upwards as well as downwards, drawing in the intercostal spaces at each systole; and that respiration does not lessen the region of cardiac dulness on percussion, and of impulse. Dr. Law, in a communication that I have not been able to find, states that change of posture does not alter the position of the impulse.

In my paper on the situation of the internal organs, I, in 1844,[1] described four cases of adherent pericardium, and gave figures showing the position of the internal organs after death, two of which figures I reproduce here (see Figs. 1, 2). In one of these cases, a young woman, the heart was small in size, and presented during life no physical sign of disease of the heart, but the pulse was very feeble; she had palpitation, dyspnœa, and anasarca; and her lips were blue.

The heart was very large in the three remaining cases, two of which had mitral regurgitation, and the third had narrowing of the mitral, aortic, and tricuspid orifices. One of the two cases with mitral disease has been already described, and is figured at page 64. In the other case of the same class, the impulse was very strong and jogging; shaking and heaving the whole chest. The apex protruded strongly; the lower half of the sternum advanced firmly at the beginning of the systole, and fell back gradually and firmly during its continuance. The lower end of the ensiform cartilage receded during the systole; the impulse was irregular, 140 to 160 (*see* figure 1, page 209).

The remaining case with adherent pericardium presented physical signs that differed materially from those observed in the two other cases. The obstructed, mitral, and aortic apertures tested by the cone, each measured half an inch, and

[1] *Prov. Med. Trans.*

the tricuspid orifice three-quarters of an inch. The heart was very large, weighing thirty-two ounces; and all its cavities, and especially the ventricles, shared in the enlargement. The following were the physical signs:—"Strong protruding impulse at the apex between the sixth and seventh ribs. During the systole, the sternum and the left and right costal cartilages over the right ventricle became steadily depressed; immediately after the systole they advanced with a shock."[1] (See Fig. 2, p. 210.)

In the general description, I thus defined the character of the impulse in the two classes of cases just given: "The sternum, costal cartilages, and xiphoid cartilage are heaved forward firmly and steadily at the beginning of the systole; and during its continuance those parts fall back steadily and quickly, coinciding with the mode of systolic contraction of the right ventricle. In some cases the sternum and costal cartilages spring forward with a jerk during the diastole."

M. Bouillaud,[2] in 1846, described a sign by which he had been able to announce the existence of adherent pericardium in six or seven cases. It consisted in evident retraction of the pericardial region; the movements of the heart not being free, but embarrassed or curbed. He does not state during what period in the revolution of the heart's action the depression of the pericardial region took place.

Skoda,[3] in 1852, published an important paper on the diagnosis of adherent pericardium, in which he gives a critical account of most of the communications just analysed, and reports of three cases observed by himself. In the first case, a youth, there was dulness on percussion, equal in extent during inspiration and expiration, from the second left space to the ensiform cartilage, and from the middle of the sternum

[1] *Loc. cit.* p. 562. [2] *Traité de Nosographie Médicale*, i.
[3] *Zeitschrift der Gesellschaft der Aerzte zu Wien*, 152, i. 306.

to the left nipple ; and fulness over the second space, which advanced during the systole and sank in during the diastole ; the third, fourth, and fifth spaces deepened with the systole and filled out with the diastole; the heart's impulse was feeble, and the apex-beat was imperceptible. The heart sounds were natural, but the second sound was split over the pulmonary artery. The pericardium was tied to the walls of the chest by filamentous bands, and was universally adherent to the heart, which was natural in position ; the right ventricle was enlarged, the right auricle was changed into a stiff-crumbling tuberculous mass, and the conus arteriosus was widened, its walls being only a line in thickness.

The second case, which passed through all its stages under Skoda's eye, a youth, was admitted with pericarditis. The friction sound, then loud and extensive, became feeble and limited to the apex on the 15th, and was lost on the 19th day. On the 37th day there was a systolic deepening of the third, fourth, and fifth spaces, and the apex-beat was imperceptible. A month later, when he left the hospital, during each systole, besides the indrawing of the spaces, there was indrawing of the lower half of the sternum, which sprang forward after the systole with a perceptible shock. He was admitted ten weeks later with pneumonia, when the heart-signs were unchanged, and he died fully six months after his first admission. The right ventricle was enlarged ; the valves were healthy; the heart, which lay in the middle of the chest, was firmly adherent to the pericardium, which was, in turn, strongly glued to the walls of the chest by a tuberculous exudation.

Skoda's third case was a man, with narrowing of the mitral orifice, ascites, and œdema. The region of cardiac dulness remained unchanged during inspiration and expiration. There was a considerable deepening of the fifth space during the

systole, after which the hollow quickly disappeared, and a shock was perceived there at the beginning of the diastole. After his death, five months later, the pericardium and pleura were found to be universally adherent, and the right side of the heart was considerably enlarged.

These cases, published by Skoda, form a valuable addition to the clinical history of adherent pericardium, for the true points of diagnosis have here been clearly observed, stated, and confirmed; and are given with force, and as the effects of the central cause, the doubly adherent pericardium. They do not, however, present any new points of diagnosis, for it will have been seen, in the previous narrative, that he has been anticipated by one or more authors in the observation of each diagnostic sign. Thus the systolic deepening of the intercostal spaces had been observed by Heim and Dr. Williams, the return shock over the previously retracted space by Sander, and the great extent of the cardiac space upwards, and the non-diminution of that space, by Dr. Williams and myself; while the retraction during the systole of the lower half of the sternum, and its advance with a shock immediately after the systole, was observed by myself in the case already given.

Great diagnostic value is to be attached to the principal points specially illustrated by Skoda's paper, namely: the systolic indrawing of the lower sternum or intercostal spaces by the contraction of the adherent heart; and the diastolic shock or back-stroke that immediately follows, given by the return elasticity of the chest-walls.

Cejka,[1] in 1855, published four cases of adherent pericardium, three of which confirm, with more or less precision, the points illustrated in Skoda's paper. In one of them, with contraction of the aortic orifice, there was systolic indrawing

[1] *Vierteljahrschrift für die praktische Heilkund*, 1855, 128.

of the third, fourth, and fifth spaces, and so strong a blow was given by the return elasticity of the chest walls that it was like the impulse of the heart. In another instance, an old man with adherent pericardium, a chronic affection of the lungs, dilatation of the aorta, and thickening of the mitral valve, the fifth and sixth spaces were drawn inwards with each systole, and became quickly even with each diastole. The impulse was not perceptible, and there is no note of diastolic back-stroke. In the third patient, with aortic aneurism, the vaulting of the sixth left space, caused by the systole, gave place towards the end of the case to a slight drawing inwards of the corresponding region. Cejka's fourth case of adherent pericardium, also with aneurism of the aorta, presented no impulse and no apparent drawing inwards during the systole.

Clinical History. (B) *Cases observed in St. Mary's Hospital and at Nottingham.*—1. *Cases examined after Death.*—The pericardium was completely adherent in fifty-one, and partially so in nine of the cases free from Bright's disease, recorded after death in St. Mary's Hospital up to the year 1870. (See the table at p. 174.) Besides these, seventeen of the cases with Bright's disease had universally, and three of them had partially, adherent pericardium.

Rheumatic pericarditis had evidently been the cause of the adhesions in more than one-half of the cases, since of those with complete adhesions, 29 in 51 were free from Bright's disease, and 9 in 17 with Bright's disease, had valvular disease of the heart; while the valves were affected in 7 out of 8 of those with partial adhesions that were free from Bright's disease, and the three cases of that class with that affection.

General adhesion of the pericardium was rarely associated with disease of the aortic valve (2 in 32), and with mitral

obstruction (1 in 21), in cases free from Bright's disease, while that affection was very frequent in such cases with mitral and mitral-aortic valve disease (13 in 33 of the former and 11 in 31 of the latter affection). Adherent pericardium was present in one case with disease of the tricuspid valve. Partial adhesions of the pericardium were noted in one case with aortic regurgitation, in two with mitral obstruction, in none with mitral, and in two with mitral-aortic regurgitation, without Bright's disease; since the aortic valve was affected in 1 in 4 of the cases, while only two had mitral and two had mitral-aortic disease. Among the cases of complete (17) and partial (3) adhesions with Bright's disease, 4 (in 21) had aortic valve-disease, 5 (in 29) had mitral and 2 (in 20) had mitral-aortic valvular disease, and 1 (in 9) had mitral contraction.

Aneurism of the ascending aorta was the evident cause of adherent pericardium in three instances (3 in 25), and cancer of the heart in one (1 in 10).

There was no other affection of the heart or aorta, excepting enlargement of the organ itself, in more than one-third of the cases with complete adhesions (19 in 52). The adhesions were not accompanied by any other affection in less than one-half of these cases (7 in 19), and they were complicated in more than one-half of them with pyæmia (in 2), apoplexy (in 1), pneumonia (in 3), empyema (in 2), phthisis (in 3), or peritonitis (in 1). All those affections, excepting the last two, were acute; and they could not, therefore, have given rise to the adhesions. Phthisis, and especially empyema, which is so often associated with phthisis, may, owing to the duration of those diseases, have induced first pericarditis and then adhesions. Notwithstanding this, the whole of those cases may be taken into account when considering the effect of pericardial adhesions on the size of the heart, for none of

them by themselves cause enlargement of that organ, excepting pneumonia, and, less often, phthisis, both of which affections tend to increase the right ventricle in size.

The heart was enlarged, its valves being thickened but competent in one instance, in fully two-thirds of the cases with adherent pericardium that were free from any other cardiac disease, and in which the size of the heart is mentioned (11 in 16); it was rather large in three of them; and in only two instances was the heart of its natural size. We may however, I think, estimate that in one-third of these cases the adhesions did not cause an increase in the size of the heart. These results do not differ materially from those arrived at by Dr. Kennedy,[1] who found that in 90 cases of adherent pericardium in which valvular disease was not present, the heart was of natural size—"healthy"—in 34, or fully one-third, hypertrophied in 51, or three-fifths—being dilated also in 26—and atrophied in 5.

It is proved that pericardial adhesions do not necessarily cause enlargement of the heart. I saw four cases in Nottingham in which the heart was of natural size and one in which it was lessened; Dr. Gairdner[2] gives brief notes of ten cases in which the heart was not morbid, and by inference was not affected in size; and Dr. Stokes[3] informs us that Professor Smith found that general adhesions of the pericardium corresponded with atrophy or with hypertrophy of the heart in nearly equal proportions.

We may, I think, safely conclude from what has gone before that adherent pericardium may, and often does, exist without influencing the size or healthy function of the heart; that in a few rare instances it may induce atrophy of that organ; and that in nearly two-thirds of the cases it tends to

[1] *Edinburgh Medical Journal*, iii. 986. [2] *Ibid*, Feb. 1851.
[3] Dr. Stokes, *Diseases of the Heart.*

cause an increase in the size of the heart, both as regards the thickness of its walls and the capacity of its cavities.

We have just seen that the heart was enlarged in the majority of the cases of adherent pericardium that were free from any other affection of the heart itself. When we take this into account it is natural to expect that the heart should be more enlarged in cases with valvular disease when they are affected with adherent pericardium than when they are not so ; and the analysis of the cases of this class that were recorded at St. Mary's Hospital by taking a simple average of the weights of the hearts with valvular disease, with or without pericardial adhesions, gives some support to this anticipation, as will be seen by the examination of the following summary of the average weight of the heart in those cases.

Average weight of the heart in cases of valvular disease with and without adherent pericardium. The cases were not affected with Bright's disease except where specified.

	Average weight.
Mitral regurgitation, pericardium adherent (4) . .	21 ounces.
Ditto, pericardium not adherent (14)	16·6 ,,
Ditto, with Bright's disease, pericardium adherent (3)	25 ,,
Ditto, pericardium not adherent (19)	19·4 ,,
Mitral obstruction, pericardium adherent (1) . . .	21 ,,
Ditto, pericardium, not adherent (14)	14 ,,
Aortic regurgitation, pericardium adherent (2) . .	26·7 ,,
Ditto, pericardium not adherent (23)	22 ,,
Mitral-aortic regurgitation, pericardium adherent (6)	26·3 ,,
Ditto, pericardium not adherent (12)	22 ,,
TOTAL of combined valvular diseases, without Bright's disease, pericardium adherent (13) . .	23·3 ,,
TOTAL of combined valvular diseases, without Bright's disease, pericardium not adherent (63) .	19 ,,

This method is far from doing scientific justice to the question before us; for cases of all ages, both sexes, and various degrees of disease, are brought together under one common heading, although in reality many of these cases differ materially from each other. Notwithstanding this, a rough and ready answer is given to us that is probably not far from the scientific truth. We find, then, that the average weight of the heart in the thirteen cases of valvular disease, with adherent pericardium, was $24\frac{1}{3}$ ounces, while its weight in sixty-three cases of a like kind, in which the pericardium was not adherent, was 19 ounces, or $5\frac{1}{3}$ ounces less than the first series. It is to be kept in view that the pericardium was included with the heart in the first set of cases, and what its average weight may be under the varying circumstances I do not know. It may, however, I think, be concluded that in the cases of valvular disease of the heart the existence of adherent pericardium tended to increase the size and weight of the heart, but not to a great extent.

The size of the heart, as we have seen, has been usually described; its weight being often given, in the cases with adherent pericardium observed in St. Mary's Hospital. The relative size of the different cavities of the heart has, however, only been described in 11 of these cases. I have, therefore, with a view to discover the influence that the presence of adherent pericardium may have on the size of the various cavities of the heart and the thickness of their walls, brought together 18 additional cases from various sources—or 29 in the whole—in which the general condition of the various cavities of the heart was described, and which are given in the following summary:—

Cases with adherent pericardium in which the size of the different cavities of the heart was described:—

1.—Cases in which both ventricles were enlarged (hypertrophy and dilatation)	16
Of these, 6 were free from valvular or other heart disease (1 had Bright's disease); 10 had valvular disease (3 aortic, 2 mitral, 3 mitral-aortic, regurgitation, 2 mitral contraction).	
2.—Cases in which the right ventricle was enlarged, the left being not so (in 1), or small (in 1), or not described (in 3) . .	5
Of these, 3 were free from valvular disease, 1 had mitral regurgitation, and 1 aneurism of the aortic sinuses.	
3.—Cases in which the left ventricle was enlarged, the right being small (in 1), or not described (in 7)	8
Of these 3 had no valvular disease, 1 had aortic, and 3 mitral, regurgitation, and 1 had aneurism of the apex of the left ventricle.	
TOTAL	29

There was valvular disease of the heart (15), or aneurism of the heart (1) or aorta (1) in 17 of these cases, and as those affections exercise a definite influence of their own on the size of the cavities of the heart, they must be left out of view in considering the direct effect of adherent pericardium on those cavities. The same must be said of one instance with Bright's disease among the remaining 12 cases in which there was no valvular or other affection of the heart or aorta. Hypertrophy and dilatation of both ventricles existed in 5; of the right ventricle in 3; and of the left ventricle in the remaining 3, of these 11 cases. From this it would appear that adherent pericardium, when it produces enlargement of the heart, tends to affect both ventricles to an equal but varying degree.

2. *Physical signs observed during life in cases with adherent pericardium admitted into St. Mary's and the Nottingham Hospitals.*—I have observed nine cases with adherent pericardium in St. Mary's Hospital, and have added one recorded there by Dr. Markham; and have examined seven such cases at Nottingham, four of which I published in 1844, and have

given briefly above. There was no valvular disease of the heart in three of these seventeen cases, while in the remaining fourteen, one or more of the valves was affected, mitral regurgitation being present in nine of them, mitral-aortic regurgitation in three, and mitral obstruction in two, of those cases.

In one of the three cases in which the valves were healthy, in which case Bright's disease was present, the sounds of the heart were natural but weak, and the presence of impulse was not noted. In another of them, a man, with empyema and lardaceous disease of the kidney, the heart being only slightly enlarged, the impulse was at one time imperceptible, but afterwards, when it could scarcely be felt over the ribs, it was perceived over the ensiform cartilage. In these two cases, and in that of the same class already alluded to at page 204, in which the heart was small, the presence of adherent pericardium could not, I think, have been discovered during life.

The signs of the heart were not noticed in one of the cases in which adherent pericardium was associated with mitral regurgitation, an old man who presented various sonorous noises over the lungs. In one of two cases, both men, with mitral disease, observed at Nottingham, in which the heart was very greatly enlarged, the left ventricle was greatly hypertrophied and dilated, the right being so to a minor degree; and the impulse was feeble, the second sound, distinct over the sternum, was scarcely audible at the apex, and the lungs were œdematous. In the other case, with hypertrophy of both ventricles, the impulse was inconsiderable, but was diffused over the whole left mammary region.

The next case is an important one, reported by that careful and accurate observer, Dr. Markham, for it shows that the

apex-beat may be strong, and far to the left, in some unusual cases of adherent pericardium. In this patient, a girl, the impulse was heaving and extensive, and was violent far to the left of the nipple line, and beneath the sixth rib. The second sound was very loud over the pulmonary artery, but was absent at the apex. M. Aran likewise describes a case of adherent pericardium, in which the apex-beat was present in the sixth space, three-and-a-half inches from the sternum, and the systolic impulse was strong and progressive, and was not followed by a diastole impulse. Skoda takes exception to my observation that the apex protruded extensively to the left in two of my cases published in 1844, given briefly above at pp. 204–206. We shall see that the apex-beat is usually feeble, and does not extend far to the left in cases of adherent pericardium; but it was certainly otherwise in this case of Dr. Markham, in that of M. Aran, and, I would say, also in my two published cases. It appears to me that in this patient and in the other cases just given, there was no sign characteristic of adherent pericardium.

The next instance was too ill for careful physical examination, and presented a feature unusual in cases with pericardial adhesions. The healthy impulse was much more diffused than natural, being present in the epigastric space and four or five intercostal spaces, and the lower ribs retracted during the diastole, which is a rare occurrence. The apex-beat, which was felt in the fifth and sixth spaces, did not extend outwards so far as the nipple line. The two following instances present features that were sufficient to characterise them during life as being affected with adherent pericardium. In the first of these cases, the left ventricle was hypertrophied, the right ventricle was small, and both the auricles were very large. The apex-beat was seated in the sixth space, an inch

to the left of the nipple line, and $5\frac{1}{2}$ inches from the sternum, and in spite of the great and extensive hypertrophy of the left ventricle, was feeble. The second sound, which was heard over the right ventricle, was faint at the apex. There was, on the 54th day after admission, a diffused impulse chiefly over the cardiac cartilages, extending down to the seventh costal cartilage, and to the ensiform cartilage. The impulse advanced quickly and fell back suddenly during the systole, and was followed with a sharp sudden shock or jerk over the whole region of the impulse. There was slight pulsation of the liver below the ensiform cartilage. Breathing was rather high, the movement being chiefly at the upper part of the chest, with retraction at its lower part. The other case, equally remarkable, and the last of the series with mitral incompetence, had points of close resemblance to the last, with points of marked difference. In this case the front of the heart adhered strongly to the inner surface of the sternum through the medium of the pericardium. The walls of the right ventricle and auricle were much hypertrophied, while the left ventricle was only somewhat thickened; thus reversing the conditions that were present in the former case. There was some fulness over the region of the heart. The impulse over the heart, and especially over the right ventricle, was very extensive, spreading from the third to the seventh cartilage; and from the right cartilages, across the sternum and ensiform cartilage, to the sixth left space, an inch-and-a-half beyond the nipple line. The impulse was peculiar, and told remarkably on the sternum, first heaving that bone forwards with sudden force, and then drawing it backwards with great strength. "The heart" (or rather the front of the chest) "seemed to be dragged backwards during each systole. The apex-beat was feeble, low down, and far to the left, in the sixth space, an inch-and-a-half beyond the nipple line.

There was some pulsation of the liver in the epigastric region. The second sound was loud and plunging over the right ventricle, and feeble at the apex, where a mitral murmur was loud and extensive. Afterwards the fulness over the heart, and the extent and force of the impulse lessened, but the beat of the heart retained its remarkable character, first advancing, and then forcibly retracting, during the systole. Later still the apex-beat, which was very weak, extended only a very little beyond the nipple line. Notwithstanding this contraction of the region of the impulse, it extended from right to left over a width of six inches. A deep inspiration caused a marked lowering of the upper and lower borders of the region of the impulse, in spite of its great extent. After a few days he became drowsy, felt tight in the chest, and died three weeks after his admission." It is to be remarked that while in the previous case a diastolic shock or back-stroke followed the systolic retraction, which was preceded by a systolic advance; in this case there is no note of back-stroke, though I cannot vouch for its absence; but the sudden systolic heave followed by a forcible systolic retraction of the sternum and cartilages, as if those parts were dragged backwards by the heart clinging, as it were, to its buckler, pointed definitely to adherent pericardium as the cause of the chain of signs.

The two cases of adherent pericardium with mitral-aortic incompetence present, like the last two cases, physical features that denote the presence of the adhesions, though not perhaps with the same emphasis as the two first related. In the first case, a youth, the heart was of very great size, so as completely to cover the left lung. On his admission, three months before his death, the impulse was gradual, but ended abruptly with a shock; and extended from the third cartilage to the sixth, but scarcely beyond the nipple line; there was

also a marked general pulsation over the whole liver, both in front and at the right side. A month later the impulse had extended itself to the left, being diffused, and shaking the whole of that side of the chest, the apex-beat being an inch-and-a-half to the left of the nipple line. Afterwards the impulse extended more to the right and was felt in the epigastrium, but its characteristic features are not again described The other instance was a boy, and in him the heart, which was considerably enlarged, clung so close to the sternum and cartilages that it was found best to remove the viscera *en masse* from behind. There was fulness over the cardiac region, and the beat of the heart, which was extensive, reaching down to an inch-and-a-half below the sternum, and extending thence to the seventh cartilage, was of a peculiar character, beginning with a diffused heaving impulse, which gave way to a sudden and sharp retraction. He always said, after this examination, that he felt better, though he really was not so, and eight days later he died.

The two remaining cases with adherent pericardium had mitral contraction. In one of them, a young woman, the heart was very large ; the impulse extended from the second space to the seventh costal cartilage and the ensiform cartilage, and, even when she lay on the left side, the apex-beat was feeble. As in the last case, there was strong pulsation over the whole liver, extending from the front to the back. The remaining case with adherent pericardium and mitral contraction was observed by me in Nottingham in 1835, and although it presents no signs characteristic of the adhesions, is perhaps of interest, as being, so far as I know, the earliest case in which the so-called presystolic murmur was described. The size of the heart is not given, but there was no hypertrophy of either ventricle. The mitral opening was half an inch in diameter. A thrill, extending over a large space, was

communicated to the hand when applied over the apex, which was terminated by a jerk. A peculiar purring sound was heard at the apex, the vibrations being longer and louder as the time progressed, the sound ending in a strong loud clear jerk, synchronous with the pulsation. The sound occupied two-fourths of the time, no other being audible at the apex.

Resumé of the Physical Signs observed in Cases of Adherent Pericardium.—The steady retraction of the lower half of the sternum during the whole of the systole of the ventricles, and the sudden starting forwards of the lower half of the sternum at the beginning of the diastole with a return shock or blow, was observed in my own case, published in 1844, and in one of Skoda's given in 1852.

The drawing inwards of the cardiac intercostal spaces during the systole was first observed by Heim, and afterwards by Dr. Williams, by Skoda in three cases, and by Cejka in three more.

This sign, which is sometimes present in other cases, renders the existence of adherent pericardium probable, and especially if this sign is still present when the patient draws a deep breath ; but if it is followed by a diastolic shock the diagnosis of that affection is certain. The existence indeed of a diastolic back-stroke taken by itself pronounces that the heart is adherent. This sign, which generally gives the impression of a double impulse, was first noticed by Sander ; afterwards by Dr. Hope in four cases of adherent pericardium ; in the two typical instances just given and described respectively by myself and by Skoda, who observed it in another instance ; by Cejka in one, and by myself in two others given above.

A double movement of the systolic impulse, first forwards with a heaving motion, then backwards with a forcible retraction, was observed by myself in a case in the Nottingham

Hospital, to the description of which Skoda takes exception, and afterwards in three other cases in St. Mary's Hospital. The outward pressure, equal in every direction, of the blood contained in the ventricle during its contraction naturally forces forwards the walls of the chest in front of it at the beginning of the systole. During the continuance of the systole, the adherent sternum resists the contraction of the heart, but in the struggle the bone yields, and is drawn forcibly inwards by the active ventricle.

The non-diminution of the region of pericardial dulness and of the impulse was observed by Dr. Williams; and the absence of change in the position of these signs when the patient lay on the left side was noticed by Dr. Law.

The non-diminution of the area of pericardial dulness and impulse is undoubtedly a valuable sign of adherent pericardium; in one of my cases, however, the impulse below was unusually strong at the end of expiration, and in another of them the upper and lower borders of the impulse palpably descended during a deep inspiration. This is indeed different from the diminution of the extent of dulness and impulse and, what is still more important, from the bodily transfer during a deep breath of the seat of the dulness and impulse from the cardiac cartilages and the fifth space near the nipple, to the epigastric region, including the ensiform cartilage and the adjoining seventh costal cartilage. One of my cases illustrates in its own manner the other point just referred to—the non-shifting of the seat of the impulse when the patient turns on the left side. In that case, when the patient lay on the left side, the apex-beat, which was an inch and a half to the left of the nipple line, and in the sixth space, was very feeble. This is very different from the great transfer of the position of the apex-beat from the fifth space, a little lower than the nipple, and within the mammary line, to the sixth or

seventh space, two inches to the left of that line, which was observed to be the case in several patients, in whom the chest was healthy, by Dr. Humphreys, Dr. Coupland, and myself, in the Middlesex Hospital.

These, so far as I know, are the only signs that are characteristic of adherent pericardium; but there are certain other signs that, without ranking in precision with those just named, have their significance.

The drawing inwards during the systole of the space between the ensiform cartilage and the seventh costal cartilage, was noticed by Sander in a case of adherent pericardium; and in another case, I observed that the tip of the ensiform cartilage was retracted during the contraction of the ventricle.

There was pulsation of the liver in four of my cases, which was limited to the epigastric space in two of them, but in the two others extended over the whole organ, in front, at the side, and in one even behind. Burns considered that the impulse so often present in the epigastric space in cases of adherent pericardium is due not immediately to the heart itself, but to the pulsation of the liver.

It is evident, from the brief recital of the cases that has just been given, that a great variation in the extent, force, character, and position of the impulse exists in cases of adherent pericardium.

The impulse was imperceptible in one of Cejka's, and at an early period in one of my own cases of adherent pericardium; and it was feeble in one of Skoda's and two of my own cases; it was heaving during the systole and very extensive in one of Dr. Markham's cases, and in one of my own; it was tumultuous and very irregular in one of my cases; it was strong and very greatly extended, both upwards to the second space, and downwards to the epigastric space and the seventh

cartilage, and to the right and left, across the chest, from a full inch to the right of the lower half of the sternum, to a full inch to the left of the nipple line in the sixth space, in cases observed by Dr. Hope, Dr. Markham, and myself; and in two of Dr. Hope's cases the violent action of the heart was observed over the whole front of the chest.

The apex-beat is, as a rule, feeble, even when it extends from an inch to an inch and a half to the left of the nipple line, being felt in the sixth space. Sometimes indeed, as in one of Skoda's cases, it is imperceptible; and at others it is situated, even when there is general enlargement with hypertrophy of the ventricles, to the right of the nipple line, as occurred in one of M. Aran's cases, in which the apex-beat was in the fifth space, two and a half inches from the sternum; and in two of the cases given by Dr. Gairdner, who points to this restraint of the apex as a probable element in the diagnosis of adherent pericardium.

There are, however, important exceptions to the rule that the apex-beat is usually restrained in its action and sometimes in its position by adherent pericardium, for in two cases published by me in 1844, the apex-beat was far to the left and low down, strong, gradual, and protruding; and as we have seen, the apex-beat presented the same condition in Dr. Markham's, and to a less degree in M. Aran's important cases.

The impulse was found in the epigastrium in Mr. Burn's cases, in two of Dr. Hope's, and in four of my own.

M. Aran, in 1844, gave the extinction of the second sound as the unique sign of adherent pericardium, on the strength of the absence or great feebleness of that sound in those cases reported by him. He does not distinguish between the second sound over the pulmonary artery and right ventricle, and that over the left ventricle. Dr. Markham describes the

second sound as being, in his case with mitral incompetence, very loud, heard like a beat, over the pulmonary artery, while there was no second sound over the apex. In one at least of my cases observed in Nottingham the second sound was loud or natural over the right ventricle, while it was indistinct and dull at the apex, and in two of the cases given above the second sound, loud over the pulmonary artery and right ventricle, was feeble at the apex of the heart.

The last physical sign that I shall consider is the movement of respiration in relation to adherent pericardium. In two cases of adherent pericardium observed by myself in Nottingham, the inspiratory movement of the abdomen at its centre was equal to that at its sides: although in health, the central movements are from two to three times as great as the lateral movements of the abdomen. At the same time in both those cases the lower half of the sternum fell inwards, or was drawn backwards, and the left ribs, from the fourth to the sixth, either retracted or were stationary, or had much less movement during inspiration, than the corresponding right ribs. The retraction of the sternum was caused by the forcible displacement downwards of the central tendon of the diaphragm, where it forms the floor of the pericardium; and as under these circumstances the lungs could not interpose themselves between the heart and the sternum, that bone was partly forced backwards by atmospheric pressure, and partly dragged backwards by the adherent heart, when drawn somewhat downwards by the diaphragm.

XVIII.

ENDOCARDITIS.[1]

ENDOCARDITIS, to a greater extent even than pericarditis, is chiefly associated with acute rheumatism. The extent to which this is the case will be seen by the study of the accompanying table, at page 239, from which it may be seen that endocarditis without pericarditis was established in one-third of the cases, or in 107 out of a total number of 325. If to these we add those cases with pericarditis that were also affected with endocarditis, amounting to 54, we find that endocarditis attacked one-half of the cases of acute rheumatism, or 161 in 325. In addition to these cases, in which the presence of endocarditis was rendered certain by the character of the signs and symptoms observed during the attack, there was a considerable proportion of the cases, amounting to one-fourth of the whole (76 in 325), in which endocarditis was either threatened (in 63) or very probable (in 13). Endocarditis is not however limited to acute rheumatism, being also present in a considerable proportion of cases affected with chorea, and in a small but uncertain number of those with pyæmia and Bright's disease. Cases, also, of established valvular disease of the heart are subject to intermitting attacks of endocarditis affecting the diseased valves.

I shall, in this article, (1) first give a brief account of the

[1] From Vol. iv. of Reynolds's *System of Medicine*.

anatomical appearances that present themselves after death in endocarditis, and then (2) a clinical history of rheumatic endocarditis, as it presented itself in the cases with acute rheumatism under my care in St. Mary's Hospital, during the years 1851 to 1869-70; those cases being divided into two series, an earlier series from 1851 to 1866, and a later series, treated by means of rest, from 1867 to 1869-70.

I.—The Anatomical Appearances Observed in Cases of Endocarditis.

The anatomical appearances found after death in cases of endocarditis have been well described from actual observation in the excellent and readily available works of Rokitansky,[1] Hasse,[2] and Rindfleisch,[3] which have been well translated; and in the original and interesting lectures of Dr. Moxon,[4] and manual of Dr. Payne.[5]

The inflammation of the interior of the heart is as a rule limited to the left ventricle, this being evidently due to the great labour to which that ventricle is subjected when it drives the blood into the arteries of the system, and to the comparatively slight effort with which the right ventricle sends its blood through the vessels of the lungs. In the fœtal state, the right side of the heart, which is then the most powerful side, and has the greatest amount of work to do, is subject to endocarditis, judging by the frequency with which the pulmonary valves are adherent, so as to contract the orifice of the

[1] Rokitansky, *Pathological Anatomy*, Syd. Soc. iv. 175.
[2] Hasse, *Pathological Anatomy*, Syd. Soc. 124.
[3] Rindfleisch, *Pathological Histology*, New Syd. Soc. i. 279.
[4] Dr. Wilks and Dr. Moxon, *Pathological Anatomy*, 125.
[5] Dr. Jones, Dr. Sieveking, and Dr. Payne, *Pathological Anatomy*, 348.

pulmonary artery. Dr. Norman Chevers finds that sixty such cases have been observed by various authors. The mitral and aortic valves are the chosen seat of endocarditis, and especially the mitral valve. It is not, however, the whole of either valve that is the immediate seat of the inflammation; which, as a rule, is limited to the lines and surfaces of contact of the valves, close to the edges of their flaps where they come together and press against each other so as to close their respective apertures. The aortic valve is shut by the blood quietly filling the sinuses towards and at the end of the systole and during the diastole. The blood, when the sinuses are filled, presses the sides of the flaps against each other with a diffused and equal but firm pressure. This pressure is made on the first closure of the valve at the end of the systole, by the blood filling the sinuses; but this pressure is suddenly reinforced by the back-stroke or return wave of blood, caused by the recoil of the distended aorta and arteries, which propels the blood equally in every direction, forwards and sideways, as well as backwards with a return stroke, which beats on the aortic valve sinuses, and the ascending aorta, and which causes the second sound, which follows the closure of the valve by the tenth of a revolution of the heart's action. Afterwards the pressure of the aortic flaps upon each other is kept up during the diastole by the pressure of the blood, due to the steady contraction of the coats of the aorta and its branches. The pressure upon the aortic flaps bears, not upon their exact margins, but upon their surfaces of contact, a little within those margins, and upon the sesamoid bodies; and the endocarditis affects, not the exact margins of the flaps, but their surfaces of contact.

The mitral valve is shut on exactly the same principle as the aortic valve, by the pressure of the blood driven during the systole into the small open cells on the under or ventricular

surface of the valve, in the manner described and figured at page 89, Vol. III. The force with which the blood presses upon the closed mitral valve, owing to the contraction of the ventricle, is much greater than the force with which the blood presses upon the aortic valve, owing to the recoil of the previously distended walls of the aorta. The flaps of that valve are pressed together by the backward portion only of the effect of the recoil of the aorta walls, which expands itself in every direction; and that force of recoil is itself but a portion of the original propulsive force of the left ventricle, which presses with its full power upon the closed mitral valve. The surfaces or lines of contact and closure of the mitral valve extend along and just within the borders of its two flaps. This border of contact is not a mere edge, but a surface or line of adaptation, made up of the small bead-shaped cells, that dove-tail into each other along the margins of the flaps; those flaps being held in their place by the simultaneous contraction of the papillary muscles, acting on their tendinous cords; the result is that the margins of contact of the mitral flaps press against each other when the valve is shut with much greater tension, force, and concentration, than the margins of contact of the aortic valve; under the triple agency of a finer margin of contact, greater pressure of blood, and the muscular force and tendinous traction proper to the valve. The mitral valve, which is situated in the muscular centre of the ventricle and in the focus of its internal inflammation, is more immediately and frequently subjected to endocarditis than the aortic valve, which has broader surfaces of contact, less pressure of blood, and no muscular and tendinous traction.

Endocarditis, as I have said, does not therefore attack the very rim of the flaps of the mitral valve at the attachment of their out-spreading tendinous cords, but the line or margin of

contact just within the edges of the valves. When the mitral valve is inflamed, a frill of small bead-like granulations lines the whole proper border of contact and closure of the valve; and tends to prevent their perfect adaptation, and to cause regurgitation through the valvular aperture when the ventricle contracts. These prominences consist of a swelling and granular disintegration of the connective tissue, with softening of the intercellular structure. Each of these prominences is covered by a cap of fibrin deposited from the blood in the manner well represented by Rindfleisch.[1] Endocarditis affects the surfaces of contact of the aortic valve in the same way that it affects those of the mitral valve.

This is the usual manner in which endocarditis affects the mitral and aortic valves, whether the parent affection, rendering those parts prone to inflammation, be acute rheumatism, chorea, or pyæmia. Sometimes, however, the inflammation deepens at its original seat on the surfaces of contact of the mitral valve, and extends beyond those surfaces, so as to affect a large portion of the flaps of the valve on their ventricular surface. Under these circumstances, the inflamed, softened, and thickened structures may undergo granular degeneration, and its ventricular layer may become broken or ulcerated. The auricular layer of the valve thus tends to yield before the pressure of the blood, which forces its way through the breach in the ventricular layer, and to form pouches or aneurisms protruding into the left auricle. The auricular layer may then be involved in the inflammation, and become in turn subjected to granular disintegration and breaking up of tissue, so that the flap of the valve may become perforated. The fibrin of the blood deposits itself everywhere on the inflamed surfaces, often in the form of

[1] *Loc. cit.* p. 281, fig. 87.

vegetations, which may become extensive; and thus the fibrin often lines, closes, and conceals the perforation.

We have already seen how many points in its favour, as regards its tendency to endocarditis, the aortic valve presents over the mitral; and it presents another in this respect—that while the pressure of the blood bears directly upon the inflamed surface of contact of the mitral valve during its closure at the time of the systole, the pressure of the blood does not bear upon the inflamed ventricular surface of contact of the aortic valve when it is closed at the time of the ventricular diastole, but upon the uninflamed upper or aortic surface of the valve. Although this condition, favourable to the aortic valve, exists, I have seen preparations in which a small aneurism, or aneurisms, of one or more of the flaps of the aortic valve protruded downwards into the ventricle.

The advantages are not, however, entirely on the side of the aortic valve when it is affected with endocarditis; for a serious counterbalancing disadvantage exists under such circumstances, as I shall now mention. The sesamoid body, and the margin or surface of contact of the valve on each side of the sesamoid body, which are the seat of endocarditis when it affects the aortic valve, receive the direct pressure of the column of blood in the aorta; and those parts, which are softened by the inflammation, tend therefore to be pushed downwards towards the ventricle during the ventricular diastole; with the effect of sometimes producing retroversion of the sesamoid body, and to a greater or less extent of the softened flap, of which it is the centre. We here see the great disadvantage in which the inflamed aortic valve is placed from the want of tendinous cords and papillary muscles to support its flaps when rendered soft and yielding by endocarditis.

Another special evil accruing to the aortic valve from a

similar class of cause, is the tendency of the sesamoid body, and the adjoining portion of the flap affected with endocarditis, to lay hold of deposits of fibrin from the regurgitating stream of blood, with the effect of establishing a chain of fibrinous vegetations, which form one upon another, and which hang pendant into the left ventricle, being forced in that direction by the return current of blood. When this chain of fibrinous concretions forms upon either the right or the left posterior flap of the valve, it is driven downwards and backwards by the stream of regurgitation, so as to beat against and rest upon the anterior flap of the mitral valve, with the effect of causing ulcerative endocarditis of that flap. As the blood regurgitating from the aorta into the ventricle beats upon that flap, it parts with its fibrin, which clings to the inflamed surfaces of the mitral valve, and forms on these a second chain of fibrinous concretions.

The flaps of the mitral valve are, as we have seen, the principal seat of endocarditis, but inflammation may also attack the papillary muscles, and especially where they are brought into contact with each other towards the end of the systole, and cause fibroid degeneration of those muscles. The tendinous cords may also sometimes become inflamed, softened, and disintegrated, when the grave result of rupture of the cord may ensue.

I have just given a series of notable instances of the occurrence of endocarditis, locally excited by the contact with each other of the two opposing surfaces of the valve; of two adjoining papillary muscles; and of a pendant chain of fibrinous concretion beating against the anterior flap of the mitral valve. These are not the only parts of the interior of the heart that may be inflamed from this cause, for wherever two surfaces of the endocardium come into contact with and rub against each other, endocarditis may be excited in both

of those surfaces. The influence of the labour of the left ventricle and the mutual contact of its internal surface in tending to produce endocarditis is illustrated in an original and able manner by Dr. Moxon. I would refer to his work and to the others already named for the study of the various effects of endocarditis.

Among the effects of endocarditis, I would here simply name the formation of vegetations on the inflamed valves, already in part illustrated ; the production of embolism by the washing away from the vegetations of fibrin into the current of the blood ; the ulceration of the surface of the endocardium ; the establishment of valvular disease from the thickening and enlargement of the valves ; the contraction, adhesion, or retroversion, and perforation of their flaps ; the rupture of the tendinous cords ; the formation of aneurisms of the valves ; the fibroid and atheromatous degeneration of the fibrous and muscular structures of the ventricle ; the production of aneurisms of the heart ; and other effects that will be found described in the works to which I have referred.

II.—Clinical History of Rheumatic Endocarditis.

The accompanying analytical tables of 325 cases of acute rheumatism under my care in St. Mary's Hospital during the years 1851-66, show the proportion in which those cases were free from endocarditis, and were threatened with or attacked by that affection ; and the number that were attacked by pericarditis, distinguishing those with established endocarditis ; also those in which endocarditis was doubtful, and those in which it was absent.

The analyses contained in the tables sufficiently indicate the reasons for arranging the cases in the manner adopted.

TABLE SHOWING THE CONDITION OF THE CASES OF ACUTE RHEUMATISM, WITH ESPECIAL RELATION TO THE ABSENCE OR PRESENCE OF ENDOCARDITIS.

I.—Cases of Acute Rheumatism in which there was no Endocarditis.

Affection of joints somewhat severe or moderate, no general illness, no palpitation, signs over heart not named 2
Joint affection slight, some general illness, heart not named 13
Joint affection not, or scarcely severe, some or little general illness, heart sounds healthy . . 10
Joint affection not, or somewhat severe, some or considerable general illness, heart not named 5
Joint affection not severe, some or considerable general illness, heart sounds healthy 10
Joint affection severe, some general illness, heart not named 6
Joint affection somewhat severe, considerable general illness, heart sounds healthy, or loud and ringing 7
Joint affection severe, some general illness, heart sounds healthy 11
No description of state of joints, or general illness, heart sounds feeble 1
Joint affection not, or rather severe, slight or no general illness, slight prolongation of first sound 7
Joint affection rather severe, slight or no general illness, doubtful occasional obscure murmur 1
Previous valve disease, mitral regurgitation ⇒ 2
Death, delirium 4

I.—Total 79

II.—Cases of Acute Rheumatism in which Endocarditis was threatened.

Some general illness, pain over the cardiac region, heart not named 1
Great general illness, pain left side, or region of heart, signs of heart not named 2
Great general illness, pain left side, heart sounds healthy 3
Great general illness, pleurisy, heart sounds healthy 1
Great or considerable general illness, pain left side, or region of heart, heart sounds healthy . 8
Great general illness, delirium, pain left side 1
Considerable general illness, first sound very loud 3
Considerable general illness, doubling of first sound 1
Considerable general illness, first sound or heart sounds feeble or indistinct 3
General illness, pain over region of heart or left side, first sound indistinct or muffled . . . 2
Slight general illness, prolonged first sound 13
Great general illness, prolonged first sound 3
Great general illness, lung affection, prolonged first sound 4
General illness, pain in region of heart or chest, prolonged first sound 10
Little general illness, faint or obscure murmur early or late in the attack 5
Considerable general illness, obscure murmur after cessation of attack (endocarditis probable) . 1
Previous valve disease, considerable general illness 2

II.—Total 63

III.—Cases of Acute Rheumatism in which Endocarditis was probable.

Great general illness, pulmonary apoplexy in 1, prolonged first sound (situation unknown), almost a murmur in 1, a pulmonic murmur in 1 2
Great general illness, severe cough in 2, prolonged first sound at apex, almost a mitral murmur in 2, almost a tricuspid murmur in 1, a pulmonic murmur in 3 4
Great general illness, prolonged first sound at right ventricle, almost a tricuspid murmur, and a pulmonic murmur 1
Slight general illness, tricuspid murmur ⇐, ending in prolonged first sound in 1 2
Slight general illness, previous or established mitral regurgitation ⇒ murmur did not vary materially in 1, murmur became louder in 1 2
Considerable general illness, previous or established mitral aortic regurgitation ⇒ ⇩, aortic murmur absent at first in 1, mitral murmur became musical in 1 2

III.—Total 13

IV.—Cases of Acute Rheumatism in which Endocarditis was present without Pericarditis.

Prolongation of first sound, almost a murmur, pain in heart, 1, in chest 1, extreme general illness 2
Tricuspid murmur ←, murmur absent on recovery ←o 7
Tricuspid murmur ←, murmur lessening on recovery ← 6
Tricuspid murmur ← TOTAL 13
Mitral murmur →, murmur disappearing on recovery o→ 25
Mitral murmur →, murmur lessening on recovery → 10
Mitral murmur →, murmur established on recovery → 14
Inflammation of mitral valve →, died, murmur in 1, no note of murmur in 1 . . ⬭ 2
Mitral endocarditis → TOTAL, mitral murmur in 50, no note of murmur in 1 . . 51

Aortic murmur ↓ murmur disappearing on recovery o↓ 5
Aortic murmur ↓ aortic regurgitation established on recovery ↓ 5
Aortic murmur ↓ TOTAL 10

Mitral-aortic murmur ↓→ murmurs disappearing on recovery o→o↓ 3
Mitral-aortic murmur, mitral murmur established, aortic murmur disappearing →o↓ . . 2
Mitral-aortic murmur, mitral-aortic regurgitation established →↓ 4
Mitral-aortic murmur, →o↓ TOTAL 9

Previous valvular-disease, mitral regurgitation ⇨ 6
Previous valvular disease, mitral and tricuspid regurgitation ⇨ 3
Previous valvular-disease, mitral regurgitation, adherent pericardium aortic regurgit. ⇨↓ 1
Previous valvular-disease, aortic regurgitation ⇩ 3
Previous valvular-disease, mitral-aortic regurgitation (tricuspid murmur 2) ⇨⇩ . . 9
Previous valve disease.—TOTAL 22

IV.—Total cases of Endocarditis *107

V.—Cases of Acute Rheumatism with Endo-Pericarditis.

Heart previously healthy, 46.
- Tricuspid murmur ← 3 . . { Murmur disappearing on recovery ←o . . 1; Murmur established on recovery ← . . . 2 } 3
- Mitral murmur → 36 . . { Murm. disappearing on recov., mitral o→ 17, aortic o↓ 1, mitral-aortic o→o↓ 1 = 2 } 19; Murmur lessening on recovery, mitral → . 8 } 43
- Aortic murmur ↓ 1 . . .
- Mitral-aortic murmur →↓ 6[1] { Mur. estb. on rec., mitral → 11, mit.-aor. →↓ 5 = 16 }

Total cases of endocarditis in which the heart was previously healthy 46
Cases of endocarditis, with prev. valv. disease, mitral ⇨ 5, mitral-aortic ⇨⇩ 3 . . 8
Total cases with endo-pericarditis 54

IV., V.—Total with endocarditis 161

VI.—Cases of Acute Rheumatism with Pericarditis; Endocarditis being doubtful 3

VII.—Cases of Acute Rheumatism with Pericarditis in which there was no Endocarditis 6

V., VI., VII. Cases of Acute Rheumatism with Pericarditis.—TOTAL 63

GRAND TOTAL of Cases of Acute Rheumatism 325

* 108 cases of Endocarditis appear in the Tables at pages 242, 244, Vol. iii. I find that one of those cases has been accidentally enumerated twice over, a woman, aged 23.

Correct errors of press in the Table at page 282 thus—(1) line 9, for 1 *read* 6; (2) line 12, for mitral-aortic *read* mitral-aortic 5; (3) line 16, for total 10 *read* 19.

I have considered the cases of endocarditis according to the character of the valvular affection of the heart due to the inflammation of the interior of the ventricle, and have arranged these cases into those (I.) with an uncomplicated tricuspid murmur; (II.) with mitral regurgitation; (III.) with aortic regurgitation, (1) not accompanied by a mitral murmur, and (2) accompanied by a mitral murmur; (IV.) with prolongation of the first sound without a murmur; (V.) with endocarditis supervening upon previous valvular disease.

I.—Cases of Rheumatic Endocarditis with an Uncomplicated Tricuspid Murmur. *Symbol* ←

In a moderate proportion of the cases of rheumatic endocarditis under my care in St. Mary's Hospital during the fifteen years ending 1866—amounting to 13 out of a total number of 107, or one in eight—there was a murmur over the right ventricle from regurgitation through the tricuspid valve, without a mitral murmur. In nearly all of these cases there was a greater or less amount of general illness, and in one-third of them (4) there was pain in the region of the heart. A tricuspid murmur was present also in 2 cases, in which endocarditis was probable, and in 2 that have been included, with a little doubt, among the cases of pericarditis.

In the majority of these cases the murmur had disappeared when recovery was established; and in the remainder the murmur was then diminishing in loudness, extent, and clearness.

This tricuspid murmur is usually present over the body of the heart, or, in other terms, over the right ventricle; and extends from the lower half of the sternum to a line a little

within the left nipple, which line corresponds with the ventricular septum, and from the third to the sixth cardiac cartilage. The presence of this murmur in these cases over the right ventricle in the early stage of endocarditis, and that, too, when no other murmur prevails, naturally suggests to the mind at first sight that it is due to endocarditis affecting the right ventricle and the tricuspid valve.

This inference is, however, forbidden by the following considerations.

(1) Endocarditis, and disease the result of endocarditis of the tricuspid valve, are very rarely discovered on dissection in those who have died from rheumatic inflammation of the interior of the heart, or from valvular disease, the effect of such inflammation.

(2) The tricuspid murmur, when uncomplicated with disease of the mitral valve, was not established in any of my cases, but had either ceased altogether, or was steadily declining on the recovery of the patient.

(3) The tricuspid murmur was frequently associated with a mitral murmur, and less often with a mitral-aortic or an aortic murmur of recent origin.

A tricuspid murmur was present over the right ventricle in one-half, or 27 in 50, of the cases with recent mitral murmur. In 7 of those 27 cases the presence of a tricuspid murmur was somewhat doubtful. In eight of those cases the mitral was preceded by the tricuspid murmur, and in six of these the tricuspid murmur had ceased to be audible when the mitral came into play. In thirteen other cases both murmurs were present when they were first noticed, which was at the time of admission, in fully one-half of those patients. The mitral murmur appeared before the tricuspid in five cases. The tricuspid murmur disappeared when the mitral murmur was still audible in two-thirds of the cases (16 in 27); both

murmurs ceased at the same time in seven instances ; and in four the tricuspid murmur outlived the mitral. A tricuspid murmur was also present in one-third, (3 in 10), of the cases of endocarditis with mitral disease of old standing.

A tricuspid murmur was present in two or three of the eight cases of mitral-aortic, and in about four of the ten cases of aortic, regurgitation of recent origin ; and in two of the five cases with aortic, and none of the seven instances with mitral-aortic valvular disease of old standing affected with endocarditis.

(4) I have observed tricuspid regurgitation as a marked and lasting feature in a case of button-hole contraction of the mitral valve ; in several instances in which the tissue of the lung was permanently condensed, owing to repeated attacks of bronchitis ; in patients affected with contracted granular kidney, in whom obstruction of the pulmonary circulation, with enlargement of the right ventricle, had followed upon obstruction of the systemic circulation, with its attendant tension, dilatation, and thickening of the systemic arteries, and hypertrophy of the left ventricle.

These circumstances point irresistibly to the conclusion that the tricuspid regurgitation is usually due to the so-called "safety-valve" function of that valve, and not to endocarditis of the right side of the heart. In all these cases resistance to the flow of blood through the lungs has induced tension of the pulmonary artery, and distension of the right ventricle and auricle, with, as a result, incomplete closure of the tricuspid valve. The pent-up blood flows back through that aperture, and upon the veins of the system ; with the effect of distending those veins, and of giving proportionate relief to the blood gathered up in excess in the pulmonary vessels. At each contraction of the right ventricle, indeed, instead of the whole of the blood flowing forwards into the over-charged pulmonary

artery, a portion of it flows backwards into the right auricle, and venæ cavæ.

Inflammation of the left side of the heart, even when there is no regurgitation through the mitral orifice, impedes the flow of blood from the lungs into that side of the heart; and the accumulation of the blood in the pulmonary vessels, thus caused, induces and is relieved by the tricuspid regurgitation.

The tricuspid murmur was present on admission in two of the thirteen cases of endocarditis in which that murmur existed without mitral regurgitation. In nine of the remaining cases, the tricuspid murmur was not observed until from two to seven days after admission, and generally on the fourth or fifth day. In one case the murmur did not appear until the 26th day after admission.

In nine of these instances the duration of the illness before their admission is stated. In one of them the murmur appeared on the 7th day; in five, from the 10th to the 12th; and in two, from the 14th to the 16th day after the beginning of the attack of acute rheumatism; and we may therefore infer that the tricuspid murmur generally comes into play about the 10th or 12th day of the primary attack.

In four instances the murmur was preceded by a prolonged first sound over the right ventricle, and in one by a very loud, and in another by a peculiar booming first sound.

In five of the cases there was direct evidence of endocarditis at the time of admission, in the shape of pain in the heart, and a prolonged first sound; although the murmur did not pronounce itself fully until several days had elapsed. In two of them, indeed, the murmur did not appear until there was a marked improvement in the general symptoms.

The duration of the tricuspid murmur in these cases was very variable. In two instances it was only observed once,

and in eleven it disappeared in from two to nineteen days ; in eight the murmur when last noticed had become much more feeble, and in three of these the first sound became prolonged at the apex, at the time that the tricuspid murmur was diminishing. In three cases a pulmonic murmur, which indicates lessened tension of the pulmonary artery, appeared when the tricuspid murmur was lessening.

From these observations we are entitled, I consider, to infer :—1. That the appearance of a tricuspid murmur over the body of the heart, extending from the sternum to the nipple, and limited to that region, which corresponds to the right ventricle, is usually the effect and the evidence of endocarditis affecting the left side of the heart. 2. That when this murmur is neither coupled with nor replaced by a mitral murmur, we may safely foretell that when the inflammation leaves the heart, the valves will be perfect and the organ free from disease.

A tricuspid murmur, as I have already remarked, is often the prelude, and for a time the accompaniment, of mitral murmur in cases of rheumatic endocarditis. The latter murmur, however, in two-thirds of the cases (16 in 27) outlives the former, which is essentially a transient murmur. I have already given the proportion in which mitral regurgitation is accompanied, preceded, or followed by a tricuspid murmur (see p. 242).

The duration of the tricuspid murmur in these cases, in which it was associated with a mitral murmur, though variable, was usually short. In ten instances it was only heard once, and that generally on the day of admission, but in one half of these the existence of the murmur was doubtful ; in six cases it was audible for from two to seven days, and in seven from nine to sixteen days; while in three, after a short duration, it vanished and reappeared

after about twenty days, and in another case after a much longer period.

The tricuspid murmur appeared much earlier in a large proportion of those cases in which it was associated with mitral regurgitation than in those in which it was the only murmur audible. The murmur was present at the time of admission, or on the second day—in two-thirds of the cases (19 in 27), in which there was both a tricuspid and a mitral murmur, and in only one-sixth (2 in 13) of those in which the tricuspid murmur was alone audible. This contrast between the two sets of cases is more striking if we date the murmur from the beginning of the attack. The tricuspid murmur appeared on or before the eighth day in at least nine cases in which there was both tricuspid and mitral regurgitation ; and in one only in nine of the cases in which the tricuspid murmur was alone audible. In one of the cases in which both murmurs were in full play on the day of admission, the patient had been ill only two days, in two others four days, and in three others a week. These cases of combined mitral and tricuspid regurgitation, in respect to the more rapid development of the murmur, and not in that respect only, present greater intensity, energy, and rapidity of inflammation in the left cavities of the heart, than in the cases in which the tricuspid murmur was alone audible. In almost all the cases of tricuspid incompetence there was at the time of admission great general illness ; but this and other points of clinical interest must be reserved until mitral regurgitation is specially considered. In four of these cases the tricuspid murmur was replaced on its disappearance by a transient prolonged first sound over the right ventricle. The tricuspid regurgitation reappeared after being absent for a longer or shorter period in five of the patients. In four of these the renewed tricuspid murmur was conjoined with mitral murmur, but in the remaining

one it cropped up alone 47 days after it had disappeared, and 34 days after the cessation of the mitral murmur.

The tricuspid murmur is easily recognised by its position and character. It is distributed over the right ventricle from the sternum to the nipple and from the third cartilage to the sixth, it usually stops at the septum, occasionally extends over the right auricle, to the right of the lower sternum, and is sometimes audible over the epigastrium below the lower boundary of the heart. The tricuspid murmur is usually grave or even vibrating in tone, and superficial, and it begins with an accent or shock, and ends with the second sound.

In cases of extensive mitral regurgitation without tricuspid murmur, the first sound is feeble while the second is intensified over the pulmonary artery, owing to the tension of that artery, the second sound being usually loud over the right ventricle, and sometimes even at the apex.

When, however, mitral is coupled with tricuspid regurgitation, the blood is thrown back upon the right auricle and the venæ cavæ, the tension of the pulmonary artery is relieved, and the first sound over that artery is moderately loud, or prolonged, or even murmuring ; and the second sound, though perhaps rather loud, ceases to be intensified.

The mitral murmur is usually softer and less grave in tone than the tricuspid, being more like a bellows sound ; it appears also to be deeper ; and its point of greatest intensity is situated to the left of the nipple, and, in endocarditis, towards the axilla. When the mitral murmur is loud and vibrating, and especially if accompanied by a thrill over the apex, perceptible to the finger, it is heard very extensively, radiating in every direction. It then becomes audible over the right ventricle. This transmitted mitral murmur over that ventricle is readily distinguished from the tricuspid murmur originating within the right ventricle itself; for the

transmitted or mitral murmur is accompanied and more or less masked by the normal first sound of the right ventricle ; while the immediate or tricuspid murmur, besides being grave and shallow, begins with an accent, and is inseparably incorporated with the first sound of the right ventricle.

When the mitral and tricuspid murmurs coexist, it is usually easy to distinguish them from each other upon the principles just stated ; for the tricuspid murmur over the right ventricle is then palpably more superficial than the apex murmur, instead of being less so, as it is when the mitral is alone audible ; the first sound of the right ventricle does not mask or muffle the murmur ; and the difference in tone of the two murmurs is perceptible, the mitral being soft and smooth, the tricuspid grave or vibrating. Two cases were typical instances of this difference in tone of the two murmurs when thus coexisting ; in one of them the mitral murmur was a soft bellows sound, while the triscupid murmur was grave ; and in the other the tricuspid murmur was harsh and grating, while the mitral was soft. When the mitral murmur is rasping and vibrating in character, the difficulty of distinguishing the presence of a conjoint tricuspid murmur is increased. An instance of this was presented by a patient in whom the apex murmur was short and rasping, while there was a bellows-sound over the right ventricle. Here the rasping mitral murmur might have become softened by its transmission through that ventricle.

It is sometimes difficult to distinguish between a tricuspid murmur and a friction-sound, especially when the latter is murmur-like in character, as it frequently is at the beginning and towards the end of an attack of pericarditis. The chief points of distinction are—that the friction-sound is usually double or to-and-fro in character ; the tricuspid murmur being single : the friction-sound is not exactly rhythmical with

the heart-sounds, those sounds being readily heard distinct from the friction-sound when that sound is not loud and grating, so as to extinguish every other noise; the tricuspid murmur is incorporated with the heart-sounds; the friction-sound starts off without a shock, and retains the same tone throughout; the tricuspid murmur begins with an accent or shock. The pressure test usually clears up every doubt. When the stethoscope is applied over the right ventricle with increased force, the tricuspid murmur may be intensified, but is not materially changed in character; while the friction-sound is usually both intensified and changed in tone, it ceases to be murmuring, and becomes grazing, rubbing, grating, or creaking in character.

When pericarditis supervenes upon a tricuspid murmur, the pressure test is sometimes in the early stage almost essential to the discovery of the friction-sound; sometimes, however, the patient under these circumstances is so ill that you cannot make pressure. Local pain will then usually guide the treatment, and time will clear up the obscurity.

In five of my cases, aortic regurgitation was accompanied by a tricuspid murmur; and in two of these by a mitral murmur also.

Cases of endocarditis with aortic regurgitation present obstruction to the flow of blood through the lungs, and so may cause tension of the pulmonary artery and tricuspid regurgitation; more, however, owing to the inflammation of the interior of the left cavities and the mitral valve itself, than to the aortic regurgitation, which is rarely sufficient in volume to induce congestion in the lungs. This is shown by the clinical fact that there were four instances with tricuspid murmur in the sixteen cases of endocarditis in which there was recent aortic regurgitation, in seven of which there was mitral regurgitation also; while there was no instance of

tricuspid murmur in the fourteen cases of endocarditis in which there was aortic regurgitation owing to the previous disease of the valve, in one-half of which cases there was mitral regurgitation also.

A tricuspid murmur was present in three cases of endo-pericarditis; and in two of those cases the murmur was persistent; while in one of them it disappeared, after the recovery from acute rheumatism.

I will give here the proportion in which a tricuspid murmur was present in cases of acute rheumatism with endocarditis under my care from October, 1866, to 1869, treated by means of rest.

There were altogether 31 cases of endocarditis in a total of 74 of acute rheumatism, and in none of those thirty-one cases was one tricuspid murmur present without a mitral or other murmur.

While the tricuspid murmur unaccompanied by another murmur was absent in those cases; although it was present in the proportion of one in eight of such patients treated during the previous fifteen years; the proportion in which the conjoint tricuspid and mitral murmurs were present was fully maintained in the cases treated by rest. Mitral regurgitation was present without aortic regurgitation in twenty of those cases, and of these, tricuspid murmur was present in nine, or, if we add two doubtful cases, in eleven instances.

In none of these instances did the tricuspid murmur precede the mitral; in four the two murmurs appeared at the same time; in four the mitral preceded the tricuspid murmur by from one to three days, and in one (45) by nine days.

In three of these cases the mitral murmur outlived the tricuspid; in two it was the reverse; in three they were combined to the last, and in the remaining case the mitral murmur probably lasted beyond the tricuspid.

The relation of prolongation of the first sound over the right ventricle to tricuspid murmur will be considered at pages 258, 289.

II.—Cases of Rheumatic Endocarditis with a Mitral Murmur. *Symbol* →

The mitral and the tricuspid valves, while they correspond in general structure and function, differ essentially in the construction and arrangement of their flaps and in the whole setting of the valve.

The tricuspid valve, as I have already stated, is composed of three great flaps and several intervening small ones, which meet somewhere about the centre of the valve; and the aponeurotic ring which forms the base of those flaps is surrounded on all sides by muscular walls. (See figs. 1, 2, pp. 73, 74, vol. iii.; and figs. 13, 14, 15, pp. 96, 97, 99, vol. iii.)

In health, when the ventricle is not over-distended, the flaps of the valve adapt themselves to each other perfectly, and close the tricuspid aperture completely during the contraction of the ventricle.

When, however, the cavity is over-distended, as it is under the various circumstances which I have already described, the flaps of the valve adapt themselves only partially to each other, especially, so far as I have observed, at the meeting-point of the three great flaps, and regurgitation ensues. The so-called "safety-valve" function of the valve is thus brought into play, with the effect of relieving the tension of the vessels of the lungs, and throwing the blood backwards upon the veins of the system.

The result is that the tricuspid murmur is, with rare exceptions, not a sign of inflammation of that valve,

but of the over-distension of the right ventricle, caused by obstruction to the flow of blood through the lungs.

The mitral valve is formed of one great semilunar or convex flap, the base of which is incorporated with the powerful aponeurotic structure that is continuous with the two posterior sinuses of the aorta; and of a crescentic or horse-shoe flap, complex in structure, being formed of three segments, set in the muscular walls at the base of the left ventricle. The setting of the base of the valve is therefore two-thirds muscular and one-third aponeurotic. There is no tendency in the aperture to widen outwards at the base of the valve equally in all directions, for the aponeurotic structure, when healthy, though elastic, is practically unyielding. The single anterior semilunar flap, held in check by its proper chords and fleshy columns, fills up the posterior crescentic flap with perfect adaptation. The edges of the opposed flaps press against each other with increasing force in proportion to the increasing pressure of the blood on their under surfaces; and the over-distension of the left cavity does not, owing to the structure to which I have alluded, readily tend to widen the orifice and open up the valve. The healthy mitral valve, therefore, when the left ventricle is not greatly enlarged, possesses only under circumstances of extreme backward pressure or forward resistance a function like the "safety-valve" function of regurgitation with which the tricuspid valve is endowed. Such a function of the mitral valve would indeed be the opposite of a "safety" valve function, for it would immediately endanger the lungs by throwing the blood backwards upon their vessels. (See figs. 1, 2, pp. 73, 74, vol. iii.; and figs. 7–12, pp. 88–91, vol. iii.)

The result is that when the right ventricle is over-distended, it relieves itself backwards through the tricuspid aperture upon the veins of the system: and that when the

left ventricle is over-distended, it, with rare exceptions, relieves itself directly forwards upon the arteries of the system, and so the lungs are spared in both instances.

I derive the more important evidence of the correctness of this view from the well-understood pathological history of aortic regurgitation from widening of the orifice of the aorta, owing to atheroma of its walls. In those cases the cavity of the left ventricle becomes greatly, sometimes enormously, enlarged, and yet I know of comparatively few instances of this kind in which the mitral valve was therefore incompetent.

Mitral regurgitation, without disease of the structure of the valves, occurs most frequently among cases in which there is great arterial tension owing to Bright's disease, and great consequent distension of the left ventricle ; in which cases there is often also an atheromatous, or thickened state of the mitral valve, with, as an effect, widening of the fibrous portion of that aperture, and possible regurgitation.

Mitral murmur is, as a rule, neither a sign of over-distension of the left ventricle, nor of a supply of blood to that cavity too small in amount, or too thin in quality.

The existence then of a mitral murmur in a first attack of acute rheumatism is a direct sign of inflammation affecting the left side of the heart.

Mitral regurgitation, not connected with previous disease of the valve, and without aortic regurgitation, was present in 50 out of 107 cases of rheumatic endocarditis under my care in St. Mary's Hospital, from 1851 to 1866, and in 20 of 31 such cases treated by rest from 1866 to 1869. In twenty-five of the earlier series of cases the murmur had disappeared, and in ten others it was lessening at the time of the patient's recovery, while in fourteen of them the murmur seemed to be established ; and it was absent in one and present in the

other of two fatal cases of mitral endocarditis at the time of death. In the cases of the later series the corresponding numbers were thirteen, four, and three, the latter being the only cases in which the murmur was established at the time of the patient's recovery.

In one-half of the cases of both sets the mitral murmur was heard on the day of admission or the next day; the numbers being 28 in 50 of the first set, and 9 in 20 of the second set. The murmur presented itself within six days of admission in three-fourths of the remainder, or seventeen of the earlier and nine of the later series, and from 8 to 17 days after admission in the remaining cases, amounting to one-seventh of the whole.

Among the thirty-seven cases of endocarditis, combining the two series, admitted with mitral murmur, one-third, or eleven, had been ill from 2 to 7 days, nearly one-half, or fifteen, from 8 to 14 days, six from 2 to 4 weeks, two for a longer time, and three for an unknown period.

The mitral murmur became audible after admission in thirty-six cases, and of these the murmur appeared in six during the first 7 days, in eleven from 8 to 14 days, and in eight from 15 to 28 days after the beginning of the attack of acute rheumatism; in six at a later period; and in three at a time unknown.

The mitral murmur may be present in full force on the third day of the attack, or its appearance may be delayed until the fortieth day. In a fair proportion of the cases, amounting to one-fourth, it is developed during the first week, and in the larger number, or two-thirds, before the end of the second week.

General illness.—In nearly every case of endocarditis the patient presents great or considerable general illness. Thus in sixty-two of the seventy-one cases of mitral endocarditis the

illness was great or considerable, in two it was definite, and in five it was slight; while in two there is no description of the general state of the patient.

In most of the few exceptions to this rule of the presence of great general illness in these cases, the murmur was established at the time of their admission, and the severity of the attack was already mitigated or passing away.

Those cases in which there was no endocarditis present a very different aspect, since in scarcely one-third of them was there considerable general illness.

As might be expected, constitutional illness was more severe and frequent in those instances in which there was a threat of endocarditis, though its existence was not actually demonstrated by valvular incompetence, since in nearly two-thirds of them the general illness was either great or considerable.

The illness in cases of endocarditis is peculiar. It differs from and is superadded to that due to simple rheumatic inflammation of the joints, and is such as to call the attention of the physician to the state of the heart.

The face may be flushed all over, the forehead, nose, lips, and chin being of as high a colour as the cheeks, a state that is usually associated with profuse perspiration, drops of sweat standing in beads on the surface—a condition, however, that may be present in cases with severe affection of the joints without endocarditis. Thus when endocarditis exists the face loses the brightness, glow, and smoothness, and the variety of hue and tone of health, and becomes clouded, being dusky, dull, or ashy in hue, or glazed, or unduly white, or even of a bluish tint. The countenance, no longer expressive of interest in things and persons around, or even of pain in the limbs, is marked by internal trouble. The aspect of the patient is altered, often profoundly so, being anxious, depressed, or

indifferent. The eye loses its lustre and expression, and becomes heavy and dull.

Sleep is often absent, the nights being restless; but this is perhaps more often due to the inflammation of the joints than to that of the interior of the heart.

The nervous system is often gravely affected. Delirium at night, the patient wandering, muttering, and complaining, is occasional, but rare; it occurred in two instances, in which the affection of the heart was evidently the primary exciting cause of the mental trouble. In another patient the head was confused on the third day.

Choreal movements, as we have seen, are in some instances a definite effect of endocarditis, especially of the non-rheumatic kind, traceable frequently to cerebral embolism; but choreal movements, and indeed embolism, were of very rare occurrence in my cases of rheumatic endocarditis uncomplicated with pericarditis. In one instance the patient, previously anxious, and with sordes on his teeth, was nervous and fidgetty; and in another, starting appeared on the sixth day, having been preceded on the fourth day by pain in the heart.

Sickness is occasionally present. It was so in four of my cases. These cases, however, point not to the stomach as the cause of sickness, but rather and usually to the state of the nervous system, and more immediately to that of the brain itself; as in a case in which giddiness and sickness appeared together, and in another in which sickness was preceded by restlessness.

Failure in the power of the heart is an occasional occurrence in cases of endocarditis. Thus, two of my patients were attacked with fainting. One of these fainted on the day of admission, and again on the thirteenth, and on the following day was sick, so that failure of the heart may be a cause

of sickness. In the other case pain in the heart and fainting appeared on the seventeenth day after admission. We may fairly attribute the fainting in these cases to the actual failure of the heart itself, caused by the internal inflammation of that organ.

The pulse is often quick, feeble, and fluctuating. I believe that it is dicrotous, but I have not employed the sphygmograph in any case of endocarditis, being perhaps deterred by the state of the wrist.

Perspiration is often especially profuse and of long continuance; sudamina being also present in some of the more severe cases.

The breathing is usually affected, being more or less quickened. In rare instances pulmonary apoplexy or extravasation is the result of the difficulty to the flow of blood through the lungs, which is the general effect, varying in degree, of endocarditis.

The chain of symptoms here described points mainly to the affection of two great functions. The nervous power is lowered; and the circulation of the blood through the fine vessels of the lungs and the body is enfeebled.

Pain in the Region of the Heart.—Pain in the region of the heart, sometimes severe and lasting, sometimes slight or transient, amounting perhaps only to uneasiness, was present in about one-fourth of the cases of tricuspid and of mitral murmur belonging to the earlier series, and in one-half of the later series, treated by rest. If to these we add other cases having mitral or tricuspid murmur in which there was pain in the left side, or in the chest; the proportion thus affected reaches to nearly one-half in the first series, and to fully one-half in the second.

The pain in the heart was sometimes, but not generally, severe. In a few instances the pain was increased or excited

by pressure. We may fairly infer that in those cases pericarditis was imminent or was actually present, though not, except in rare instances, with such intensity as to cause even a transient friction-sound.

Palpitation was very rarely complained of, but fainting, as I have already stated, occurred in two instances.

Prolongation of the First Sound occurring during the Early Period of Mitral Endocarditis.—In one-half of my patients affected with mitral regurgitation, as we have just seen, a murmur was established at the time of admission. In one-half of the cases in which the murmur was not thus established, prolongation of the first sound preceded, and was merged into, the murmur. In all but one of those cases the first sound was prolonged at the time of admission, and in that case and two others a tricuspid murmur was then in full play.

The tricuspid murmur was likewise heralded by prolongation of the first sound in one-half of the cases in which that murmur was not already present at the time of admission.

In a number of the cases, the exact position of the prolongation of the first sound was not defined; but wherever it was so, the mitral murmur was preceded by prolongation of the first sound at the apex; and the tricuspid murmur by prolongation of the first sound over the front of the heart, or the right ventricle.

I think that no cardiac sign is more readily recognised than prolongation of the first sound, and yet there is none so difficult to define. That this is so, however, is natural, for it is a transition sound. It forms, as we have just seen, the transition from a clear healthy first sound to a murmur; and as we shall see, at a later period, in a large proportion of the cases, it forms a transition between a mitral or tricuspid murmur when dying out, and the restoration of the healthy first sound. In one-half of the cases in which the prolongation

preceded the murmur, there was a double transition, the murmur being both preceded and followed by prolongation of the first sound. This prolongation is sometimes so like a murmur that it is difficult to make the distinction, and this is especially the case just before the time of transition, when the prolongation precedes the murmur; and just after that time, when it follows the murmur.

Prolongation of the first sound is the absence of silence and the presence of the wavering, grave, feeble sound during the interval between the first and second sounds. It is not the prolongation of the shock of the first sound which is itself significant, being sometimes a precursor of the more telling signs of endocarditis. The prolongation of the first sound is not the same as the natural loud vibrating character of that sound over the superficial cardiac region which is almost always present in cases of anæmia, when the muscular force of the ventricles is maintained, and even in excess, but when the blood is scanty and thin, being deficient in red corpuscles.

Prolongation of the first sound is, I repeat, a feeble, indeterminate, wavering sound, that fills up the space between the first and second sounds, which space is silent in health. It presents every gradation, from a sound so feeble that it is with difficulty discovered, to a sound so murmurlike that it can scarcely be distinguished from the murmur into which it so often ripens. Prolongation of the first sound was noticed on the first day of observation in fourteen cases; the prolongation developed into a murmur in two-thirds or nine of those cases before the seventh day after admission; and in the remaining third, or five, between the seventh and fourteenth days. In two other instances the prolongation, absent on the day of admission, appeared on the following day, and in the other after a lapse of four days.

It is evident that in all these cases the endocarditis was present before the appearance of the murmur for a period of time at least as long as the previous period of duration of the prolongation of the first sound.

There are other modifications of the first sound, besides its prolongation, that point to endocarditis, if they do not indicate it, which have been, in a few instances, the precursors of murmur. It will be sufficient if I simply name them. They are—1. Loud heart sounds, the first being sharp, the second ringing; or both sounds may be ringing. 2. Healthy sounds with powerful action of the heart. 3. Roughness of the first sound. 4. A humming noise over the right ventricle, and in one case at the apex, where it was associated with murmur. 5. Doubling of the first sound (over the ventricle), which occurred in two cases associated with a prolonged first sound, which was not followed by a murmur in one of those cases. 6. Feeble first, loud second sound, followed by tumultuous action of the heart and mitral and aortic murmurs. 7. Extensive presystolic murmur (*rrrp*) present in one case for five days, followed in succession by loud heart sounds (6th day), doubling of the second sound (15th day), and a faint mitral murmur, not limited to the apex. 8. Loud "plunging" first sound over both ventricles, present on the 4th day, followed by prolongation of the first sound on the 6th, and mitral murmur on the 8th; and 9. muffling of the first sound, which in one case succeeded the murmur, which was extinguished by an attack of pain in the heart, followed by fainting.

All the above varieties in character of the first sound were, in the instances referred to, followed within a very few days by a mitral murmur.

The only one of these varieties of the first sound that I would speak of is the last: the peculiar "plunging" sound. I call it so for want of a better name. The sound is some-

thing like what I have heard in the working of a steam-engine. It was as if the piston made a peculiar plunging sound when it dipped down and reached the bottom of its play. I have heard this sound in at least three cases. One of them was attacked afterwards with delirium, long torpor, almost coma, extreme depression, and pericarditis, but no murmur. In all the cases, the constitutional symptoms more or less threatened endocarditis.

Besides these peculiarities of the first sound preceding mitral murmur, there is one other affection of the sounds of the heart that I would name; and that is a complete silence of both sounds; which occurred in one case threatened with endocarditis, in which a mitral murmur did not appear. In that case there was tenderness over the heart, fighting for breath, a piercing pain between the chest and back, and great depression, lasting for some days. On the 8th day she looked more bright, on the 9th the sounds of the heart were audible, on the 14th its impulse had returned and was gaining power, and on the 26th day the sounds were of natural loudness, and there was no murmur.

In most of the cases of endocarditis with mitral murmur there is undue, but not great, strength of the impulse of the right ventricle, which may be seen and felt between the cardiac cartilages to the left of the lower sternum. This is found even in the earlier stages, and before the appearance of the mitral murmur.

It is evident from what has just been stated, that while in some cases that murmur bursts into full play at the commencement of the attack, being audible on admission, and on the 3rd, 4th, 5th, 6th, or 7th days after the seizure; in others it is not audible until a period varying from the 8th to the 30th day, although there is unequivocal evidence that the inflammation in the left side of the heart was present before and at

the time of admission. This evidence consists in the existence of a tricuspid murmur, or a prolonged first sound, or pain in the region of the heart or in the chest, with great or considerable general illness.

The inflammation of the valve cannot cause regurgitation until perfect adaptation is prevented by the formation of small prominences, covered with a deposit of fibrin upon the surfaces or lines of contact of the margins of the valve, or by the softening and yielding of its flaps.

In three of the cases tricuspid or mitral murmur became audible after admission, when the patient's illness increased. In ten other cases, however, it was the reverse, for in all of them the murmur came into play when the patient's health began to improve.

We are therefore, I conceive, warranted in assuming that in a considerable number of the cases, the active stage of the endocarditis is passing away at the time of the appearance of the murmur.

Progress of Cases of Endocarditis with a Mitral Murmur.— Cases with a mitral murmur from endocarditis affecting a valve previously healthy, may usually be readily distinguished from those in which the murmur is due to established disease of the mitral valve by the character, seat, and area of the murmur, its changes, duration, and transition, its cessation or establishment; by the size of the heart and the force, extent, and position of its impulse; and by the nature of the first and second sounds over the right ventricle, the pulmonary artery, the aorta and great arteries in the neck. The mitral murmur is always situated over the apex and body of the left ventricle, and the ventricular septum. The centre of the murmur and its point of greatest intensity and purity is usually just below the left nipple. Sometimes it is limited to this point, but in

general it covers a larger area, spreading inwards towards the right ventricle, outwards and upwards towards the axilla and over the lung, and downwards over the stomach. This area is rarely extensive, being usually limited by a diameter of from two to three inches.

When the heart is high, owing to the elevation of the diaphragm, and when the left ventricle is exposed in consequence of the shrinking of the overlapping portion of the left lung, the murmur extends upwards towards the axilla, and even above the mamma, and a little outwards, rather than downwards. The direction of the murmur upwards towards the axilla is peculiar to the mitral murmur of endocarditis, for when disease of the valve is established, the lungs expand downwards to an unusual extent, and so muffle or arrest the murmur in its course towards the axilla.

The extent of the area of the murmur depends much upon its character. A smooth, soft, bellows-murmur, especially if it is rather feeble, is in general limited to the apex and left ventricle; so also is a weak, grave murmur. But when it is vibrating, loud and almost musical, and especially if a thrill is felt by the finger over the apex—then the area of the murmur is extensive. Sometimes, indeed, it is so all-pervading that it may be heard over the whole cage of the chest, front and back, and even upwards into the neck and downwards over the abdomen.

It is only in established mitral disease, or in very rare cases of endocarditis with extensive mischief to the valve, that we find this pervading vibrating murmur with perceptible thrill.

In cases of established mitral disease the murmur is usually audible to a greater or less extent over the region of the stomach, often coming quite down to its lower boundary. The vibration in the left ventricle, which rests immediately upon the stomach, the diaphragm alone interposing, awakens

a corresponding vibration in the stomach, and as this takes place in a hollow sac, its tone is often metallic, and it thus sometimes imparts a musical character to the murmur at the apex.

In cases of endocarditis with mitral regurgitation, the murmur is often so feeble that it is limited to its birthplace, and is unable to generate corresponding vibrations in the adjoining organs. In these patients the murmur is inaudible over the stomach ; but in other cases of endocarditis, according to the loudness and penetrating quality of the tone, the murmur makes itself heard over a greater or less portion of the stomach, at that part of it nearest to the apex of the heart.

The murmur was heard over the lower part of the back of the chest in only two of the fifty cases of endocarditis with mitral murmur of the first series, and in one of the twenty cases of the second series. In one of these cases the murmur was audible over the lower part of the back, the lungs being condensed, on the 4th day, but it was not again heard in that position. In another such case the murmur was heard over the back of the chest from the 27th to the 34th days after admission, but ceased to be so on the 30th ; and in the third case the murmur was heard below the shoulder blades for the first time on the 18th, and for the last time on the 42nd day. After that date the murmur was less loud, and its area was correspondingly lessened.

I have to add to these, one case of death with inflammation of the mitral valve ; the anterior flap was softened and enlarged, its edge and that of the posterior flap were covered with lymph or fibrine, and the valve permitted extensive regurgitation through the mitral aperture. The patient, a young man previously in good health, had been ill a fortnight with acute rheumatism ; when admitted, he had an anxious

expression, hurried and difficult breathing, and sickness. A loud mitral murmur, beginning with a sharp shock and followed by the second sound, extended forwards almost to the sternum, where the heart sounds were healthy, and backwards to below both shoulder blades. From the 9th day to the 11th he raised phlegm tinted with blood, he was propped up in bed, and there was dulness and fine crepitation over the left lower lobe. On the 14th he sat forward in bed in great distress, breathing with difficulty. In the course of that day he died, and on dissection he presented the inflammation of the mitral valve and the extensive pulmonary apoplexy that were evidenced during life.

The patients usually lay flat in bed, their pain being increased by movement, and as the back was not examined, some of these might have presented a murmur over the lower lobes of the lungs behind; but when we regard the limited area over which the murmur was usually heard in front and at the side, it is evident that it could scarcely have been audible behind. I think it probable that three cases, in addition to those just named, may have been exceptions to this rule, and perhaps two others, for in them the murmur was loud, while in the first three it was vibrating in tone.

The mitral murmur at the time of its first appearance, or of its transition from prolongation of the first sound, is as a rule either weak and grave; or it is a soft, feeble, bellows-murmur, and is therefore limited in area.

The mitral murmur invariably begins with an accent or shock, which corresponds with the shock of the impulse, and it generally ends with the second sound. It fills up, in fact, the space between the first and second sounds, that space being often lengthened so as to admit of greater prolongation of the murmur, with the effect of altering the rhythm of the heart. Sometimes the murmur does not quite fill up this

space, so that there is a distinct silent pause between the end of the murmur and the second sound. The presence of the accent or shock at the beginning of the first sound distinguishes an endocardial murmur from an exocardial or friction murmur.

The pressure test comes in to settle the difficulty of distinguishing one condition from the other. If the noise be endocardial, the sound may become louder from the closer application of the stethoscope, when pressed upon the walls of the chest ; but the quality of the noise is unaltered, it is rhythmical with the heart sounds, it retains its accent or shock, it fills up the space between the first and second sounds, and it ends exactly with the second sound.

But if the noise be frictional, it usually loses its murmur-like tone when the pressure is made—and becomes rustling or grazing, grating or creaking in character ; it extinguishes the first and second sounds of the heart, which were previously heard side by side, but not incorporated with the murmur ; it brings out a double sound where there was but a single one before, a sound to-and-fro in character, or a noise not unlike that made by the sharpening of a scythe, with a single down-stroke during the beat of the heart, and a double up-stroke during its pause. Sometimes the mitral murmur is masked or confused at the apex by the co-existence of a vibrating systolic noise. The interposition of a piece of paper or cloth between the stethoscope and the surface of the chest annihilates this vibrating noise, and the mitral murmur is then heard with perfect purity and clearness. The interposition of the lung effects the same end—for this vibratory noise is heard only where the heart is in direct contact with the walls of the chest; and hence, when using the naked stethoscope, we meet with cases in which the murmur is more smooth and bellows-like just to the left of the apex or towards the axilla,

than it is over the apex itself. For this effect, however, the layer of lung must be thin and the tone of the murmur must be penetrating. In cases of endocarditis with mitral regurgitation, the murmur is often muffled by a rumble, or a comparatively feeble vibration. The interposed paper or the intervening lung extinguishes this vibrating noise, and brings a pure, soft, bellows-murmur into play.

The changes that the mitral murmur of endocarditis undergoes during the progress of the case are remarkable, and they vary in almost every instance. These changes consist in alterations of its tone, loudness, and area ; in its transition from a true murmur to prolongation of the first sound ; in the substitution of a tricuspid for a mitral murmur, or the reverse, or the companionship of the two murmurs ; in the suppression and reawakening of the murmur ; and frequently in its final extinction, either directly or by passing again into prolongation of the first sound, which precedes the restoration of the healthy sounds of the heart.

In one-fourth of the cases (18 in 70) the mitral murmur was only heard on one occasion.

Of 50 cases, in all of which the mitral murmur was heard more than once, that murmur was of equal loudness during the successive observations in one-fifth (11) ; became gradually weaker in one-third (17), but in six of these it passed through a double oscillation and increased and lessened a second time ; became gradually stronger in one-fifth of the cases (11), in one-half of which it again gradually declined ; was suspended and then renewed for a time in one-fourth of the cases (12), when the murmur again faded away; and it sometimes yielded to the healthy sounds of the heart, and sometimes to prolongation of the first sound. In two instances, already included in the abstract just given, there was a double disappearance and reawakening of the mitral murmur, which in

one of them met with final extinction, while in the other it became established.

The changes in the area of the murmur corresponded in a considerable degree to the changes in its loudness, the former widening as the latter increased and narrowing as it diminished.

In the great majority of the cases, and especially in those in which the murmur disappeared, the tone of the murmur underwent but little change. It became progressively louder and feebler, more clear and more obscure in almost every instance, but it usually retained its original character.

The murmur was observed to be soft and smooth, approaching to the character of a bellows sound, in less than one-half of the first series of the cases of endocarditis with mitral regurgitation, and in less than one-third of the second series; the cases in each series in which the murmur was not characterised amounting to fully one-third of the whole.

In a small proportion of the first series and a large proportion of the second series of cases, the murmur was grave in character, being in some of them feeble, and in a few, loud and almost vibrating.

Musical, sawing, and rasping murmurs formed but a small proportion of the total number of cases, and these were they that passed through a series of changes in tone and character.

One case, a youth, was a notable and rare instance of the variety of changes in tone through which the mitral murmur may pass. He had been ill a fortnight and had suffered from pain in the heart. On admission he presented a tricuspid murmur. To this a loud mitral murmur was added on the 3rd day, when he was very ill. On the 8th he was better, and from that day to the 15th the murmur was weak, soft, and smooth. On the 21st it was louder

and on the 29th it altogether changed its tone and became musical. After this, without apparent cause, it underwent two variations, having first the character of a sawing and then of a bellows sound. The tone of the murmur then again altered, and it became grave, and finally on the 52nd day it had regained its lost musical character.

We must now answer the important practical questions suggested by these observations, what are the character and progress of the murmur, when the attack tends to end in perfect restoration of the efficiency of the valve? And what, when it tends to become permanently incompetent, owing to the establishment of mitral disease?

The answer may be already almost gathered from what has gone before. When the murmur is permanently feeble, soft, and smooth, with an approach to, or even the formation of, a gentle bellows sound; or when it is feeble and grave, the complete restoration of the efficiency of the valve may be anticipated. In illustration of this statement we find that the murmur was feeble, soft, and approaching to a bellows sound in 14 of the 25 cases of the first series that ended in recovery of the valve; and in 4 of the ten that left with a lessening murmur, the corresponding number in the two like classes of cases of the second series being 5 in 17, while of the 17 cases that ended in established valve disease out of a total of 71, the murmur was feeble in none, and was smooth or soft in 6, most of which presented a definite bellows-murmur.

The feeble grave murmur was more frequently developed in the later than in the ealier series of cases, but in both its presence was almost always followed by the restoration of the function of the valve.

When the loudness of the murmur steadily diminished, or when it first rose and then fell, or when after disappearing it

reappeared and again faded away, the integrity of the valve was generally regained.

When the murmur was loud, its area being extensive ; when it presented a sharply-defined loud, bellows, musical, sawing, or rasping sound ; when it was vibrating in tone ; when it steadily increased in loudness, or only slightly rose and fell to rise and fall again, without a temporary disappearance ; then valvular disease was, as a rule, though not invariably, permanently established. One patient, a nurse in the Hospital, left with a loud mitral murmur, but after a time, when she resumed her work, the murmur had given place to healthy heart sounds.

Condition of the Heart and the Great Vessels in Cases of Endocarditis affecting the Mitral Valve.—In these cases there are, as I have already illustrated, many affections of the heart besides imperfection of the mitral valve with its attendant murmur. When inflammation affects the great central cavity of the heart, the pivot of its action, the whole organ is involved, and every part of it becomes, in succession, modified in its action ; and in the force, movement, and sounds by which it makes that action known.

Inflammation of the fibrous structure of the left side of the heart is as essentially a part of acute rheumatism, as is inflammation of the fibrous structure of the joints. The inflammation may commence in the heart at the same time that it commences in the limbs. It attacks that part of the heart that is working with the greatest force, just as it attacks those parts of the limbs that are subjected to the greatest labour. The increasing inflammation of the joints calls forth increasing force in the action of the left ventricle, and so stirs up and adds to the inflammation that may have already existed in that cavity from the commencement of the attack.

This inflammation of the ventricle, like the inflammation of

every other organ, lessens the power of the muscular cavity to expel its contents, and to propel the blood round the vessels of the system. This imperfect transmission of blood to the system, the demand for which is increased by the inflammation in the limbs, causes distension of the left auricle, and impedes the transmission of the blood through the lungs. This induces distension of the pulmonary artery and its branches, with, as its effects, accentuation—or loudness and sharpness, or shock—of the second sound, with relative feebleness, or even absence, of the first sound over that artery; and distension of the right ventricle, with increase in the action of its walls and in the force and extent of its impulse.

We have, thus, two ventricles beating side by side, the left one, the seat of the inflammation, beating with lessened power; the right one, with increased force.

The increased fulness and force of the right ventricle tend, when they pass certain limits, to reverse the flow of a portion of the blood, and to send it from the right ventricle back into the right auricle; with the effect of relieving the distension of the arteries of the lungs, increasing the fulness of the veins of the system, and producing a tricuspid murmur.

After a time, the whole volume of the blood is diminished, and the proportion of its red corpuscles is lessened; and then appear as later and secondary effects, a murmur over the pulmonary artery, and sometimes a murmur over the aorta and its great branches—murmurs that are due to the lessening of the contents, and relaxation of the walls of those vessels.

Such murmurs in the great arteries appear, however, also in the early stages of the affection, in the aorta more frequently, owing evidently to the lessened power of the inflamed left ventricle, and the diminished supply of blood

that is therefore sent into the aorta, the walls of which are thus relaxed; and in the pulmonary artery occasionally, for reasons that have yet to be ascertained.

The close study of the condition of the heart and great vessels generally throws more light upon the degree of the inflammation of the heart, and its effect on the vital powers of the organ, than does the simple observation of the mitral murmur.

I shall now rapidly review the conditions of the heart and great vessels as they presented themselves in the cases of endocarditis with incompetence of the mitral valve—that valve being previously in the virgin state and uninjured.

The Impulse of the Heart.—I find that I have taken notes of the state of the impulse in one-half of the cases with mitral incompetence, or in 25 out of 50 of the first series, and 9 out of 20 of the second.

The beat of the heart was, as a rule, not extensive or strong. It showed itself rather in the higher than the lower cardiac intercostal spaces, being present in only one instance below the fifth space, less frequently in that space than in the fourth, and sometimes even in the third space. While the impulse at the apex was in general feeble or absent; that of the right ventricle, though rarely powerful, was usually somewhat increased in strength, being present in the third and fourth, and even the fifth spaces between the cartilages. This impulse of the right ventricle was not as a rule marked or strong, but it could be felt diffused over those spaces when the ball of the palm of the hand was applied over them, or when the fingers were pressed gently into the spaces.

In a few instances the action of the heart, and especially the impulse of the right ventricle, was strong and diffused or powerful, or even tumultuous and violent, soon after admission; and then the size of the heart, which was not in general

notably affected, became enlarged, the chest over the cardiac region being more prominent than over the corresponding space on the right side.

In one or two patients the impulse presented a peculiar shock.

But the distinctive feature with regard to the impulse in a fair proportion of the cases was its variation during the successive periods of the disease. Thus, in one instance, it was feeble on the 1st day in the fourth space, very strong on the 3rd day, moderate in strength in the fifth space on the 8th day, and in the third and fourth spaces on the 12th day. In another patient the impulse was felt in the second and third spaces on the 2nd day, when there was pain in the heart; on the 5th, the pain still continuing, the heart beat violently; from the 6th to the 18th the pulsation was strong in the second space, and from the 28th to the 34th it was diffused from the third to the fifth spaces. In this case mitral disease was established, and the gradual extension of the impulse of the right ventricle told with precision the story of the increasing valvular disease in the left ventricle.

The study of the impulse conveys the most important lesson in all cases of endocarditis. Its absence may tell of the want of vital power; and its excess in the right ventricle, while it is wanting in the left, shows lessened power from inflammation in the latter cavity, and consequent increased labour in the former. Its gradual increase in force, and enlargement in area, with persistence of mitral murmur towards the period of the termination of the attack of endocarditis, and after its cessation, mark advancing and established valvular disease: and its extent and force point out the amount of the back-flow of blood from the left ventricle into its auricle, and the obstacle to the on-flow of blood through the lungs induced thereby. The impulse of the right ventricle

is, in short, a measure of the extent of the injury to the mitral valve, and of the consequent resistance to the circulation through the lungs.

The impulse of the right ventricle was diffused and strong, extending out to the nipple, in a considerable proportion of the cases in which there was a tricuspid murmur.

In a few instances the impulse of the right ventricle was so high as to be present in the second space ; but generally the pulsation felt in that space was due to the presence there of the distended pulmonary artery, when that pulsation was double, the second impulse being more smart and shock-like than the first. In these cases the pulmonary artery was distended, the first sound was feeble or absent, while the second was unusually loud and strong, penetrating the ear with a shock.

The apex beat is, in cases of endocarditis with mitral regurgitation, usually slight, sometimes absent—during the early period, before the mitral murmur is developed, owing to the weakened muscular power of the inflamed left ventricle ; and after the appearance of the murmur, owing to the relief afforded to the organ by the greater ease with which its surcharge of blood is sent backwards into the auricle than forwards into the aorta.

There are, however, certain exceptional cases of great interest, several of which have come under my observation, in which the left ventricle beats with great force, and unduly to the left.

In three of these cases there was extensive pulmonary apoplexy, or pneumonia of that type, in the lower portion of the left lung.

One was a youth, with hurried and difficult breathing, tinted phlegm, and dulness over the lower portion of the left lung, which was solid and lessened in size owing to pulmonary

apoplexy. The condensed and solidified lung shrank away from its natural position between the walls of the chest and the apex of the heart; and the apex was therefore completely exposed, beating with all its force upon the fifth space more than an inch beyond the left nipple. At that time there was no mitral murmur, but as soon as the lung began to recover itself, the murmur came into full play. When the lung again expanded, it covered the apex of the heart, and its beat was no longer perceptible. The whole heart in this case was displaced to the left; and its displacement was still greater in the sister case, in which the apex beat was situated three inches beyond the nipple line; the impulse of the right ventricle was placed to the left of the costal cartilages; and the double pulsation of the pulmonary artery, with a strong second shock, was present in the second space above the mamma.

A fourth case, when admitted, had pain in the region of the heart, and the apex beat was situated an inch and a half to the left of the nipple. Five days later the extreme limit of the impulse had shrunk one inch, being seated half an inch to the left of the nipple.

Accentuation of the Second Sound, with Silence, Feebleness, or Prolongation of the First Sound over the Pulmonary Artery.—Accentuation of the second sound over the pulmonary artery, in the left second space, is a well established sign attendant upon mitral regurgitation, and it may be present in every degree.

The second sound may be more or less loud and sharp or ringing—or it may penetrate and strike the ear with a loud shock; when a double impulse is to be felt over the pulmonary artery, the first being gentle and gradual, while the second gives to the hand a smart shock.

This increase in loudness and sharpness of the second sound is due to distension of the pulmonary artery, owing to

the difficulty with which the blood travels through the vessels of the lungs.

Whenever the blood thus accumulates in the lungs, whatever be the cause, the same effect is induced. In cases of phthisis, and notably when there is hæmorrhage from the lung and shrinking of its tissue, the pulmonary artery, enlarged and tense, displaces the lung superficial to it, and presses against the second space; where there is a double impulse, the first gentle, the second felt and heard as a shock. In bronchitis, emphysema and pneumonia, there is the same distension of the pulmonary artery, but greater in degree. The interposition of the lung, enlarged owing to the disease, screens the pulmonary artery from the hand and the ear, so that over it the second sound is often not unduly loud; but it is so in some instances over the right ventricle.

Whenever the tension of the pulmonary artery is thus so great as to cause a strong and loud shock with the second sound, the first sound is either almost silent, or feeble, or faintly prolonged.

When the blood is sent into a tight and full artery, it makes but little, often no sound, either in the shape of shock or murmur; but the second sound caused by the smart and strong reflux of the blood upon the walls and closed valves of the artery, makes a loud, sometimes a ringing or metallic sound. The same occurs in the aorta when it is enlarged and rendered tense owing to the difficulty with which the blood leaves the arterial system in advanced cases of contracted granular kidney. When you listen over the aorta a single sound is often heard, a loud ringing metallic second sound, the first being almost or absolutely silent. Sometimes in these cases the artery is so large and tense that it presses against the second right intercostal space, producing there a

double pulsation, the first gentle and gradual, the second smart and with a shock.

I find that I have described the second sound as being loud or sharp or ringing in about one-half of the 50 cases of the first series and 9 of the 20 of the second series of cases of endocarditis with mitral murmur, and in 5 of 13 of those of the first series with an uncomplicated tricuspid murmur. This does not of course include all of this class.

It was noticed that the second sound was sharp or loud in the early period in a large proportion of the cases in which that sign was observed, or in 13 out of 25 of the first series, and 7 out of 9 of the second series.

In all but six of the patients in whom it was noticed that the second sound was intensified, it continued to be loud down to a late period, to the time in fact of approaching recovery.

Loudness of the second sound may be associated with each of the signs, singly or in combination, that are habitually found in cases of endocarditis with inflammation of the mitral valve. It accompanied a mitral murmur either alone or in combination with a tricuspid murmur in 22 of the cases ; in about 15 cases it was allied with prolongation of the first sound over the left and sometimes the right ventricle ; and in 8 cases it was joined to tricuspid regurgitation, which was however combined with other important signs in every instance but one. The first sound of the pulmonary artery was affected, when the second sound over that artery was loud or sharp, on ten occasions, in different patients: in 4 of these there was a pulmonic murmur, in 4 the first sound was prolonged, being generally free from shock, and in 2 it was silent or scarcely audible.

These numbers, however, taken by themselves give a very inadequate idea of the relation of the first to the second sound of the pulmonary artery in cases when that second

sound is intensified. Thus, as we have just seen, pulmonic murmur was followed by a sharp second sound in four instances, but there were altogether 32 cases in which a pulmonic murmur was heard, and in only four of them was it stated that the second sound was thus affected at the time when the pulmonic murmur was audible. In one of the cases in which there was a pulmonic murmur on admission, the second sound was free from accent; while on the 3rd when the pulmonic murmur had disappeared, that sound was slightly accentuated over the pulmonary artery. Again, in only two of the cases is it noted that the first sound of the pulmonary artery was silent or scarcely audible when the second sound was loud. Since, however, my attention has been drawn to the relation of the first to the second sound of the pulmonary artery, in every instance that I have observed accentuation of the second sound, especially with, but even without shock, the first sound has been either very feeble, being occasionally prolonged, or almost or even quite silent. This condition was signally marked in a case of chorea under my care in the hospital, a boy, who on admission, presented no mitral or other murmur over the heart. After gaining ground steadily he became rather worse, his temperature rose, he had pain in his chest, and the second sound was loud, the first feeble over the pulmonary artery; and six days later a mitral murmur came into play. At the same time the right ventricle, previously quiet, beat with great force, and a strong shock was felt over the pulmonary artery with the second sound. On listening over that vessel, a loud second sound penetrated the ear and struck it as it were with a shock, and the first sound was silent, the second sound being alone audible to all who listened. After a short time he became very ill, and for two days he passed his evacuations involuntarily in bed. He kept both hands fixed on his wrists, and

his fingers on his hands. He soon began to improve, and gradually as this boy gained strength, speech, power to move, and freedom from irregular movements; and as his lungs enlarged, the mitral murmur being still audible, the second sound though still loud lost its shock, the second impulse ceased to be felt over the pulmonary artery, and the first sound, though feeble, become more and more audible.

In a fair proportion of the cases in which the second sound was sharp and loud at the early period of the disease, that sound retained its character unaltered through all the surrounding changes in the sounds of the heart. Let us take one case. At first there was a tricuspid murmur, the second sound being sharp; on the 6th day there was a mitral murmur, and the second sound was loud; next day the murmur was less marked, but the second sound was still loud; and on the 11th the murmur had given place to prolongation of the first sound over the right ventricle, and yet the second sound still remained loud. In another instance on the 9th day there was an obscure mitral murmur, on the 16th there was mitral, tricuspid and direct aortic murmurs, on the 19th these had all vanished, and on the 23rd the tricuspid and direct aortic murmur had returned; and yet on each occasion there was the same sharp second sound over the pulmonary artery. I could give several instances of this kind and would refer to four cases. In these instances the sharp second sound went on drumming, like the tom-tom in the streets, whatever was the variety of the surrounding noise, or even when there was freedom from murmur or prolongation of the first sound.

The intensified second sound is, however, by no means always so unvarying in its note. Thus, in one very interesting case on the 11th day the second sound was very loud over the pulmonary artery, the first being scarcely

audible; on the 34th both sounds were loud over the ventricles, the second being very loud; the next day all the sounds were natural.

I must refer to one other case, in which on admission the first sound was faint, the second loud over the pulmonary artery, the first sound being prolonged over the ventricles; on the 13th day the two sounds were equal over the artery and there was a feeble murmur at the apex; on the 27th the second sound was again louder than the first; and on the 40th a singular change took place, the first sound being louder than the second over the pulmonary artery—while over the aorta it was the reverse, and on the 50th day the natural standard was regained, the second sound being louder than the first.

The close study of the second sound and of its relation to the first over the pulmonary artery, is of practical importance in cases of endocarditis affecting the mitral valve. It may foretell the coming murmur in the early, and betray the recently extinct murmur in the later, period of the disease; and during its progress, it points by the degree and force of its accent to the amount of the resistance to the pulmonary circulation, the intensity of the internal inflammation of the ventricle, and the extent to which the function of the ventricle is impaired. It is, in short, a tell-tale sound pointing to the agency in the central cavity of the heart which gives it birth. The intensified second sound of the pulmonary artery, or that of the aorta, is associated as we have seen with a corresponding feebleness, or even silence, of the first sound of each of the vessels respectively. The observation of the one sound demands a corresponding observation of the other sound. When the artery is distended, it enlarges, lengthens, and advances, and comes gradually into contact with the second intercostal space, displacing the intervening lung from

before it. You can then feel the double pulsation of the great artery; the first movement is gentle, gradual, barely perceptible to the touch; the second strikes the walls of the chest and the applied hand with a sudden smart shock or tap. When you listen to it the ear takes in the same effect through another sense; the first sound is in extreme cases silent, or is soft and gentle, feeble and perhaps somewhat prolonged; while the second penetrates and strikes the ear with a loud shock, often ringing and metallic. Over the pulmonary artery, as I have just said, that subdued sound or even silence, and this shock, betoken tension of the artery, and obstacle to the flow of blood through the vessels of the lungs; whether that obstacle be caused by a back flow, due to inflammation or disease, with incompetence, of the mitral valve; or directly to disease of the lung itself, whether from phthisis, contracted lung, pneumonic bronchitis, or emphysema; the shock being in these last cases shielded from the hand and muffled to the ear by the interposition of a couch of lung, thickened by the undue expansion of the air cells induced by the disease.

When the aorta is thus distended, pushing aside the lungs, beating with a double pulsation upon the second right intercostal space, over the ascending aorta, the first gentle and gradual, the second, a smart shock, the first feeble or even silent, the second, a loud ringing, metallic shock, you know that the blood forces its way with difficulty through the fine vessels of the system, and that the cause of this is the contamination of the blood, induced by advanced granular contraction of the kidney.

Two conditions are needed for the production of this double effect, one, that just spoken of, the obstacle to the onflow of the blood; the other, the force with which the pulsating ventricle sends its blood into the artery. Lessen that force,

and the supply of blood is lessened, the proportion of blood in the vessels and the power to pass it on is brought more into equipoise; the tension of the blood being relieved, the first sound becomes again audible, and the shock of the second sound is subdued, so that it becomes merely unduly sharp or loud.

Additional observations are wanted on this important practical point of the relative loudness of the first and second sounds over the pulmonary artery and aorta; combined with information as to the poisoning and accumulation of the blood, structural change in the walls of the arteries, and vital power. The two sounds must be listened to, and their relative intensity noted, which I do by the ready method of figures of varying size written on a diagram of the body on which the outline of the ribs is traced. The size of each figure denotes the relative intensity and actual loudness, judged of by the ear, of the two sounds. When the first sound is silent, and the second is loud and with a shock, I mark it thus, °/2; when two sounds are equal, thus, 1/2; when the first is louder than the second, thus, 1/$_2$; and when the second is louder than the first, thus, ^{1}/2. Every shade can be thus rendered. Combined sphygmographic and cardiographic tracings, some of which I have made, in these cases, will give positive and scientific accuracy to our knowledge.

Doubling of the Second Sound.—In two of the cases of endocarditis with mitral murmur, there was doubling of the second sound. One of these came in with doubling of the first sound, or almost a murmur at the apex; on the 4th day a peculiar plunging first sound, with scarcely any second sound, appeared over the ventricles. On the 6th day there was doubling of the second sound. On the 8th day mitral and pulmonic murmur appeared, followed by a tricuspid murmur, and on the 10th these murmurs had all vanished.

In the other case the doubling of the second sound appeared late and was very tenacious. There was a mitral murmur up to and on the 23rd day, when the second sound was prolonged over the pulmonary artery. On the next day there was doubling of the second sound over that artery. The second sound was louder than the first—and this proved that the later sound was the pulmonic, the earlier the aortic sound. In this instance the doubling of the second sound, which lasted to the 60th day, disappearing on the 69th, was due, I consider, to the longer time occupied by the right ventricle than the left in emptying itself, owing to the resistance to the flow of blood through the lungs.

Pulmonic Murmur.—Symbol ⇖.—A systolic murmur over the pulmonary artery, at the second left space, was heard in a considerable number of the cases of endocarditis with mitral murmur. This number amounted to one-third of the first series, or seventeen in fifty-two, and to one-half of the second series, or ten in twenty. This murmur was present also in one-third of those cases of endocarditis affecting the left side of the heart, in which there was tricuspid, but not mitral, murmur. In more than one-half of those cases the pulmonic murmur appeared towards the close of the attack, when all the acute symptoms had gone by, when the period of convalescence was approaching or established, when the patient was pale and thin, having lost a large proportion of the red corpuscles from the blood, and was weak from the exhausting nature of the disease. In nearly one-half of the remaining cases this murmur appeared at the middle period, and in one in four of the whole number it was audible soon after the admission of the patient.

The murmur almost always occupied a well-defined limited area at the edge of the sternum in the second space, just over the pulmonary artery. It never extended as far as

the right edge of the sternum, but it could be heard very feebly in the first space, and occasionally in the third. When the position of the pulmonary artery was unusually low, the murmur moved downwards, being then heard strongly over the third space, and feebly over the second and fourth spaces.

The pulmonic murmur rarely presents a smooth soft bellows sound, but is usually grave and superficial, without however being large in character or very loud. The murmur appeared as a peculiar scratching noise in one-half of the cases, or 4 out of 8, in which the sign appeared soon after admission, and besides these in one on the 8th and in another on the 21st day. The scratching nature of the sound when I first observed it (I found it noticed in one case as early as the year 1852) was very puzzling. It strongly suggested friction-sound. But it differed in these respects: it was always systolic, being never to-and-fro; pressure sometimes highly intensified, but never altered it in tone; it clung to one spot; and it gradually disappeared without passing into a wide-spread double friction-sound. Its noise was exactly like that made by scratching slowly and gently with a pin on a deal table.

The cause of the pulmonic murmur is exactly the same as that of the aortic "anæmic" murmur, which is audible only during the systole. It is due to the blood being very thin and lessened in quantity, and propelled into the vessel when its walls are relaxed, with undue force, by the ventricle.

When the pulmonary artery is flaccid, its contents have free room to vibrate as they move onwards in the current of the circulation, and therefore pulmonic murmur is engendered. The pulmonic murmur thus indicates that there is relaxation of the pulmonary artery, or a condition the opposite to that of tension of the artery. The second sound following the

murmur may be loud, but it is usually feeble. It is loud if during and towards the end of the contraction of the right ventricle, the pulmonary artery becomes tense; its walls then recoil with force upon their contents and propel them with equal pressure in two directions, forwards into the vessels, and backwards upon the ascending pulmonary artery, its sinuses and valve, where the back-stream strikes with a sudden shock, the shock of the loud second sound. The second sound is, on the other hand, feeble if the flaccid artery does not become distended during the systole; when the recoil of the walls is therefore weak, and when the back-wave breaks with only moderate force upon the roots of the artery.

Silence or feebleness of the first sound is the opposite in character and cause to pulmonic murmur. If the artery is distended when the ventricle begins to contract, the column of blood moves steadily forwards, the walls of the vessel and its contents are not thrown into vibration, and the first sound is either absent or feeble. The extreme tension of the pulmonary artery thus induced, leads, when the blood has ceased to enter it, to the recoil of its walls with excessive force upon their contents, which are driven with a strong back-stroke or shock upon the walls, sinuses, and valve of the artery. When the lung is displaced from before the pulmonary artery, thus distended, this shock is felt by the hand and heard striking against the ear with a loud metallic sound.

Pulmonic murmur, as we have just seen, came into play most frequently when the disease was passing away. It was therefore rarely, or only once or twice, associated with a mitral murmur when at its zenith, and uncomplicated with other murmurs. In fully one-half of the cases (13 in 24) it accompanied prolongation of the first sound, or a feeble mitral murmur; in nearly one-half of them (9) it appeared

with a conjoint mitral and tricuspid murmur; and in one-fourth with a simple tricuspid murmur, a companion sign that was therefore present in three-fourths of the cases. In one-fourth of the cases (6) it was traced side by side with an anæmic murmur over the aorta or carotid artery; and thrice it was unaccompanied by any murmur. Nearly all these instances point to a state in which the tension of the pulmonary vessels was either not yet established or was passing away.

A pulmonic murmur was audible in a large proportion (or 5 in 13) of those cases that I have classed as being probably affected with endocarditis. In all of these cases there was prolongation of the first sound. In three of them it was noticed soon after admission, and in the two others at a late period of the illness.

A pulmonic murmur was heard in a small proportion of the cases in which endocarditis was either threatened or only transient, amounting to 7 in 63 of the first series, and 2 in 22 of the second, or one-tenth of the cases. In all of these but one it appeared at a late period, when the intensity of the disease was passing away.

Pulmonic murmur is not then a direct sign of endocarditis. Its presence, however, in the early period of acute rheumatism usually points to endocarditis, and to the actual or approaching presence of a mitral or tricuspid murmur.

Its existence at a late period in a case of endocarditis generally points to relief in the severity of the disease, to the cessation of the inflammation of the heart, to a definite removal of the tension of the pulmonary artery, due to congestion in the lungs, and to the establishment, for a time, of the opposite state of that vessel, its walls being relaxed and the quantity of its blood diminished.

The pulmonic murmur, then, while it is a sign threatening

inflammation of the interior of the left side of the heart in the early stage of acute rheumatism, is a sign of the passing away of endocarditis when it appears at a time when that affection has been established. Pulmonic murmur never becomes permanent. It generally diminishes rapidly when the patient leaves the bed, and gains colour and strength, and in the convalescent patient it is often inaudible when standing or after walking, when it may be still heard if the patient is lying down.

I have heard the pulmonic murmur in several cases of enteric fever, when it indicates the condition of which I have just spoken, or relaxation of the pulmonary artery.

The pulmonic murmur usually, I believe, tends to become less vibrating and more feeble during the progress of the systole, when the artery is becoming less relaxed, and to die out of the end of the systole when the vessel is becoming tense, and the stream of blood is being gradually brought to a stand-still.

Tricuspid Murmur in Cases of Endocarditis with a Mitral Murmur.—I have already illustrated this sign when I described tricuspid murmur in cases of endocarditis of the left side of the heart without mitral murmur. I refer to that part at pages 242—245, and shall here therefore only state generally the conditions under which this murmur is found.

A tricuspid murmur is not, as we have already seen, a sign of inflammation of the right side of the heart, and of the tricuspid valve; but of inflammation of the left side of the organ and of the mitral valve. When the left ventricle is weakened by that inflammation, it sends less blood into the vessels of the system, and an undue amount of blood therefore accumulates in the vessels of the lungs. The pulmonary artery is over-filled, and the left ventricle is distended. The "safety-valve" function of the tricuspid valve is then brought

into play, regurgitation takes place, and by throwing a portion of the blood backwards upon the veins of the system, it lessens the pressure of the blood forwards upon the arteries of the lungs.

Tricuspid regurgitation, then, while it declares the presence of inflammation of the left ventricle and the mitral valve, relieves the congestion in the lungs, which is one of the worst effects of that inflammation.

A tricuspid murmur is present in nearly one-half of the cases of endocarditis with mitral murmur. A tricuspid murmur may precede a mitral murmur, accompany it, alternate with it, or waken up after it has disappeared. A tricuspid murmur, then, is a friendly sign—it warns you of inflammation elsewhere, and relieves the ill effects of that inflammation. It is a danger signal, and a brake, lessening the mischief.

Aortic Systolic Murmur (*Anæmic*).—A direct aortic murmur was noticed in twelve of the seventy cases of endocarditis with mitral murmur, and there were others in which its presence was doubtful. This murmur appeared in the early period of the disease in eight of the cases, and in the later period in four. In three of the patients in whom the murmur appeared early, it lived through the whole of the attack ; and in one other of them, after vanishing for a time, it again appeared when the patient was recovering.

In all the cases but one, the aortic murmur was associated with conjoint mitral and tricuspid murmurs, and in fully one-half of them, seven, the aortic was coupled with a pulmonic murmur, usually at a late period of the disease. These twin murmurs, the aortic and pulmonic, are due to the same cause, a deficient supply of blood in the great arteries, which are therefore imperfectly filled. Their walls are consequently flaccid, and their contents have free room

to vibrate as they move onwards in the current of the circulation.

The direct aortic murmur is much less frequent than the pulmonic murmur in cases of mitral endocarditis. But the aortic murmur appears early in the attack much more frequently in proportion than the pulmonic murmur. The reason of this would appear to be that in the early stage the inflamed left ventricle sends its blood with insufficient force and volume into the aorta, and vibrations with their consequent murmur therefore ensue. At a later period, the lessened volume of the blood circulating through the body, and the diminution of its red particles, lead to the formation of the murmur.

The question is an interesting one, and is not easy to answer, why the pulmonic murmur is so much more frequent than the aortic at the later period of the affection? May it not arise from two influences? (1) The increased size to which the pulmonary artery has attained during the period of its tension, when the disease was approaching to and at its acme; and (2) the greater relative influence that the diminished supply of blood has upon the comparatively restricted area of the arteries of the lungs, when compared with the much larger area of those of the body?

Prolongation of the First Sound occurring at a late Period in Cases of Endocarditis with Mitral Regurgitation.—We have already seen that in a considerable proportion of those cases of endocarditis that are admitted before the appearance of a mitral murmur, that murmur is preceded by prolongation of the first sound.

Prolongation of the first sound (as we have seen at page 258) may develop into a tricuspid or mitral murmur, and when the murmur fades away, it may give place to a renewal of the prolongation of the first sound. This is precisely what

occurred in one case, a patient in whom, when admitted, the first sound was prolonged; on the 10th day a tricuspid murmur was audible, which was replaced on the 19th by prolongation of the first sound. In another case the sounds were at first healthy, but the first sound was prolonged at the apex on the 4th day, a tricuspid murmur appeared on the 6th, which yielded on the 9th to prolongation of the first sound over the right ventricle, and on the 48th day the sounds were again healthy. In five cases with a mitral murmur a similar chain of transformations took place. In one of these a mitral murmur, which appeared on the 5th day, superseded prolongation of the first sound at the apex; that murmur became weaker on the 10th, and was joined on the 12th by three other grave feeble murmurs, a tricuspid, a pulmonic and an aortic. On that day the murmurs were audible when the patient lay down—but they passed into prolongation of the first sound when he stood up—and on the 20th day that prolongation was only audible when he lay down, the sounds being healthy when he stood up; owing evidently to the greater amount of blood that was then demanded by the body and the lungs, and was consequently supplied to the aortic and pulmonary artery.

In a few of the patients the murmur during the illness yielded for a time to prolongation of the first sound, and then reappeared. One case, a female patient, was a notable instance of the variety of transformation sounds that may occur in this disease. When admitted, she presented a mitral or tricuspid murmur; on the 3rd day the first sound was prolonged, and on the 6th the sounds were natural. But on the evening of that day a mitral murmur set in which remained for several days, being joined by other murmurs. On the 14th those had vanished, the first sound being prolonged. On the 16th a tricuspid murmur appeared, which was exchanged

for a mitral murmur on the 27th, which from that date became permanently established.

In many instances the position of the prolongation of the first sound is not specified, but when it is, the situation of the murmur as a rule corresponded with that of the prolongation of the first sound out of which it grew and into which it faded—both being present at the apex when the murmur was mitral, and over the right ventricle when it was tricuspid.

The passage from murmur to prolongation and the reverse was often very gradual; they often each glided insensibly into the other. The prolongation was often murmurlike in character, and the murmur was often so obscure as to be quite as fitly ranked with prolongation.

In several of the patients, prolongation of the first sound over one ventricle was accompanied by a murmur over the other. Thus in three cases a tricuspid murmur was associated with prolongation of the first sound at the apex; and in another instance a mitral murmur was coupled with prolongation over the right ventricle. One case is an example of both kinds in succession. At the time of admission, when the patient was very ill, the sounds were loud, the first being sharp. From the 2nd day to the 7th there was a tricuspid murmur with prolongation of the first sound at the apex; and on the 21st there was a double exchange, a mitral murmur being joined by prolongation of the first sound over the right ventricle. Sometimes there was a double prolongation of the first sound, at the apex, and over the right ventricle, as occurred in four cases. I have noticed this coupling of the sign only in cases observed at a later period, and I am certain that it occurs much more frequently than my earlier notes would indicate. In a large proportion of the cases the murmur passed into prolongation of the first

sound towards the period of convalescence. This was noticed in six of the thirteen cases of endocarditis with tricuspid murmur; in sixteen of the forty-one cases of endocarditis with mitral murmur of the first series, in one of which that sign gave place finally to a permanent mitral murmur; and in twelve of the twenty of the same class of the second series.

Prolongation of the first sound is the first whisper of an approaching murmur, the last of a departing one. It is a sign of coming danger, and it usually betokens, towards the conclusion, a favourable issue.

Prolongation of the first sound, or an obscure murmur, was heard in seven of the seventy-nine cases of the first series, and in none of the fourteen cases of the second series that were classed as having had no endocarditis.

Of those patients in whom endocarditis was threatened or probable, the first sound was prolonged, or there was a doubtful murmur, in forty-three of the seventy-six cases of the first series, and eighteen of the twenty-six of the second series. In more than one-half of the cases thus affected there was great general illness (35 in 61), and of these in fifteen there was pain in the region of the heart.

We must look then upon prolongation of the first sound as a sign of actual, or probable, or threatened, inflammation of the heart; whether we regard its presence in those cases of pronounced endocarditis with a mitral or a tricuspid murmur, or in those of probable or threatened endocarditis, in which the murmur was not declared.

The duration, the degree, and the progress of endocarditis is not to be estimated by the presence of a mitral murmur alone, but by the effects also of the inflammation upon the body, the lungs, and the heart. The face is anxious and

dusky; there is sometimes pain in the heart; the breathing is quickened and oppressed; the impulse of the left ventricle is weak, while that of the right is unduly strong; the circulation through the lungs is impeded; the pulmonary artery is distended, its first sound is silent or feeble, and its second is accentuated; a tricuspid murmur is often present, sometimes alone, sometimes conjointly with a mitral murmur; prolongation of the first sound precedes and follows the mitral and tricuspid murmurs; and anæmic murmurs are often heard both over the pulmonary artery and the aorta, during the early and also the late period of the disease, but rarely during its acme; the pulmonic murmur being more frequent at the period of convalescence, the aortic murmur during the early stage of the disease.

III. Cases of Rheumatic Endocarditis with Aortic Regurgitation. (1) Not Accompanied by Mitral Murmur. (2) Accompanied by Mitral Regurgitation.

Symbol ↓.

(1) *Aortic Regurgitation, not accompanied by Mitral Regurgitation.*—Incompetence of the aortic valve is much less frequent in rheumatic endocarditis than incompetence of the mitral valve. There was a diastolic aortic murmur not accompanied by a mitral murmur in ten; and there was a mitral murmur without a diastolic murmur in fifty of the first series of cases—while there was mitral regurgitation in twenty, and aortic regurgitation in none of the later series of cases. This brings up the cases of mitral in relation to aortic regurgitation to the proportion of seventy of the former to ten of the latter. Besides these, eight of the first series and one of the second presented both mitral and aortic incompetence. This makes the total number of cases in which there

was aortic regurgitation eighteen, and the total number in which there was mitral regurgitation seventy-nine.

In more than one-half of the cases of endocarditis with aortic regurgitation, there was no mitral murmur (10 in 18). The mind naturally infers that in these patients the inflammation was limited to the aortic valve, and did not extend to the mitral. The close examination of the cases, however, leads I consider to the conclusion that in all of them there was inflammation of both the mitral and the aortic valves.

A mitral murmur appeared in one of the ten cases for a single day and was not again heard. That was the only case in which this, the central and immediate sign of mitral endocarditis, was noticed. In the others, however, the more important secondary signs of inflammation of the interior of the left ventricle were present.

In five of the cases a tricuspid murmur was audible over the right ventricle. In three of these that murmur was heard before the murmur of aortic regurgitation came into play ; in one, the two murmurs were present on the day of admission ; and in the fifth case the tricuspid murmur appeared a week later than the aortic, but the aortic murmur was preceded by prolongation of the first sound, which was present on the day of admission.

The first sound was prolonged over one or both of the ventricles in six of the cases ; in three of which there was, and in three there was not a tricuspid murmur. In two of the three in which there was no tricuspid murmur, prolongation of the first sound preceded the aortic murmur.

Thus eight of the ten cases of endocarditis with aortic incompetence, without mitral murmur, presented either a tricuspid murmur, or prolongation of the first sound over the ventricles, or both signs. In six of them, one or other of those signs preceded the appearance of the aortic incompe-

tence ; in one other the patient came in with both aortic and tricuspid regurgitation murmurs ; and in the remaining one only did the aortic murmur precede by three days the prolongation of the first sound. The ninth case was admitted with aortic regurgitation, and he suffered from pain in the region of the heart.

The tenth case, a female patient, was an anomalous and doubtful one. She was very ill when admitted, when she had pain in the left side, and the sounds of her heart were rough. On the 12th day a soft double murmur was audible in the second left space which was probably due to aortic incompetence.

(2) *Cases of Rheumatic Endocarditis with Aortic Regurgitation accompanied by Mitral Regurgigation.*—In eight cases mitral and aortic incompetence were combined, and in six of these the mitral murmur preceded the aortic. Both murmurs were present on admission in one of the two remaining cases, and they appeared together in the other one on the seventh day after admission.

These illustrations, and the considerations that I have just advanced, appear to me to render it conclusive, that the inflammation always commences in the interior of the left cavities, affecting primarily the mitral valve ; and that it extends at a later period, and in a limited number of cases to the aortic valve.

These facts lead us to expect that in cases of endocarditis the aortic diastolic murmur appears at a later period than the mitral murmur. In two only of the cases was the aortic murmur heard on the day of admission. One of these had been ill a week, and that was the earliest date of the appearance of the murmur. In three of the patients the aortic murmur appeared from the 7th to the 10th days, in one-

fourth of them (5) from the 10th to the 15th days, and in more than one-half (10) from the 22nd to the 88th days, after the beginning of the attack of acute rheumatism.

We have seen that aortic regurgitation is preceded with rare exceptions by a mitral or tricuspid murmur, or a prolonged first sound over the ventricle, or in other words by evidence, immediate or secondary, of inflammation of the left cavities of the heart and the mitral valve.

In a small proportion of the cases, amounting to three in eighteen, the murmur of aortic regurgitation was preceded by prolongation of the second sound over the aorta or the carotid artery. This prolongation of the second sound over the aorta before the appearance of the aortic diastolic murmur, has evidently the same relation to that murmur, that prolongation of the first sound has to a mitral or tricuspid murmur. It is a transition sound, and is the immediate herald of the coming complete murmur of regurgitation.

An anæmic systolic aortic murmur sometimes precedes the appearance of the diastolic murmur made by aortic regurgitation; but it more often comes at the same time or later, when the two sounds combine to form a true double murmur. This double murmur was present in eleven of the eighteen cases of endocarditis with aortic regurgitation, in four of which the systolic murmur was audible before the diastolic murmur, in five they appeared together, and in two the latter murmur came first into play.

The situation of the aortic diastolic murmur of endocarditis is ruled by the position of the aperture of the aorta, and the direction of the back current flowing through it into the left ventricle.

The murmur is more loud and intense to the left of the middle of the sternum, just over the root of the aorta, than elsewhere. It takes there a direction downwards and to the

left, and is audible to the left of the lower three-fifths of the sternum, becoming feebler as it descends, and is lost usually before it reaches the limit of the lower end of the sternum. The murmur was heard also in five cases as high as the lower end of the manubrium, and indeed over that portion of the sternum. In rare cases it is audible at the apex.

In my cases of endocarditis with aortic regurgitation, the most frequent position of the murmur was to the left of the lower portion of the sternum, a space that extended from the middle of the sternum to its lower end, and from the third left costal cartilage to the sixth; a space that is immediately in front of the right ventricle, where it is denuded of lung. The murmur was audible over this space in thirteen of the eighteen cases. In four others it was heard at or to the left of the mid-sternum, a position that is included in the space noted as being to the left of the lower sternum, and which is, therefore, the position at which the aortic diastolic murmur of endocarditis is heard most frequently and with the greatest intensity.

In two of the cases the murmur was audible just below, and in one of these over the manubrium. In none of them is it stated that the murmur was heard to the right of the upper portion of the sternum, a position in which the direct aortic murmur was audible in five of the cases. In the exceptional and doubtful case, the double murmur was restricted to the left second space. There was certainly no regurgitation in that case from the pulmonary artery into the right ventricle, and we are, therefore, I think, entitled to consider that it was, like the others, a case of aortic endocarditis, with regurgitation. In a patient under my care in St. Mary's Hospital an exquisite musical plaintive diastolic murmur sprang up at a late period just over and below the lower portion of the manubrium, and over the pulmonary artery

in the second space, and was limited to that region. In this case the position of the heart was high and the murmur was heard over a correspondingly high and limited area.

In four, and in four only, of the cases the diastolic murmur was heard at the apex.

When we consider that the current of blood flows from the aorta back into the left ventricle, it seems natural to expect that the murmur of aortic regurgitation should be heard over the left ventricle, into which the stream of blood falls; and not over the right ventricle, which, with its double wall and its full contents, is interposed between the stream of return-blood and the ear. But the fact is the reverse of this. The murmur is always heard in front of the heart, over the right ventricle, and rarely over the left ventricle, to the left of the septum.

After a little reflection, the reason of this curious deviation of the direction of the sound becomes apparent.

When the aortic valve is incompetent, two streams pour side by side into the left ventricle. One of these comes down, in a large volume of blood, from the left auricle, through the mitral orifice, into the left ventricle; and this large living stream of blood occupies and completely fills the whole of the outer portion of that cavity, which is the part that is in contact with the walls of the chest at and beyond the septum, and at the apex. The other stream is that of regurgitation from the aorta. It is a small and an active stream which plays downwards into the innermost portion of the cavity, or that portion of it which lies immediately behind the right ventricle. The large living stream of blood that pours down from the left auricle into the outer part of the left ventricle, through the mitral orifice, cuts off the inner, deeper, and finer current flowing back from the aorta into the left cavity, and so silences it. This answers the question, why do you not hear the murmur of aortic regurgitation at the apex and

over the left ventricle? The answer, however, to the second question is still to seek, why do we hear that murmur through the right ventricle, with its double walls, and its large volume of blood entering freely through the tricuspid orifice? When thinking out the answer to this question, we must steadily come back upon the facts as to the position of the aortic orifice, the nature of that part of the ventricle immediately in front of the aortic aperture, the direction of the return-current of blood into the right ventricle, the point of the greatest intensity of the murmur, and the bearing of the fading away of the murmur.

The aortic valve lies behind the middle of the sternum, at its left edge; in front of it is the conus arteriosus, which is the shallowest part of the right ventricle, its cavity being there wider than it is deep, and its posterior wall being there pushed forwards by the left ventricle and the root of the aorta and the aortic orifice through which this back-current flows; the walls of the conus arteriosus are here thin, especially the front wall; the blood contained in this part of the right ventricle is not in lively motion during the diastole, for it is above the current of blood from the right auricle into the right ventricle; and that current pours across from right to left, low down into the larger, deeper, and lower portion of the ventricle behind the lower part of the sternum and upper part of the ensiform cartilage. The murmur rapidly loses loudness and intensity as it approaches the lower part of the sternum in front of the tricuspid current, and it is lost before we reach the top of the ensiform cartilage.

We now see that the murmur of aortic regurgitation has a shorter way to travel, and passes through a less troubled blood, by passing straight through the arterial cone of the right ventricle, immediately in front of the aortic aperture; than it would if it were to force its way through the large and deep

living current of blood that flows from the left auricle, through the mitral orifice, into the left ventricle, and that completely occupies the body and outer or left side of that cavity, where it presents itself at and beyond the septum and at the apex.

When active endocarditis passes away and leaves the aortic valve permanently incompetent, the murmur becomes more intense, and its area more extensive. The diastolic murmur may then be present over the whole length of the sternum, extending to the right of that bone at its upper portion; and slightly to the right, and to a great extent to the left of that bone at its lower portion; the area of the murmur sometimes extending as far outwards as the region of the apex of the heart.

The murmur of aortic regurgitation in cases of endocarditis is usually soft, smooth, and like a bellows sound. Sometimes it is musical, the note being fine and plaintive, limited in area to the middle of the sternum, or a little above that point, not penetrating, and easily obscured by the other sounds of the heart, and by respiration. It was thus in one case—a very pale woman aged 49. On her admission she presented tricuspid, carotid, and loud mitral systolic murmurs, and a musical diastolic murmur over the middle of the sternum. On the fourth day she was better, and all the murmurs were less marked; and on the sixth they were gone. Next day there was an obscure musical diastolic murmur, which also disappeared in a few days. In one case, on the 101st day after admission, there was a double musical murmur to the left of the lower sternum. In another case, already alluded to, an exquisite musical murmur appeared just below the manubrium, extended to the left during the time of convalescence, was limited in area, and disappeared in about a week.

In another patient, a man, who came in with a mitral murmur, which established itself, a distinct double murmur

appeared for the first time on the 69th day. Six days later the diastolic murmur appeared as a long whistle, but it resumed its usual character on the following day. One other patient that presented a peculiar musical diastolic murmur was a woman, aged 40, ill with acute rheumatism for four days, who came in with a faint blowing tricuspid murmur, which went on the third day, when she had pain in the heart. On the tenth she was better in every respect, but a peculiar diastolic murmur appeared to the left of the lower sternum, like the twang of a harp-string, which was still audible next day; but this was soon replaced by an ordinary short diastolic murmur to the left of the mid-sternum, which ceased after a few days, when both sounds were a little prolonged. Dr. Broadbent observed this case with me.

In another patient, a man affected with acute rheumatism and endo-pericarditis, a loud, grave musical murmur sprang up in the course of the illness, a vibrating murmur, with a perceptible thrill over the aorta, in the second right space, where the murmur was most intense; but the sound was heard to a great extent over and even below the chest. This murmur became established.

Of the remaining cases (14), in nearly one-half (6) the murmur was soft, or like a bellows sound, and this was undoubtedly its predominant character in the rest, although in them the precise nature of the murmur is not stated.

In about one-half of the cases of rheumatic endocarditis with aortic regurgitation, the murmur disappeared when the patients were under observation; while in the greater proportion of the remaining half, the murmur became fixed, being associated with established mitral regurgitation in two-thirds of those cases.

It was interesting and a source of anxiety to watch the progress of the murmur, dwindling and disappearing in

the former set of cases, and ripening into permanent valvular disease in the latter set.

We have already seen that the fine musical diastolic murmurs with a limited area disappeared, while the louder ones of that class became established.

The character of the early murmur of aortic regurgitation gave little ground for foreseeing whether the incompetence would be permanent or transient. Thus in three, if not four, instances, a diastolic murmur, obscure, faint, feeble or confused at first, ripened later into an established aortic valve disease.

The history of the murmur, its development or decay, the widening out or contraction of its area, and the presence or absence, the increase or diminution of the characteristic signs of aortic regurgitation attendant upon the murmur; give more information as to the actual state, progress, and probable future of the patient than the exact character of the murmur on any particular day.

A statement of the duration of the murmur, and of the secondary signs in the cases in which the valve completely regained its function; and a brief recital of the leading points in one or two of the cases that ended by producing aortic valve disease, will illustrate practically the probable future prospect of the affection in these important cases.

The diastolic murmur was short-lived in all but three of those cases that ended in restoration of the function of the valve, its duration being from one to eight days. In the three others the murmur, which diminished steadily in loudness, or sometimes remitted, lasted from fifteen to fifty days.

We shall be the better able to understand the extent to which these cases depart from health, and approach to disease, of the aortic valve with regurgitation, by rapidly reviewing the characteristic signs of the established disease, so as to obtain a standard of comparison.

The characteristic signs of permanent aortic regurgitation are —enlargement of the left ventricle, fulness over that ventricle, and undue force of the apex-beat, which extends beyond and below the left nipple; strong visible pulsation of the carotid arteries; sudden hammering stroke and collapse of the pulse, especially when the arm is raised, when the pulse is visible, and is audible with a loud shock that gradually lessens and disappears when the arm is lowered beneath the level of the heart; diastolic bellows-murmur over the whole sternum, its maximum intensity being to the left of the middle of the bone; the murmur extending to its left at the lower portion of the sternum, becoming more feeble downwards, and to its right at the upper portion becoming more feeble upwards; a direct aortic murmur, generally audible over the manubrium, and to its right, where there is a true double aortic murmur; and a grave vibrating systolic murmur in the neck, over the visibly pulsating carotid artery, which is not followed either by a second sound or a diastolic murmur.

When the patient sits up, the extent of regurgitation and the collapse of the artery increases; and as a consequence, the diastolic murmur often becomes louder and more intense and extensive over its proper region; and the systolic murmur becomes more grave over the aorta and carotid artery, or is replaced there by a local and sudden shock when the regurgitation is very great so as to empty the ascending aorta during the diastole, the shock being occasioned by the blow with which the advancing column of blood is impelled by the stroke of the left ventricle upon the walls of the empty aorta and carotid artery.

If the incompetence of the aortic valve is caused by great enlargement of the aperture of the aorta, owing to dilatation of the vessel from atheroma, the artery extends to the right of the upper sternum, displacing the lung, and may present

there a thrill and a loud vibrating musical murmur, heard, perhaps, at some distance from the surface, and extending over the whole chest, front and back, the neck, and even the abdomen.

My cases of endocarditis with aortic regurgitation ending in complete restoration of the valve, presented, with the exception of the double murmur, to a very slight degree the characteristic signs of disease with incompetence of the aortic valves. The diastolic murmur was present at the mid-sternum, and a little higher, and extended downwards, and to the left, becoming gradually feeble ; but it was never heard upwards, over and to the right of the upper sternum, unless it was joined to a mitral murmur. The area of the diastolic murmur was thus limited ; and it was feeble, very soft, and like a bellows-sound, or plaintively musical.

A systolic murmur was present over the aorta, or the carotid artery, or both, in two-thirds of the cases, this being an anæmic murmur, and not one caused by obstruction. It was due, in fact, to the flaccid state of the aorta, caused primarily by the comparatively small amount of blood sent into it by the inflamed and weakened left ventricle, and increased by the reflux of a portion of that blood sent back again into the left ventricle through the inflamed and insufficient aortic valve. This flaccid state of the aorta allowed the blood contained in it to play freely to and fro in a series of noisy vibrations, with the effect of inducing a grave systolic aortic murmur.

The impulse was rarely notably strong. It was observed in four of the nine cases of this class. The apex-beat was felt close to the nipple in one of these patients ; and in another, in whom the murmur lasted long, it was present on admission in the fifth space, outside the mammary line, and was stronger than usual on the 7th day ; but it retreated within the nipple

line from the 12th day, varying in position from the fourth to the fifth space.

The second sound, which is usually lost over the carotid artery in disease of the aortic valve, was audible in the neck in seven out of the nine cases of endocarditis in which the incompetence of the aortic valve was only temporary. In several of these cases the second sound was at one time or other less clear than natural over the neck, being feeble in two, grave in a third, and in a fourth, first prolonged, then silent, and afterwards natural, but feeble.

Although, then, in these cases, the second sound is still audible, perhaps, over the aorta, and certainly over its branches, the innominate and carotid arteries, it is often palpably modified in character. The presence of a second sound over the great arteries at the root of the neck, and over the ascending aorta, where it is, however, rendered doubtful by being blended with the transmitted presence of the pulmonic second sound, is due to the slight degree of the imperfection of the aortic valve. The shock of the second sound is therefore caused over those parts by the recoil of the walls of the distended arteries after the end of the systole, which sends the blood not only forwards into the arteries, but with a pressure equal in every direction, also backwards with a return-stroke upon the inner walls of the ascending aorta, including its sinuses, and slightly imperfect valve. The aortic second sound, although present, is often modified in tone and blunted, owing to the force of the back-stroke of the blood being impaired; (1) by the reflux of a small portion of the blood into the left ventricle through the inflamed and slightly insufficient valve; and (2) by the lessened supply of blood to the aorta and arteries from the left ventricle, the action of which is weakened by the inflammation of its inner surface. The degree to which the second sound over the neck is rendered feeble, blunted,

prolonged, or almost or quite silenced, is a key to the knowledge of the amount of regurgitation, and of the defective supply of blood from the left ventricle. This important element of diagnosis is farther illustrated by what is found in cases of Bright's disease with contracted granular kidney, when the aortic valve is rendered slightly insufficient by the great distension and enlargement of the aorta. Here the blood is sent by the powerful left ventricle into the aorta and the arteries, already rendered tense by the difficult onflow of the poisoned blood through the small vessels ; and the relief afforded to the tension by the reflux through the insufficient valves is so slight, that the back-stroke of the blood caused by the recoil of the arterial valves is still made with so much force, that the second sound usually retains the metallic ring, and the first sound the feeble note, so characteristic of aortic tension from Bright's disease.

Some of the cases of endocarditis with aortic regurgitation, ending in disease of the aortic valve, acquired step by step the characteristic signs of the permanent affection.

One case of this class, a man, ill a week, came in with quick breathing, a slightly prolonged second sound, and a rather extensive impulse. On the 5th day a soft mitral murmur appeared, which was loud on the 7th, when a diastolic murmur was also audible over the sternum, which extended next day slightly both to the apex and the neck. A week later there was a combination of mitral, tricuspid, and double aortic murmurs, and an obscure second sound was heard in the neck. At the end of the third week the disease was settling into its permanent form, the impulse being extensive, the carotid pulsation visible, and the second sound absent from the neck. The diastolic murmur, feeble on the 24th day, was loud on the 34th, when it was combined with a mitral murmur, and the apex-beat was strong.

Another patient, a labourer, ill eight weeks, was admitted with profuse perspiration, tremulous hands, rather quick breathing, and a double murmur to the right of the upper half of the sternum. On the 4th day the murmur was louder, and was audible over the right ventricle; but on the 6th he was faint, and the murmur was again limited to the aorta. On the 8th day he felt better, and the aortic murmur was again audible to the left of the lower portion of the sternum, as well as to the right of its upper portion. Variations followed, renewed diminution of the aortic murmur over the right ventricle being joined to renewed illness; but after this the systolic murmur became rasping, especially over the third right cartilage, and the diastolic bellows-sound became again widened in area.

The third case of this class, a woman, ill a week, came in with prolongation of the first sound, but no murmur. On the 3rd day an obscure diastolic murmur was audible at the left nipple, and on the 7th this murmur was present along the whole sternum, especially from below the manubrium, and to the right of its upper portion. The second sound was heard in the neck, and the pulse was not distinctly audible at the wrist. On the 15th the diastolic murmur, smooth and prolonged, was not extensive downwards; the second sound, feeble at the apex, was audible in the neck; and a mitral murmur was present for the only time. On the 29th day the pulse was visible at the wrist, and on the 52nd, when she was almost well, there was some fulness over the region of the heart, its impulse being stronger over both ventricles, and especially at the apex. The diastolic murmur was most intense at the fourth cartilage, but was audible along the whole sternum, except its summit. The second sound was still present in the neck, and the pulse was not audible.

In these three cases of endocarditis, the affection of the

aortic valve advanced steadily, but with variations, under my notice, and during the evolution of the disease its characteristic signs came into play one by one.

The next case, a man, stands alone; the aortic regurgitation, after being suspended for a time, returned, and again lessened, without disappearing.

In the last group of four cases of endocarditis with aortic regurgitation, ending in disease of the aortic valve, the murmur appeared at a late period of the disease.

In one of these patients, a man, the murmur appeared suddenly without warning and in full force on the 88th day, being heard loud along the lower sternum. He had previously presented a variable mitral and an occasional tricuspid murmur. This mitral murmur was suspended during a period when the patient was ill with enteric fever, and when prolongation of the first sound was its temporary substitute.

A second case of this class, a boy, ill a week, came in with pain in the heart, a friction-sound, and a mitral murmur, which was still present on the 5th day. After this there is a gap in the narrative until the 49th day, when there was still a mitral murmur. On the 69th day a double aortic murmur suddenly appeared for the first time, and already the pulse at the wrist was audible when the arm was raised. This diastolic murmur varied, increased, and extended to below the ensiform cartilage, but not to the top of the sternum; was once a long whistle, but generally a bellows sound; was accompanied by a mitral murmur at the apex, probably by a tricuspid, and certainly by a direct aortic murmur, there being no aortic second sound. The impulse of both ventricles became extensive, strong, and peculiar, pointing to adherent pericardium; it presented a double shock, one during the systole, and the other at the commencement of the diastole.

In the third case, a woman, one of remarkable interest,

a faint diastolic murmur appeared to the left of the lower sternum on the 47th day, having been preceded and accompanied by varying mitral and tricuspid murmurs. In this case the thyroid gland became very large on the 64th day; was a good deal smaller on the 74th, and finally resumed its natural size. There was a distinct double murmur on the 101st day.

The last case presented healthy heart-sounds on the 17th day after admission, and on the 22nd a soft diastolic murmur came into play to the left of the lower sternum, and a double aortic murmur just below the manubrium. The pulse was audible when the arm was raised, and the impulse was normal in extent.

These interesting cases of aortic regurgitation, coming on by surprise at a late period in cases of endocarditis, usually with a persistent mitral murmur and extensive and deep-seated inflammation of the interior of the left cavities; show that the aortic valve, though it suffers rarely and slightly when compared with the mitral valve, may silently and without warning, and when the patient appears to be well, break down in its functions by the steady and long advance of a latent inflammation.

When we consider how remote the aortic valve is from the focus of the inflammation, how passive and rigid the structures at the outlet of the left ventricle are in which that valve is embedded, how gently the flaps of the valve come together, how comparatively slight is the force exerted upon the valve by the back-flow of the blood in the artery, due to the recoil of the walls of the aorta—that vessel being imperfectly supplied with blood by the inflamed and weakened left ventricle—a force that spends itself mainly in driving the blood forwards, and secondarily in impelling it backwards on the valve, it is only natural that the aortic valve should be

rarely incompetent during the attack of endocarditis, and more rarely permanently crippled. These cases perhaps point to a gradual extension of the inflammation on the ventricular surface of the valve, and to the gradual yielding of the inflamed and softened valve; which at length gives way suddenly at its margin, and so admits of regurgitation from the aorta into the left ventricle.

IV.—Cases of Rheumatic Endocarditis with Prolongation of the First Sound.

The examination of the cases of endocarditis in which there was tricuspid, mitral, or aortic murmur, alone or in combination, show, I think conclusively, that prolongation of the first sound at the apex or over the right ventricle points to actual or imminent endocarditis.

Thus prolongation of the first sound both preceded and followed a temporary tricuspid murmur in three cases, preceded the appearance of that murmur in two other cases, and followed its disappearance in two additional ones. The first sound was therefore prolonged in one half of the cases (7 in 13) in which a tricuspid murmur was present without a mitral murmur.

Again, a mitral murmur when present without aortic regurgitation was preceded and followed by prolongation of the first sound in seven cases; and was preceded by it in nine, and was followed by it in twenty other instances. The first sound therefore was prolonged in fully two-thirds (36 in 50) of the cases of endocarditis with mitral murmur in which there was no aortic regurgitation.

Finally, the first sound was prolonged in six of the ten cases of endocarditis with aortic regurgitation in which there

was no mitral murmur; and in four of the nine in which there was both aortic and mitral regurgitation, or in more than one-half (10 in 19) of the cases of endocarditis with aortic diastolic murmur.

If we combine the three series of cases with tricuspid, mitral, and aortic regurgitation, we find that in a little more than three-fifths of the whole number (53 in 82) the first sound was prolonged over one or other or both of the ventricles, and that this proportion held its ground in each of the three classes of valvular murmur from endocarditis. If we deduct from the 29 patients in whom there was no prolongation of the first sound, those who both came in and went out with tricuspid or mitral murmur, amounting to fully twelve cases, and who could not therefore present prolongation of the first sound preceding or following a murmur, we naturally increase the proportion in which the first sound was prolonged; and this proportion would necessarily be still further increased if we could deduct the unknown quantity of cases in which the prolongation of the first sound escaped observation.

It is evident then that prolongation of the first sound is a sign of transition; that it tends to expand into a mitral murmur when situated over the apex, into a tricuspid murmur when over the right ventricle, and occasionally into a systolic aortic murmur when situated over the aorta; and that when either of these murmurs passes away, it naturally glides into prolongation of the first sound over the region of the lost murmur.

Prolongation of the first sound over one or both of the ventricles in a case of acute rheumatism is in itself then a sign, actual, probable, or threatening, of endocarditis affecting the left cavities of the heart. If it is present when the face is covered with a diffused flush, or is dusky and anxious,

when the breathing is quickened or oppressed, or when pain is seated in the region of the heart, and the second sound is intensified over the pulmonary artery, we may at once conclude that the patient is affected with endocarditis.

I have included among the cases of endocarditis two of the patients affected with acute rheumatism, who had prolongation of the first sound without murmur, but in both of whom that sound was murmur-like ; and who had also several important symptoms pointing to internal inflammation of the heart, including pain over the heart in one, pain in the chest in the other, and very great general illness. I have ranked seven of these cases with prolongation of the first sound apart, among a class in which endocarditis was probable, and I may say, almost certain.

In more than one-half, or five, of these nine cases, including both those in which endocarditis was present, and those in which it was probable, the prolongation of the first sound was murmur-like in character. In six of these cases there was a pulmonic murmur; in four the face was dusky; in three there was restlessness or delirium ; in two others the sleep was bad ; in three there was pulmonary apoplexy, or cough, with phlegm; in one there was pain in the heart ; and in two there was pain in the chest.

It is more difficult to settle the exact position of those cases with prolongation of the first sound that I have ranked among those threatened with endocarditis. Among the cases of this class belonging to the first series, amounting in the whole to 63, almost one-half (30) presented prolongation of the first sound ; and in five more there was a double murmur ; while in nine others the sounds of the heart were affected, the first sound being very loud in three, and doubled in one ; while both sounds were feeble or indistinct in five.

Of the 30 patients in whom there was prolongation of the

first sound, in one-half (14) there was great or considerable, and in 16 there was slight, general illness. I think that we may consider that the fourteen patients with great or considerable general illness, nine of whom had pain in the region of the heart, were probably, or almost certainly, affected with endocarditis. To these perhaps may be added the four patients who presented an obscure murmur. Three of these, however, had but slight general illness. If we add to the fourteen with great general illness and prolongation of the first sound, the case with an obscure murmur and also with great general illness, we may conclude that fifteen of those who were threatened with endocarditis were almost certainly attacked with that affection.

Among the 79 cases that are ranked among those who had no endocarditis, seven had prolongation of the first sound, and one had an obscure murmur. All of these had but slight general illness, and I think that they have been properly assigned to their present place.

If we examine the cases of the second series, or those treated by means of rest, we find that out of twenty-two cases threatened with endocarditis fourteen presented prolongation of the first sound. Of these nine had pain in the region of the heart, or great general illness, or both, while in one of them the general illness was slight. Eight of these cases may therefore, I think, be almost ranked with the cases of endocarditis.

In two of the remaining cases threatened with endocarditis there was a transient murmur.

V.—Cases of Rheumatic Endocarditis with previous Valvular Disease of the Heart.

Previous valvular disease of the heart was present in 22 of the 107 cases of endocarditis of the first series, and in 7 of the 28 of the second series of cases admitted into St. Mary's Hospital under my care during the years 1851—1869-70. Among the cases of the first series, ten had mitral, five had aortic, and seven had mitral-aortic regurgitation, and the seven of the second series had mitral incompetence. Sixteen additional cases with previous valvular disease appear among my 325 cases with acute rheumatism of the first series; and of these eight had endocarditis combined with pericarditis, four had "probable" endocarditis, two were "threatened" with that affection, and only two presented no sign or symptom of endocarditis. We thus see that of the total number of cases of acute rheumatism with established valvular disease (amounting to 38), 30 (or 79 per cent.) had endocarditis; in 6 (or 16 per cent.) endocarditis was probable or threatened; and 2 (or 5 per. cent.), had no endocarditis. Compare these cases broadly with the rest of the cases of acute rheumatism in which there was no previous valvular disease. Of the total number, amounting to 287, 161 (or 56 per cent.), had endocarditis, of which 54 had pericarditis also; in 73 (or 25 per cent.) endocarditis was probable or threatened, including 3 with pericarditis; and in 83 (or 29 per cent.) there was no endocarditis, including 6 with pericarditis. We thus see that previous valvular disease of the heart, in cases of acute rheumatism, exercised an all-powerful influence in exciting endocarditis. Nor can we wonder at this important result. It has been the key-note, underlying the whole of this long clinical history of pericarditis and endocarditis, that whatever part, liable to be affected by the disease, was

exposed to the burden of labour, was exposed, in exact proportion to that labour, to the attack of inflammation, the severity and extent of the inflammation being proportioned to the amount of labour.

The presence, then, of established valvular disease, which adds very seriously to the labour of the heart in cases of acute rheumatism, adds very seriously to the probability, the almost certainty, of endocarditis in such cases. We have just seen that the influence of valvular disease, which tells with such force in the production of endocarditis, has but little effect in exciting pericarditis. The reason is, I think, obvious. The great extra work is thrown upon the interior, and not upon the exterior, of the left ventricle, and especially upon its mitral valve. A second local influence, in the altered apertures and roughened surfaces of the mitral and aortic valves, and especially at their margins, comes in to heighten the effect of the local labour in the production of endocarditis.

The two conditions that prevailed through the whole series of cases of established valvular disease with endocarditis are —the variability of the murmur from day to day ; and great general illness. That chain of signs distinguished every case, and that chain of symptoms affected all but two of the whole series of instances of endocarditis with disease of one or more of the valves of the heart.

The variability of the murmurs showed itself not only in their greater or less loudness during the successive phases of the disease, but also in their transformation from one tone to another quite different ; their extinction, suspension, and reappearance ; and their extended, contracted, and shifted areas. This variation in the nature, character, and field of the murmur, is governed mainly by three leading influences :—(1) the changes to which the valves themselves and

the interior of the heart are subjected by the inflammation; (2) the varying power of the heart under the influence of increasing general weakness, and returning strength; and (3) the tumultuous action of the heart owing to local pain, or the struggle to pass the blood onwards through the obstructed orifices; or its intermission and failure from the exhaustion of previous overwork.

I shall illustrate the variable character of the murmur in these cases of endocarditis with previous valvular disease by the brief notes of a few cases, first selecting from among those with mitral regurgitation, then those with aortic, and finally those with mitral-aortic valvular disease.

The first instance with *mitral* disease that I shall quote was a young woman who had left the hospital four days previously with a mitral murmur, due to a primary attack of acute rheumatism. She came in suffering from a fresh attack, with a distressed, anxious look, a dusky face, rather livid lips, and accelerated breathing. She had pain over the heart, its action being rapid and tumultuous, and an indistinct murmur. On the 3rd day there was a loud systolic murmur at the apex, and the second sound was sharp over the pulmonary artery: and on the 4th she had agonising pain in the heart, its action was tumultuous, and its sound could not be defined; she struggled violently and perspired profusely. Next day a loud systolic murmur, tricuspid as well as mitral, was audible over the whole region of the heart. On the 10th day the tricuspid murmur was audible along the sternum, and a second impulse, with a loud second sound, were present over the pulmonary artery in the second left space. On the 18th she was bright and cheerful but a cough was still present, and the murmur was softer. On the 23rd day she walked about the ward, but on the 29th there was a return of pain on movement, and the murmur was louder. After this she did well, there was a

thrill over the heart, the murmur was loud over the apex, and was heard over the left scapula. Here the mitral murmur was obscured when the heart was tumultuous ; and was loud and smooth, and joined by a tricuspid murmur, when the health improved and the heart was steady in its action. Another case, with previous mitral regurgitation, had, when admitted, tightness of the chest, pain over the heart, and a loud systolic murmur. Three days later, with less pain, the murmur was almost musical at the apex, and quite so below it over the stomach ; two days later she looked better, and the murmur presented a third change, being not nearly so loud ; but next day, with returning tightness of the chest, there was a fourth transformation of the murmur, which was rasping or almost musical over the heart ; the 10th day, however, with renewed improvement, showed a fifth variation in the murmur, which was no longer rasping ; but on the following day there was a sixth change, and the murmur was musical around the apex ; after this, on the 13th day, the murmur was grave, this being its seventh variation ; its eighth occurring on the 18th day, when it was again musical over the stomach, and when it was joined by a systolic murmur over the aorta. After this, with steady improvement, the murmur was no longer variable. A third case illustrates the variations of the murmur during the convalescent period.

These two cases are typical, but their successive snatches of ever-varying murmur, contrast with the murmur, now swelling, now dwindling, that is found in other and more simple cases. I will just quote one of these. A youth, a carpenter, came in with pain in the chest and a prolonged musical systolic murmur at the apex. This murmur was persistent, but it varied in tone, being grave on the eighth day, when pain was present. The heart's beat was strong.

Each of the remaining seven cases presented features of

its own; the variations of the murmur being great and complicated in four of them, and in three of them comparatively simple. In four cases, if not five, the mitral murmur was associated with a tricuspid murmur, in one with a pulmonic, and in one with a direct aortic murmur; while in one the first sound was prolonged over the right ventricle. In one of the cases just enumerated, a diastolic aortic murmur appeared and disappeared, reappeared and was finally extinguished, the mitral murmur being permanent throughout.

The *aortic* murmurs of established valvular disease scarcely vie with the mitral murmur in variety of tone, loudness and area, and alternate extinction and return, in cases of rheumatic endocarditis; but I may state that the study of the five cases that I can cite shows that in all these points the diastolic-aortic murmur presents frequent variation; though the systolic murmur of aortic contraction is much less subject to change.

In one case with aortic regurgitation, probably of some standing, tricuspid and mitral murmurs were added temporarily to the diastolic murmur, which varied much and was not always audible during the attack of endocarditis. At the cessation of the illness a double aortic murmur was alone audible. In the other case a double aortic murmur, which went and came again during the illness, was apparently joined on the 28th day by a tricuspid murmur, which had departed on the 34th, leaving a double aortic murmur.

The remaining seven instances had previous *mitral-aortic* valvular disease. Two of the cases belonging to this last group were admitted twice with mitral-aortic endocarditis, so that the actual number of patients belonging to it is reduced to five. One of those two patients who were thus admitted twice with endocarditis, had left the hospital six months

previously, after an attack of rheumatic endocarditis, and came in with double aortic and mitral murmurs; which varied somewhat in loudness and extent, but were substantially unchanged during this illness. Four years later she returned with severe acute rheumatism and endocarditis, and died after a very long illness, albuminuria having been finally added to her ailments. The murmurs underwent several oscillations, sometimes the mitral, sometimes the aortic diastolic murmur, being very loud, while at other times one or other of those murmurs was almost or quite extinguished at the heart; the mitral murmur being however generally distinctly though feebly audible over the back of the chest.

In the three remaining cases the variations in the murmurs were rather in loudness and extent of area, than in the tone and character of the sounds.

The extent and strength of the impulse, and their variation during the attack, are among the most decisive tests of the previous presence of valvular disease in cases of rheumatic endocarditis. As a rule, the impulse in such cases is unduly diffused, strong, and propulsive; and this applies more in degree to cases with mitral aortic, than to those with simple mitral regurgitation. The extent of the impulse in a case of valvular disease without endocarditis, is a test of the undue amount of labour to which the heart has been put to overcome the obstacle to the circulation of the blood caused by the affection of the valves. The supervention of endocarditis sometimes, by rendering the heart's action tumultuous, increases the impulse; but sometimes its effect is the reverse, and by lowering the power of the heart, it lessens the impulse.

Among the ten cases of endocarditis with previous mitral incompetence, including one in which aortic incompetence sprang up temporarily during the attack, in five the impulse

was strong, in one it was diffused, in two it was moderate, in one it was feeble, and in one it was not described. In three of those cases the impulse was stronger during the attack of endocarditis than after it, and in two it was the reverse. The impulse of the left ventricle was usually increased in the cases of established mitral incompetence, but that of the right ventricle was, in proportion, more affected in those cases.

Among the five cases of previous aortic incompetence with endocarditis, including the two that were joined during the attack, one by mitral, the other by tricuspid incompetence, in three the impulse was strong and extensive, especially towards the apex ; in one it was diffused but rather feeble ; and in one it was of moderate force and extent. The impulse was more extensive during the attack of endocarditis than after it in one case. The impulse was strong, extensive, and unduly far to the left, in five of the seven cases of previous mitral-aortic incompetence with endocarditis ; it was diffused but rather feeble in one ; and in one it was feeble. The impulse appeared to be strengthened during the period of the endocarditis in four instances, while in one case it was the reverse.

Pain was present over the region of the heart in four of the ten cases of endocarditis with previous mitral incompetence, in four of the five with aortic incompetence, and in four of the seven with mitral-aortic incompetence. There was pain in the side or chest, or tightness of the chest, not including those with pain in the heart—in four of the ten with mitral ; in one of the four with aortic ; and in three of the seven with mitral-aortic valvular disease. There was no pain either in the heart, chest, or side, in two of the ten cases with mitral ; in none of the five with aortic ; and in one of the seven with mitral-aortic valvular disease, or in only three of the twenty-two cases under consideration. We have seen that pain in

the heart, side, or chest, occurs in by far the largest proportion of such cases; and that pain in the parts named is much more frequent in cases of endocarditis in which the heart was previously affected with valvular disease, than in those cases of endocarditis in which the heart was previously healthy.

The respiration was seriously affected in a very large proportion of the cases of valvular disease with endocarditis. This condition in such cases is inevitable, for the effect of all the diseases of the valves is to interfere with the efficient onflow of the blood towards the system, and therefore to throw the blood backwards upon the lungs. This applies of course with primary and immediate force to incompetence of the mitral valve, which throws a portion of the blood just received back again upon the lungs, with the effect of overcharging the pulmonary vessels. The return of the blood back again from the aorta, owing to aortic incompetence, into the left ventricle from which it has just been sent, is, however, only one short stage forwards from the seat of mitral incompetence; and the almost immediate effect of the aortic incompetence is to produce a back-flow of blood upon the pulmonary vessels, and to delay the blood in those vessels and congest them. The presence of this surplus amount of blood in the lungs, which upsets the healthy balance of the circulation through the lungs and the body, compels the respiratory organs to exert themselves to the top of their power, so that they may, if possible, expel forwards into the body the weight of blood that oppresses them. Hence result laborious, difficult, and rapid breathing, pulmonary apoplexy, pleurisy, catarrh, and bronchitis.

The respiration was rapid in four, the chest was painful or tight in two, and cough with pulmonary apoplexy occurred in another of the cases with mitral valve disease; while in two

of those cases there is no note of the state of the lungs, and in one they were healthy in function. The breathing was quick, or there was cough, or pain in the chest, in four of the five cases with aortic, and in six of the seven with mitral-aortic valvular disease. More than three-fourths, therefore, of the cases of valvular disease with endocarditis had serious disturbance of the respiratory functions.

CLINICAL HISTORY OF ENDOCARDITIS IN CASES OF CHOREA.

The association of chorea with endocarditis has long been known, both clinically and from examination after death; and it has already received illustration in this volume, at 391, 392, Vol. III., where two important cases of chorea are alluded to that have been published by Dr. Broadbent and Dr. Tuckwell, in both of which there was endocarditis, and minute cerebral embolism; and in one of which there was acute rheumatism as well as chorea. I had also occasion, in this article on endocarditis, to give at page 278 a case which illustrates the association of chorea with endocarditis. I shall now give a brief account of the cases of chorea treated by me in St. Mary's Hospital, with especial relation to their association with endocarditis.

Clinical History of the Cases of Chorea in relation to the presence of Endocarditis, observed by the Author in St. Mary's Hospital.—I find notes of 40 cases of chorea that were under my care in St. Mary's Hospital, and in 34 of them the signs of the heart are noted, while in 6 of them they are not so.

CASES OF CHOREA IN RELATION TO THE PRESENCE OR ABSENCE OF ENDOCARDITIS.

1.—Cases in which there was no endocarditis, heart sounds healthy		10
2.—Cases in which there was probably no endocarditis :—		
a. Slight prolongation of the first sound	5	
b. Anæmic murmur over the pulmonary artery	1	
	—	6
3.—Cases in which there probably was endocarditis :—		
a. Prolongation of the first sound	3	
b. Murmur, tricuspid or pulmonic	2	
	—	5
4.—Cases in which there was endocarditis :—		
a. With mitral regurgitation →		
⇉ Ending in restoration of valve	2	
⇉ Lessening of murmur on recovery	2	
⇉ Mitral regurgitation established on recovery	8	
	12	
b. ⇊ With aortic regurgitation ↓	1	
	—	13
		34
Cases in which the heart was not observed		6
TOTAL		40

Association of the Cases of Chorea with Rheumatism.—The well-established association of chorea with articular rheumatism, renders the study of the connexion of rheumatism with these cases of chorea necessary before we consider the occurrence of endocarditis in chorea. Acute rheumatism, as we have just seen, is so very frequently accompanied by endocarditis that we must be careful, when ascertaining the frequency of endocarditis in chorea, not to attribute the internal inflammation of the heart too readily to chorea, when it may be caused by the rheumatism associated in certain cases with that affection.

Articular rheumatism, in a subacute form, was definitely present during the attack in six of the forty cases of chorea. In five of these cases the rheumatic affection immediately

preceded the occurrence, and continued for a short time after the supervention of the attack of chorea. In one of the cases in which there had been no previous rheumatic attack, the joints became inflamed in the course of the choreal affection.

In addition to these six cases of chorea with pronounced articular rheumatism, there were five cases of chorea in which there was pain in all the limbs (in 1), or in the shoulder and hips (in 1), or in the legs (in 1), or in the hands (in 1), or there was stiffness of the arms and legs, and of the left ring-finger (in 1). In none of these cases, however, was there swelling or redness over the joints; but this does not apply to the redness which affected the wrists, elbows, and face in one patient from violent friction. There were also five cases of chorea that were free from rheumatism during the attack, that gave a history of antecedent acute rheumatism, occurring from two years to two or three months, and in one instance for an uncertain period, before the occurrence of the chorea.

The proportion in which endocarditis appeared in those cases will be given presently.

Proportion of Cases of Chorea in which Endocarditis was present.—In nearly one-third (10 in 34) of the cases of chorea in which the sounds of the heart were observed, those sounds were healthy; in one-sixth of them (5) there was slight prolongation of the first sound, and in one case there was a pulmonic murmur. I have classed the six latter cases among those in which there was probably no endocarditis, and I think we may infer that those sixteen cases, amounting almost to one-half of the whole, were free from inflammation of the interior of the heart.

In three cases in which there was marked, almost murmur-like, prolongation of the first sound, and in two with a tricuspid or pulmonic murmur, amounting to almost one-sixth

of the whole (5 in 34), the presence of endocarditis was probable.

The remaining cases, amounting to fully one-third of the whole (13 in 34), gave complete evidence of the existence of endocarditis, in the presence of a mitral murmur in twelve instances, and of a diastolic-aortic murmur in one.

I think it probable that the majority of the six cases of chorea in which the heart was not observed, ought to be added to those in which there was no endocarditis.

Cases of Endocarditis with a Mitral Murmur.—The cases of choreal endocarditis with mitral regurgitation, considering the comparatively small number of those cases, offered as great variety in character, mode of commencement, course, and result, as the cases of rheumatic endocarditis with mitral regurgitation.

Endocarditis with mitral regurgitation ended more than twice as often in established mitral disease in chorea than in acute rheumatism. Mitral regurgitation became permanently established in two-thirds of the cases of chorea with mitral murmur (8 in 12) ; and in less than one-third of the cases of acute rheumatism with mitral murmur of the first series (14 in 49), and in only one-sixth of those of the second series treated by rest (3 in 20). The integrity of the valve was restored in one-sixth of the cases of chorea with a mitral murmur (2 in 12) and in another sixth of them, the murmur was becoming feebler when the patient left the hospital (2 in 12).

The mitral murmur in fully one-half of the cases (7 in 12) was situated in the region of the apex, and was not described as extending beyond that region ; but was simply entered as a systolic mitral murmur, or a systolic murmur at the apex.

The five remaining cases, compared with those just

dismissed, presented greater breadth of area; variety in intonation and volume of sound; and individual life.

In two of these cases the mitral murmur was very extensive, being audible over the back of the chest, above and below the scapulæ, and the greater part of the left side. One of them, when admitted, had been ill with chorea in a severe form for some weeks, but the affection was now but slight. A loud systolic murmur centred itself at the apex; and was audible along the sternum, and far to the right of its lower portion, though feeble at its upper part; from the third to the seventh left costal cartilages; in front of the epigastrium and the liver; and all over the dorsum, especially on the left side. The impulse of both ventricles was immoderately strong and extensive, the apex-beat being present an inch to the left of the nipple-line. These signs underwent little change after the admission of the patient, and it was evident that the endocarditis had ceased.

The other case came in with acute endocarditis, a mitral murmur being audible at the apex and over the right ventricle. A few days later it could be heard towards the axilla, and over the back, as high as the upper part of the scapula. At the end of the seventh week the murmur was grave and musical, and a fortnight later it appeared as a prolonged bellows sound. After this it was hardly so loud, but towards the end of the fourth month after admission it was grave and vibrating.

This case had an interest much broader than the simple relation of chorea to endocarditis; for it had interwoven with it from its commencement, and throughout an important part of the early period of its course the relation of acute rheumatism to chorea, and of acute rheumatism to endocarditis also. It began with inflammation of the ankle, conjoined with chorea. Six weeks later, when admitted, the knee was inflamed, chorea being the most pronounced disease, and the

two affections being accompanied by endocarditis. Was this endocarditis the direct offspring of the subdued attack of acute rheumatism, or of the chorea, or of the two conjoined affections, each taking its part in giving a combined birth to endocarditis ?

During the third week the arms were slightly rheumatic, as well as the lower limbs, and the patient lay motionless in bed, apparently stilled by the affections of the limbs and joints, the chorea being almost or quite latent. After this the rheumatism insensibly disappeared, the chorea insensibly reasserted itself, and for the remainder of the patient's long history, the chorea, modified in form and severity, was the only apparent affection ; accompanied throughout, however, by endocarditis.

The other three instances of which I have to speak were cases of chorea, unalloyed, during the attack, by rheumatic arthritis ; but two of them had suffered some time before from acute rheumatism.

One of them, a girl, had been long ill with chorea, and had gone through a rheumatic attack two years before. She came in with a loud, smooth, systolic murmur at the apex, which was audible over the right ventricle. After this the murmur underwent minor transformations, being like a bellows sound on the 4th day, and almost musical on the 8th, when the apex-beat extended furthur outwards, the murmur being faintly, if at all, audible below the angle of the left scapula. The apex-beat extended a little beyond the nipple. This case came in with endocarditis, which was evidenced by the varying character of the murmur ; but there is nothing to show whether or not this patient had acquired mitral disease from the old attack of acute rheumatism.

This last question does not complicate the next case, for though this patient, a girl, had twice been affected with acute

rheumatism, yet she had no murmur, but a prolonged first sound, on admission. A murmur, however, appeared at the apex on the 4th day, which was grave on the 6th and the 8th, and was loud on the 14th day, when it extended towards the axilla. The apex-beat was strong on the 6th day, three-and-a-half inches from the sternum; but on the 8th it could scarcely be felt.

The last case, a boy, was free from rheumatic taint, and presented no murmur during the first six weeks; but at the end of that time he had pain in the chest, and a week later a smooth bellows murmur appeared at the apex, which three weeks later spread upwards towards the axilla, and downwards over the stomach. After this, during a long period, extending from first to last over five months, the murmur underwent various changes, being a very smooth bellows murmur on the 62nd day, audible upwards towards the axilla, and downwards over the stomach. On the 76th the murmur was very loud and superficial, being heard towards the axilla, but for a very short way below the heart. The first sound was very feeble, while the second was very loud over the pulmonary artery, in the manner already related at page 247. After this the mitral murmur underwent various modulations, being moderately loud on the 102nd day, very loud at the apex on the 105th, but scarcely audible over the lung to the left, or towards the axilla; much weaker on the 126th; but on the 135th day it was loud below, especially on expiration, and was not heard outwards during inspiration. On the 146th day, and the last report, there was very slight fulness over the heart, the impulse of the right ventricle, which seven weeks previously was strong, extending from the third cartilage to the sixth, and from the sternum to the nipple, was on the last observation less strong to the right of the lower sternum, and extended from the second to the fourth

cartilages. The mitral bellows-murmur was not so smooth as before, and was again heard up to the axilla. The double impulse of the pulmonary artery, previously marked, was no longer perceptible. There was no murmur over the back. He went out comparatively well, being free from choreal movements.

In this case, as in that just related, during the attack of endocarditis, when the patient lay speechless in bed, the heart became enlarged, and the lung shrank away from before the heart, exposing its increased impulse over a large area; and the mitral murmur was heard extensively over the region of the contracted lung, and that of the stomach. At a later period, however, with returning health, strength, and exercise, the lung expanded freely, and interposed itself between the greater part of the heart and the walls of the chest, so as to cut off the extended border of the area of impulse, and to lessen that of the murmur by damping and silencing its sound.

Case of Endocarditis with a Diastolic-Aortic Murmur.— This patient, a boy, came in with a second attack of chorea, which began three weeks previously with pain in the legs of a rheumatic character, followed, a week later, by choreal symptoms, which became gradually more severe. On his admission the heart sounds, so far as they could be made out, were healthy, but on the 3rd day a diastolic murmur was audible over the centre of the sternum. Ten days later this murmur was heard, very prolonged and loud, over the whole length of the sternum; being audible to the right of the upper part of the bone, and to the left of its lower portion, but becoming weaker towards the apex of the heart. On the 86th day the diastolic murmur was still loud, and maintained its ground everywhere; and it was joined by a systolic murmur, loudest at the sternum and not mitral. Three weeks

later the diastolic murmur was inaudible at the middle of the sternum, and was feeble at its upper and lower portions; but on the 79th day, the last observation, it had apparently resumed much of its loudness and extent, and the systolic murmur was silent.

In this case, as in one of those just told, the question must be put, Was the endocarditis caused by the primary auticular rheumatism, or by the resulting chorea, or by the combined influence of the two affections?

ENDOCARDITIS IN PYÆMIA.

There was only one instance among the 71 cases of pyæmia or secondary inflammation examined after death in St. Mary's Hospital in which the appearance of endocarditis was observed and reported. That case, a man, who was under my care, presented a spot in the right lung, an inch long, consisting of pus, and apparently broken down lung-tissue, and superficial to this a patch of dry fibrinous deposit on the pleura; and numerous spots, similar but smaller, through the back of the middle and lower lobes of that lung. There was also a large globular and fluctuating tumour on the upper and inner part of the left kidney three inches in diameter. On cutting into it highly offensive blood-like fluid escaped, and on laying it freely open there was a clot of blood and a little pus. The sac was lined with a delicate, highly-organised, chorion-like membrane, with numerous prominent blood-vessels ramifying on its surface. There was a large black spot of apoplectic effusion in the substance of the kidney near the membrane. The structure of the kidney was healthy.

The heart was of natural size, and there was a patch of recent roughness on the surface of the left auricle. Several nodules, from the size of a split pea to that of a millet-seed,

were situated on the free margin of the mitral valve. The corpora Arantii of the aortic valve were enlarged. The patient was admitted in a state of great depression, his mind wandered, and mucous and sonorous noises were audible over the chest. The state of the heart was not observed.

The attack of endocarditis was in this case the marked secondary effect of the pyæmia, but the solitary occurrence of this instance with endocarditis in 71 cases of pyæmia shows that the inflammation of the interior of the heart, so common, as we have seen, in acute rheumatism and chorea, is rare in pyæmia, though less so, as we shall see, than in the fatal stage of Bright's disease.

The signs of the heart affection were not observed in this case of pyæmic endocarditis. I have had, however, frequent opportunities of examining a patient affected with pyæmia, in the course of whose very serious illness the signs of endocarditis appeared and held their ground. Pleurisy first showed itself, and the evidence of inflammation in both lungs; and after a time a systolic murmur became audible at the apex. This murmur was constant, but it varied in loudness, tone and area during the course of the illness. After this patient's recovery a mitral murmur was established.

ENDOCARDITIS IN BRIGHT'S DISEASE.

I have only been able to find one instance with evidence after death of endocarditis in the whole of the cases of Bright's disease described in the post-mortem records of St. Mary's Hospital, amounting to 207, excluding those in which there was regurgitation through the mitral or the aortic valve, or through both valves, or obstruction of the mitral orifice. That case was one of fatty disease of the kidney in

a man, aged 41, who was under my care. His heart was rather large, weighing 12½ ounces, and was dilated and flabby. The structure of the valves was healthy, with the exception of a patch of white deposit on the anterior flap of the mitral valve, which did not appear, after death, to interfere with the function of the valve.

This man, when admitted, presented a yellowish pallor and puffiness of face. He had been a healthy man until he took cold, nine months previously, after which he became gradually weak and pale, and had palpitation and frequent vomiting, symptoms with which he was still troubled. There was some albumen in his urine. The right veins of his neck were rather swollen and pulsating, and there was pulsation of the temporal artery. The heart's impulse was very feeble, and diffused over the cardiac space during expiration only, but it could be felt between the ensiform cartilage and the left seventh costal cartilage. The liver was firm and low, and presented a diffused pulsation in the epigastric space.

A soft systolic bellows-murmur was audible at the apex, and a peculiar short double murmur between the nipple and the sternum, which was obscured by the natural heart sounds. These mumurs varied considerably from day to day, but they were generally audible, though the diastolic noise was more or less obscure. About a week after his admission a peculiar humming venous murmur was heard to the right of the sternum when he sat up, but not when he lay down, which, sometimes, disappeared without apparent cause, when it could be brought back by pressure over the jugular vein.

On the 42nd day he presented considerable general dropsy, and for the first time the murmurs were very faint and obscure, and two days later they were lost. After this the mitral murmur was sometimes audible, but was generally not so, and the diastolic murmur was only heard once, corresponding

with a thrill near the apex. The last observation was made on the 77th day, when a faint systolic murmur was heard over the seventh cartilage, and feeble doubling of the first sound over the sixth cartilage. The urine was then scarcely albuminous, and it had been so during a considerable period of the history of this patient, who died on the 98th day.

I have ranked this case as one of endocarditis, because of the presence of a white deposit on the mitral valve, which was otherwise healthy, and of the history of varying murmurs, pointing to changing affection of the mitral and aortic valves. The long duration of the case, and the small amount of change to which the valve had been subjected, make it doubtful whether the endocarditis was present in more than its effect, the white deposit on the mitral valve, at and before the time of death; but if we take that appearance, and the varying signs of double regurgitation into account, I think we may infer that this case was one of endocarditis. It is true that both mitral and aortic regurgitation may be present in Bright's disease when there is very great tension of the arteries, and great hypertrophy, with dilatation of the left ventricle; that in such cases those murmurs usually vary in character, according to the varying intensity of the causes that gave them birth; that they may be suspended, restored, and again lost, even permanently; but this case did not present those conditions, for the heart, though dilated, was not greatly enlarged, and was not hypertrophied, since it only weighed 12½ ounces.

Admitting, then, that this was a case of endocarditis occurring in a patient affected with Bright's disease, it is evident, that as this was the solitary instance of that kind that was noticed among so many cases of Bright's disease without disease of the valves, that although endocarditis may occur in that disease, yet that it is rare. This becomes more marked

when we compare the cases of acute rheumatism, and of chorea, with those of Bright's disease; for in the two former affections, from one-half to one-third of the cases were affected with inflammation of the interior of the heart.

The frequent presence of thickening of the mitral valve, and occasionally of the aortic valve; and the large proportion of cases of valvular disease without a previous history of acute rheumatism; perhaps point to the occurrence of endocarditis in those cases during the earlier period of their history. If so, endocarditis, and pericarditis, behave very differently from each other in Bright's disease, for while pericarditis is common towards the fatal period of this disease, especially when the kidney is granular, and is rare during its earlier history, endocarditis is very rare towards its fatal period, but is not very infrequent during its earlier history; that is—if the thickening of the valves, and especially of the mitral valve, and complete valvular disease, have their origin in Bright's disease itself.

CLINICAL HISTORY OF ENDOCARDITIS OCCURRING IN CASES OF VALVULAR DISEASE OF THE HEART.

The influence of previous valvular disease in rendering endocarditis more frequent and severe in cases of acute rheumatism has been already seen at page 315. We then observed that the presence in that affection of disease in the valves of the heart, by adding to the labour of that organ, and by rendering its internal apertures more rough and irregular, increased the danger of the occurrence of internal inflammation of the heart, and intensified that inflammation when established.

So great, indeed, is the influence of valvular disease in exciting and intensifying inflammation of the diseased valve,

that we find that endocarditis is apt to occur in such cases, even when free from acute rheumatism, chorea, or any other general disease.

I would refer here to some interesting remarks by Dr. Moxon on this important subject.

The accompanying table (p. 336) will show at a glance the proportion in which endocarditis was present at the time of death in the cases of valvular disease of the heart treated in St. Mary's Hospital.

Pathological Evidence of Endocarditis in cases of Valvular Disease of the Heart.

It is difficult, even impossible, in every case to say, from the appearances presented after death, whether or not endocarditis is present in the affected valves, and the adjoining surfaces of the ventricle and auricle. This is due to the readiness with which, in certain cases, a deposit of fibrin from the blood as it streams backwards and forwards through the mitral and aortic apertures, attaches itself to the surfaces of the imperfect valves, roughened by disease. This is equally the result, whether those surfaces be roughened by the slow degeneration of the diseased fibrous tissues, which, although they may have been generally inflamed at the starting-point of the disease, yet they may have long ceased to be so; or whether the surfaces of the valve be inflamed by a recent and renewed attack of local endocarditis. In many instances, however, it is self-evident that inflammation actually affects the valve, for the appearances presented are precisely those that are found in cases of recent endocarditis, owing to acute rheumatism, chorea, or pyæmia. Those appearances in these cases are to be confided in, for the diseased valves have been described, without, however, as a rule being defined as being

inflamed, by a succession of able and careful pathologists, including the distinguished names of Dr. Markham, Dr. Burdon Sanderson, Dr. Murchison, Mr. Gascoyne, Dr. Charlton Bastian, and Dr. Payne.

Table showing the number of cases with established valvular disease, among those not affected with acute rheumatism, in which endocarditis was present at the time of death.

		Affected with Bright's disease.*
I.—Cases with established mitral regurgitation :—		
a. Cases with endocarditis, not affected with Bright's disease	9	5
b. Cases with fibrinous concretions on the valve, probably not affected with endocarditis	2	3
c. Cases in which no description of the valve was found	1	2
d. Cases without endocarditis or concretions	22	19
I.—TOTAL	34[1]	29[1]
II.—Cases with aortic regurgitation : (A)—from disease of the aortic valve :—		
a. Cases with endocarditis, not affected with Bright's disease	5	1
b. Cases with fibrinous concretions, endocarditis doubtful or absent	5	5
c. Cases in which there was no description of the valve	2	2
d. Cases without endocarditis or concretions	13	12
TOTAL	25	20
(B)—From great dilatation of the aorta, the flaps of valve being healthy but insufficient	5	1
II.—TOTAL with aortic regurgitation.	30	21

* I find a difficulty in reconciling the heading "Affected with Bright's disease" with the insertion of five cases "not affected with Bright's disease." The apparent discrepancy occurs several times in this table, and I note it without being able to explain it.—ED.

[1] I am not certain that these numbers include the whole of the cases with mitral regurgitation, since most of the original copies of those cases

		Affected with Bright's disease.
III.—Cases with mitral-aortic regurgitation :—		
a. Cases with endocarditis, not affected with Bright's disease	5	3
b. Cases with fibrinous concretions, endocarditis doubtful or absent	4	0
c. Cases in which there was no description of the valve	3	0
d. Cases without endocarditis or concretions	16	16
III.—Total	28	19
IV.—Cases with obstruction of the mitral orifice :—		
a. Cases with endocarditis, not affected with Bright's disease	1	1
b. Case with roughness and ulcer at edge of valve	0	1
c. Cases with vegetations or concretions on valve, endocarditis doubtful or absent	2	1
d. Cases without endocarditis or concretions	18	6
IV.—Total	21[1]	9[1]

Among the cases of *mitral regurgitation*, five presented "fringes" and one a ring of small papillary elevations or granulations around the free edges of the valve, and two others had warty or rough excrescences, and another had nodules of lymph on those free edges ; and in one of these the auricular surface of the valve was roughened. One of those instances described, I think, by Dr. Payne, presented also yellow succulent elevations, almost resembling a false membrane, but seated under the epithelium. I have also included among the cases of endocarditis four instances with

have been lost or misplaced, and I have taken them from a detached tabulated abstract of those cases.

This note applies also to the cases of mitral regurgitation given in the Table at page 323.

[1] In 5 of these cases the size of the mitral aperture is not described ; in 5 it was contracted to a moderate extent, and in 19 to a great extent ; and in 1 it was almost closed by a ball of organised fibrin.

vegetations on the auricular surface of both flaps of the mitral valve, and one with extensive ulceration of its anterior flap, in which case the adjoining surface of the ventricle was inflamed; five other cases presented large excrescences, or concretions and smaller vegetations, but these I have not included among those with endocarditis, although some of them may have had that affection. This may be said also of a doubtful case in which the posterior flap of the valve was attached to the wall of the ventricle by adhesions readily separated.

Five of the fourteen cases that I have classed among those with endocarditis were affected with Bright's disease, and nine of them were not so.

Forty-one cases with mitral regurgitation were free from vegetations, and of these, nineteen had Bright's disease, and twenty-two were free from that affection.

The cases with *aortic regurgitation* presented comparatively few instances or severe, with endocarditis, but these presented great variety in their features. One of them showed deposits of red vegetations towards the edge and centre of each flap of the aortic valve. In another, the flaps of the valve were cemented together, and their free margins were roughened, by fibrinous deposit. In a third the aortic aperture was converted into a mere chink by adhesions; and there was an irregular deposit of lymph, forming vegetations, about the basis of the conjoined flaps, some being hard, some cheesy, and others apparently quite recent. The united flaps projected like a funnel into the aorta in the fourth instance, and a little above the valve, and therefore on the inner surface of the aorta was an oval patch, half an inch long, with a red highly vascular flocculent surface. The aortic valve, in the fifth case, was enlarged but soft. One of the flaps had ulcerated away at the sides, and a large nodular mass was

appended to its sesamoid body. The sixth case was one of great interest, with contraction of the descending aorta below the subclavian artery so as scarcely to admit a probe, and embolism, blocking up the left brachial artery. The valve was universally red, soft, pulpy, and formless, and the aperture was contracted. I had originally only ranked five of these cases as being affected with endocarditis, but I think that the whole six may safely be so classed. Only one of these six cases with endocarditis had Bright's disease, the remaining five being not so affected. Ten other cases presented concretions of various size, some being large, one like an alpine strawberry, attached to the aortic valve ; these cases being affected, and unaffected, by Bright's disease in equal numbers. Twenty-five of the cases with aortic regurgitation were free from concretion, and of these, thirteen had Bright's disease, and twelve were free from that affection. In six cases, aortic regurgitation was due to great enlargement or dilatation of the ascending aorta, the flaps of the aortic valve being healthy in structure, but of insufficient size to close the widened orifice of the aorta.

It will I think be sufficient if I state the proportions in which the cases with *mitral-aortic regurgitation* were affected with endocarditis, presented concretions, without distinct evidence of endocarditis, and were free from concretions, without entering into details. I consider that eight of those cases had endocarditis, five being free from, and three being affected with, Bright's disease ; four of them had concretions on the valves, none of which had Bright's disease ; and in thirty-two there was no concretion on the valves, one half of these being free from, and the other half affected with, Bright's disease.

I shall deal with the cases with *obstructed mitral orifice* in the manner that I have just dealt with those having mitral-

aortic regurgitation. Two of them had endocarditis, one being free from, and one affected with, Bright's disease, and another case having that disease presented roughness and ulceration of the edge of the contracted mitral valve; three had vegetations, one of those only having Bright's disease, and twenty-five of them had neither endocarditis nor concretions in any form on the obstructed mitral orifice, only seven of which cases had Bright's disease.

It is evident that while cases with mitral regurgitation are affected in a rather large proportion, or nearly one-fourth (14 in 63), with endocarditis, only one, or at most two, in twenty-nine of the cases with obstruction of the mitral orifice gave evidence after death of that affection. Cases with aortic regurgitation occupy a middle position between the two classes just considered, 6 in 51 (or 1 in 9) of these cases being affected with endocarditis. The cases of aortic regurgitation that were free from Bright's disease were much more frequently affected with endocarditis (5 in 30 or 1 in 6) than those that were affected with that disease (1 in 21).

Cases with mitral-aortic regurgitation have endocarditis rather more frequently (8 in 47 or 1 in 6) than those with aortic regurgitation (6 in 51 or 1 in 9), and less frequently than those with mitral regurgitation (14 in 63 or 1 in $4\frac{1}{2}$).

Valvular disease was less frequently attacked with endocarditis in those cases that were affected with Bright's disease (11 in 78 or 1 in 7) than those that were free from that affection (20 in 105 or 1 in 5·2); and, as we have seen, this tendency in Bright's disease to lessen the frequency of the occurrence of endocarditis in cases affected with valvular disease, prevailed through the whole of the varieties of disease of the valves that we have been investigating, excepting in cases with mitral obstruction.

The Signs and Symptoms of Endocarditis affecting Cases with Valvular Disease.

The signs and symptoms of endocarditis when it occurs in cases of valvular disease of the heart, not affected with acute rheumatism, do not differ essentially from the signs and symptoms of endocarditis, when it attacks cases of acute rheumatism affected with valvular disease of some standing. I have already given a brief clinical history of a series of cases of that class at pages 314-322, and it will, I think, be sufficient if I here refer to the narrative and *résumé* of those cases. As in those cases so in these, the two great distinguishing features of the supervention of endocarditis upon valves already affected with regurgitant or obstructive disease are (1) the great variability of the valvular murmurs, and of the size of the heart, as indicated by the alternate extension and contraction of the area of the impulse, and the alternate increase and diminution of its force; and (2) the great general illness with which the patient is affected, an illness not marked by dropsy, but by elevation of temperature, over-action or failing power of the heart, and pain in the cardiac region, side, or chest, hurried, difficult, and laboured respiration, connected often with a congestive affection of the lungs, showing itself sometimes in the form of bronchitis or of pulmonary apoplexy with its attendant pleurisy. I would again refer to the illustrations I have given with regard to those vital symptoms in a previous part of this article.

I would here remark that the occurrence of a special fever, such as enteric fever, may, as we have already seen, suspend a mitral or an aortic regurgitant murmur for a time, but this occurrence proclaims itself by its own distinctive symptoms.

I have not given any account of the temperatures of the body in the above clinical histories of pericarditis and endocarditis; for the thermometer was only employed in the later cases, and therefore in an insufficient number to enable us to arrive at general results.

Endocarditis affecting the Tricuspid Valve.

Endocarditis and structural disease of the tricuspid valve are admitted to be so rare in the adult, that there are few clinical or pathological records describing affections of that valve.

I have examined the whole of the cases of valvular and other diseases of the heart, and of Bright's disease, contained in the post-mortem records of St. Mary's Hospital, from 1851 to 1869-70, with the special object of ascertaining the frequency, extent, and character of any affection of the tricuspid valve that might occur in those cases, and the result is given in the accompanying Table.

Cases with Affection of the Structure of the Tricuspid Valve, not including instances in which the valve was incompetent owing to the great size of the tricuspid aperture; but including all those in which the edges of the valve were thickened, but the function of the valve was unaffected.

		Affected with Bright's disease.
a.	Cases with endocarditis, not affected with Bright's disease	1	1
b.	Case with fibrinous concretion on valve	0	1
c.	Case with contraction of mitral valve	0	1
d.	Cases with thickening and corrugation, or roughness of valve (1 with mitral-aortic reg., 1 with mitral obstr.) .	2	0
e.	Cases with thickening of valve, valve not incompetent .	11[1]	7[2]
		14	10

[1] Of the 11 without Bright's disease, 2 had mitral, 2 aortic, 3 mitral-aortic regurgitation; 2 mitral obstruction, and 1 had no valvular disease.

[2] Of the 7 with Bright's disease, 2 had aortic regurgitation, and 5 had no valvular disease.

The tricuspid valve was affected with endocarditis in two instances; one of these patients was a woman, aged 40, who had been subject to acute rheumatism when a child, and had palpitation on slight exertion. She had been a patient in the hospital ten months previously with dropsy, ascites, albuminuria, and a mitral murmur. The ascites and dropsy disappeared, but they were greater than before when she was readmitted, when the lips and nose were blue; and the urine was scanty and very albuminous. The mitral murmur was louder than before, and dyspnœa appeared in paroxysms. The heart was rather large (12 ounces), and presented patches of lymph on its surface; the walls of the right ventricle were half an inch thick, being thicker than those of the left ventricle. Warty, rough, irregular fibrous excrescences were present around the margin of the mitral orifice; looking towards, and being entirely in, the left auricle; the ventricular surface being free from deposit: and there was a smooth fibrinous deposit on the (auricular) surface of the tricuspid valve.

The other case with endocarditis of the tricuspid valve was a woman aged 42, who had contraction of the mitral orifice, which allowed of the passage of but one finger. The heart was of very great size, and its cavities contained twenty ounces of blood, although it only weighed 13½ ounces. The tricuspid valve had all its flaps thickened with excrescences along their margins, but the valve itself was competent. She became subject to palpitation twelve months previously after a shock or fright. Three days before admission, she raised half a pint of bright blood. The legs and feet were swollen, she had pain in the chest, the heart's action was violent, and there was a confused rumbling sound at the apex. There was no albumen in the urine. She became gradually worse, and finally palpitation and dyspnœa were superseded by drowsiness.

In both of these cases, the right side of the heart was excited to excessive and continuous labour by the diseased condition of the mitral valve, which in one instance was affected with regurgitation, and in the other with great obstruction.

In one remarkable case a large concretion was attached to the tricuspid valve. This patient was a man, aged 69. The heart was large, weighing 16 ounces, the tricuspid valve was universally thickened, and a fibrinous deposit, the size of a nut, was present on the anterior surface of one of the flaps. The tendinous cords were hypertrophied and atheromatous. One of the valves of the pulmonary artery was converted into a hard concrete mass. There is no account of the left side of the heart.

These were all the instances that I can find in which there was endocarditis of the tricuspid valve, or the presence of concretions on its flaps; but the inquiry into the number of other cases in which the tricuspid valve was affected may throw some light on the probable frequency of antecedent endocarditis of the tricuspid valve, as a probable cause of disease of the valve.

I may briefly state that in one case there was contraction of the tricuspid orifice, so as barely to admit two fingers; and thickening round the margins of the valve; and although the other valves were stated to be healthy, a mitral murmur was audible during life. In another case, with mitral obstruction, the edges of the tricuspid valve were thick and corrugated; and in a third patient, who had been affected with acute rheumatism six months previously, which was followed by mitral-aortic regurgitation, the tricuspid valve, which was not seen, felt rough and thick. These are the only cases that permit definite evidence that in them the tricuspid valve had been previously affected with endocarditis. There were

however eighteen other cases, as may be seen in the Table, in which there was some thickening of the tricuspid valve, in two of which it was stated to be atheromatous ; but in none of these cases did it appear that the tricuspid valve was incompetent. Twelve of those cases had mitral, aortic, or mitral-aortic regurgitation or mitral obstruction ; and of the remaining six cases that were free from valvular disease, five had Bright's disease.

It does not appear to me that any of these cases present definite evidence of the previous existence of endocarditis of the tricuspid valve as the cause of the thickening of its flaps, although it is probable that in some of them the valve had been originally inflamed and especially in those cases that presented aortic, mitral, or mitral-aortic regurgitation, or mitral obstruction.

Treatment of Endocarditis.

Endocarditis is so completely an affection associated with those important diseases, acute rheumatism and chorea, in which it is rare, with pyæmia and Bright's disease, in which it is common, and with established valvular disease, that the proper treatment of the parent affection must in all such cases be the proper treatment of the associated inflammation of the valvular structure of the heart. The treatment of those diseases, however, should be modified in the form of additional precautions when endocarditis appears ; and the general treatment of acute rheumatism and chorea must, from the first, be mainly governed by the consideration that in both of them endocarditis is the most serious natural complication of the general disease. What I have said with regard to the treatment of acute rheumatism in relation to the prevention of pericarditis, applies also to the treatment of acute rheumatism in relation to the prevention, if possible,

and the alleviation of endocarditis. We have already seen that one-half of the first series of cases of acute rheumatism are affected with endocarditis (165 in 325); and that in one-half of the remainder (79 in 164) the occurrence of endocarditis is either threatened (in 63) or probable (in 13). This treatment may be summarized in the brief but effectual rules of (1) the absolute rest of every limb and joint, and of the whole body, during the attack of acute rheumatism; and the maintenance of this absolute rest, especially in the limbs and joints that have been most recently affected, for a period of several days after the complete disappearance of the local inflammation; and (2) the application of the belladonna and chloroform liniment, sprinkled on cotton-wool, over the affected joints, and the support of those joints by the application of flannel over the affected parts so equally adjusted as to give relief and comfort to the patient. We have already seen that the great cause of the inflammation affecting the interior of the left ventricle is the powerful exercise and overwork of that ventricle in maintaining the circulation through the vessels of the inflamed parts, which at the same time call for a greater supply of blood. The fibrous structures of the heart, in common with the fibrous structures of the joints, are prone to inflammation in acute rheumatism; and in the struggle to which the left ventricle is subjected, the valves of that ventricle readily become inflamed at their surfaces and lines of contact. When endocarditis threatens, or first discloses itself, and especially if there be pain in the region of the heart, the application of three or four leeches over that region may be of essential service in lessening the inflammation, and so perhaps permanently saving the valve. It will be well also to cover the region of the heart with cotton-wool, sprinkled with the belladonna and chloroform liniment.

The influence of the treatment of acute rheumatism by means of rest, and the employment of soothing applications and comfortable support to the joints, on the occurrence, severity, and permanent ill-effects of endocarditis, will be best illustrated by comparing the clinical history of the 74 cases treated by rest, with that of the 325 cases not so treated.

There was endocarditis alone, or combined with pericarditis, in one-half (161 in 325) of the first series of cases that were not treated upon a system of absolute rest; and in two-fifths (34 in 74) of the series that were so treated.

Valvular disease became established in 43 of the 127 cases (or 1 in 3·1, or 34 per cent.) of endocarditis, with a cardiac murmur, including those with pericarditis also (18 in 46), but excluding all those that had previous valvular disease, of the series not treated by rest; and in 3 of the 24 (or 1 in 8, or 12·5 per cent.) of the same kind of cases, of the series that were treated by rest. If we extend the comparison to the whole of both series of cases, excluding those that had previous valvular disease, we find that 43 in 281, or 1 in 6·6, of the series that were not treated by rest, and 3 in 61, or 1 in 20, of the series that were treated by rest, had established valvular disease, indicated by a permanent murmur after their recovery from acute rheumatism, and at the time of their last examination.

There was no murmur, and therefore no valvular disease, when the patient recovered from the attack of acute rheumatism, in 60 of the 127 cases with endocarditis, and without previous valvular disease (or 1 in 2·1, or 44·4 per cent.), that were not treated by rest; and in 17 of the 24 (or 1 in 1·4, or 71 per cent.) of the cases of the like kind that were so treated.

The murmur was lessening in intensity at the time of the last observation, when the patient had recovered from acute rheumatism, in 24 of the 127 cases just spoken of (or 1

in 5·4) that were not treated by rest; and in 4 of the 24 (or 1 in 6) of the analogous cases that were treated by rest.

We here find that, in the series of cases of acute rheumatism that were treated by a system of absolute rest, the proportion of those that were attacked with endocarditis was slightly less than that of those that were not so treated. Thus far the comparison is but slightly in favour of the treatment of acute rheumatism by a rigid system of rest; and this would seem to suggest that a certain, and a very large proportion of cases of acute rheumatism are habitually and intrinsically attacked by endocarditis. When, however, we extend the comparison, and ascertain the proportion in which those cases of endocarditis, not previously so affected, acquired permanent valvular disease, so as to injure health during the remainder of life, and to shorten life itself, we discover that the series of cases not treated by a system of absolute rest were thus permanently injured in a far larger proportion of cases, amounting to more than twice as many, or in the ratio of 8 to 3, than in those that were treated by rest.

If we pursue the inquiry further, so as to discover the relative extent to which the interior of the heart was inflamed in the two series of cases, we discover that there was but one instance, or 1 in 24, of those with endocarditis and without previous valvular disease, of the series treated by a rigid system of rest, that gave definite evidence of inflammation of both the aortic and mitral valves; while in 19 instances in 127, or 1 in 6·7, of the same kind of cases that were not treated by a rigid system of rest, there was direct evidence of aortic regurgitation. In nine, or rather ten, of those cases that were not treated by rest, there was a mitral murmur, and therefore direct evidence of inflammation of the mitral valve; but in the remaining nine cases there was also evidence of mitral endocarditis in the shape of a tricuspid murmur, or

prolongation of the first sound, with intensification of the pulmonic second sound, and obstacles to the flow of blood through the lungs. The whole chain of evidence points then, I think, irresistibly to the conclusion that the extent, severity, and permanent ill-effects of the endocarditis were much greater in the series of cases that were not rigidly treated by rest than in the series that were so treated.

Pericarditis, also, attacked a much larger proportion of the cases not treated by a system of rest, or 63 in 325, or 1 in 5·2, than of those that were treated by rest, or 6 in 74, or 1 in 12·2. Thus more than twice as many of the former series of cases, that were not treated by a rigid system of rest, were attacked with pericarditis, than of the latter series of cases that were treated by a rigid system of rest.

I am of opinion, however, from a careful revision of the clinical history of those cases, that the treatment by opium, which was pursued in a considerable proportion of the first series of cases that were not treated by rest, had some influence in increasing the frequency and severity of inflammation of the heart, and especially of its exterior. Taking this into account, however, I consider that the clinical evidence here afforded shows, that the severity and permanent ill-effects of endocarditis, and the frequency and severity of pericarditis, are greatly lessened by a system of treatment by rest absolutely maintained; and combined with the use of local means in the shape of the application of the belladonna and chloroform liniment, and of equal and comfortable support to the affected joints, and the employment of leeches applied over the region of the heart, when that organ was attacked by inflammation, and especially on its exterior, and when accompanied by pain.

The clinical evidence in favour of the treatment of acute rheumatism by rest is conclusively supported on the patho-

logical grounds stated at the commencement of this article (see page 232), and in Dr. Moxon's very striking, important, and convincing lecture on endocarditis, to which I have there referred. We have there seen that the surfaces or lines of contact, pressure, and friction of the valves, and chiefly of the mitral valve, are the parts that are especially affected with endocarditis. Thus the overwork of the left ventricle of the heart, and the resulting friction, pressure, and tension of its valves, in cases of acute rheumatism and chorea, tend to augment the primary influence of the parent disease, and to excite and intensify the inflammation of the interior of the heart, and especially of the mitral valve.

XIX.

INJECTED SPECIMENS OF LUNG AFFECTED WITH PLEURITIS.[1]

In some instances the portions affected with pleuritis exhibited themselves in small red spots, like spots of red ink, scattered over the surface of the pleura, preferring chiefly the base of the lung; in addition to these spots long red lines frequently fringed the margins of the lobes. These red spots and lines consisted of groups of minutely injected capillaries which were numerously divided, tortuous, and irregularly dilated. They were always traceable to one or more irregularly enlarged tortuous capillaries, which were continuous with the healthy small contiguous capillaries. It was manifest that, under the influence of inflammation, the walls of the capillaries had become relaxed, softened, and yielding; and that, under the pressure from behind of the heart's action, the capillaries had become elongated, irregularly dilated, twisted, and looped and distributed in numerously branched, more minute varicose capillaries. Some of these spots were nearly flat, others were raised and projecting, and others, especially where they were seated at the margins of the interlobular fissures, were drawn out into vascular filaments. These filaments formed vascular bands of connection between the edges of the adjoining lobes. That these vascular pleuritic spots may be formed in a very short period was evidenced in a patient who died twenty-seven

[1] From *The Transactions of the Pathological Society*, vol. ii. p. 180 (1850).

hours after being extensively burned: in this case minute violet vascular spots were scattered over the base of the lung.

In some instances, instead of spots, there were vascular prominent patches; and in others, diffused vascularity over an extensive surface.

In those cases of pleuritis where the structure bearing new tortuous capillaries rises above the surface of the pleura, that structure may be torn away in webs from the cellular surface beneath. In doing so, the enlarged capillaries, springing from the healthly capillaries, are torn across, and numerous open mouths of vessels are left on the inflamed surface. This exposed denuded surface is not properly the old serous covering of the pleura, but is the fibrous or aponeurotic structure over which the serous covering was spread.

In emphysema, pneumonia, and phthisis, the serous surface immediately over the diseased portion of lung is very frequently affected with pleuritis. In these instances the character of the vascularity of the inflamed membrane differs from that in ordinary pleuritis. In emphysema the new or developed capillaries are usually small, nearly straight or slightly curved; they do not form elevated groups, but are usually on the same plane with the healthy pleura. In pneumonia there is in some cases a diffused, soft, smooth vascular couch; the new capillaries are tortuous, and irregular in size; but they do not form raised groups. In phthisis the character of the pleuritis does not materially differ from that of pneumonia, but sometimes the new capillaries group into a round bulging patch, corresponding with a tubercle underneath.

The degree of softening of the walls of the capillaries appears to regulate the mode of their distribution. The current of the parent capillary appears to give the law to the current of the new capillaries. If the parent capillary be

straight and small, the new capillaries are straight and small: they ramify at right angles, and the group formed by them is scarcely elevated. If the parent vessel form a curve, the new capillaries form curves also. If the parent vessel be irregularly dilated, first small and then bulging, tortuous, spiral and rising in loops, the new minute capillaries have exactly the same form and direction.

NOTE.—This paper ends in the original with a reference to a further account of the subject, to be found in the paper on the Situation and Structure of the Internal Organs in Health and Disease which heads this collection. The paper is inserted more for its value in indicating the way in which Sibson's work was developed, than for its intense importance. With it ends the series of papers relating to the respiratory organs.—ED.

XX.

ON THE TREATMENT OF FACIAL NEURALGIA BY THE INHALATION OF ETHER, AND ON A NEW INHALER.[1]

In the following cases of neuralgic affection of the face and head, the inhalation of the ether was attended with immediate relief from pain :—

Martha Tansley, a pale fair-haired young woman, married, aged 21, of a remarkably soft fine skin, very susceptible of the influence of cold, applied at the hospital as an out-patient (under Dr. Storer) on the 2nd of January, suffering from an "aching, springing, jumping pain" in the left side of the face : she felt as if a sharp instrument were run into the cheek. Violent paroxysms usually came on after each meal, and lasted about two hours. The pain increased and subsided gradually, but never disappeared except during sleep. She had suffered from the pain in the left side of the face during three weeks, and during the previous eighteen months from neuralgic pain in the right side of the face.

She took, on her first application, a dose of castor oil and turpentine ; after the action of which she had the third of a grain of the extract of belladonna three times a day, in the manner detailed in Dr. Hutchinson's paper on the Employment of Belladonna in Neuralgia. She persevered with the belladonna, with but little relief, until the 8th, when the

[1] From the *London Medical Gazette*, vol. iv, 1847, p. 350.

sesquicarbonate of iron, following another purgative dose of castor oil and turpentine, was substituted for it. She continued the use of the iron until the 29th, when the face being worse, she was directed to have half a grain of the extract of belladonna three times a day.

On the 30th she came to the hospital, suffering from agonising pain in the right side of the face. She inhaled the ether. In about two minutes the pain disappeared. She was quite conscious, had no agreeable or exciting sensations, but felt "rather numbed all over her." The object being merely to obliterate the pain—not to annihilate consciousness or general sensibility—the inhalation was discontinued. In about ten minutes, the pain, which had returned to a slight degree, was again removed by the inhalation. A few minutes after its discontinuance she felt faint. She soon recovered, and walked home an hour afterwards.

About three hours after the administration, a "feeling of jumping" came on for an instant in the left side of the face, and after supper the same side "ached and jumped" for a few minutes.

Next morning she was quite free from pain. She resumed the belladonna three times, and sometimes twice daily, with the effect of exciting a dry prickling sensation in the mouth and fauces, dimness of vision, "numbness all over her," and general sleepiness. She was quite free from pain until the 4th of February, about 2 o'clock, when she felt a slight aching in the left side of the face for an hour. She ceased taking the belladonna on the 5th of February. To-day, the 6th, she is quite free from pain, she feels much better in general health, and her appearance is much improved.

On the evening of the 8th, the neuralgia returned, first on one side of the face, then on the other. The pain was not

very severe : it lasted until the afternoon of the 9th. She was free from neuralgia until the 13th, on the afternoon of which day she had a severe return of the "jumping" pain. After inhaling the ether through the nose and mouth for a short time, the pain ceased. The face ached a little in the night.

Pain returned on the evening of the 14th. After inhaling the ether a short time, she lost consciousness ; she gradually became sensible, but remained some time in a drowsy state.

15th.—She had some return of pain from 10 A.M. until 2 P.M., when it went off.—Capt. Bellad. Ext. gr. ss. ter die.

Mary Peach, a rather dark hazel-eyed girl, having a smooth skin, usually but little susceptible of cold. She had very good health until last August, when, after cessation of the catamenia, she was attacked with "aching jumping" pain in the face, coming on at night, and affecting the warm side of the face. Two months since, she suffered from pain in the chest. About three weeks since, she suffered from neuralgic pains in the temple and forehead : sometimes on one side, sometimes on both sides.

On the 2nd of February she suffered much from a very severe "leaping" pain in both temples. She inhaled the ether through the nostrils, and in about a minute the pain disappeared. She felt rather dizzy, but was not otherwise affected. In a few minutes the pain in the right temple returned. She renewed the inhalation both through the nostrils and the mouth. She was not rendered unconscious, and the pain, though much subdued, did not entirely disappear from the right temple.

The pain in the left temple and in the face did not return, and in the course of the night the pain in the right temple disappeared. She had not been free from pain for a fortnight.

On the 3rd of February she took a purgative dose of castor oil and turpentine, and afterwards half a grain of extract of belladonna three times a day. She experienced no sensible effect from the belladonna, neither dimness of vision nor dryness of the fauces.

She continued free from pain until the 7th, when she had a "jumping" pain in both temples, which made her hot and feverish over the whole body. The pain in the temples gave place to aching pain in the face, which disappeared this morning, the 8th February.

9th.—The pain returned yesterday evening in the right temple as bad as ever, and lasted until dinner time to-day ; it has now (one o'clock) nearly disappeared.—Capt. Bellad. Extr. gr. ss. ter die.

Feb. 8th.—M. A. Corbett, an out-patient of Dr. Hutchinson's, a dark-complexioned young married woman, who has usually had very good health. During the last six months she has suffered from pain in the stomach after food, and from leaping pain in the top of the head striking through to the temples ; the left temple being the most frequently and severely affected.

This morning, Feb. 8th, she suffered from a "leaping pain in the top of the head, striking through to the left temple." After inhaling the ether through the nostrils about a minute, the pupils dilated considerably, she breathed convulsively, and became restless and obstinate. The inhalation was discontinued. When she became quiet and rational she was free from pain, but felt dizzy and sick. She states that it is two months since she has been so free from pain as she now is.

9th.—The pain in the head and temple has not returned ; she has been dizzy and rather sick since the inhalation. She still suffers from pain in the stomach.

Feb. 8th.—Catherine Spencer (an out patient of Dr. Hutchinson's), a florid sensitive young woman, aged 19; she has suffered during the last five weeks from pain in the left temple, as if a knife were running into it, and from a leaping pain in the left cheek, also from epigastric pain and abdominal tenderness.

She is now weeping, owing to pain in the left temple and cheek.

After inhaling ether a very short time through the nostrils, almost unwillingly and in a sobbing manner, the pupils dilating and contracting, and finally dilating, she suddenly became unconscious, the eyes fixed and open. When consciousness returned she was free from pain, and she is so now, three hours after the inhalation.—To remain in the hospital.

It is six weeks since she has been free from pain. She has scarcely slept during that time; if she dropped asleep, in about ten minutes the pain came again "as if it were killing her."

9th.—Nausea lasted about half an hour after the inhalation. During the evening she felt pain in the legs, and while walking to bed the use went suddenly from her limbs, and she dropped on the floor. She had a rigor after getting into bed. The pain continued in the legs all night; indeed she still complains of it. She has no pain in the face, nor, indeed, anywhere, save in the legs.

10th.—The pain went from the legs yesterday evening; she has no facial pain, and she feels better in every respect; but she is unable to sit up, owing to weakness in the back.

14th.—Pain in the right side of the face returned this afternoon: after ethereal inhalation producing unconsciousness she was free from neuralgia.

16th.—She has been free from neuralgia since the last inhalation; she now suffers somewhat from epigastric pain.

M. Eggleston, æt. 37, an out-patient of Dr. Storer, by whom she was sent on the 13th of Feb. to inhale the ether. She has suffered from pain on the left side of the face for two years. During the last six weeks the pain has been unusually severe, accompanied by complete aphonia, and by constant convulsive movements of the jaw to the right side.

On inhaling diluted ether (the ferrule being open), through the nose and mouth for 75 seconds, she became unconscious, the eyes being turned up, the pupils contracted. She recovered consciousness in 65 seconds, the pupils at the same time dilating. In a few minutes, there being slight return of pain, re-inhalation was practised, and again repeated with the effect of removing the pain. On one occasion, when the full volume of ether was admitted, she could not inspire: when the ferrule was opened, so as to dilute the ether with air, she fetched two deep inspirations, and immediately became unconscious.

At the beginning of the inhalation the jaw was in constant rapid convulsive movements: about ten minutes after the fourth and final inhalation her voice returned. She remained perfectly free from pain, and the convulsive movements of the jaw ceased.

15th.—Pain in the face, and convulsive movements of the jaw, returned yesterday.

In the evening, the convulsive movements of the jaws and head were constant: after inhalation they ceased, and she became unconscious. The inhalation at first irritated her, and increased the convulsive movements; but for the nature of the nose and mouth-piece the inhalation could not have been exhibited. In a few minutes consciousness returned, and, though she was still drowsy, the pain and the convulsive motions returned. A short inhalation again produced unconsciousness; on recovering from which the pain and

movements recurred. Twice was this process repeated. After the last time she remained some time in an unconscious state ; and on recovery she was free from pain and the convulsive movements.

Feb. 16th.—She remained free from pain until this morning, when it returned, though in a mild form.

Phœbe Godby, a patient of Dr. J. C. Williams. A full-faced dark girl, aged 17. She has suffered during the last seven weeks from violent pain on the left side of the face. The pain became worse after the incomplete extraction of some carious teeth : the molars are carious, and the alveolar sockets irregular.

After inhaling the ether some minutes, the facial pain went away. The pain returned in the night.

Dr. Williams directed the repetition of the ethereal inhalation from time to time.

On the 15th, after inhaling the ether a short time, she became unconscious: on coming to herself she was free from pain.

Mr. Attenburrow, one of the surgeons of this hospital, administered ethereal inhalation in a case of anæmic hysterical neuralgia, with the effect of removing the neuralgic pain.

The above cases, in addition to a case of neuralgia in which Mr. Lonsdale, of Bristol, treated a paroxysm successfully with ethereal inhalation, and to several cases of a like character, similarly treated by MM. Menière and Honoré, prove that the paroxysms of neuralgia can be cut short with something like certainty by the inhalation of ether. They also show that the neuralgic pain will usually not return until some time has elapsed after consciousness is restored. This period of freedom from suffering varies ; on some occasions extending only over a few hours, on others over some days.

It would be irrational to expect the cure of neuralgia by the unaided means of ethereal inhalation, but we certainly possess in it the means of cutting short the paroxysms of that distressing malady, and of rendering the system more amenable to a scientific treatment directed either to the sources or the symptoms of the disease. We must regard the ethereal inhalation, in fact, as the precursor and handmaid of a more enduring treatment.

We have more reason to hope for relief and benefit from its employment in those cases of neuralgia, due to a reflected morbid sensation in the facial nerves, excited either in the nerves of the deranged digestive organ, skin, or uterus, than in those cases of neuralgia depending on disease of, or pressure upon, the nerve.

Neuralgia is so capricious, and in many persons so peculiarly mental a disorder, that I feel that the above cases rather indicate the course of an important inquiry, than prove that ethereal inhalation is of further value in the treatment of neuralgia than as an almost certain means of putting an end to a paroxysm.

Since the above cases and remarks were written, the following is a sketch of the further history down to this day, Feb. 22nd.

Martha Tansley. Left the hospital on the 16th. I learned from her husband on the 20th that she is much worse; the pain is more violent, and she feels weaker. She has not persevered with the belladonna.

M. A. Corbett. She has suffered but once, for about ten minutes, from the leaping pain since the inhalation: this was about ten days since. She is now, Feb. 23rd, free from pain in the head and temples, but she still complains of sickness and pain in the stomach, and she has occasional attacks of

pyrosis.—Powders of bismuth, rhubarb, and ammonia, to be substituted for a mixture hitherto taken, containing prussic acid, soda, and spirits of lavender.

Catherine Spencer. The facial pain has not returned. On the 21st of February she began to take half a grain of extract of belladonna three times a day. To-day, Feb. 22nd, she has had leeches to the pudenda.

M. Egglestone. On the 17th of February she was directed half a grain of extract of belladonna three times a day. She has continued to take the belladonna since, and has had a belladonna plaister applied behind the left ear.

On the 17th, pain and convulsive movements returned. They were removed on the 18th by four successive ethereal inhalations.

Pain and convulsive motions again returned on the 19th, and were again removed on the 20th by four repeated inhalations.

The pain and convulsive movements returned to-day, and were easily removed by three inhalations administered in immediate succession.—Continue the belladonna.

Phœbe Godby. The pain remained away until the 17th, when she was obliged to go into the town; she caught cold, and shortly after she re-entered the hospital: the neuralgic pain returned. Ethereal inhalation removed the pain on the 18th, and again on the 20th.

She now, Feb. 22nd, suffers from occasional neuralgic pain in the face, and from pain in the epigastric region.

In addition to the above cases, the following has been observed :—

Thomas Head, æt. 25, a framework-knitter, a pale brown-haired man, of firm texture, whose habits have been steady, and whose health has been good, applied on the 18th of February with pain in the course of the left frontal nerve,

which had been periodical for ten days, lasting from about nine o'clock every morning until four in the afternoon.

He inhaled the ether for about ten minutes. Is excited; occasional coughing and dyspnœa. The neuralgic pain became much less severe; indeed, at one time he stated it was gone, but afterwards he found that there was still a little pain, which went off about four o'clock.

A dose of castor oil and turpentine brought on frequent and painful micturition.

On the 19th, the neuralgic pain, hitherto periodic, did not return. He has not been to the hospital since the 19th.

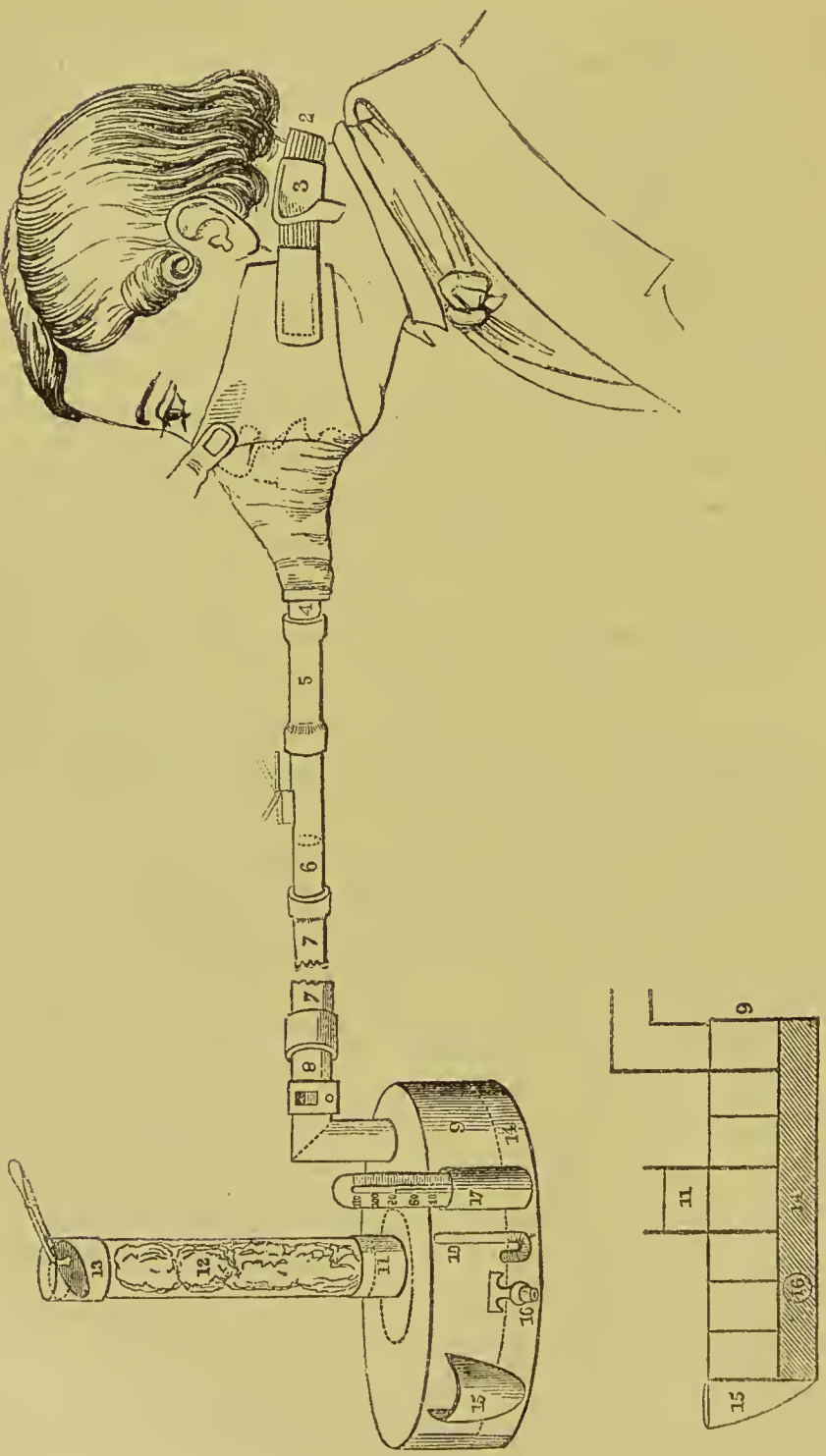
The last case, and the continued history of the previous cases, corroborate the remarks made above.

In the case of Tansley, all treatment ceased, and she has become much worse; while in the cases of Corbett, Spencer, and Egglestone, general treatment has been carried out, and Corbett and Spencer are altogether free from neuralgic pain: and Egglestone enjoys longer and longer periods of intermission between the paroxysms.

The case of Godby shows the importance of retaining cases of neuralgia treated by ethereal inhalation under supervision in the comfortable wards of a hospital. Her case, which up to a certain point proceeded so favourably, has been complicated, and its treatment rendered difficult, by the injurious effects of exposure to cold.

In the case of a man, Joshua Gibson, aged 60, a collier, affected for ten weeks with sciatica, Dr. J. C. Williams, on the 13th of February, directed the inhalation of ether. After the first inhalation he became unconscious; on coming to himself he had a rigor: he was free from pain. The pain returned in the course of the night.

He re-inhaled on the 15th, and again on the 18th; each time unconsciousness was produced, followed by a rigor. The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1, Nose and mouth-piece—a funnel fitting, as closely as possible, over the nose and mouth, made of macintosh, lined with oiled silk. The thumb compresses it over the nose, the fingers, if needful, over the chin.
2, Vulcanised India-rubber strap to keep the nose and mouth-piece in its place by means of
3, A buckle.
4, 5, Two inches of vulcanised India-rubber tube attached to the mouth-piece.
6, Brass tube with valves. The outer valve is almost poised by a lever; the inner valve is closed by a weak spring.
7 7, Eighteen inches of flexible tube.
8, Brass tube, with an opening closed by a ferrule.
9 9, Spiral ether chamber.
10, Glass tube showing the quantity of ether in the chamber.
11, Tube opening into the ether-chamber.
12, Tube containing a sponge saturated with ether.
13, Valve admitting the air and confining the ether.
14, Chamber containing warm water.
15, Funnel to pour in warm water.
16, Tap to draw off the water.
17, Thermometer to indicate the temperature of the water.

Amendment since the apparatus was figured.

The piece of brass (4) is dispensed with.
Dr. Snow's ether-chamber, instead of being round, is square. The ether-chamber is half an inch, the warm water chamber is one inch, in height.

pain, necessarily annihilated during the unconscious state, returned each time with the return of consciousness. He thinks he is worse since he inhaled the ether.

In sciatica the trunk of the nerve is affected in a more distressing manner than the extremities; while in the above cases of facial neuralgia, the extremities of the nerves were the chief seat of pain. Sciatica is a very different disease from that kind of facial neuralgia due to reflective morbid sensation excited in the nerves either of the deranged digestive organs, skin, or uterus.

We cannot infer that because ethereal inhalation is a serviceable aid to treatment in neuralgia, that it will therefore be of service in sciatica.

The above case, though not benefited by the ether, does not, however, prove that in other cases of sciatica ethereal inhalation may not be of value — a point that extended observation can alone decide.

The apparatus here figured and described presents the following advantages :—

The spiral inhaler (9) of Dr. Snow is attached to a chamber (14) capable, if needful, of holding warm water. The ethereal evaporation is further increased at will by the current of inspired air passing through a sponge in a tube (12) before it enters the spiral chamber. This principle is adopted from Mr. Attenburrow's inhaler. Air can be admitted in any proportion through the opening at 8 to dilute the ethereal vapour during the first inspirations. As this opening is at a distance from the mouth and nose, the air and the ethereal vapour are thoroughly mingled before they enter the larynx. If the opening is near the mouth-piece, the ether and air enter in two separate adjoining streams. The supply of ether can be regulated. The various parts of the apparatus can be joined together and disjoined with rapidity and ease. The inhalation can be carried on either in the recumbent or sitting posture, by turning the valve-piece (6) on the mouth-piece (5) and elastic tube a quarter of a revolution. The lever on the valve at 6 can be pushed below the edge of the vulcanised India-rubber tube, and so the patient can breathe air without removing the nose and mouth-piece. The current of respired air is very easy, the tubes through their whole course being wide, indeed nowhere narrower than 5-8ths of an inch.

All the above points are important, but the most important feature in this apparatus is the nose and mouth-piece (1). This is brought over the nose and mouth, and the person inhaling cannot help breathing through it; he breathes, too, without annoyance, and without pressure on either the nose or mouth. He can breathe at will either through the nose or mouth, or through both conjointly. Mr. White, one of the surgeons of this hospital, threw out the idea.

The difficulty, the baffling difficulty, in every other apparatus, is the almost impossibility of ensuring the inhalations when the patient is on the verge of unconsciousness, or when there are convulsive or intoxicated strugglings and resistance to the inhalation. The ethereal vapour is largely mingled with atmospheric air, the patient is re-awakened to consciousness, the work has to be begun afresh, and the difficulty of influencing him increased, if not rendered impracticable. Besides, restless, irritable, excitable, hysterical, sobbing persons, and, above all, stupid persons, often cannot be induced or taught to inhale properly. In certain cases of tic, where the jaws are in constant convulsive movement, as in the case of Egglestone, it would be quite impossible to get them to inhale through a mouth-piece. Through the nose and mouth-piece figured above, inhalation can be carried on in the most refractory. It passed through several stages before it came to its present simple form. I first constructed a nasal inhaler in the earlier cases of tic ; it answered very well, they being easily excited. I afterwards tried to combine a nasal and an oral inhaler in one instrument : at length I bought a common sixpenny mask, lined it within with oiled silk, cut away the septum of the nose, the lips, and the whole circumference of the mask to within an inch of the nose and mouth. I then pasted a funnel of macintosh cloth over the nose and mouth, and over this a piece of macintosh to go over the cheeks ; to these I attached a vulcanised India-rubber strap and buckle. When this is fastened, the nose and mouth-piece fit delightfully. Usually all that is needed is to compress the nose-piece with the finger and thumb against the sides of the nose ; and in the most difficult subjects it is only needful to bring both hands together, the fingers under the chin, the thumbs to each side of the nose, and the head pressed back against one's own body.

I have tried this mouth-piece on a remarkably lantern-jawed shrivelled old man, a crying boy, on several girls, and on every variety of subjects, and in all the adaptation was so complete that they could not breathe at all when the opening for air was closed.

XXI.

REMARKS ON THE ACTION OF NARCOTIC POISONS; THE EFFECTS OF CHLOROFORM, AND THE STAGES OF INSENSIBILITY PRODUCED BY IT.—TREATMENT OF CASES OF POISONING BY THE VAPOUR.[1]

THE death of Hannah Greener, under the influence of chloroform, painfully reminds us that chloroform is not only an admirable anæsthetic, but that it is also a narcotic poison of great power and rapid action. Chloroform, ether, and alcohol are closely allied, both as anæsthetic agents and as narcotic poisons. In small repeated doses they produce agreeable exhilaration and pleasing hallucinations. A friend informs me that he has frequently produced, by merely inhaling chloroform from a bottle, the most delightful thrill over his whole frame. These narcotics, carried by the blood into every capillary, excite simultaneously every part of the frame. The action on the capillaries themselves is universal; they enlarge and are distended with blood, which now circulates through capillaries previously invisible. The conjunctiva becomes injected, and the lips and cheeks are turgid. The secretions are consequently increased, the eyes become watery, and saliva collects in the mouth; while the visible capillaries are turgid, the capillaries in the brain, the lungs, and the other viscera, are turgid likewise.

Turgidity of the capillaries is an effect characterising the

[1] From the *London Medical Gazette*, vol. vi. p. 267. 1848.

narcotic poisons. Alston observed this in frogs poisoned by opium. I dipped the limb of a frog into a watery infusion of opium; all the capillaries of the limb were soon injected with blood. Mr. Nunnely has noticed the remarkable reddening from the distension of the capillaries produced by prussic acid. Stramonium, hyoscyamus, and belladonna often excite universal redness of the skin. The experiments of Mr. Wakley show that ether and chloroform cause distension of the capillaries in the lungs and other internal organs.

The action of chloroform, ether, and alcohol, pervades the whole frame. In the first stage the excitability of every organ and fibre is exalted, but, throughout, their marked action is upon the brain. Exhilaration is followed by excitement, excitement by cerebral disturbance. At this stage the person affected revels in the absurdities of social intoxication: consciousness still exists, but it is deranged. The mind is intent in its own way on many things, but does not now observe all personal realities. The mind often at this stage does not feel pain, simply because it is intently taken up with other things, just as a man in battle often does not feel a wound. This stage of deranged consciousness is also that of staggering intoxication, the muscular power and control being enfeebled, and sensation blunted.

This stage of cerebral excitement and disturbance is speedily followed by the stage of cerebral sleep. There is unconsciousness but not coma; the person sometimes can be roused, the eye turns up as in sleep, and the iris contracts. Now in all persons free from cerebral disease the pupils contract during sleep. I have opened the eyes of many sleepers, and, the brain being sound, I invariably found the pupils contracted. The sounder the sleep the smaller the pupil. In all persons the pupil dilates as soon as they awake, and if their sleep be sound the dilatation is gradual.

I first noticed the contracted pupil, during sleep, in a patient under the general influence of belladonna; his pupils, when awake, were largely dilated. I lifted his eyelids, when asleep, and found them contracted; he awoke, and instantly they dilated. Opium produces an almost characteristic contraction of the pupil; it causes, in fact, a deep but rousable sleep—a true sopor.

Opium is not the only narcotic that induces contracted pupils. Dr. Ogston found that of 22 persons poisoned by alcohol six had contracted pupil. In one case, on evacuating the stomach, dilatation of the pupil gave immediate place to contraction; and, according to Bedingfield, who witnessed many cases poisoned with rum, the patient will recover if the iris be contractile, but if it be dilated and motionless, recovery is improbable.

Under the increasing influence of ether and chloroform the pupils first contract, then oscillate between contraction and dilatation, and finally dilate. So long as the pupil is contracted a dreamy state often exists, and the patient, when operated upon, frequently manifests an unremembered consciousness; he is, in fact, in the state of sopor. When the pupils dilate, and the iris is immoveable, consciousness is extinguished, and the patient is in the state of coma.

In fifteen dogs in which Dr. Percy injected alcohol into the stomach, veins, or arteries,

The pupil was contracted in	. . . . .	4
„	first contracted then dilated in . . .	2
„	contracted and dilated alternately in .	3
„	first dilated and then contracted in .	1
	and dilated in	5

I have met with notices of one case of poisoning by carbonic acid, one by aconite, and one by oil of bitter almonds, in which the pupils were contracted; and in one dog, poisoned

by prussic acid, Mr. Nunneley found that the pupils were contracted.

Contracted pupil is not, then, absolutely characteristic of poisoning by opium, for it may exist in poisoning by alcohol, ether, chloroform, carbonic acid, aconite, and oil of bitter almonds, and *prussic acid?* (in one dog). In all of these, save opium, the pupils are dilated in the extreme action of the poison ; and even in poisoning by opium, although contracted pupil be the almost invariable rule, yet now and then a case is met with in which the pupil is dilated. I have, indeed, found notices of two cases of poisoning by opium, and three by morphia, in which the pupils were dilated. I do not include any case before that of Dr. Kinnis, who was, I believe, the first distinctly to report that opium caused contraction of the pupil.

The remaining narcotics always cause dilatation of the pupil : viz.—belladonna, hyoscyamus, stramonium, characterised by cerebral excitement and disturbance ; tobacco ; digitalis ; conium ; wourali ; *prussic acid*, with the exception noted above ; parsnip-root ; œnanthe crocata ; nux vomica, and strychnia.

Valentin considers, from anatomical grounds, and from observation on rabbits, that the contraction of the pupil is due to the reflex function of the brain [1] through the ciliary filaments from the motor oculi, and that the dilatation of the pupil is due to the reflex function of the spinal marrow through the ciliary filaments from the cervico-spinal nerves. I conceive that this view is borne out by physiological and pathological observations.

A boy was brought dead to this hospital from a recent

[1] By the reflex function of the brain is meant the excito-motory function of that portion of the true spinal system of Dr. Marshall Hall which is seated above the medulla oblongata.

destructive injury to the brain : the pupils were widely dilated. Here the reflex function of the brain was destroyed, while that of the spinal marrow still existed. Next day I found the pupils of a medium size—a proof, I conceive, that the reflex functions of both spinal marrow and brain were then destroyed.

In sleep or sopor, consciousness and volition are absent, but the cerebral reflex function is still active, and induces contraction of the pupil; but in coma or cerebral disease the reflex function of the brain is often destroyed. I opened the eyelids of a boy asleep and ill of fever, who had passed a delirious night : the pupils were dilated. Next morning, after passing a good night, I found them contracted during sleep : when he awoke they dilated.

It is not easy to explain the comparative dilatation caused by arousing a person from sleep. I conceive it to be thus :— During healthy consciousness, the reflex functions are controlled by the mind, so that the spinal action of dilatation of the pupil, and the cerebral action of its contraction—each prevails in turn according to the present need. If the control of the mind be removed, as in sleep, the reflex action of the brain predominates, and the pupils are contracted.

Certain poisons—viz., belladonna, hyoscyamus, and stramonium—act immediately upon the brain, disturbing and progressively destroying both its mental and reflex functions. Such poisons invariably cause dilatation of the pupil, owing to the spinal reflex functions being still active.

On the other hand, strychnia so excites the reflex functions of the spinal marrow that the brain loses control over them : tetanic convulsions ensue, and the pupils are dilated. When the pupils become fixed, it is a proof that the cerebral function is suspended.

So long as respiration continues, it is manifest that the

spinal marrow, from the medulla oblongata downwards, is still alive. The nerves that supply the diaphragm, and all the muscles of respiration that expand the chest, come off from the spinal marrow below the medulla oblongata. It is, then, erroneous to say that the medulla alone lives at this stage; the spinal marrow is alive also, its functions being necessary to support life. As the action of chloroform increases, costal respiration ceases, and the diaphragm only acts, the functions of the spinal marrow being gradually destroyed from below upwards.

During the stage of sopor there is frequently rigidity of certain muscles, as those of the jaw; this I conceive to be due to the gradual withdrawal of the control of the mind from the reflex functions.

This rigidity is speedily followed by complete relaxation of all the voluntary muscles, save those of respiration. Mr. Tracey found that even galvanism had no effect in exciting muscular action in the stage of coma. In addition to their influence on the brain, chloroform and ether now have a local action on the voluntary muscles, paralysing their irritability.

Finally, the respiratory muscles are paralysed, and absolute death ensues.

The action of the heart, according to the observations of Mr. Wakley, continues two or three minutes after respiration has ceased, and Dr. Percy observed the same phenomenon in the majority of the dogs he destroyed by alcohol, but, in four of them, respiration and the heart's action ceased simultaneously. In these cases the heart, especially its right side, was greatly distended. The heart contracted when the blood was withdrawn.

Dr. Glover injected chloroform into the veins. Respiration and the action of the heart ceased at the same time. The heart was found gorged with clotted blood, and its irritability

destroyed. The lungs were congested to a surprising degree, and the bronchi filled with frothy serum.

Dr. Lonsdale poisoned animals by prussic acid. Generally the heart beat some minutes after respiration had ceased : in some instances the heart's action ceased shortly, owing to the enormous distension of its cavities. On the withdrawal of a little blood the heart's action was renewed. When he injected the acid into the trachea the heart's action ceased with respiration, and the right cavities were found to be enormously gorged.

Sir Benjamin Brodie found the heart at rest and enormously distended after poisoning an animal by tobacco : on irritating the heart its action was renewed, and it was kept up for some time by artificial respiration.

I observed that the peristaltic action of the intestines still continued in a chloroformized ass after its destruction by pithing.

The stages, then, of the increasing influence of chloroform, ether, and alcohol are :—

Cerebral excitement.

Cerebral derangement ; staggering intoxication.

Cerebral sleep (sopor) ; pupils contracted ; dreams ; reflex functions of brain and spinal marrow still active.

Cerebral death (coma) ; reflex functions of medulla and spinal marrow still active.

Death of spinal marrow ; cessation of respiration (heart's action still generally present).

Cessation of heart's action.

It is very important to be able to tell easily when the stage of safety—sopor, is about to emerge into that of danger—coma. The action of the pupils is the key to this knowledge. Chloroformization ought not to be continued one instant after the pupils, previously contracted, have begun to dilate. If

unconsciousness can be secured by sopor, the inhalation should not be carried on to produce coma.

If complete muscular relaxation be sought for, as in hernia to facilitate taxis, in dislocation to make reduction easy, and in tetanus, then it will be needful in general to urge the patient from sopor into coma, but as soon as the muscular relaxation is secured the inhalation should cease.

When chloroform or ether is employed in chorea, delirium tremens, or other affections, it should never be urged beyond sopor. In neuralgia it is not usually needful to produce unconsciousness. Whenever the pain disappears the inhalation ought to cease.

Dr. Snow has made this important observation, that the effects of chloroform and ether increase after the administration of it has ceased: this he calls the cumulative property of those vapours. This increase of effect he has observed to last for twenty seconds. Mr. Wakley, in his experiments, observed the same thing. I observed this effect the other day in a woman whose lip was extensively pared under the influence of chloroform. I gave up the employment during sopor, the pupils being still contracted: and in a few seconds coma supervened, the pupils being permanently dilated.

On this ground Dr. Snow objects to rapid chloroformization. He conceives that the complete effect ought not to be gained in less than two minutes. I own I cannot see how a slow, but effective administration of the vapour can lessen the danger arising from the still increasing effect after the administration has ceased. The longer the inhalation is continued the greater the total amount of chloroform received into the system; and this chloroform, already circulating in the blood, will still, I conceive, under either circumstance of quick or slow inhalation, continue to produce an increased effect. Indeed, in the case in which I observed this action, the inhalation had

been carried on, with intermission, some minutes. In another case, in which unconsciousness was produced quickly, the pupil, which had been dilated during one or two seconds, contracted immediately after the cessation of the inhalation.

I think, with my friend Mr. Fearn, that it is important to dilute the chloroform vapour largely with air during the first few inhalations, so as to avoid the sudden shock on the nerves of the lungs, and accustom them to its presence.

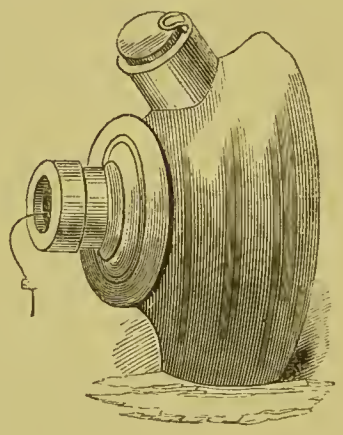
After the death in Newcastle, and after the alarming case at Guy's Hospital related by Dr. Gull, in which respiration appeared to be suspended for some time, we must feel alive to the proper means for restoring animation should it be suspended.

If natural respiration ceases, there is nothing for it but to establish immediately artificial respiration. Ammonia, cold water, bleeding, will be resorted to in vain.

We ought, then, whenever we administer chloroform, to be ready, in case of need, to perform artificial respiration. I have constructed a chloroform inhaler, which can be immediately so applied on the face that, by breathing through the external opening, and pressing back the larynx, artificial respiration can be established. The basis of this inhaler is the mask that I invented for the inhalation of ether, which mask Dr. Snow employed in May last.

"This inhaler is made of copper, brass, or white metal. It has a border or face-piece of thin flexible lead lined with oiled silk, covering the nose and mouth, and from its ductility easily adapted to any face. The lower, or inspiring valve, as seen in the cut, is constructed on the principle of Arnott's ventilators, having a counterpoise weight which keeps it shut, unless acted on by pressure from without. The upper, or expiring valve, is a plain metallic lid always closed unless acted on by pressure from within. The tube to which this

valve is attached may be drawn out so as to expose an aperture for the admission of air when desired."[1] To perform artificial respiration with this inhaler, draw out the expiratory tube: imbed the mask firmly on the face, press back the larynx against the œsophagus and spine—inspire deeply, and distend the chest by blowing through the upper tube. Renew the artificial respirations in rhythmical succession, about sixteen in each minute.



As the action of the heart usually continues after the respiration has ceased, artificial respiration will generally restore animation.

It is possible that the cessation of the heart's action and of respiration may be simultaneous, owing to the distension of the right cavities of the heart. In such a case, in addition to artificial respiration, the abstraction of two or three ounces of blood from the jugular may relieve the distension of the heart, and permit the renewal of its action.

[1] *Pharmaceutical Journal*, Feb. 1848.

XXII.

ON THE USE OF CHLOROFORM IN NEURALGIA.[1]

I ATTEMPTED, in the *Medical Gazette* of Feb. 18, 1848, to explain the action of chloroform as a narcotic poison. The poisonous action of chloroform, far from being against its use as a therapeutic agent, is one of the strongest arguments in its favour. Indeed, almost all our really undoubted remedies are poisons.

In a previous paper (*Gazette*, Feb. 25, 1847) I gave some cases of etherization in facial neuralgia. Following up the treatment there advocated, chloroform has been employed here in the same class of cases. In almost all the cases the pain speedily gave way to the action of chloroform; indeed, usually before unconsciousness was produced. Before the inhalation, the patient was desired, if the pain disappeared, to lift the hand. As soon as relief was obtained the chloroform was withdrawn. If the pain returned shortly, the administration was renewed.

CASE I., *Feb.* 11, 1848.—Robert Buddon, aged 21, a butcher; he is fleshy, fair, and has a thick flabby skin. He complains of an aching, jumping pain, over the right orbit, the right temple, and the right malar bone; this pain has lasted twelve days. He has suffered from it every year, generally

[1] From the *London Medical Gazette*, vol. vi. p. 535. 1848.

in winter, and on one occasion in summer. It is usually, he conceives, brought on by exposure to cold. The pain, which is violent, usually comes on about eight in the morning, disappearing about six in the evening.

After he had inhaled the chloroform, much diluted with air, during nine or ten inspirations, he signified, by holding up his hand, that the pain had ceased. The inhalation was discontinued. He then raised his head, previously resting in the arms of an assistant, and became immediately, and for a few seconds, almost unconscious. He felt low and weak for about an hour after, and was sleepy until the evening. The pain did not return.—℞ Pil. Hyd., Ext. Hyosc. āā. gr. v. st. sum. Ol. Ric., Ol. Tereb. āā. ℥ss. capt. nocte maneque. Ext. Bellad. gr. ss.; Quin. Disulph. gr. vj.

Feb. 18th.—He has been free from pain or uneasiness of any kind, save a slight return yesterday morning. He now feels quite well.

This case illustrates well the favourable action of chloroform. The relief was immediate, and the pain did not return. The immediate relief was undoubtedly due to the chloroform; the permanent relief to the treatment subsequently adopted, and to the natural progress of the case. I have often seen neuralgia removed by the use of the extract of belladonna. Chloroform and belladonna, in their therapeutic influence on neuralgia, have a similar action; they neither of them remove the cause of the neuralgia; they both of them remove the pain by obliterating sensation. Patients under operation, recovering from the action of chloroform, often become conscious while still insensible to pain.

To sensation attention is necessary. A wound in battle is unfelt. The familiar tick and stroke of a clock in one's room

[1] See a paper by Dr. Hutchinson, on Belladonna in Neuralgia, in the *Lancet* for 1843-44.

may go unheard. Toothache often ceases at the sight of the tooth forceps. I have known neuralgia to cease on merely fitting the dry chloroform inhaler to the face. The hysterical female feels pain first in one part, then in another; indeed wherever the attention is drawn. Pain will often be felt for years in an injured part, although the part be perfectly restored, the recollected pain being felt. Of many simultaneous impressions, one only may be perceived if the attention be concentrated on that one. In auscultation, the practised ear, when it chooses, hears over the same place either only the heart's sounds, or only the breath sounds, although both sounds be present. Cheerful society, the so-called mesmerism, opium, belladonna, alcohol, ether, and chloroform, all remove pain by withdrawing the attention from its seat.

Whether we use chloroform or belladonna, or a combination of both, in neuralgia, we must use other means to remove the disorder of which neuralgia is a symptom. The state of the bowels, the skin, and the urine must be regulated. If the disease be of a periodic character, as in this case, quinine is indicated. It is not, however, my object to speak of the general treatment of neuralgia: what I insist on is this, that we must not wholly look to chloroform in the treatment of neuralgia. By its use we remove for a time the pain, and enable those means to tell on the system that during the presence of and because of the pain, produced no effect.

CASE II., *Feb.* 24.—Jane Fox, aged 34, a person of a dark unhealthy complexion. For several years she has been subject to occasional attacks of severe neuralgic pain on the right side of the forehead and right temple. This pain, which is always present, though sometimes it is much worse, has been much aggravated during the last four days.

She inhaled chloroform, much diluted with air, for seventeen

seconds, when she became sleepy, but not insensible, and perfectly free from pain.—Capt. st. Ol. Ric., Ol. Tereb. āā. ℥ss. ℞ Ext. Bellad. gr. ⅓; Quinæ Disulph. gr. iv. nocte manequc.

Feb. 27.—She has had no return of neuralgia.

CASE III., *Feb.* 8.—Mary Roper, aged 23, a widow, of dark, somewhat florid complexion. She suffers from a shooting pain in the left cheek and over the left eyebrow, which appeared forty-eight hours since. Half a drachm of chloroform was used. The pure air was never completely shut off, but in forty seconds sopor was produced (the pupils being contracted), which lasted one minute and twenty seconds. On coming to herself she expressed relief, but in a little time the pain came on again slightly, which was again relieved by merely inspiring the chloroform once or twice.

Feb. 11.—Is now quite free from facial pain. The pain returned in the forehead with much severity, but it did not return in the cheek. In the afternoon the pain disappeared and has not since returned, except for a short time when she has been looking down intently at her work. She had no unpleasant feeling after the chloroform.—Cap. Dec. Cinchonæ, ℥j.; Acid. Sulph. Dil. ℥xv. ter die.—Pil. Hyd. Pil. Al. c. Myrr. āā gr. v. horâ somni.

Feb. 16.—Has had no return of neuralgia.

This case differs from the preceding cases in that, although unconsciousness was excited, a short second inhalation was necessary before the neuralgia disappeared. The pain returned too in the brow with some severity for a few hours, but after disappearing, did not again recur.

CASE IV.—Ann Bennett, aged 22, a florid young woman, having been for a week afflicted with darting neuralgic pain on one side of the face: she inhaled chloroform three minutes, when the pain was removed. Complete insensibility was not produced, but she afterwards felt sick and

giddy, and was obliged to lie down. The sickness and giddiness, with some headache, continued till the afternoon, when she went home, the pain being then as bad as at first.—Cap. st. Ol. Ric. et. Ol. Tereb. āā. ʒiij.—℞ Belladonna gr. ½, Quin. Disulph. gr. v. nocte maneque.

She attended a short time afterwards and said that the pain was altogether much lessened. In this case the unpleasant effects of the chloroform were unusually lasting, and it may, indeed, be questioned whether the chloroform was of service.

Three minutes, an unusually long time, elapsed before the inhalation produced its specific effect. It would hence appear desirable not to produce the effect slowly.

The following case differs from the four preceding, in that the neuralgia was the result of a local injury.

CASE V., *Feb.* 23.—Jno. Whitworth, aged 22, a stout young man, accustomed to work in the field, and much exposed to changes of temperature. He received an injury about six days since from an axe which divided the lower lip and knocked out two of his teeth. His face has ached ever since the injury, but became worse yesterday.

On inhaling chloroform the pain left him in one minute and twenty seconds, but returned again in about two minutes, and was again relieved by two or three inhalations.—Cal. gr. v. P. Jalap. Co. gr. x. st. sum.

March 17.—He did not return until to-day, when he was sent for. He then said the pain returned in about an hour after the inhalation; it was violent at first, but did not last so long, nor was it so bad, as before. In about a week the pain left him.

In the above cases, if we except No. IV., the relief was immediate and decided, and the administration of the chloroform was followed by no injurious or unpleasant effect.

The following case is interesting in that the chloroform, though it removed the neuralgia, was otherwise injurious.

CASE VI., *Jan.* 31.—William Marriott, aged 50. Pale and thin. He suffers from intense neuralgic pain of the left cheek and temple, and dimness of sight in the left eye. He has had neuralgia for three years. Under the administration of chloroform he did not become quite unconscious, but he moved about as if in delirium tremens. After the inhalation the pain in the temple and cheek disappeared, but he complained of pain in the frontal region.

Feb. 19.—Has appeared this morning, after being sent for. He states that the neuralgia did not return for three or four hours after taking the chloroform. He had a cough, and headache over the frontal region for three weeks, and tinnitus aurium for three years before he first applied. These symptoms were worse when he lay down at night. After the chloroformization the pain in the head became worse than it had ever been before ; when he coughed the head was "as if it would cleave in two." The tinnitus aurium produced deafness. His cough was worse for a week after the inhalation.

The neuralgia is now much better ; the pain, which is trifling, being only occasional. He still complains of singing in the ears, and deafness.

In this case the neuralgic pain masked the head symptoms that contraindicated the use of chloroform, and which were not elicited by careful inquiry until they were aggravated by the inhalation.

In a subsequent case the same class of symptoms existed : the man was pale, and in addition to severe neuralgia he suffered from headache and tinnitus aurium : the chloroform was consequently not employed.

Marriott's case is, I conceive, the most important I have given ; it points out the necessity of a careful inquiry into the

less manifest symptoms, and indicates the class of cases in which chloroform is injurious.

In all cases where the neuralgia is due to, or attended by, cerebral affection, chloroform and belladonna are contra-indicated.

In my paper in the *Gazette* of Feb. 18, I traced the progressive action of chloroform, ether, and alcohol on the brain, and classified thus their increasing effects :—Cerebral excitement ; cerebral disturbance ; cerebral sleep, sopor, the pupils contracted ; cerebral death, coma, the pupils dilated.

At p. 433 of the *Medical Gazette* for March 10, Dr. Miller, of Louisville, relates an interesting case where phrenitis with delirium, resulting in death, was caused by the repeated inhalation of ether at an evening party.

It may be laid down as a fixed principle, that ether and chloroform are contra-indicated in persons labouring under cerebral disease, as both these agents act injuriously on the brain.

In those cases where the neuralgia is caused by disease in the nerve itself, or by the pressure of diseased bone or other diseased tissue—in those cases, indeed, in which, from their very nature, the affection must be unyielding to any treatment, chloroform can only afford very temporary relief indeed—probably only during the time of unconsciousness ; in such cases belladonna or aconite may possibly be of service.

The inhalation of chloroform is especially indicated in those cases of neuralgia that are due to the reflex morbid sensation[1] excited by disordered stomach and bowels, exposure of the skin to cold, and other like causes.

[1] In the same way that an impression on a distant nerve may excite through the spinal marrow, and through a motor nerve, reflex motion, so an impression on a distant nerve may excite in a sensory nerve reflex sensation. The spinal system of nerves have excito-sensory as well as excito-motory functions.

In conclusion, I would insist on the importance of a careful inquiry into the whole of the symptoms before deciding on the use of chloroform in neuralgia, and on the difficulty of estimating the beneficial or injurious effects of chloroform, or indeed of any other remedy, on the system and on the disease, as the two following instances illustrate. On questioning a female after the use of chloroform for neuralgia, I found that the pain had ceased just before the administration. In this case, without close inquiry, the relief might have been ascribed to the chloroform. A man, suffering from neuralgia, applied the other day to have the chloroform, he having heard of its good effects. He had a carious tooth, the possible cause of the neuralgia. The chloroform was withheld that the tooth might be extracted, and other means be used. While he was waiting he was attacked by a severe rigor. In this case, had the chloroform been administered, we should unquestionably have attributed the rigor to its action.

XXIII.

ON DEATH FROM CHLOROFORM.[1]

THE death from chloroform of Mr. Badger, related in the *Lancet* of July 8th, is an interesting illustration of the effect of enlarged liver on the space for the lungs and heart, and on respiration and circulation. The liver in Mr. Badger weighed eight pounds, and the summit of it was behind the third intercostal space. The liver evidently pressed upon the heart and the lungs, interfering with their function. In this case the heart was flaccid and somewhat fat. The flaccid state of the heart showed that the death was immediately caused by paralysis of the heart's action: there were clots of dark grumous blood in both cavities. The lungs were not materially congested.

Since the death from chloroform of Greener, near Newcastle, three additional fatal cases have unfortunately occurred. They are all well recorded, especially in the post-mortem appearances, and we are, consequently, better able to say from experience what is the immediate cause of death from chloroform, what treatment ought to be pursued when death is imminent, what precautions ought to be adopted in the administration, what class of persons ought not to be submitted to its action, and in what cases its employment cannot be sanctioned.

In the lower animals, from the experiments of Dr. Percy, Dr. Glover, and Mr. Wakley, the first fatal effects of chloroform,

[1] From the *London Medical Gazette*, vol. vii. 1848, p. 108.

ether, and alcohol, are usually the cessation of respiration, the heart's action continuing for some minutes. I say usually, for Dr. Percy and Dr. Glover both noticed that, in some dogs, the respiration and the heart's action ceased simultaneously.

One continental experimenter found that, by artificial respiration, he could always restore the lower animals when animation was suspended by the action of chloroform.

If anything can restore the human subject when animation is suspended by excessive chloroformization, it is artificial respiration. The experiments on the lower animals hold out much hope of success by this means; but, unfortunately, from the four recorded cases, I fear that, in man, even artificial respiration will seldom succeed.

In the case of Greener the lips became suddenly blanched; blood would not flow freely from the arm; the lungs were excessively congested; the heart quite healthy, containing in both cavities dark fluid blood. It is not stated whether the heart was flaccid.

In Maria S., the fatal case at Boulogne,[1] the last appearances of life were two deep and laborious inspirations. The lungs were healthy, not congested, except at the depending part. Heart flaccid, like an empty bag; fat; cavities quite empty. Fluid blood black as ink in the large veins. Large bubbles of air were found everywhere in the veins; one or two ounces of bloody serum in the pericardium. Artificial respiration was adopted.

In Mrs. Simmonds, the American case (who enjoyed excellent health), whilst inhaling in a chair, the *face became pale;* respiration and pulsation ceased about the same time. About half an hour later electro-magnetism caused muscular contractions, but no effect on the heart's action. Artificial

[1] *Lancet*, June 6th.

respiration and other means were used. Limbs rigid ; lungs not intensely congested, bronchi being stained with blood. Pleura highly injected. Heart flaccid ; all its cavities and the great vessels entirely empty; their inner surface deeply stained. Only a little blood in the abdominal cava. Six drachms of bloody serum in the pericardium. Some bloody serum in both pleural cavities and in the right hypochondrium.

In the case of Mr. Badger the heart was flaccid, and contained coagulated blood.

A man died at Auxerre last August under the influence of ether.[1] In him the pulse and the respiration ceased about the same time. The countenance just before death was deeply livid. The lungs posteriorly, and the bronchial lining throughout, were highly congested; blood fluid ; state of heart not specified.

In the three later fatal chloroform cases the heart was quite flaccid. In the case of Greener the state of the heart is not specified, but the countenance became suddenly blanched. In all the four cases it is manifest the immediate cause of the instantaneous death lay in the heart. The heart, influenced by the poison, ceased to contract, not from the cessation of respiration, for the heart in asphyxia will beat from one to three minutes after respiration has ceased, but from immediate death of the heart.

There is no doubt a combination of causes operating to destroy the heart's contractile power : the mental influence, the congestion in the systemic and that in the pulmonic capillaries, will all have a material influence. In poisoning by prussic acid in a dog, Dr. Lonsdale found the heart's action ceased with respiration ; the heart was distended ; on puncturing the cava the heart renewed its action. In poisoning by tobacco a similar state occurred to Sir Benjamin Brodie. By artificial respiration he kept up the action of

[1] *Gazette*, March 10th, 1848.

the heart, which had been renewed by local stimulus after its complete cessation.

But, besides these causes, all co-operating to arrest the heart's action, there is indisputably the direct action of the poison on the muscular tissue of the heart. The poison penetrates to the heart from the lungs in a single pulsation; and at the beginning of the next systole, the blood is sent through the coronary artery to the whole muscular tissue of the heart. The blood passing into the coronary artery is less diluted—is more strongly impregnated with chloroform—than is the blood in any other part of the system, except the lungs. The experiments of Dr. Simpson, Mr. Nunneley and others, have shown the action of chloroform to be local. Those of Allston, Fontana, Whytt, and Monro, in the last century, completely demonstrated the local action of opium. Dr. Whytt destroyed the contractility of the frog's heart by steeping it in a watery solution of opium.

I fear, from the experience of these fatal cases, that we must regard chloroform as one of the most uncontrollable narcotic poisons when its action is pushed so far as to suspend *circulation* and *respiration*. It is very manifest, that in the American and Boulogne cases the heart's action was not arrested from its over-distension, as it was in the cases of poisoning by prussic acid and tobacco previously cited. In both those cases the heart was absolutely empty of blood. A question arises here, Was the heart arrested from want of blood? The experiments of Dr. Kay and others have proved, that any limb or muscle will be paralysed if it be deprived of blood. Indeed, the action of rhe right side of the heart, which usually continues long after that of the left has ceased, will be the first to cease if the left side be supplied with blood while the right is deprived of it.

It is very possible, that in these cases the want of blood

for the heart to act upon had to do with its ceasing to act; this would be due to the arrest of the blood in the lungs and in the system generally, and the supply of blood to both sides of the heart.

It is, however, to be remarked, that in both of these cases the blood was quite fluid; that in both of them there was bloody serum (about an ounce) in the pericardium; that in the American case there was a deep red stain on the interior of the empty heart and great vessels. In these cases I do not doubt that some blood was in the heart at the time of death, but made its way out by imbibition and otherwise, owing to its fluidity, during the twenty-four or thirty hours that intervened between death and the examination.

In ordinary death, the right ventricle, which contains blood, is flaccid, while the left, which is quite empty, is rigid. This proves, that the flaccidity of the walls of the heart in the cases under review was not due to the empty state of the ventricles.

We are obliged then, from the experience of these cases, to conclude, that in man the death is usually instantaneous, and due, as every instantaneous death is, to paralysis of the heart. In animals, the death is usually due to paralysis of the muscles of respiration.

It is chiefly owing to the superior control of the mind over the body in man, that in him the poison acts on the heart more than in dogs. It is from the same cause that opium, which so usually produces convulsions in the lower animals, so seldom produces them in man; and it is for the same reason that opium produces convulsions so much more frequently in children than in adults.

These cases suggest some important considerations on the *mode of chloroformization*. In three out of the four fatal cases the chloroform was given in the sitting posture. This

posture requires much greater power in the heart to carry on the circulation than the recumbent. Chloroform should not, if possible, be administered in the sitting posture.

In three out of the four fatal cases, the chloroform was administered by the operator: this should never be. Chloroformization is the exhibition of a subtle poison, and ought to be watched by its administrator with undivided attention during the whole of its operation.

During chloroformization, the state of the eyes, the lips, the pulse, and respiration should be continually watched. Since my paper on the action of chloroform, in a former number, I have never in any case, however prolonged the operation, allowed the action to proceed so far as to cause dilatation of the pupil.

As soon as the eyes turn up, and the eyelids cease to quiver and resist, draw up one eyelid, and keep the eye constantly open; watch the pupil closely—it is usually contracted, and ought never to proceed to dilatation, excepting, perhaps, in the reduction of dislocation and in the reduction of hernia. If the eyeball begin to move, and the eyelids to quiver, apply the inhaling mask again for a few seconds until they again become fixed: thus, with the inhalation of very little chloroform, a person may, at will, be kept long under its influence, and yet not a minute longer than is needful, as you have the patient just on the margin of unconsciousness.

The inhaler should be so constructed that every inspiration be made palpable by it. The tell-tale valve of my inhaler does this perfectly, and may be, and indeed has been, adapted to other inhalers. Without some such precaution, the patient might cease to breathe unnoticed.

The chloroform should be administered gradually, much diluted with air at first, and less so afterwards. The effect

should neither be produced too quickly nor too slowly; in either case, the accumulative effect pointed out by Dr. Snow may endanger the patient after the chloroform has been withdrawn.

If the respiration ceases before the pulse, artificial respiration must be immediately resorted to: it may be performed instantly by breathing into the lungs through the inhaling mask, described in a previous paper.

If the heart has ceased to beat, the case is almost hopeless. If the veins of the neck be swollen the right cavities of the heart are distended, and an ounce of blood taken from the jugular may relieve the distension of the heart, and lead to the renewal of its action. Under any circumstances, artificial respiration should be resorted to as the last resource.

In each of the four cases,[1] the operation, though painful, was not serious. In such cases, the mind usually fears the chloroform more almost than the operation. It is otherwise when the operation is serious.

In dental surgery (except in extreme cases) and in trivial operations, the use of chloroform is not justifiable.

As the heart is subject to paralysis from the action of chloroform, its use should not be lightly resorted to when there is affection of the heart. I do not speak so much of organic disease of the heart as of those cases where palpitation

[1] Since this was written, a fifth case of death from chloroform (in India) has been reported in the *Gazette*. Like the four other cases, the operation was trivial; like three of them, chloroform was administered by the operator himself, and in the sitting posture. During the operation scarcely a drop of blood escaped. The patient was probably already dead! There was no post-mortem. I observe that the Boulogne case is reported anonymously. One cannot, therefore, confide in the post-mortem report of that case; but if it be shut out altogether from this paper, it will not affect the remarks contained in it. The authentic account of that case in the *Gazette* fully justifies the editorial remarks on it.

and dyspnœa are easily excited, either from abdominal distension or from mental emotion. To such persons chloroform is, I conceive, more likely to prove destructive than to those with organic disease of the heart, when they do not suffer from palpitation. Mr. Walshe, of Worcester, has shown, that the dread of a serious operation often does more harm than the operation itself; and hence the real value of anæsthesia is often not so much to save the immediate pain, as the bad effects of the dread of the pain. This, which applies to persons enfeebled by disease, applies with equal force to persons the subject of heart disease, and they, when the dread of a severe operation is great, may sometimes be peculiarly benefited by the careful and *short* production of anæsthesia during the *cutting* part of an operation.

It is to be hoped that Mr. Robinson, to whom the profession owes so much for his communication on ether and chloroform, will favour us with an account of Mr. Badger's case. It would appear, from the evidence of the servant, that the mask was never brought in contact with the face: this may be a mistake from imperfect observation. It would be interesting to know from Mr. Robinson whether this was so; and whether he finds that the effects of chloroform can be usually thus induced. One disadvantage in Mr. Robinson's mask is the inability to admit pure atmospheric air through the mask: for this he appears to make up by not bringing the inhaler in contact with the face. Another disadvantage is the want of a tell-tale valve.

These fatal cases, unfortunate in themselves, will be of service in checking the employment of chloroform in trivial cases, and in persons well able to bear an operation, and in drawing attention to the proper mode of its administration; but they ought not to interfere with its judicious use in severe

operations, when the patient, from long-continued disease or the shock of an accident, is unable to bear the pain and shock of an operation. The employment or non-employment of chloroform is a balance between two evils.

These cases ought not to interfere with the judicious medicinal use of chloroform. In neuralgia and chorea it is not necessary to push the chloroformization to the extent of unconsciousness. As in such cases the full effect ought not to be induced, it is perfectly safe to administer the chloroform in the sitting posture, the head being supported.

XXIV.

ON THE NARCOTIC POISONS, PARTICULARLY OPIUM, AND THEIR ANTIDOTES.[1]

It is my object in this, and the following papers on the same subject, to develop the results of an inquiry into the therapeutics of poisoning by narcotics, and more particularly by opium. That the most proper means of treating such cases are as yet either not agreed upon, or not generally known, is evidenced by the great variety of practice and the frequent want of success in their treatment. The importance of the inquiry is shown by the very great number of persons that annually fell victims to poisoning by narcotics, especially by opium.

In the years 1837-1838 the deaths by poisoning amounted to . . .	543
Of these, the total number poisoned by opium, laudanum, morphia, and opiate cordials were	198
A number greater than that by arsenic	185
Poisoned by other narcotics	44
Viz. :—	
By prussic acid	27
Oil of bitter almonds	4
Nux vomica	3
Strychnia	2
Belladonna	2
Carburetted hydrogen	2
Hemlock, monkshood, wolfsbane, and gin, of each 1 . . .	4

These returns[2] show that the largest proportion of cases of

[1] From the *London Medical Gazette*, vol. vii. 1848, pp. 578, 792.
[2] *Medical Gazette*, xxv. 284, and Taylor on poisons, 186.

poisoning in this country are by opium, exceeding even those by arsenic.

There can be no doubt, as Mr. Taylor remarks, that the number of deaths from poisons which annually occur in England and Wales are much greater than this table represents.

I may add, that this remark, applicable to all kinds of poisoning, is especially applicable to poisoning by opium, that drug being used so extensively by the ignorant, and acting so silently, and with so many of the appearances of natural death. The extent to which this is so may be surmised from the fact, that of the 198 cases poisoned by opium, 106 were either from overdose or by mistake; 64 of the remainder being suicidal, and in three only was it "wilfully administered." So long as this is the only country in Europe where the sale of poisons is indiscriminate, we must expect that the number of persons poisoned by opium will be immeasurably greater in this country than in any other.

Since opium is the preponderating cause of death from narcotic poisoning in this country, I shall devote the chief portion of these papers to an inquiry into the action of opium as a poison, with the view of ascertaining the best means of averting its poisonous and fatal effects.

On the Local Action of Opium.

Before endeavouring to ascertain the action of opium on the complicated human organism, I shall inquire into the evidences of its local action on separate portions of the animal organism.

During the last century, especially towards the latter part of it, many of the great physiologists of that day busied themselves with this very question, of the local action of

opium and other agents. Amongst these were Whytt, Monro, Fontana, Alston, Valli, and Humboldt.

The numerous experiments and observations of Humboldt[1] convinced him that opium, like other stimuli, exhausts only in consequence of excessive excitation. He exposed muscle shortly after its removal from the living frog to oxymuriatic acid: the effect was first to stimulate, and then to exhaust, muscular contractility. This exhaustion may be removed, he found, by opium, which re-excites and then again exhausts contractility.

"This exhaustion may be removed (he found) by oxymuriatic acid or oxide of arsenic, while opium also is capable of removing the inexcitability produced by them," p. 272. I ought to state that these are the words employed in the copious abstract of Humboldt's work in the *Annals*—the previous details being inferred, rather than actually extracted from the statements in that abstract.

From the observations, then, of Humboldt, and of Michælis quoted by him, we may infer that the action of opium, on the direct application of it either to nervous or muscular tissue, is first to augment, and then to exhaust their excitability.

These inferences are corroborated by the more recent experiments of Dr. Wilson Philip (on the Vital Functions, 133), who observed, that when opium or tobacco are applied in very small quantity to a muscle, they tend to excite muscular action; in larger quantity they immediately destroy the muscular power. They produce these effects in the hollow muscles, as the heart and the intestines, chiefly when applied to their internal surfaces. They produce the same effect when applied locally to either the nervous or the sanguiferous systems.

In all these cases the stimulant effect of the opium is more

[1] *Annals of Medicine*, iv. 223-271.

considerable than that of the tobacco, and the sedative effect of the latter is more considerable than that of the former.

These observations of Dr. Wilson Philip differ in this respect from those of Humboldt, that while the latter noticed that opium first stimulates and then exhausts excitability, the former noticed that the application of a small quantity of the poison immediately excites, whilst that of a large quantity immediately exhausts, excitability. He does not state that the opium first excites and then exhausts.

Humboldt's and Wilson Philip's experiments taken together, illustrate the whole question, first in the application of a small quantity or dose—that is, in a therapeutical point of view—a small dose of opium being a true stimulant, as Sydenham said, almost the only true stimulant; while the application of a large quantity or dose exhibits the poisonous action.

Humboldt's experiments show that these two opposite actions are not really opposed to, but are dependent upon, and consecutive to, each other; and that, when the application of a large quantity of opium appeared to be immediately followed by exhaustion, that exhaustion was in reality preceded by the excitation, just as lightning, in destroying vital contractility, primarily and violently excites it.

Valli (on Animal Electricity, 73) was much puzzled on finding that when he applied opium to the isolated tibial nerve of a frog, the excitability of the muscles of the leg was in some experiments destroyed, and in others increased. These experiments are explained by those of Humboldt and Wilson Philip. The cases in which excitability was increased were evidently in the earlier stages, and those in which it was destroyed in the latter stages of the action of opium.

The great experimenters before alluded to afford ample and interesting illustration of the local effect of opium in destroying excitability.

Whytt immersed the heart, still pulsating, of a frog in water, and another pulsating heart in a watery solution of opium. The heart in the watery solution ceased to pulsate before the heart in water.

In ten minutes both hearts were taken out: they were then motionless. That which had been in the watery solution of opium could not be stimulated to contract, and it never moved again; that which had been in water could be stimulated to contract—and in a few minutes it of itself resumed its pulsation.[1]

Monro poured ten drops of a watery solution of opium underneath the skin among the muscles of the left thigh. After ten minutes that leg seemed to be weaker, and in ten minutes more the muscles lost their power, and the toes had little sensibility: the animal seemed now to be a great deal stupified, and its heart gave now only twenty-five strokes in a minute. An hour and a half after the beginning of the experiment, the toes seemed to have quite lost their sensibility, and the muscles their motion; but the animal jumped by the help of the other hind extremity. Two days thereafter this leg had recovered both its sense and motion, and the animal seemed quite well.[2] Fontana says,[3] "I plunged the body of a leech into a watery solution of opium, and found in a little time that this part had lost all motion, whilst the other half continued in action. I looked upon it as something very extraordinary, that one half of the creature should become dead, whilst the other half continued in the state of not having undergone any change or suffered any injury."

The interesting experiments detailed above prove that almost every and any organ of the body may be affected by the local action of opium; and that the organ affected by it has its excitability first increased and then exhausted.

[1] *Physical Essays*, vol. ii. [2] *Physical Essays*, xiv. 827.
[3] *On Poisons*, ii. 364.

If the poison be applied to the voluntary muscles, or to the heart or intestines, the muscular contractility is first excited, and then exhausted. If it be applied to the individual nerves, or to the whole limb, sensation and motion are first excited, and then paralysed.

There is now no occasion to bring forward proof that the narcotic poisons as well as all other soluble substances, when received into the stomach or into the rectum, and in some animals, and with some agents, when applied to the skin, pass into the circulation.

As the opium enters the blood when it acts upon the system, it necessarily follows that the opium admitted into the circulation is carried with the blood to every organ and portion of the frame. Every part of the organism is subjected to the immediate and characteristic influence of the poison; and, as Whytt says, opium destroys by rendering the several organs insensible to the stimuli destined by nature to excite them.

As the opium when admitted into the system is first diffused through and applied to the whole circulating apparatus, I shall first inquire into the effect of opium on the circulation in the capillaries, arteries and veins.

The Effect of Opium on the Circulation.

Dr. Allston, that he might observe the effect of opium on the circulation in frogs, performed the following beautiful experiments, which he thus details:—

(*a*) "In the physic garden at Holyrood House, I one evening put a strong big paddock into a pot of water, wherein a small quantity of opium was dissolved; it soon appeared to be uneasy, by making strong efforts to get out of it, but in

a short time it flagged or grew dull, making very little motion, and next morning it was dead, and much swelled." This experiment proves that opium can enter the circulation through the integuments, and so destroy life. This had not hitherto, I believe, been demonstrated.

(β) "Assisted by Mr. Robert Fullerton, a curious gentleman, and very dexterous in microscopical observation (in August, 1733), I conveyed, through a small glass tube, a few drops of a solution of opium in water into a frog's stomach, and putting the animal into a glass cylinder, adapted it so to a good microscope that we had a distinct view of a part of the membrane betwixt the toes of its hinder foot, where the circulation of the blood may easily be seen. My design was, since I found opium killed frogs, to observe if there was any visible change made by it in the blood itself, or in its motion; neither of us could, indeed, see any alteration in the blood as to its consistence, colour of the serum, magnitude, figure or colour of the red globules; but we very distinctly saw a surprising diminution of the blood's velocity, for it did not move half so swiftly as it uses to do in these creatures. We alternately looked at it again and again, and in less than half an hour saw the velocity of the blood gradually increase, the uneasy frog recover its wonted vigour, and the blood its common celerity; upon which we took out the paddock, put it in a bason of clean water, and allowed it half an hour to refresh itself—then gave it another dose of opium—fixed it to the microscope with all expedition—and viewed it as before. The blood then moved yet slower than it did the first time, and its velocity gradually decreasing, at length it stagnated, first in the smaller, then in the larger vessels, and in about a quarter of an hour the animal expired.

"One thing was very observable all along—viz. that notwithstanding the diminished velocity of the blood, there was

no sensible diminution of the frequency of the pulse: yea, when there was no circulation or progressive motion of the blood in this part, the pulse was visible by an undulatory motion—that is, the blood returned as far back at every diastole of the heart as it was protruded by the preceding systole; this continued till the frog was quite dead, or at least appeared to be so. When we had lost all hopes of its recovery, I opened it, and found nothing in its stomach but a clear mucus like a jelly, a little coloured with the opium of which it was full: everything else seemed perfectly natural. This experiment we frequently repeated, and it had always the same appearance and event. The recovery, however, of one of the frogs which for a considerable time seemed to be dead, is not to be omitted. My friend and I one evening killed as before, a couple of frogs with opium: one of them, which was the strongest, I laid half in water on a tile in the bottom of the water-pot, that if it recovered it might sit either wet or dry as it liked best; the other I left on the earth, dry, under a hedge. Next morning when I returned to the garden, I found the one under the hedge dead, as I left it; but the other in the water-pot was alive, and appeared to be in perfect health."[1]

These two very interesting experiments, which for their real illustrative value, in elucidating the action of opium on the system, as well as for their historical interest, are well worth repeating here, I combined in one experiment, which I devised in the following manner. The experiment is easily performed:—

Experiment.—I attached a frog to Mr. Goadby's frog-holder, so that either web could be placed under the microscope. I plunged the left leg into a test-tube containing a watery solution of opium—the right leg into one containing

[1] *Medical Essays*, v. 130.

water. The tubes were so arranged, that they could be withdrawn, and either web be placed under the microscope without disturbing the frog.

Before the immersion of the left leg into the solution of opium, the circulation was very rapid : the corpuscles in the arteries shot past so rapidly that they could scarcely be distinguished. Those in the veins and the large capillaries moved rapidly, while those in the small capillaries moved slowly.

After the left leg had been immersed in the solution of opium for ten minutes, the motion of the blood in the smaller capillaries of that leg was quickened, and the blood circulated through many capillaries previously devoid of corpuscles. The movement of the blood in the artery and vein was less rapid. The circulation in the right leg was not altered.

After a further immersion of ten minutes the circulation in the left leg was further modified, that in the right leg being not perceptibly changed.

After the left leg had been replaced for half an hour, the frog was again observed. Whenever the skin was touched, either on the *left* or *right* leg, the frog cried out in a peculiar manner, the creature being universally convulsed. The skin was touched repeatedly, and in rapid succession, with the effect of producing convulsions, which became less and less strong each time they were excited. At length the convulsions could be no longer excited by touching the left leg, and after a time they ceased also to be excitable in the right leg. After a little rest the convulsions could again be excited.

The left leg was swollen, being evidently more vascular than the right.

The capillaries in the left leg were now much enlarged

several corpuscles moving slowly, side by side, through capillaries that were previously empty. The blood moved much more slowly both in the arteries and the veins.

The circulation in the right leg was now very perceptibly modified, and as nearly as it could be observed, to the same extent as that in the left leg was affected after being immersed in the solution of opium for ten minutes.

After a re-immersion for an hour and a half, quick feeble tetanic spasms of both limbs were excited by the slightest motion—by walking across the room, or touching the microscope, or by touching the skin of either leg. These convulsions ceased when the legs were touched alternately and in rapid succession : the left leg first lost its excitability, and then the right. The convulsive motions were the least in the left leg.

It was found during the last observation that the animal was quite unconscious, and had ceased to breathe.

The capillaries were now very much enlarged in the left leg, being greatly distended and almost blocked up with the accumulation of blood corpuscles, the motion of which was but just perceptible. The movement of the blood in the arteries and veins was exceedingly sluggish. The right leg was similarly affected, but the capillaries were not so much distended, and the circulation was not so slow, as they were in the left leg.

The circulation became progressively slower, and convulsions were no longer excitable. About four hours after the first immersion in opium the heart was exposed, pulsating slowly, emptying itself on each contraction, and receiving and sending out but little blood. After the heart was cut out, the movement of the blood in both webs continued, though it was very sluggish, and in the left leg was only observed in the large artery and vein.

Alston's and Monro's Experiments proving that Opium can be absorbed into the Circulation through the Skin of the Frog, and that it can only act on the whole Organism through the medium of the Circulation.

1. I particularly solicit a re-perusal of the very interesting experiments by Dr. Alston, and the experiment by myself, in which I combined both of those by Dr. Alston, in the foregoing pages.

Before studying the effect of opium on the circulation, I shall inquire into the proofs that that poison, and indeed other poisons are received into the circulation, wherever they are applied, before they can act on the system.

In Dr. Alston's first experiment, he put a frog into a pot containing an aqueous solution of opium, and the result was that next morning the frog was dead.

This experiment tended to prove that the opium might penetrate and poison the system through the skin. Dr. Monro remarks, that in this way of making the experiment, the water with the opium might enter the mouth. To obviate this objection, and to prove that the animal is affected in the same way, whether the opium is applied inwardly or outwardly, Dr. Monro performed the following experiment:—

"About a hundred drops of a watery solution of opium were applied on scraped linen to the skin of the belly and hind legs of a frog. After two hours the animal began to be convulsed and extended, and the blood had entirely ceased from motion in the hind legs. After three hours it was more violently convulsed, and unable to move its body out of the place where it lay. After five hours it showed no

outward sign of life; and though the solution was removed, and the legs washed, the animal did not recover." [1]

I tried the plan adopted by Dr. Monro, but did not observe the results described by him until I plunged one leg into a test tube containing a solution of opium as I detailed in the previous paper. Dr. Monro's and Dr. Alston's experiments, of which my own is merely a modification, prove that animals "may be affected by the absorption of opium, and its mixture with their circulation."

That he might ascertain whether opium could act on the whole system through the medium of the nerves alone, Dr. Monro performed the following experiment:—

"I cut all the organs at the pelvis, except the large nerves that go to the hind extremities. Then I tacked the hind legs by threads to the trunk of the body, to prevent the nerves from being over-stretched or torn; after which I injected thirty drops of the solution of opium under the skin of both hind extremities.

"Ten minutes thereafter, the hind legs were less sensible, and much weakened, and, in a quarter of an hour more, they were quite insensible and motionless; yet the fore part of the body was not observably affected six hours thereafter, and the animal lived until the next day."

This experiment proves that opium applied to the hinder limbs will not act on the anterior part of the body, when the current of the circulation is cut off, although the nervous connection be preserved.

The following experiment by Monro is the reverse of the last one: in that he divided the vessels, leaving the nerves intact; in this he destroyed the spinal marrow, leaving the vessels intact:—

"I cut the spinal marrow across at its middle, and then

[1] *Physical Essays*, iii. 305.

destroyed, with a red hot wire, the hind half of it; and, twenty hours after this, applied about a hundred drops of the solution, on scraped linen, to both hind legs. After three hours, the animal cried when the fore part of it was touched. After four hours it began to be convulsed, and the blood had ceased from motion in the hind feet. After eight hours it showed no outward sign of life.

"This experiment demonstrates that opium can affect animals universally after all communication of the nerves to which the opium is primarily applied, with the rest of the nervous system, is cut off by destroying the origin of those nerves, or cutting them through in their progress. Whence it follows that the opium must have produced these deleterious effects by being absorbed and conveyed with the blood to organs of the body distant from those to which it was primarily applied." [1]

I make no apology for introducing these experiments and remarks; so clear are they, compact and conclusive, that they spare any extended reference to the many valuable and more recent experiments which go with them to establish these laws—that narcotics may be absorbed into the circulation from the external surface, and that they can only act on the *whole* organism through the medium of the circulation.

Whytt's Experiment, apparently opposed to the Universality of the last Inference.

2. Dr. Whytt details an experiment—it was confirmed by Dr. Monro,—which apparently upsets the universality of this law, that poisons can only act on the whole organism through the medium of the circulation.

[1] *Loc. cit.* 337.

"A frog continued to move its limbs and leap about for above an hour after I had cut out its heart, and was not quite dead after two hours and a half.

"Five minutes after taking out the heart of another frog, I injected a solution of opium into its stomach and guts. In less than half an hour it seemed to be quite dead, for neither pricking nor teasing its muscles produced any contraction in them, or any motion in the members to which they belonged!" [1]

Fontana repeated and contrasted this experiment in the following manner:—"I made twelve frogs swallow each about twenty drops of the aqueous solution of opium, and instantly separated the heart from the thorax. I opened the thorax in twelve others, but did not remove the heart: all of these, as well as the others, had previously swallowed opium. I noted the time of its action on all the twenty-four, and found that the effects of the opium discovered themselves much sooner in the frogs, the heart of which remained in the thorax, than in those from which I had removed it. The difference in time was more than one half."

The above experiments of Whytt's, I own, puzzled me for a long time, as it apparently contradicted what was otherwise so completely established. But I imagine that the difficulty may be got over thus. From the observations of Dr. Wilson Philip and others, and as was instanced in my own experiment detailed in the previous paper, the movement of the blood through the capillaries, from the arteries to the veins, continues for some minutes after the removal of the heart. By imbibition some of the poison would probably find its way into the aorta, and thence to the capillaries of the limbs. By imbibition also the poison would come into direct contact with the internal organs and possibly with the

[1] *Physical Essays*, ii. 282.

limbs. At all events, Fontana's repetition of, and contrast to, this very interesting experiment, show that the poison acts much more speedily when the heart is not removed, and the circulation is entire.

Absorption into the Circulation from the Stomach and Intestines.

3. The absorption of narcotic poisons into the circulation through the skin, so conclusively proved by the above experiments, is necessarily much less rapid than the absorption from the stomach and intestines into the circulation.

Pannizza, quoted by Matteucci,[1] injected a small portion of strong hydrocyanic acid into a small portion of the small intestine of a horse ; the blood which was immediately afterwards taken from the vein returning from the intestine, was already charged with hydrocyanic acid.

Solutions of most of the organic salts, and several of the inorganic salts, have been detected in the urine some time after being received into the stomach. Dr. Percy detected alcohol in the blood and urine after the injection of that fluid into the stomach. Monro says, "I applied camphor to the hind legs of two frogs for an hour and a half, and then I cut off the hind legs and the whole of the skin ; and on opening the abdomen I distinctly perceived the smell of the camphor in it, and after infusing the bowels in spirits and in water, I plainly perceived its taste in those liquors." For the details of the chemical proofs, that inorganic salts and some organic salts have been discovered in the urine after being received into the stomach, I refer to Simon's Chemistry, Dr. Day's translation, and to Dr. Taylor's work on poisons.

From the physiological proofs detailed above, and from the

[1] *Sur les Phenomènes Physiques des Corps Vivants*, p. 76.

chemical proofs referred to, it may be laid down as a received law, that narcotic poisons are received into the circulation, whether they are presented to the stomach and intestines, the skin, or the lungs, and that it is only through the medium of the circulation that they can act on the whole system.

On the Treatment of Narcotic Poisoning by the Elimination of the Poison from the Blood.

4. I shall scarcely be premature if I insist here on the importance, in a therapeutical point of view, of recognising habitually and practically that every poison is diffused through the whole circulating mass of the blood, so long as the blood is taking into itself fresh supplies of poison, whether from the stomach and intestines, the rectum, the skin or the lungs (as in the inhalation of ether and chloroform); and that the blood is constantly and necessarily eliminating from itself the poison diffused through it; and that every means should be resorted to, short of exhausting the vital energies of the patient, to hasten the elimination of the poison from the blood, through the usual channels, the kidneys, the skin, the liver and intestines, the salivary glands, and when the poison is vaporisable (prussic acid, ether), the lungs.

This is not the place to enter into details on what may be termed the treatment by elimination, but a recognition of the importance of that branch of the treatment here is well, as it fixes the attention on the broad fact, that poisons are detectable by the chemist in the liver, the lungs, the brain, the heart, the kidneys, the blood itself, the bile, the urine, the serous fluid in the serous cavities, and that their removal during life from each and all of these places in their aggregate from the whole system, may be materially aided by a rightly directed system of treatment.

Having gained a practical recognition of the fact, that the poison is admitted into the circulation, and carried by it through the whole frame, the ground is prepared for an inquiry into the effects of the poison on the circulation.

Action of Opium on the Capillaries, Arteries, and Veins.

5. In the experiment performed by myself, detailed in the former paper, and which was a modification of those of Allston and Monro, I found it of very great advantage to have the animal so arranged on Mr. Goadby's frog holder, that I could withdraw the test tube containing the solution of opium, or that holding water, from either of the legs respectively immersed in those fluids, and examine the progress and changes in the circulating currents, without in the least degree disturbing the creature, and so modifying the flow of the blood.

The first change observed was manifestly a dilatation of the capillaries: these capillaries which were previously so small that the corpuscles could only press slowly through in single file, each thinned, lengthened, and bending in the narrow tube, were now so much enlarged, that the corpuscles moved quickly through them; and the blood circulated through many capillaries previously too small to admit corpuscles. The opium was in contact with, and impregnated the texture of the capillaries, and the evident effect was a yielding of their walls, and the necessary result the enlargement of the area of the capillaries. While the blood in the capillaries moved more quickly and extensively, that both in the arteries and veins moved manifestly more slowly. In the progress of the experiment, the capillaries became much farther distended, their enlarged walls yielding to the *vis a tergo* from the heart; the quantity of blood in them was much

increased, several corpuscles moving side by side in capillaries that were previously empty; and the motion of the blood, both in the arteries, capillaries, and veins, was very materially diminished.

The cause of the diminished motion in the arteries and the distended capillaries is rendered clear by an experiment of Dr. C. J. B. Williams, which proves that "flaccidity, and increased length and size of a tube, afford impediments to the passage of liquid through it." Dr. Williams attached a tube with two arms to a syringe; to one arm was fitted a brass tube two feet long, having several right angles in its course; to the other arm was fitted a portion of rabbit's intestine, four feet long, and of calibre (when distended with water) double that of the brass tube. The tubes were both filled by successive strokes of the piston; and when they both began to discharge, the quantity discharged from the small brass tube was from two to five times the quantity from the larger but membranous tube.

The experiment proves, that the increased tortuosity, and number of vessels in a congested part, the greater mass of their contents, and the atonic flaccidity of their coats, do truly form additional obstacles to the passage of blood through them.[1]

This experiment explains perfectly certain of the successive phenomena observed in the circulation of the foot of the frog which was immersed in a solution of opium. When the coats of the finest capillaries became flaccid, they admitted corpuscles freely, although, under exactly the same pressure, many of them admitted none before. As the capillaries increased in size, and their coats in flaccidity, the increased mass of corpuscles in them, having still only the original amount of pressure from behind, moved forward much more slowly, a great portion of their moving force being expended

[1] *Principle of Medicine*, p. 144.

in expanding the yielding walls. The arteries, as well as the veins, partook of the diminished motion : the former, because the blood in them had to push before it a greatly increased mass of corpuscles ; the latter received less blood from the swollen capillaries, because a great part of the blood which they received was detained in them, and because less blood entered them from the arteries. The phenomena are in part illustrated by the rapidity with which soldiers in single file can pass through a narrow doorway, which, on the other hand, becomes almost blocked up by the striving pressure of a disorderly crowd.

It was very interesting to remark, during the progress of the experiment, that the circulation in the capillaries of the limb in water, which was at first unchanged, became gradually affected precisely in the same manner with the circulation of the limb immersed in opium. The changes were exactly the same in character, but they were at any given time less in amount, even to the end of the experiment.

This change in the circulation of the limb in water, was manifestly due to the opium, which, having found its way through the circulation, over from the left limb to the right, produced on that limb the same characteristic effects. The opium acted on the capillaries of the left or medicated limb, to use Fontana's expression, from without, while it acted on those of the opposite limb from within, their walls being bathed with the blood holding in it the opium in solution.

Perspiration in Poisoning by Opium.

6. From this observation, we learn that opium causes congestion in all the systemic capillaries ; and we may infer with certainty that the same effects extend to the pulmonic capillaries, in which the resistance to the circulation is probably

greater than it is in the systemic capillaries. There is one interesting, and in a therapeutic point of view, important symptom in poisoning by opium, which is referable to the congestion induced by it in the systemic capillaries, and that is, profuse perspiration, which usually breaks out over the whole surface of persons under the poisonous influence of opium. Neither morphia nor meconic acid have as yet been detected in the perspiration; but there can be no doubt that the perspiration is one of the channels by which the poison is eliminated from the system. I am, indeed, convinced, both from the consideration of the matter, and from experience, that it is of very great importance to promote by external warmth copious perspiration in cases of poisoning by opium. I need scarcely say, that this must not interfere with other steps in the treatment, and must not be pushed so far as to exhaust the system; and that the perspiration must not be allowed to remain on the surface, so as to chill it by evaporation.

Opium is not the only narcotic poison that produces congestion in the capillaries; indeed, I feel assured nearly all, perhaps all, the narcotic poisons have the same effect, though the effects are doubtless somewhat modified in each instance. It would form an interesting and valuable series of experiments to observe the effect of each of the narcotic poisons on the circulation in the web of the frog's foot, adopting in each the plan employed in the experiment detailed above.

Monro, in fact, adopted this plan with alcohol and with camphor; Dr. Wilson Philip with tobacco (I have not seen details of his experiments); and Mr. Wharton Jones with carbonic acid gas, directing a stream of it on the web of the frog's foot: in all these instances (I cannot speak with certainty as to Dr. Wilson Philip's) the circulation was retarded and checked by the action of the poison.

The experiments of Mr. Nunneley, of Leeds, show that the invariable action of prussic acid is to cause congestion of the capillaries of the surface to which it is applied; and Professor Simpson has observed the same with regard to chloroform. The invariable effect of chloroform and ether, when it has penetrated from the lungs into the systemic capillaries, is at first to produce active congestion of those capillaries, as may be always witnessed on the conjunctiva.

Dr. Blake has made some interesting experiments, which exhibit, by a different mode of inquiry, the resistance to the circulation in the systemic capillaries. He observed by the hæmodynamometer that in animals poisoned by injecting tobacco, digitalis, and euphorbium into their blood, that the pressure on the hæmodynamometer is about double: this increase in pressure appears to be due to the resistance to the circulation in the capillaries.

Obstruction to the Circulation in the Capillaries caused by other Narcotic Poisons.

7. In addition to the narcotic poisons just instanced, the following showed their effect in increasing the volume of the blood in the capillaries, and obstructing the capillary circulation by certain symptoms during life:—

Belladonna—in many of the recorded cases the face, and in some the whole surface, was rendered red.

Stramonium—the face was reddened in the majority of cases.

Hyoscyamus, I believe, has the same effect, from its general analogy to the action of belladonna, but I cannot meet with any note of such a symptom in an analysis of cases poisoned by it.

In one or more instances the following symptoms were excited by the following poisons :—

Cicuta virosa	the face was bloated.
Œnanthe crocata . .	the face was bloated and livid.
Aconite	the face was swollen.
Strychnos	the face was swollen.
A poisonous fungus .	the face was swollen.

It may be considered, then, that the following narcotic poisons cause congestion in the capillaries :—opium, alcohol, ether, chloroform, carbonic acid gas, prussic acid, tobacco, digitalis, belladonna, stramonium (hyoscyamus), cicuta virosa, œnanthe crocata, aconite, and strychnos.

Asphyxia, producing Obstruction to the Circulation in the Systemic Capillaries.

8. In asphyxia there is congestion and obstruction in the pulmonic, and also in the systemic capillaries. This congestion is of the same character, alike, indeed, in its essential phenomena, with that produced by opium and the narcotic poisons ; and in Mr. Wharton Jones's experiment, in which the capillary circulation in the web of a frog's foot was retarded or even checked by directing on it a stream of carbonic acid gas, it may be said that local asphyxia was produced.

It has long been understood that in asphyxia the pulmonic capillaries are obstructed, but Bichat was, I believe, the first to point out the obstruction in the systemic capillaries as an essential feature in the chain of morbid changes, instancing in support of this view the fulness and livid colour of divers parts, such as the face, tongue, and lips, and also putting it partially to the test of experiment by exposing the mesentery in the living animal during asphyxia.

Dr. Reid found that in asphyxia, there was increased

distension in the arterial system, as indicated by the hæmodynamometer, evidently due to the resistance to the circulation in the systemic capillaries. Mr. Erichsen has observed under the microscope the resistance to the circulation in the capillaries during asphyxia. I repeated his experiment, and found where the air was shut off from the lungs by a stop tap in the trachea, that the circulation in the arteries and veins, which was previously quick, became gradually slow, and that the flow of blood through the capillaries was at first increased and then obstructed: on again permitting respiration, the circulation speedily quickened.

In asphyxia it appears that the obstruction is, in part at least, due to the increased size and adhesiveness to each other of the red corpuscles ; those being the changes effected by carbonic acid on those corpuscles, according to that accurate observer, Mr. Gulliver. Besides this, the tonicity of the capillaries is doubtless diminished, and their relaxation consequently increased, by the continued presence of unoxygenated blood. May we not say, and say truly, that asphyxia is, in part at least, the action on the capillaries of a narcotic poison diffused through the blood?

Inflammation, producing the same effect.

9. The phenomena presented by the circulation in the capillaries in congestion and inflammation are very similar to those in narcotic poisoning and asphyxia. For interesting details on this subject, I refer to Dr. C. J. B. Williams's *Principles of Medicine.*

XXV.

REMARKS ON THE FEVER OF NOTTINGHAM AND THE NEIGHBOURHOOD, THAT PREVAILED IN THE SUMMER AND AUTUMN OF 1846, AND WAS IDENTICAL WITH THE "FIÈVRE TYPHOIDE" OF PARIS, AND DIFFERED FROM THE USUAL WINTER TYPHUS OF EDINBURGH AND DUBLIN.[1]

DURING the last five months, from the beginning of July to the 29th of December, 223 patients with fever were admitted into the General Hospital near Nottingham, and 676 fever cases were treated as out-patients. This number of fever cases, 899 in six months, is enormous, especially if contrasted with the number of cases usually admitted into the institution. The total number of fever cases during the years ending Feb. 28th, 1844, 1845, and 1846, being respectively only 54, 43, and 36.

It is not my intention to give a scientific analysis of this fever; indeed, I want the materials for so doing. My aim is to give a general account of the symptoms and pathology of the fever, its causes, the varying phases of it as the weather and the *locale* of the fever changed, and its analogy and contrasts to the epidemic fevers of other places and seasons.

The fever was characterised by abdominal disturbance; a want of energy in the nervous system, sensation being blunted, the muscular actions enfeebled, and the intelligence

[1] From the *London Medical Gazette*, vol iv., 1847, p. 59.

either torpid or disturbed ; and by a feeble, often quick pulse. It was ushered in by a succession of rigors, each followed by heat of surface, ending usually in perspiration. Uneasiness and disturbance of the abdomen, and diarrhœa, were almost universal. The tongue varied much ; it was in some cases moist and pale, or furred, being always more red and clean at the margins ; in others its surface was red and glazed, either moist and of a shining smoothness, or dry as if varnished, being often marked by shallow fissures ; in other cases, and these were of the worst class, the tongue was dry, fissured, coated with a dark brown film ; in such cases sordes collected on the teeth.

The countenance always bore the stamp of the disease, having the expression in every feature of blunted sensation, feeble nervous energy, and sluggish intellect. The lips quivered ; the cheeks, nostrils, and eyelids, were tremulous ; the tongue was protruded slowly, and was in motion over the whole surface. The eyes were usually dim, and the hearing dull ; the limbs were feeble, and the movements slow. Headache was frequent, and thirst, though present, was not extreme.

A great proportion of the cases were mild ; some of them, indeed, ephemeral ; but they all partook of the same general type. In all of them there was abdominal derangement.

The worst cases were marked by extreme debility, partial unconsciousness, muttering delirium, great abdominal distension, and profuse diarrhœa—often of dark, offensive, sometimes of bloody stools. Certain of the cases—and these, which were of the most intractable sort, were fortunately but few in number—lay coiled up, almost insensible, uttering constantly, night and day, at each expiration, a loud, dismal, crying moan. The pupils were dilated. The abdomen was distended ; diarrhœa alternated with constipation. On

emptying the bowels, these cases were, for a short time, less noisy; but these intermissions were short and imperfect. Out of four such cases—they were boys—one recovered after keeping up incessant noisy cries for about a fortnight. Opium and henbane in repeated doses, combined with other treatment, seemed to silence the cries of this boy. In these cases there were ulcerations, though to no unusual extent, of the aggregate glands in the ileum. Vomiting, epistaxis, and bleeding from the gums, cough with bronchitis, hoarseness, loss of voice, and redness of the fauces, were frequent complications. One poor fellow could scarcely breathe for œdema of the epiglottis and glottis.

Of the 223 cases admitted, 19 have proved fatal. In 18 of these that I examined, the Peyer's follicles in the ileum were enlarged in 17, in 13 of which there were ulcerated patches of greater or less extent. In three of these cases there were ulcers on the mucous coat of the colon; and in one remaining case there were ulcers on the colon, although the Peyer's follicles appeared healthy. In many of these it was noticed that the mesenteric glands were much enlarged and softened.

The spleen was usually normal in size and consistence.

The lungs were gorged and solid at the posterior part in many of the cases, and in four there were patches of pneumonia.

The brain was normal in the majority of the examined cases, even when the head symptoms had been severe. In a few the brain was congested; in others there was slight subarachnoid effusion; and in one case, in whom there was a subdued maniacal delirium, with strabismus, the membranes were unusually dry, and the arachnoid at the base of the brain was thickened and opaque.

In one case there was pleuritis, with a thick, soft, new membrane over the lower lobe of the right lung. In another

case there was pleuritis combined with pneumonia of the right lung.

Two women died after abortion; in one peritonitis was present.

During the first two months nearly the whole of the cases came from two circumscribed districts in two large populous manufacturing villages within a mile of Nottingham. These districts Dr. Hutchinson and Dr. J. C. Williams, physicians to the hospital, and myself, examined with great care. In one of these districts, New Lenton, the fever prevailed in certain yards in which the privies were open to the rain and sun, and in certain streets that were neither paved nor macadamized, and in which the channels were a succession of small filthy pools. Adjoining yards, in which the privies were closed, and an adjoining clean well-drained street, were quite free from fever. This district was on a rising ground, being about sixty feet above the level of the flat of the river Leen, in the meadows of which river were fœtid, half stagnant, open ditches. These ditches were polluted by the decomposing vegetable refuse from some starch works, and by offensive animal matter from a skin yard. Close to these filthy ditches fever cases prevailed, as I was informed by Dr. Hutchinson and Dr. Williams. In one yard in Radford, the other district alluded to, in which were five privies, the filth from one of which ran into the yard, out of forty inhabitants twenty-one were attacked with fever.

In no previous summer to this, for many years, has the heat been so excessive and so early. My friend Mr. E. J. Lowe, whose meteorological observations are published in the quarterly returns from the Registrar-General of Deaths, has favoured me with his accurate meteorological observations for the past summer. From these I find that, during the greater part of June, the mean temperature was from 7 to

14 degrees above the usual mean. After falling below the mean for a month, the temperature rose again above it at the end of July; fell below the mean in the middle of August; was above it by 7 or 8 degrees during the whole of September; and was considerably below the mean during the whole of October.

The excessive heat of June acted to decompose the animal and vegetable matter in the open privies, and in the unformed streets of New Lenton and Radford. Both of the infected districts were offensive to the smell. Diarrhœa prevailed during the month of June, and was the immediate precursor of the fever. The open privies were, in many parts of the districts, the manifest foci of the fever. Sir John Pringle, in his *Observations on the Diseases of the Army*, says, speaking of dysentery in the camp:—"Of this the chief *fomes* seemed to be the foul straw and the privies: for as soon as we left that ground the sickness visibly abated" (p. 27). At p. 103, he states, that "human excrement lying about the camp in hot weather, when the dysentery is frequent," is one of the chief causes of sickness. Elsewhere, p. 353, after showing that ordinary fæces are less, if at all, infectious, owing to the antiseptic action of urine, he says, "The case is different in putrid diseases, and especially in dysentery, where the fæces are highly corrupted and contagious." It cannot be questioned that the diarrhœal fæces that abounded in the open privies immediately before the access of the fever would pollute the atmosphere, when acted upon by the sun, much more than healthy fæces.

It was delightful to see the rapidity with which bad cases rallied, when removed from the polluted districts to the wards of the hospital. As Dr. Cheyne says,[1] "The accomo-

[1] *Dublin Hospital Reports.*

dation and regimen of the house produce a favourable change in the complexion of the disease in a single night." Dr. Pritchard shows how, in the same epidemic, those cases of fever admitted into the large airy wards of one hospital improved much more than those admitted into the small close wards of another hospital. The over-crowded filthy hospitals of conquered armies, crowded and filthy gaols and ships, have ever been generators of fever, especially in hot seasons.

We noticed that the improvement in our cases, so happy at first when they were removed from the infected districts to our wards, was less marked after many cases had been admitted. We found that improving cases often relapsed when additional bad cases entered their wards. When they were removed to purer wards, improvement was again immediate. One patient was removed four times, each time to a purer ward, and each time with marked advantage. Sir John Pringle shows that in a well-aired hospital dysentery did not spread (p. 64); while in a hot crowded hospital the rest of the patients, the apothecaries, and nurses, were seized with the flux, which spread to most of the inhabitants of the village. "To this acceded a still more formidable disease, namely, the hospital or jail-fever, an inseparable attendant of foul air from crowds and animal corruption" (p. 27). Several of our convalescent cases that returned to the infected districts were speedily re-admitted suffering from relapse.

Dr. Hutchinson, Dr. Williams, and myself, joined to certify, under the "Contagious Diseases Act," that the nuisances described were dangerous to life; but the Act we found to be worse than useless. All that it warrants is, the removal of a nuisance; it does not prevent its re-accumulation. The filthy privies were emptied, but the owners were not obliged to close them from the influence of the weather.

The stirring up of the filth caused an intolerable stench, and immediately the number of fever cases increased.

Although many of the cases were from among the very poor, very many were in good circumstances. This Dr. Hutchinson, Dr. J. C. Williams, and myself carefully verified.

We felt that the early cases of fever from Lenton and Radford were conclusive as to the effect of a polluted atmosphere in exciting the fever. The case was simple and easily inquired into; we could put our fingers, as it were, on the very causes of the disease.

In August the epidemic showed itself in a few cases in Nottingham. In September and October the numbers gradually increased, until November, when they rapidly diminished.

I did not inspect those parts of Nottingham from which our cases came; but, as a general result, I know that the filthy ill-drained parts of the town were the chief centres of supply.

My friend Dr. Stiff, one of the surgeons of the Nottingham Union, showed me the places where his fever cases lay. It was remarkable to see how they clustered round exposed privies and exposed filthy drains. The houses close to six of the privies and three of the filthy open drains contained 45 cases of fever; whereas the neighbouring houses distant from the privies, and the neighbouring streets away from the filthy drains, though inhabited by persons equally poor, were comparatively free from fever.

If every fever appearing in this kingdom bore similar characters to this, we should no longer be at issue with the great French pathologists as to the nature of fever. In almost every particular the description of the "fièvre typhoide" contained in M. Louis's admirable analysis of that

disease would answer for the description of this epidemic. In all his cases, as in all ours, there were diarrhœa and abdominal distension; in every autopsy with him, as with us, with one exception, the Peyer's follicles were enlarged, and frequently ulcerated. The mesenteric glands were enlarged in all his cases, and they were so in those of ours in which they were noticed. In his experience, as in ours, epistaxis, vomiting, and inflamed fauces, were frequent complications. The rose-coloured pimples that he found seated on the abdomen I observed in 5 or 6 out of 15 or 16 cases. In these great features our epidemic and the "fièvre typhoide" were identical; the essential difference between the two epidemics being, that in M. Louis's experience, the spleen was enlarged in 36, and softened in 34 cases out of 46; whereas with us, of ten spleens that were weighed, four weighed six ounces, and the rest from five to three ounces. In one or two of our cases the spleen was softened and enlarged. While this epidemic was identical with the fever prevailing in Paris, in every essential, except enlargement of the spleen, it differed in the same essentials, along with the "fièvre typhoide," from the winter typhus of Edinburgh, Dublin, and London. Drs. Henderson and Reid, in their report of the typhus in Edinburgh,[1] state that the elliptical patches of Peyer were elevated in four cases only out of 41; on two of these there were slight ulcerations. In none of them were the mesenteric glands enlarged. Constipation was more frequent than diarrhœa; indeed, the latter was the exception; and the majority of cases—108 out of 130—had either the petechial or the measly eruption to which Dr. Peebles[2] and Dr. Roupell[3] have lately called attention. This exanthematous eruption,

[1] *Edin. Med. and Surg. Journ.* lii. 429. [2] *Ibid.* xliv. 356.
[3] *Ibid.* lii. 490.

resembling that in measles, was observed by the great Sydenham in the winter epidemic fever of 1650.[1]

Our fever, like the "fièvre typhoide," differed from the typhus fever in the invariable affection of the elliptical patches, the enlargement of the mesenteric glands, the prevalence of diarrhœa, the absence of exanthematous and petechial eruptions (we had but one patient with petechiæ) and the presence, in five or six observed cases, of rose-coloured papulæ.

This fever differs both from the "fièvre typhoide" and typhus, in the usual absence of affection of the spleen, that organ having been normal in five only out of 41 cases of typhus: and in the "fièvre typhoide" in 10 cases out of 46.

I do not take notice of the numerous points in which the three epidemics are analogous—analogies so close, that the typhus and "fièvre typhoide" have generally been confounded. Alleix, Shattuck, and Lombard, quoted by Louis,[2] have noted the chief differences, and Dr. Stewart[3] has ably analysed many of them.

As so strong an analogy, amounting almost to identity, exists between the epidemic of this neighbourhood and that of Paris, we must look for some analogous influences to produce them. The analogy of seasons this year is as striking as the analogy of epidemics. We have had the summer of a warm climate. We have had this summer, in common with all Europe, through the world-pervading influences developed by Humboldt in his great work *Cosmos*, and in his earlier works.

The diseases of hot and warm climates differ from those of temperate and cold climates, in that affections of the liver, stomach, and intestines, prevail in the former, and those of

[1] *Schedula Monitoria*, sec. 5. [2] Louis, *Fièvre Typhoide*, ii. 315.
[3] *Edin. Journ.* liv. 289.

the lungs in the latter. As the summer of this country is analogous to warm climates, so are the characteristic diseases of our summer to be classed with the characteristic diseases of warm climates.

I have extracted from the Report of the Registrar General the following comparative statement of the number in summer and winter of those diseases characteristic of the summer season, occurring in London in 1841.

	Summer Quarter.	Winter Quarter.
Diarrhœa	477	79
Dysentery	71	22
Cholera	101	0
Gastritis	410	206
	1059	307

During the same year there were of chest diseases, excluding phthisis—

Winter Quarter.	Summer Quarter.
3088	1067

It is unfortunate that the Registrar-General groups all fevers under the common head of typhus, combining, as we shall see presently, the opposite forms of fever of winter and spring, and of autumn.

As the fevers, as well as the diseases of warm countries, differ in their type from those of cold countries, so do the fevers as well as the diseases of autumn differ from those of spring.

Through MM. Louis, Chomel, and others, we know that fever with affection of Peyer's patches exists in France, and through various American pathologists, quoted by Louis, that the same exists in New England.

Dr. Cheyne states that ulcers on the Peyer's patches prevailed in all cases of the fever that appeared in the autumn

of 1816, and lasted till December.[1] In January such cases gave place to fevers in which the bowels were never affected.[2] Dr. Law, speaking of the Dublin epidemic of 1836, says: "Thus in winter and summer have we had it accompanied by bronchitis." Gastro-enteritis was then not seen. "In summer and autumn the epidemic assumed more the character of gastro-enteritis."

Dr. Christison states that affection of the Peyer's patches is more frequent in London than in Edinburgh.[3] Sydenham informs us that dysentery and dysenteric fevers, which were, I feel assured, often identical with our present autumnal fever, appeared in the autumns of 1669, 1671-2 (iv. 3, 2, 4, sec. 6), and in autumn 1675 (v. 4, sec. 5). The dysentery and diarrhœal fevers of autumn gave place, in winter, to measles in 1670 (iv. 4), 1674 (v. i), and 1676; to a winter fever, with chest affections, in 1673 (v. 1), and 1675 (v. 5), and in 1685[4] to a fever referred to above, which was doubtless identical with our typhus, having, like it, petechiæ and an eruption like measles. It is perhaps worth remarking, that measles, the eruption of which is so closely simulated by that appearing in typhus, was, in Sydenham's experience, invariably a winter epidemic. This is an illustration I conceive, of Sydenham's law, that "Whatever distemper prevails over the rest will be found to preside over them during that year, and to the disposition thereof all the then reigning epidemics accommodate themselves so far as their nature permits."[5]

Not only may the form of fever characteristic of warm

[1] *Dublin Hospital Reports.*
[2] *Dublin Journal of Med. Sc.* xii. 178.
[3] *Libr. of Med.* i. 139.
[4] *Schedula Monitoria.*
[5] Swan's Translation, i. 2.

countries appear in Britain in warm seasons, but the typhus fever of Edinburgh closely tallies with a fever observed by Dr. Peebles in Leghorn, [1] in the winter of 1817, brought on by the same causes that so often give rise to the typhus of Edinburgh and Dublin; namely, cold and want of food; aiding these causes, contagion farther produced the disease which was prolonged far into summer.

In New York and Philadelphia, a fever identical with the "fièvre typhoide," usually prevails.[2] Coventry and Willoughby describe, in the *New York Medical Journal*, iii. 9, iv. 204, the symptoms of this fever in New York and the Lake Country. Dr. Coventry attributes the fever in the half-reclaimed forest lands, and the drying swamps, to the dead decaying vegetable matter; in New York, to the moist and fœtid land reclaimed from the sea. Dr. Graham,[3] after stating that the "fièvre typhoide" is frequent at Philadelphia, describes a fever occurring in the winter and spring and cold summer of 1836, that tallies with the British typhus. In only one autopsy out of about fifty was affection of the glands of Peyer observable. Not only may the fever, then, that prevails in warm climates appear in temperate climates during the warm season, but the fever, also, of cold climates, and of the cold season in temperate climates, may appear in warm climates, when the seasons are unusually cold, and food unusually scanty. Sydenham observes, "that some diseases happen indiscriminately at any time, whilst many others, by a secret tendency of nature, follow the seasons of the year with as much certainty as some birds and plants." (Preface, ii)

The fever, accompanied with affection of the mesenteric glands and Peyer's patches, takes its rise in autumn in this

[1] *Edin. Journal*, xliv. 356. [2] Louis, *Fièvre Typhoide*, i. xvi.
[3] *Dublin Journal*, xii. 148.

country, and chiefly in summer in warm countries. The present epidemic, in the opinion of all of us who saw the source of the disease, certainly arose from the foul condition of the atmosphere due to the rotting of vegetable and animal matter. This poisonous effluvium is highly appreciable by the smell; it is undoubtedly drawn into the lungs at each inspiration, and whether it consists of animalculæ, as Drs. C. J. Williams and Henle suppose, or of particles of decomposed matter, like musk, too subtle to be detected by our present means of investigation, the effluvium certainly penetrates with the air, through the lungs into the blood, and is carried by the blood to all the nerves and to every part of the frame. Tainted food and depressing influences, and other causes, doubtless fit the frame to yield to the poison.

The epidemic was manifestly contagious in this institution. Most of the nurses, and two of the pupils, were attacked. I believe contagion acts in like manner with the original effluvia; indeed, the effluvia from the body impregnate and poison the air which is breathed into the lungs; contact may favour the action, but one nurse who slept away from the fever cases, though she suffered from bronchial affections, did not take the fever.

Through the influence of contagion the winter typhus is prolonged into summer, and the autumn fever is prolonged into winter; the two forms of fever thus overlapping each other.

Dr. Southwood Smith justly observes in his work on Fever (p. 41), that "the fever of one country is not the same as the fever of any other country; in the same country the fever of one season is not the same as the fever of any other season; and even the fever of the same season is not the same in any two individuals."

It is to the co-operation of causes that we must look to

unfold this varied aspect of fever. Blending with the pollution of the air, or with cold, or contagion, are many other causes co-operating to produce fever. Tainted food, mental depression, want of cleanliness, organic debility of constitution, previous disease, and many other causes, combine to engender fever.

Need we marvel that different epidemics and the fever in different individuals in the same epidemic present along with identifying analogies characteristic differences? Let any one element co-operating with other elements in the production of fever be omitted, or be present in different proportions, and the whole resulting effect must be modified.

While the invariable occurrence of affection of the aggregate glands, and of the mesenteric glands, exists both in the fever with which we have just been visited, and in the "fièvre typhoide;" I feel with Dr. Hodgkin "that this pathological condition is not the essence and cause of fever, but that febrile disturbance being produced, these glands are the parts particularly disposed to become the seat of local affection, as a secondary event."[1]

In this fever I conceive that the poisonous effluvia, being inhaled, pass into and gradually accumulate in the blood. The polluted blood circulating through the system acts both on the extremities of the sentient, the motor, and the secretory nerves, and on the nervous centres. Sensation is blunted, the muscular powers are enfeebled, the mind becomes torpid, influenced to this torpor chiefly by blunted sensation. The mental disturbance, the delirium, often more resembles a dream than true delirium. Rouse the patient from his muttering torpor, recall him to sensation, and for the moment he is sensible.

As Dr. Hodgkin has put forward, I believe, that fever

[1] *Lectures on Morbid Anatomy*, vol. i. p. 485.

consists "in the suspension, or, at least, very considerable interruption, of that process by which, during health, the various parts of the system are continually undergoing a change, the old materials being removed, whilst others are substituted in their place."[1] Secretion is defective, the materials that ought to be cast off by the urine, the perspiration, the bile, and probably the lungs, accumulate in the system. As Dr. C. J. B. Williams says, "the distinctive materials of the secretions of urine and bile appear to poison the system if not separated from the blood. The sudden suppression of the urine causes typhoid symptoms."[2] In fever, the flow of urine is diminished, the proportion of urea in the urine is lessened, and in Dr. Henderson's cases, in the remarkable Scotch epidemic of 1843, urea was detected in the blood. As in fever, the food is not properly assimilated, and animal heat, on the principle developed by Chossat, is not properly generated. Long-standing and severe cases of fever often perish from deficiency of animal heat, unless the heat be artificially sustained. It is manifest that whatever cause or combination of causes may arrest or impede the secretions may cause fever.

By following up the labours of Sydenham, of Hecker, of Louis, of the many historiographers of fever, by studying closely the phenomena of the various epidemics, along with the personal, the local, and the great cosmical influences, we shall after long labour with certainty discover the main causes of epidemics.

I think we are justified in concluding that the present epidemic is nearly identical with the "fièvre typhoide" of Louis. I think it may also be inferred that the autumnal fever of this country is identical with M. Louis's "fièvre

[1] *Op. cit.*, p. 490.

[2] *Principles of Medicine*, p. 80.

typhoide," and that this fever is cut short by cold weather, just as the form of fever,—typhus, caused by cold, want of food, contagion, and other such influences—is often cut short or exchanged for the enteritic type by warm weather.

It was very interesting to note, with the changes in the weather, the changes in the secondary disorders incident to the fever. When the weather became colder, early in October, bronchitis, engorgement of the lungs, or pneumonia appeared in many of the cases.

Scurvy has been banished from our ships; gaol and hospital fevers from our gaols and hospitals; we either prevent or mitigate the small-pox by vaccination; by the draining of our marshes, ague has almost become in England, like the plague, an historical disease. These revolutions, which have annihilated so much disease, have mainly been the noble work of medical men. They are now adding to this good work the removal of many of those causes that engender autumnal fevers, and the diseases caused by a tainted atmosphere; and medical men combine with statesmen in showing the need of food and warm clothing for the prevention of the winter typhus fever, and diseases of an allied class.[1]

I intended to have made a few remarks on the treatment which was followed by the physicians of the Hospital, Dr. Hutchinson, Dr. J. C. B. Williams, and Dr. Storer, but that the interest and extent of the subject have led me on to a length far beyond my original intention. In conclusion I cannot refrain from expressing a hope that other observers will make known the phenomena presented by the present epidemic elsewhere, which I have reason for believing is extensively spread over a great part of England.

[1] There is an able analysis of the British and French Fevers in Dr. Forbes's *Journal*, xii. 292.

XXVI.

ADDRESS IN MEDICINE.[1]

MR. PRESIDENT AND GENTLEMEN,—Our age is marked by experiment and exact inquiry, directness of aim, and the skilled power to do the work required of us with completeness and economy. The labour of the past is surpassed, but not superseded; and while nothing that has been done is lost, invention awakes invention, discovery discovery. Each advance is a fresh starting-point for the future labourer. It is, indeed, everywhere taken for granted that good as this or that work may be, better work, more simple and more to the purpose, remains before us.

The ship, the bridge, the rail, the telegraph, and the gun, of the present day, as compared with those of the past, are types and marks of the skill, precision, and advancing energy of the time. Nature, closely questioned, unfolds herself to us more and more every year. Force and matter, working together and moulded by science, produce new forms, and show powers hitherto unknown.

Medicine, too, partakes of this movement that is going on all around us. The knowlege of disease is becoming, at the same time, more accurate and more large. Each year gives us a better knowledge of what remedies can do, and what they cannot do. If the spirit of scepticism has shaken the belief of a few in the medicinal means at our command, that

[1] Delivered before the British Medical Association, 1873.

spirit has aroused inquiry, and converted belief, which is shifting, into knowledge, which is secure.

Those great old forms of medicine, the tincture of the muriate of iron, the sulphate of quinine, the iodide of potassium, opium, the infusion of digitalis, that have served our fathers well, serve us better. We know what they can do, when they are wanted and when they are not wanted; and with gathered power we apply them at the proper moment.

Then science adds to those recently discovered remedies of the day just gone by, chloroform and bromide of potassium, the newly-discovered agent of this very day, chloral. So our knowledge of disease ripens, and our aims in its treatment become more precise; and men, all men, ask themselves at each step they take, why they do this or that; reason takes the place of routine, and rational medicine becomes the common property of the profession.

Side by side with the use of medicine, and not second to it, is the so-called hygienic treatment of disease—the study and regulation of the vital forces. The influence that the physician exercises over the mind, and through the mind over the body; the soothing or stimulation of the nervous power; the calming of exaltation, or the stirring up of apathy; the quieting of the over-busy brain, or the spurring of the flaccid will; the repose of over-used powers, or the awaking of suspended vital functions; the subduing of the over-sensitive skin, or the stimulating of it when wan, muddy, and lifeless; the limiting of supplies to the over-fed frame, or the repair of the body wasting from sickness by the proper kinds of food and stimulants; the bringing into play, and so again into existence, muscle that after being paralysed by disease has shrunk from disuse: these are among the aims that the physician seeks to accomplish; and these are among the methods which he seeks to employ irrespectively of, but by no

means without the use of, medicine. These are among the agencies that you hold in your power in the treatment of disease, and that you, each of you, exercise daily in coping with the various forms of malady, of ailment, and constitution.

There is a method of treatment, that of rest and ease, belonging to this great class, that I have been employing with deep interest in the treatment of acute rheumatism and acute gout, for some years. I think it not impossible that, as this subject interests me so nearly in its practice, it may interest you in its telling. I shall, therefore, at once proceed to narrate to you my experience in this method of treatment.

Rest and Ease.

During the last four years I have submitted all my patients in St. Mary's Hospital, affected with acute rheumatism and acute gout, to a rigid system of absolute rest, protection from external injury, gentle pressure, equal warmth, and the removal of pain, chiefly by treatment from without. Those two diseases, so often apparently identical, differ essentially, as you know, in this—that, while acute rheumatism attacks those whose blood and tissues are previously healthy, and is produced by overwork and exposure, acute gout seizes upon persons whose blood and tissues are already affected, uric acid being present in excess. In acute gout, therefore, I gave iodide of potassium, and sometimes colchicum, in the hope of getting rid of the special poison. But in acute rheumatism I gave no internal medicine during the active stages of the disease, unless it was called for by some special reason. I gave my patients no coloured or flavoured liquid to make them think they were taking medicine when they were not doing so. I do not think it quite right; and I do not find it needful to employ such a system of fiction. If we do so, we complicate the observations, and we deprive ourselves of the

help that the patient gives us when he understands the aim of the method of treatment.

Whatever may be the line of treatment adopted for disease, the influence of that treatment on the disease itself is less than the physician is apt to think. The great majority of acute diseases tend to get well. They have, so to speak, a lifetime of their own, with its periods of growth, maturity, and decline. They are the passing tenants of the body which they occupy, often with great injury, for a limited time. Treatment cannot change their nature—cannot expel them at once—cannot quench them—cannot materially shorten or prolong their existence. But treatment can lessen the sufferings of the body occupied by disease, shield it from outer injury, repair its waste, and support and reinforce its powers; it can quicken the sluggish action of organs whose duty it is to clear out the accumulating poison from the blood; and it can ward off those causes that tend to increase or reawaken the disease, and lessen the intensity of its action, inflammatory or otherwise, especially upon the local structures. To watch, then, the treatment of a disease is to watch, not, so to speak, the remedy and its immediate effects, but the disease itself, and its behaviour during a certain method of treatment.

The thoughtful physician, while taking note of this or that change in the malady, knows that such change is due mainly to the natural growth of the disease, and does not attribute it to the means which he has employed, unless he have grounds for doing so. Having, however, a complete knowledge of the natural changes through which the disease passes from its beginning to its end, he is able, with something like precision, to say that the plan of treatment pursued has prevented the undue development of the affection, and has lessened its evil effects on the body. In studying, then, the progress of these diseases, acute rheumatism and acute gout, under this method

of treatment—by rest and ease—we are studying the disease itself, and the swaying influences upon it that this method may induce.

In the examination of these cases I shall avoid all comparison of them with those of the same diseases observed by other physicians, and even with those previously treated by myself in the wards of the hospital. How can you compare one set of cases with another set ? Each patient has his own character, and his disease presents its own peculiar features. Cases unduly severe or unwontedly mild, are apt to come in groups, extending often over a long period. The strict comparison of one series with another becomes therefore impossible.

From the summary placed in your hands, which contains a short narrative of each patient, you will see that during the two years and a half ending May, 1868, I have treated 101 patients on this method ; and that of these seventy-four were affected with acute rheumatism, twenty-three with acute gout, and four with one or other of those diseases, their characters being doubtful. During a considerable period my colleague, Dr. Cheadle, joined me in the examination of these patients.

It would be out of place for me to give you here a rigid analysis of those cases ; but, taking certain points of interest that present themselves in the series, I shall consider the affection of the joints, and the inflammation of the heart, in its interior and on its exterior, keeping steadily in view the influence of the treatment by rest and ease on the disease.

The Joints.—In a large proportion of the cases of Acute Rheumatism the affected joints soon lost their pain and diminished in size, after the patients were placed in bed and the inflamed parts were drenched with belladonna and chloroform liniment, and surrounded by cotton-wool, with a covering of flannel pinned over it so as to make a comfortable

amount of pressure. Over all the cradle was placed to keep off the weight of the bed-clothes, the toes being raised when the ankles were affected, so as to take the pressure of the tendons off their inflamed sheaths. The joints were usually at once relieved by the application of the chloroform, the belladonna keeping up that relief; and, as a rule, the swelling of the structures and the increased amount of synovial fluid diminished steadily from day to day, as you will see in the table at p. 25, illustrating this point, placed in your hands. The facts speak for themselves. In one man (411, Henry T.), the knee lessened from $15\frac{1}{2}$ inches on the fourth day to 14 inches on the sixth; and in another (424, Thomas F.), from $14\frac{1}{2}$ inches on the first day to $12\frac{1}{2}$ on the seventh. I need not mention to you other equally notable instances to the same effect, for they are printed on the table in your hands. I cannot, however, forbear naming one more case that excited some interest in the wards. In this patient (444, Jane C.), Mr. Salaman, now assistant-surgeon in our Indian army, made careful measurements of the joints without disturbing them. The right knee lessened in size more than two inches from the first day to the fourth; and, what is more remarkable, the right wrist measured ten inches on the seventh, and less than eight on the eleventh day.

Sometimes the pain and swelling of the joints remained, or even increased, raising the temperature, distressing the patient, preventing sleep, and increasing the action of the heart. Then the application of leeches, or the injection under the skin of morphia, was employed, often with great relief, and with the effect, especially after the application of leeches, of lowering the heat of the joint and the temperature of the whole body, and so of calming the action of the heart.

The inflammation of the joints entirely ceased within eleven days in almost one-half, and within twenty-one days

in five-sixths of those patients that were not affected with endocarditis, and in those who were only threatened with it.

But the inflammation of the joints was much more prolonged and tedious in a large proportion of those affected with inflammation of the heart, whether within or without. Thus in less than a third the joints were free from pain before the eleventh day, while in almost a half the pain lasted beyond twenty-one days. Of these last one-half experienced a relapse, which was often brought on by the too early use of the limbs after they were freed from the fetters of pain. In every case, with the renewal of the inflammation there was a return of the high temperature, the latter being undeniably caused by the former.

The illustration that I have given you becomes more striking when put into simple figures. Of 27 cases affected with endocarditis, the joints continued to be inflamed in 13 after the twenty-first day; while only 5 were so affected out of 33 who had no endocarditis, or were only threatened with it, as you may observe in the table placed in your hands.

This brings me face to face with the pregnant truth, that if we would treat the heart successfully we must treat the inflamed joints successfully. By doing so we remove four distinct causes, each of which requires from the heart a larger and more rapid supply of blood, and increased force of action. We lessen the amount and rate of the blood sent to the inflamed parts; we diminish the temperature of the body, and often the amount of perspiration; we assuage the pain that of itself frequently gives violence to the beating of the heart; and we remove that mental distress, that anxious sense which oppresses, and may even overpower, the action of the organ.

The pivot upon which the treatment of acute rheumatism turns is the lessening of the labour of the heart; for when

that organ is inflamed or tends to be so, each increase in the force it employs may kindle or stir up the inflammation, which may, on the other hand, be subdued or prevented by giving rest and ease to the affected limbs, and so calming the action of the heart.

Before I leave the joints, I must touch upon that class of cases that present the great difficulty in treatment—those, namely, that do not tend to get well, that cannot be classed with acute gout—and that yet differ altogether in their behaviour from those that happily form a large majority of the patients affected with acute rheumatism.

Of the 74 cases of acute rheumatism, in 19, or over one-fourth, the joints were inflamed for more than twenty-one days. In one-half of them there was a relapse, which was usually traceable either to the over-use or the exposure of the joints too soon after the passing away of pain and swelling. I find that this relapse tends to come on if the patient use the limbs before three or four days have elapsed after the temperature of the body has reached its standard, and after all the joints are free from pain. I now lay down a rigorous rule of the ward founded upon this observation; and of late these relapses have been much less frequent among my patients. And here it is that you must enlist the patients in the treatment. They nearly all desire to use their limbs, and to get up as soon as the pain has left them. When, however, you make them understand that if they do so too soon they will have a relapse, and that, if they have, the case may become much more tedious, they cheerfully submit to the discipline.

But, besides the relapsing cases, a set of patients, amounting to one in seven of the whole, retained the joint-affection for more than twenty-one days.

The younger the patient the more rapid was the recovery

of the joints. Thus, of those at or under the age of 20, in one-half the joints were well within eleven days; while of those between 21 and 25, only one-fourth were so within that time. The scale turned after the age of 20; and, while in one-third of those between 21 and 25, the joints were still affected after the twenty-first day, they were so in only one-sixth of those who had not passed that age.

With increasing years there is a greater tendency to prolonged affection of the joints, and to relapse in those affected with acute rheumatism. I have felt that these prolonged cases, while they belong to the acute rheumatism group, are partially allied to acute gout, and that they tend to develop into that disease. This view is supported by the fact that several of my patients affected with acute gout, whose habits were not such as to engender the disease, had suffered from acute rheumatism when quite young.

I have for some time tended to treat these cases, so soon as they declare themselves, in the same way as I treat gout, giving the iodide of potassium, or sometimes the bromide, accompanied by tartrated iron, with, I think, good effect.

The cases of acute gout, on the whole, did well—all, in fact, but one, who died with double valve-disease and contracted granular kidney. These patients, like others, derived immediate relief from the use of the belladonna and chloroform liniment, the application of cotton, and the protection of the cradle. While, however, those affected with acute rheumatism gained comfort from a moderate amount of pressure over the joints, made in the manner already described, those affected with acute gout could only bear very slight pressure. This pressure was always applied so as to be comfortable to the patient, and if it ceased to be so, was immediately relaxed.

All the cases improved during the first few days. The

joints were quite free from pain and swelling in twelve of them in eleven days, and in five more within twenty-one days. But the tendency to relapse in these cases was great, more so than even in those of acute rheumatism affected with endocarditis. Nearly half (eight out of seventeen) of those in whom the joints were well before the twenty-first day suffered from relapse. In some of them the relapse was caused by using the limbs too soon. The tendency to relapse was less frequent in those to whom the iodide of potassium was given early (usually the first day), than in those who took that medicine later, or not at all.

The amount of the secretion from the kidneys increased somewhat when the iodide was given, but not always, as may be seen in the table. It would therefore appear that the amount of good done by the iodide is more than is accounted for by that increase.

The results, then, of the observations of my cases of gout, shortly stated, are these: The joints rapidly improve under the influence of rest and ease, but they tend to relapse even when that method is carried out.

The tendency to relapse is lessened by the employment of the iodide of potassium. It appeared to me that the cases did well when I gave iron with the iodide. It is, therefore, desirable from the very first to combine the two, which I am now in the habit of doing; for iron unquestionably tends to increase the amount of red corpuscles, and we know how deficient they become in acute gout. There is reason also to say that the iodide itself tends to lessen, rather than increase, the red particles, unless by its administration, as in syphilis, the patient is restored to health, and so makes red blood in spite of the iodide. The form that I employ is the tartrated iron. I was desirous of seeing the effects of the iodide on the disease when treated by rest and ease without colchicum,

but the few patients to whom I gave that medicine unquestionably did well, and I feel certain that in these cases it is right to give it for a few days during the acute or inflammatory period.

The Heart.—It will have been seen, from what I have just said, that, when I am looking at the inflamed joints in acute rheumatism, I am thinking of the heart. How can it be otherwise?

You see the limbs powerless; every joint swollen, tender and painful; and the expression of deep helpless suffering on the face. The first turn of the mind is to find relief for that external pain. But you know that, within a given number of days—you may count them on your fingers—those limbs, now so inflamed and shapeless, will lose their suffering, return to the symmetry of their form, and regain their power, and with it the strong desire to escape from their enforced weariness, and to exercise that power.

But you know that, while the outworks are thus visibly attacked, the enemy is already in the citadel; and that this disease, which in its very nature attacks the limbs, in its very nature attacks the heart also, and at the same time that it seizes upon the limbs; that is to say, almost or quite at the beginning of the illness.

In my cases, exactly one-half were affected with inflammation of the heart, and of these, all but three (371, 428, 433) presented signs or symptoms of the affection at the time of admission, while two of the three did so on or before the fourth day after it. In nearly every one of these, that inflammation pronounced itself by the immediate language of the heart itself, by pain in its region, by the anxious expression of the face and its dusky or glazed hue, and by the disturbed breathing. All of these may not be present at once, but they tend to come in their order. And I would here say,

that the murmur made by the blood passing in the wrong direction through the crippled valve is by no means the first indication in point of time.

In most of the cases of endocarditis contained in the paper now in your hands, the murmur was heard from the first. But some of them presented a much more impressive and more certain chain of evidence of the inflammation going on within the heart, than if you at once heard over the organ a murmur without that disturbance of the powers of life that always accompanies the disease during its acute stage.

Let me ask you to look at the case of Mary C. (475, p. 16), On admission there was tightness in the chest; next day she was worse, her face being dusky; and on the third, her expression was heavy, but brighter, and there was no tightness of the chest; but the first sound was prolonged almost to a murmur at the apex, where on the thirteenth day there was a distinct but feeble murmur.

Then take the very next case (Charles H., 402). On his admission, the heart's action was tumultuous; there was uneasiness over that organ and a prolonged first sound, or slight murmur; but, on the fourth day the murmur spoke out, and was heard at the apex of the heart and towards the arm-pit.

In a third patient, Harriet R. (371, p. 15), there was pain over the region of the heart on the fourth day; on the tenth she felt better, and her face was not so flushed as it had been; there was then no murmur, but on the next day one appeared at the apex, showing mitral regurgitation.

Another instance is presented by Martha H. (469, p. 16), whose face was flushed on the day of admission, when she presented reduplication of the first sound at the apex. On the sixth day, mitral and pulmonic murmurs were heard; and on the seventh, diastolic aortic murmur.

It is interesting to note the successive variations of temperature of the body in this patient, which was 101·3 on the fourth day, and fell to 99·3 on the seventh, when all the murmurs came into play.

In all these instances the murmur, which was generated after the cessation of the acute symptoms of inflammation of the heart, disappeared in a varying number of days, and they all left the hospital free from heart-affection. But the result was not so happy in the last case of this class that I shall bring before you. It is that of Mary L. (457, p. 17). On the day of her admission, she was very feverish, breathing was difficult, and there was pain over the heart, but no note of murmur; the temperature was 100. On the third she was less feverish, and there was a prolonged murmur below the heart. On the following day, when the temperature had fallen to 97, there was less pain over the heart, and she was more animated. But, to overbalance this improvement, there was a mitral bellows-murmur, which was even audible, though feeble, below the shoulder-blades.

It is indeed evident that the first effect of inflammation of the valve would not be to induce regurgitation, which cannot take place till exudation is deposited on the valve, or its structure is softened by the inflammation, so as to yield before the pressure of the blood. This prelude of the murmur—of the crippling of the valve—in the form of pain, flushed or dusky face, and anxious expression, is of great practical value, for it enables us to treat the disease in its very infancy, and before it has had time to damage the structure of the valve.

Pain in the region of the heart was present in thirteen, and pain in the chest or tightness in five more, out of the total number of twenty-eight who were affected with endocarditis, the pain, as we have just seen, often preceding the murmur.

This is consistent with what we see in the affected joints, where acute pain is present in the early stage, but tends to lessen when they become swollen. It was customary, when pain showed itself at the region of the heart, to apply over its seat the belladonna and chloroform liniment, with cotton-wool, or a poultice; and if the suffering was great, or was not removed by the liniment, a few leeches were placed over the seat of pain, which invariably produced relief.

These cases were often, so to speak, at first dumb, but their silent suffering told you more than the loudest rasping or bellows sound, that would by itself merely say—Here is a heart not now inflamed, but crippled by a bygone attack—bygone, but leaving its work done on the damaged structure of the heart.

In one-half of my cases the heart was, as I have just said, inflamed; but, in addition to those, three-fifths of the patients in whom endocarditis was not established, presented unquestionable threatening of that disease; so that only a fifth of the whole number stand apart as being absolutely free from inflammation.

But it is not for me in this place to bring the striking phases of this disease before you, who are so practically familiar with them at the bedside. I will, therefore, at once say what was the result obtained in this series of cases, treated in the manner I have described to you. I have just mentioned those cases, amounting to twenty-two, in which there was a threatening of endocarditis. In all but four of these the first sound was prolonged usually at the apex, being, in some of them, murmur-like, or indeed an actual murmur. In twelve of these cases this sign was accompanied by pain in the region of the heart, disturbed breathing, flushed face, anxious countenance, or general illness. The prolongation of the first sound, when present, was generally audible on the

first day, and it disappeared in most cases on the seventh or eighth.

In the four other cases there was pain over the heart, and its sounds were either absent, or unduly loud, or accompanied by pulmonic murmur. In one only were the sounds of the heart absolutely healthy.

Why, then, under these circumstances, do I say that these patients were only threatened with endocarditis, and not actually attacked by it? It is because the suffering and the signs over the heart soon passed away, and the organ was left untouched by disease.

If we turn now to those affected with endocarditis, we shall find that fourteen, or exactly one-half, left the hospital well and free from cardiac valve-murmur. In one or two of them there was a murmur over the pulmonary artery limited exactly to that spot, at the second left space. But I need not tell you that this points, not to valve disease, but merely to too much action, too little blood, and too thin a blood in the right ventricle; and that, as the red corpuscles and the volume of the blood increase, the sound vanishes.

The murmur as it disappeared passed into a prolonged first sound in eleven of the fourteen cases of endocarditis in which the patient left the hospital free from valve-murmur. In three of those cases the murmur was preceded by a prolonged first sound.

These cases show that a prolonged first sound is closely allied to a valve-murmur, not merely by the character of its sound, but also by its actual relation to inflammation of the interior of the heart, whether established, as in the fourteen cases just referred to, or only threatened, as in the twenty-two previously considered.

Of the remaining cases, we held that seven came in with valve-murmur of some standing from previous disease. This

inference was drawn from the loudness, situation, and quality of the murmur, the increased size and force of the ventricle, usually the right one, and the history of previous attacks, accompanied by symptoms over the heart, and followed by palpitation or shortness of breath on exertion. These seven cases came in with valve-murmur, and went out with it.

The only one of the seven that is open to question as to its nature is that of Mary H. (455, p. 467); but, at the time, we made up our minds that she also had old mitral disease, after considering the many previous attacks, the history, and the murmur, changing with the changing force of the heart, for inflammation is told in these cases by this very variation.

Of the remaining seven cases, four presented, after recovery from the attack of acute rheumatism, a faint murmur or prolonged first sound at the apex, very different from the noises heard during the acme of the disease. These four sustained, I think, no permanent damage to the valves. The remaining three left the hospital with mitral murmur at the apex; and in them mitral disease was probably established. I say probably, for I have in view patients who have left the hospital with a murmur which has disappeared after a time, when they have returned as out-patients.

Pericarditis.—Perhaps one of the most striking peculiarities in the returns of these cases of acute rheumatism is the very small number of the patients who have been affected with pericarditis. They amount to only six. I find that, if I had drawn the line so as to include two more cases, one of those patients came in with pericarditis. From the date on which that patient left the hospital, June 25th, 1869, to this, I can only find one additional case of pericarditis.

The abstract of the six cases is contained in the tables with which you have been furnished. It is, therefore, only needful for me to give a sketch of them. They were all

males. Three were young men, from 18 to 21; and three ranged from 28 to 34 years of age. The two more recent cases were also men, aged 22 and 29 respectively, the last patient being affected with gout.

It will be seen, on examining the tables, that a large proportion of my cases, amounting to thirty-two out of seventy-four, were at and below the age of 20, and of these only three were affected with this disease. Besides these, two of the cases of endocarditis—John A. (435, p. 465), aged 20, and Mary L., 32 (457, p. 466)—were affected with transient friction-sound audible in each only one day, so that these cannot rightly be included among the cases of pericarditis.

As in endocarditis, the valve-murmur is ushered in by an anxious expression and a dusky hue of the face, pain in the region of the heart, disturbed breathing, and a prolonged first sound; so in pericarditis, the varying to-and-fro friction-sound, grazing, rubbing, grating, or creaking in character, is preceded by tenderness or pain on pressure over the region of the heart, or over the lower border of that region in the epigastrium, or by pain over the heart, by quickened breathing, distress in the chest, and a peculiar double murmur over the body of the organ.

This double murmur—which is shallow, limited to the exposed surface of the pericardium, accompanied by the healthy heart-sounds, from which it is distinct, and with the rhythm of which it does not correspond, and is increased or converted into a friction-noise by pressure, so as to drown the healthy sounds of the organ—bears the same relation to a friction-sound in pericarditis that a prolonged first sound bears to a valve-murmur in endocarditis. As in the latter disease, the prolonged sound precedes and passes into the murmur, and reawakens as the murmur dies away, so in pericarditis the double murmur precedes the harsher tones

of the to-and-fro rubbing noise, and re-awakens when those tones fade into a softer double murmur which is, in fact, a more gentle grade of friction-sound.

It has been seen that more than one-fourth of the cases of acute rheumatism were threatened with endocarditis, the threat in each case being shown by definite symptoms and signs, which gradually faded away, leaving the heart intact. In like manner, about one-fourth of the whole number of patients, or eighteen out of seventy-four, may be said to have been threatened with pericarditis, with more or less precision, by definite symptoms, and in two of them, already alluded to, by transient friction-sound.

The threat of pericarditis consisted in pain or tenderness on pressure over the region of the heart in five patients, and over the lower part of that region in the epigastrium in two; and in eleven more, in pain over or close to the region of the heart.[1]

The belladonna and chloroform liniment and cotton-wool,

[1] In 18 cases pericarditis was threatened, in two of which there was transient friction sound. (All of these either had endocarditis or were threatened with it.) Of these —

5 had tenderness or pain on pressure over the region of the heart.—466. Laura B., aged 18, p. 459. 443. Eliza T., 49, p. 459. 441. M.A.C., 29, p. 463. 444. Jane C., 22, p. 464. 447. Sarah S., 15, p. 468.

2 had pain on pressure at the epigastrium.—468. Eliza C., 17, p. 459. 435. John A., 20, p. 465 (had also transient friction sound).

11 had pain over or close to the region of the heart. Of these—

8 had pain over the heart.—457. Mary L., 32, p. 466 (had also transient friction-sound). 407. Lucy D., 38, p. 459. 436. Ellen E., 20, p. 467. 400. John T. p. 466. 419. George P., 24, p. 462. 371. Harriet R., 17, p. 463. 426. Matilda L., 20, p. 463. 410. Charles M., 14, p. 467.

1 had pain at the apex.—418. John S., p. 466.

2 had pain just below the heart.—348. Eliza H., 14, p. 462. 470. Sarah B., 18, p. 468.

or poultice applied over the seat of suffering, usually gave relief, and in five or six of the cases leeches to the same spot removed the pain.

I do not think that there was an actual tendency to pericarditis in all of these patients, but there certainly was that tendency in a large proportion of them; and it appears to me that, in some at least of these cases, pericarditis was arrested in its first blush.

Patients affected with acute rheumatism and acute gout are usually weakened and blanched in a remarkable manner, owing to the lessening of the whole volume of the blood and the diminution of its red particles. I gave iron and quinine, therefore, sooner or later, to a large proportion of them—preferring, as a rule, the old tried forms of the tincture of the muriate of iron and the sulphate of quinine. For a few patients, who were wanting in colour and strength, I directed the iron at once. I find that I gave it in the later stages of the disease to about four-fifths of those affected or threatened with inflammation of the valves, and to less than half of those in whom the organ did not suffer.

I am almost afraid to express the inference that I am about to draw; for I know that suddenly—to-morrow—I might begin to have a string of patients affected with pericarditis, and with endocarditis producing serious and permanent mischief to the valves; but still I do feel, after pondering over it much and often, and weighing in the scale everything that I could think of, that my inference is supported by the small number of these cases of acute rheumatism that presented actual pericarditis, and the happy result in those who were threatened and affected with endocarditis, so large a number of whom left the hospital with valves faultless, so far as we could detect. But the inference is, that we do owe to a marked extent the small proportion of cases of pericarditis

especially in the young patients, and the favourable issue in those threatened and affected with endocarditis, to the care which was taken to keep the inflamed joints at rest, to shield them from external pressure, and to give them ease.

POSTSCRIPT.

The object of this inquiry is twofold—to observe the laws that regulate acute rheumatism and acute gout, and especially the former—and to watch the influence of systematic rest on those diseases, both of which, though general in their nature, are so local in their effects that the sound physician, taking a lesson from the surgeon, employs local as well as general means in their treatment, and in doing so gives plain and effective directions to the nurse.

The local treatment by rest and ease is not put forward in this communication as the whole treatment of acute rheumatism or acute gout ; but it is maintained that whatever be the general method employed in the management of these diseases, rest, directed by knowledge, and not trusted to chance, is essential to the well-being of the patient.

CASES

OF

ACUTE RHEUMATISM AND ACUTE GOUT.

Summary of the Cases of Acute Rheumatism and Acute Gout, treated by the Author in St. Mary's Hospital, from October, 1866, *to May,* 1869.

Total number of cases of Acute and Sub-Acute Rheumatism . .		74
1. Endocarditis was absent in	14	
(One of these died with delirium and enlargement of the kidneys.)		
2. Endocarditis was threatened in	22	
Total in which Endocarditis was absent or incompletely developed	—	36
3. Endocarditis was present in	28	
Of these there was no murmur on recovery in . .	14	
The murmur was lessening, and becoming more obscure on recovery in	4	
The murmur appeared to be established in . . .	3	
And valve disease was held to be present on admission in	7	
4. Endocarditis was probable or doubtful in . . .		4
In three of these the disease was probable, in the other it was doubtful.		
5. Pericarditis was present in		6
Of these, three had Endocarditis, and two died, one with double valve-disease of the heart and purpura, and the other with delirium, resembling delirium tremens.		
Total number of cases of Gout		23
Endocarditis was absent or transient in		22
(One of these died with double valve-disease, and contracted granular kidney.)		
And Endocarditis, and probably Pericarditis, were present in		1
Cases of Acute Gout or Acute Rheumatism (their nature being doubtful)		4
Total number of cases		101

CASES OF ACUTE RHEUMATISM AND ACUTE GOUT.

Explanation of the Symbols employed in the following Cases.

→ Mitral systolic murmur. ↑ Aortic systolic murmur. | ↓ Aortic diastolic murmur. ↕ Double aortic murmur. | ↖ Pulmonic murmur. ← Tricuspid murmur.

ACUTE RHEUMATISM.

Cases in which there was no Endocarditis.

370—1.
Mary S., 18. No account of previous attack. Ill 4—5 days.

1. Affected with acute rheumatism. 4. Better. 8. Discharged well.

1. Acute rheumatism; no endocarditis. 2. Cheeks flushed; perspiration. Heart not mentioned. (Case imperfectly told.)

No Endocarditis; shown by the rapid recovery and non-mention of the heart.

379—2.
Wm. S., 24. 1st attack. Ill 8 days. Discharged well.

Attacked in all joints, especially knee and shoulder. 1. Fluid in right knee. 9. General swelling of left lower limb. Phlebitis, first of left and then of right femoral vein.

1. Nervous tremor. Heart-sounds not abnormal.

No Endocarditis; abnormal signs and pain being absent over heart.

382—3.
George M., 18. 1st attack. Ill 7 days.

2. Pain in all his joints. 4. None in any joint, except wrist. 7. Hands affected. 8, 9. Pain neck, legs, thigh; 14. shoulder. 21. No pain.

	TEMP.	P.
2.	100	100
5.	101	80
7.	99	96
12.	99	96
14.	99	84

2. Heart-sounds healthy. 9. Pain in left side; no unnatural sound of heart. 14. Heart-sounds feeble; 19, 28. healthy.

No Endocarditis; heart-sounds being throughout healthy.

385—4.
Thomas C., 23. 2nd attack. Ill 3 weeks. 11th day discharged well.

The joints have been very painful. Pains in shoulders and other joints. 9. Improving. 11. Slight pain left shoulder.

Heart not mentioned.

Case imperfect; but there was evidently no Endocarditis.

387—5.
Joseph B. 21. 1st attack. Ill 3 days. 32nd day well.

Has felt stiffness in thighs and legs. 2. Pain in knees, ankles, and hands. 3. No pain; can use hand. 5. Can stand. 7, 8. Hands painful, swollen. 9. Better; right leg painful. 14. Pain only in shoulder. 21. Wishes to get up.

	TEMP.	P.	R.
2.	99	100	20
3.	102		
5.	100	99	
7, 8, 9.	100		
14.	97		

3. Heart-sounds healthy.
16. Ditto ditto

Relapse in limbs from using them too soon; evidently no Endocarditis.

397—6.
Sarah B., 17. No account of previous attack. Ill 1 week. 33rd day well.

1. Pain in all joints. 6. Sits up in bed. 7. Feels much better; no pain; wants to get up. 10. Got up.

1. Profuse perspiration; heart-sounds natural. 6, 7. Ditto ditto

No sign or symptom of Endocarditis.

411—7.
Henry T., 27. 1st attack. Ill 5 days. 26th day discharged well.

1. Pains in wrists, hands, and knees, especially in knee; relieved by leeches. 7. No pain or swelling in knee.

TEMP.	
1. 102·4	1. Heart-sounds hard, sharp.
4. 101·8	4. Heart-sounds natural.

No sign or symptom of Endocarditis.

414—8.
Jesse R., 25. 1st attack. 23rd day discharged well.

Ill 10 days.

2. Pain and swelling of knees, ankles, wrists; better since yesterday. 6. Pain gone from hands; 9, swelling from knees; 17, attacked ankles.

1. Heart-sounds healthy. 6. 1st sound louder than 2nd.

No sign or symptom of Endocarditis.

438—10.
Mary A. B., 16. 2nd attack. Ill 1 day. 27th day discharged well.

1. Both legs affected, not swollen. 2. Pain right ankle, left shoulder; 3, left knee. 5, 6. Knee easier. 7. Shoulders easier.

TEMP.	P.
1. 99·5	110
5. 97·5	108
6. 99·3	100
7. 99·3	100

1. Great pain (hysterical) in chest, running round to armpit. 1. Heart's action and sounds natural. 15. Heart-sounds weak but distinct. Menstruating. The hysterical symptoms lasted up to the 20th day.

No sign of Endocarditis; the symptoms being those not of heart affection but hysteria.

454—11.
Sophia E. 1st attack. Ill 2 weeks. 64th day discharged well, but subject to attacks of jaundice.

1. Pain in joints of arms and legs. 3, 5. Pain worse. 6. Better. 9. Joints well. 21. A little pain in knees, not elsewhere.

TEMP.	P.	R.
1. 102°	90	22
3. 99°	105	
8. 97°	90	
18. 100·9		
19. 97·8		
28. 103·6	112	21
32. 98·4	80	17
35. 102·8	108	26
41. 98°	92	23

Jaundice. Had bilious vomiting before coming in. 3. Pain over liver. 8. Jaundice. 9. Skin regaining colour; much better. 18. Return of pain in liver and jaundice; soon well. 27. Third attack of jaundice. 34. Fourth attack.

2. Heart-sounds healthy. 12. 1st sound loud ringing. 29, 32. Heart in all its conditions healthy. 44. Doubling of 2nd sound.

No sign of Endocarditis. Constitutional disturbance due to affection of liver, jaundice.

431—9.
Wm. C., 23. Barman. No account of previous attack. Ill a week. 3rd day died.

Had pains in limbs and joints. 1. Pain of wrist better. 2. Pulse 120; tongue furred; pain of head, restless; hurried in speech. 3. He became more restless, delirious, and died quite suddenly, with symptoms of asphyxia.

Heart-sounds rapid; normal.

Took bicarbonate of potash. T. O.

The kidneys were large, 8½ oz. each. The heart was healthy. The habits of the patient and the symptoms allied this case to delirium tremens.

427—13.
Eliza N., 21. 2nd attack. Ill 1 week. 49th day discharged much relieved.

1. Pain in foot, and between shoulders. 3. Foot not so much swollen; other joints affected then or later. 13. Pain in joints much diminished. 20. Ankles regaining size, but slightly painful. 49. Left much relieved.

Had rheumatism when young; since, heart palpitates when she runs upstairs. Was never very feverish. 3. Rough irregular mitral murmur. Still rough mitral murmur. 20. Vibrating impulse from 3rd to 6th cartilage. Loud, grave, mitral murmur.

No Endocarditis. Mitral murmur retained its character. Little general illness.

439—14.
Louisa B., 19. 2nd attack. Time ill not stated. 13th day discharged well.

	TEMP.	P.	R.	
1. Some pain of wrist and knees. 4. Right wrist swollen. 11. Improves daily; complains only of weakness.	4. 99·8	80	24	2. Face cheerful. 1. Loud mitral bellows murmur; scratching pulmonic murmur.

No Endocarditis; face cheerful, ordinary mitral murmur. Well and out in 13 days.

463—12.
Ann B., 52. 2nd attack. Ill 3 or 4 weeks. 23rd day discharged well.

	TEMP.	P.	R.	
After being feverish and sick, was attacked in ankles, wrists, and other joints. 1. Pain in clavicles, left wrist, and back. 4. Limbs nearly well. 8. Much worse; very feverish; wrist and hand affected. 16. No pain in any joint.	1. 99·8	100	27	Impulse sudden. Heart-sounds obscured by cooing respiration. 5. No murmur. 1st sound perhaps prolonged; very nearly normal.
	4. 99·	100	28	
	7. 98·8			
	8. 99·	88	22	

The patient's appearance, the cooing noises, and quick breathing point to bronchial catarrh, and not to Endocarditis.

CASES OF ACUTE RHEUMATISM, THREATENED WITH ENDOCARDITIS.

I.—Cases in which the threat of Endocarditis was shown by pain about the region of the heart, or general illness, or both, but not by prolonged 1st sound or murmur.

392—2.
Emma C., 25. No account of previous attack. Ill 10 days. 56th day discharged well.

1. Pain in limbs. 6. Still suffers in hands. 11. Tenderness in right ankle. 28. Pain in right foot.

4. Great heat; sudamina over chest. 4. Heart's action quick; heart-sounds sharp, loud. 5. Pain still below left breast. 6. Faintish; has lost pain. 8. Has lost faintness. 11. Heart-sounds not so loud; no murmur.

Symptoms threatening Endocarditis lasted 8 days after coming in; pain below heart; quick loud action of heart; no murmur.

443—3.
Eliza T., 49. 3rd attack, gradual. Ill for 3 months. 36th day discharged well.

1. Ankles swollen. 4. Ankles more painful. 5. Pain in back. 9. Joints better. 14. Some pain at acromion.

	TEMP.	P.	R.
2.	101	100	36
5.	100·2	90	42
6.	100·4	92	48
9.	100·9	90	52
11.	98	66	54

1. Pain in chest, affecting breathing; heart-sounds distant, natural in character; great tenderness over region of heart. 6. Heart-sounds inaudible (organ pushed up by stomach); fights for breath. 8. Looks brighter. 9. Piercing pain chest and back. 9. Heart sounds audible. 14. Impulse much stronger. 25. The sounds are normally loud; no murmur.

Threat of Endocarditis shown by silence of heart-sound; pain in chest; tenderness over the heart, and fighting for breath, caused also by distention of stomach.

407—1.
Lucy D., 38. 2nd attack. Ill 6 weeks. 11th day discharged nearly well.

1. Acute pain in joints.

1. Eyes much protruded; perspires very much; acute pain in region of heart; sounds healthy. 9. Still has occasional cardiac pain, going through to the back.

Threat of Endocarditis on admission, lasting 9 days, shown by acute pain in region of heart.

468—4.
Eliza C., 17. 1st attack. Ill 4 days. 56th day discharged well.

1. Pain in knees, ankles, and hands. 4. Joints going on well. 7. Hands swollen. 12. No pain.

	TEMP.
2.	101·2
5.	101·2
7.	102·3
9.	100·4

1, 2. Almost a systolic murmur over pulmonary artery; pain on pressure at epigastrium. 4. Pain much relieved by leeches. 8. Sounds almost normal. 9. Epigastric pain gone; no murmur. 17. A prolongation of 1st sound.

Threat of Endocarditis shown by pain over the pit of the stomach and by almost a pulmonic murmur.

466—5.
Laura B., 18. 2nd attack. Ill 4 days. 33rd day discharged well.

1. Pain in knees and wrists. 5. Joints better. 11. Left knee still affected. 16. Sits up, free from pain.

	TEMP.	P.	R.
1.	103·8	100	24
4.	102	104	28
5.	102·7	112	32
6.	101·3	108	27
9.	100	96	27
11.	97·5	93	24
13.	99·2		
14.	97·5		

1. Pain in epigastrium. Cheeks somewhat congested; expression rather heavy. Herpes on lip. Sounds somewhat prolonged, 1st being very feeble, 2nd loud. 5. Pain increased on pressure over the heart; its sounds, not notably morbid, are obscured by sonorous respirations.

Threat of Endocarditis shown by pain over cardiac region and in epigastrium, congestion of cheeks, prolonged heart-sounds.

II.—Cases in which the threat of Endocarditis was marked by prolongation of the 1st sound.

424—6.
Thos. F., 21. 1st attack. Time of illness not stated. 30th day discharged well.

1. Pain in knees and elbows. 4. Pain has gone from legs and attacked hands. 7. None in any joint; left knee stiff.

1. Sudden plunging noise at end of 1st sound, which is prolonged; no murmur. 4. Sudamina cover chest. 5, 7, 8. 1st sound still prolonged, and plunging. 1st sound very loud, its plunging character gone; 2nd sound weak; slight pulmonic murmur.

Threat of Endocarditis, lasting fully 8 days, shown by prolonged 1st sound, which ends with a sudden plunging noise.

429—7 Robert A., 25 2nd attack. Ill 3 days. 20th day discharged well.	1. Œdema of hands, small joints affected; blister, but gave no uric acid. 4. Less suffering; hands regaining natural size. 17. Slight pain in knees.		1. Not much expression of pain in face; heart-sounds healthy. 4. 1st sound longer than when he came in. 10. 1st sound prolonged. 20. Discharged quite well.

No sign or symptom of Endocarditis on admission, but threatening of it 4—10 days after admission (7—13 after onset of attack), when the 1st sound became prolonged.

425—8 Mary B., 25. 2nd attack. Ill 6 days. 13th day discharged well	1. Several joints affected. 2. No better. 5. No pain anywhere. 6. Wishes to get up. 7. Up.		1. Slight systolic murmur; 2nd sound healthy. 2 p.m. prolonged 1st sound.

No additional note of heart trouble. Threat of Endocarditis shown by murmur-like prolongation of 1st sound.

462—9. Hannah P., 34. 4th or 5th attack. Ill 5 weeks. 25th day discharged well.	1. Pain in knees, calves, and left hand. 4. Joints all better. 8. Shoulders only affected. 18. Up, free from pain.	TEMP. 2. 99° 4. 98·4 5. 98·7 8. 99°	P. 92 92 105 80	R. 23 23 26 22	1. 1st sound prolonged, doubtful whether murmur. 3. Sound prolonged, indistinct murmur. 5. Sound prolonged, not amounting to murmur. 14. Heart-sounds natural.

Threat of Endocarditis on admission, lasting fully 6 days, shown by murmur-like prolongation of 1st sound, gradually less murmur-like and more natural.

377—10. John M., 13. No account of previous attack. Time ill not stated. 19th day discharged well.	1. Swelling in knees and arms. 8. No swelling in hands now.	1. Grave prolonged 1st sound. 8. "Murmurish" 1st sound. 15. Convalescent.

Threat of Endocarditis shown by grave prolonged and "murmurish" 1st sound.

III.—Cases in which the threat of Endocarditis was indicated by prolongation of the 1st sound, with pain about the region of the heart, or with general illness, or with both.

401—11. Martha W., 36. No account of previous attack. Ill 1 week. 31st day discharged convalescent.	1. Pain in knees, ankles, wrists. 3. Knees better since leeches; elsewhere pain great. 4. Pain less. 6. It lessens daily. 7. Pain only in right shoulder; feels much better.	TEMP. 2. 102·8 4. 99·8 6. 98·4	2. Heart-sounds weak but natural. 4. Headache; sickness. 6. Much thirst; profuse perspiration; prolonged 1st sound. 8. No heart affection.

Threatened with Endocarditis, shown by general illness and prolonged 1st sound, coming on 4 days after admission, and 10 after the onset of the attack.

393—12.
Anne N., 25. No account of previous attack. Ill about a month. 12th day discharged well.

5. Shoulder painful; swelling of joints. 9. Has no pain.

	P.	R.
3.	90	48
5.	60	30
7,	70	25

3. Herpes on lips; face flushed; 1st sound prolonged near apex. 4. Herpes better; flush gone. 6. Feels much better. 10. Is quite well.

Threat of Endocarditis, lasting 4 days, shown by herpes, flushed face, prolonged 1st sound, weak breathing.

432—13.
James C., 21. 1st attack. Ill 2 days. 37th day discharged well.

1. Acute pain in knees, ankles, feet; 5, wrists; 8, and hands. 15. Pain in joints much less. 22. No joints affected.

1. 1st sound prolonged. 5. 8. Breathing difficult; troublesome cough; face flushed. 8, 10, 15 No heart affection.

Slight threat of Endocarditis on admission, 1st sound being prolonged. and on 5th and 8th days face being flushed, breathing difficult, cough troublesome, but heart unaffected.

456—14.
Fanny R., 15. 1st attack. Ill 4 days. 31st day discharged well.

1. Puffiness of feet and arms. 4. Pain in wrists. 7. Knee and ankle worse. 10. Ankle easier since leg raised. 16. Shoulder affected. 21. Free from pain.

	TEMP.	P.	R.
2.	99°	100	
3.	100·5	100	
4.	99·5	104	
5.	100°	104	
10.	98°	94	26
11.	99·5		
12.	100°		
15.	98·5		

1. 1st sound prolonged at apex, almost a murmur. 7. ↖ Pulmonic murmur. 3. More feverish. 8. Heart-sounds nearly normal. 10. Looks anxious. 15—23. Heart-sounds healthy. 16. Cheerful.

Threat of Endocarditis on admission, shown by murmur-like prolongation of 1st sound and anxious look.

398—15.
Nathan H., 21. 2nd attack. Ill 3 days. 41st day discharged convalescent.

1. Elbows and hands affected. 13. Pain only in right shoulder; and on 16th in wrist and elbow. 18. No pain. 29. Up. 31. Pain in hands. 34. Stiffness; otherwise well.

1. Prolonged first sound. 5, 7. Heart-sounds feeble: 15. healthy.

Threat of Endocarditis on admission, ceasing within 5 days; shown by prolonged 1st sound and by pain in chest before coming in. Slight relapse after getting up.

391—16.
Charles Q., 21 No account of previous attack. Ill 6 days. 32nd day discharged well.

1. Pain in all joints, especially knees. 6. Slight pain only in shoulders. 13. Free from swelling. To get up.

1. Prolonged 1st sound at apex, slight murmur: reduplication of 1st sound: slight occasional pain in region of heart. 7. Heart's action feeble, sounds healthy. 27. Heart-sounds almost normal; 1st slightly prolonged.

Threat of Endocarditis on admission, ceasing within 6 days, shown by prolonged 1st sound and slight pain at heart.

419—17.
George P., 23. No account of previous attack. Ill 10 days. Discharged 15th day, well.

2. Slight pain in knees. 5. Less pain. 7. None.

Had pain in cardiac region before admission. 1. Pain in region of heart; 1st sound prolonged. 4. Sounds healthy. 6. 1st prolonged; no murmur.

Threat of Endocarditis, shown by pain in region of heart, and prolonged 1st sound, which recurred 5 days later.

396—18.
Jane R., 28. No account of previous attack. Time ill not stated. 55th day discharged well.

1. Suffering from acute rheumatism. Pain in the hands on the 6th; left them on the 7th, and attacked the legs. 9. Better, no suffering except on movement. 14. Has caught cold; pains in shoulders, arms, and knees. 35. Easy everywhere, except shoulder. 42. Better.

4. Face very much flushed. 15. Took cold, cough. 22. Prolonged 1st sound. 38. Heart-sounds natural.

Threat of Endocarditis shown by flushed face soon after admission; later after taking cold; 1st sound was prolonged.

434—19.
John J., 14. 2nd attack. Ill 4 days. 15th day discharged well.

1. Whole of joints affected, great pain in elbows and knees. 7. Only slight affection of joints. 9. Pain in left hand (last note).

	TEMP.	P.
1.	102·4	
7.	102·2	88
9.	101·11	84

Attack began with pain in the chest. 1. Perspiration very profuse. 1st sound prolonged at apex; almost a murmur. 7. No cardiac affection.

Threat of Endocarditis, ceasing before the 7th day, shown by early pain in the chest, and murmur-like prolongation of 1st sound.

IV. Cases in which the threat of Endocarditis is shown by old-standing murmur, variable in character, and pain about the heart; or by recent transient murmur.

348—20.
Eliza H., 14. 2nd attack. Ill 2 weeks. 29th day discharged well.

1. Hands and knees affected. 6. Much better, except left wrist. 12. No pain.

	TEMP.	P.	R.
4.	101	102	32
6.	99	98	
7.	101	102	
9.	100	106	
11.	101	110	
13.	—	88	

1. → Mitral murmur of old standing. 3. Short cutting pain under region of heart. 4. Painless; murmur distinct. 13. → Murmur louder.

Threat of Endocarditis, lasting 4 days, shown by quick breathing; cutting pain under heart; varying loudness of old mitral murmur.

437—21.
Jeremiah W., 22. No account of previous attack. Ill 3 days. 32nd day discharged well.

1. Knees swollen and painful. 10. Knee 1 in. less. 19. No pain, only stiffness. 23. Hands not so well, owing to pressure.

	P.	R.
2.	78	33

1. Pain under left collar bone; blowing noise over apex, where 1st sound is lost. 4. 1st sound feebler than 2nd, over heart. 10. Breathing has not improved. 28. No murmur at apex: no pulmonic murmur.

Threat of Endocarditis shown by quickened breathing; blowing sound over apex on admission.

376—22.
Frederick W., 12. 2nd attack. Left hospital a fortnight ago. Ill a few days. 13th day discharged well.

1. Knees, and 2, wrists swollen. 4. Wrists very painful; cannot move legs. 8. Better; no pain in limbs. 11. Wants to get up.

1. 1st sound prolonged. 4. Bellows murmur more marked. 6. Pulmonic murmur.

Endocarditis on 1st and 4th days, when sound prolonged and murmuring. Out; recovery rapid.

CASES OF ACUTE RHEUMATISM WITH ENDOCARDITIS, TERMINATING IN RECOVERY WITHOUT CARDIAC MURMUR.

371—1.
Harriet R., 17. 1st attack. Ill 2 weeks.

1. Pain and swelling of knees and ankles. 10. Hands only swollen. 24. Hands and legs still affected. 28. No pain.

4. Pain over region of heart. 10. No murmur, better, face not so flushed as it has been. 11. → Systolic murmur at apex, feeble at sternum. ↖ Faint pulmonic murmur. 28. No murmur. 32. Prolonged 1st sound. ↖ Slight pulmonic murmur.

409—2.
Elizabeth M., 23. 1st attack. Ill 10 days. 30th day discharged well.

1. Pain in knees. 4. Pain in limbs gone. 17. Shoulder stiff. 24. Shoulder well.

	TEMP.	P.
2.	99·32	72

2. → Mitral murmur at apex; 2nd sound loud. 4. Grave 1st sound at apex. 11. Prolonged 1st sound less marked. 12. Pain in the left breast. 17. Prolonged 1st sound, but no murmur. 24. Muco-purulent expectoration tinged with blood.

426—3.
Matilda L., 20. 2nd attack. Ill 2 weeks. 25th day discharged well.

1. Pain and swelling of joints, knees, hands. 7. No pain anywhere.

	TEMP.
1.	102·
3.	101·4

2. ↑ Grave aortic systolic murmur. 3. → Obscure grave mitral murmur. ↖ Pulmonic murmur. 4. Pain over heart. No murmur. 8. Heart sounds healthy.

428—4.
James H., 20. 1st attack. Ill 2 days. 21st day discharged well.

1. Pain in all large joints; those of right leg and left arm worst. 1–10. Knee lessened from 15½ in. to 14 in. 13. Very little affection of joints.

	TEMP.
1.	103·1
4.	101·8
7.	98·6
10.	98·3

1. Much perspiration; very thirsty. 4. Slight systolic murmur at apex. 10. Prolonged 1st sound.

433—5.
Maria S., 18. 1st attack. Ill 5 days. 44th day discharged well.

1. Pain in ankles, knees, wrists, elbows and back. 11. Limbs less painful. 22. Joints slightly affected; sat up; felt better.

1. Heart-sounds healthy. 3. Head confused. 6. → Mitral murmur. 11. Murmur gone. 22. Murmur has returned. 24. Prolonged 1st sound; no murmur.

441—6.
M. A. C., 29. 2nd attack. Ill one week. 33rd day discharged well.

5. Pain ankle and back. 19. Shoulder and wrist still slightly painful.

	TEMP.	P.	R.
1.	99·4	120	
6.	101	120	48
10.	101·8	108	42
17.	99·8		

3. → Loud mitral murmur: tenderness over heart. 6. Pain in chest, relieved by leeches. 10. No distinct murmur over heart. 16. No murmur audible. 23. Obscure murmur.

444—7.
Jane C., 22. Ill 10 days. 1st attack. 24th day discharged well.

Pain in knees and ankles. 6. Joints less painful. 17. Up yesterday; felt weak.

	TEMP.	P.	R.
1.		120	
3.	101·2		26
4.	101·	108	22
10.	100·	92	24
17.	99·		

1. Grave murmur over heart, and tenderness on pressure. 4. Faint rough murmur at apex and base. 10. Prolonged 1st sound; pulmonic murmur. 15. Heart sounds distant; no murmur.

446—8.
Ellen O., 22. 5th attack. Ill 8 days. 20th day discharged well.

2. Pain of wrists and knees. 4. Knees better. 7. Shoulders painful. 9. No pain in joints.

	TEMP.	P.	R.
2.	102·	120	
3.	101·4	120	32
4.	100·	120	
6.	99·2		30
7.	98·8	72	35

1st sound at apex prolonged; doubled, almost a murmur. 3. Pain in chest. 4. Face slightly glazed. 6. Murmur at apex and base; pulmonic murmur; doubling of 2nd sound. 16. No murmur audible.

450—9.
Alice M., 19. 1st attack. Ill 3 weeks. 26th day discharged well.

1. Pain in left shoulder and arm. 13. Pain of arm relieved by leeching.

TEMP.	P.
99·2	108

A week since had stinging pain in chest. 1. 1st sound at apex somewhat prolonged. 11. Peculiar shock; prolonged 1st sound. → "Systolic murmur, most distinct at apex." 13. Murmur less distinct 21. Heart sounds ringing; no murmur.

451—10.
William B., 19. 1st attack. Ill a week. 25th day discharged well.

1. Pain in joints. 11. Up. 14. Some pain in right ankle.

TEMP.	
3.	99·2
5.	99
9.	98·2

1. Systolic murmur at apex, not elsewhere; heart-sounds weak. 10. Grave murmur (not bellows, barely audible) from apex to lower sternum. 21. Murmur getting weaker. 20. Standing; neither murmur nor prolonged 1st sound: lying; prolonged 1st sound at apex, not a murmur.

469—10.
Martha H., 25. 2nd attack; perfect health since. Ill 2 weeks. 64th day discharged well.

1. Knees and ankles affected. 6. Knees and ankles better. 18. Knee better. 29. Scarlet fever. 32. Slight return of rheumatism in knee. 34. Affection of hands. 37. Hands better.

	TEMP.	P.	R.
4.	101·3	112	28
5.	100·2		
7.	99·3		
9.	98·2		
29.	97·2		
31.	100·		
32.	100·		
33.	100·2		
37.	101·		
40.	99·2		

1. Face flushed; 1st sound double at apex; prolonged over pulmonary artery. 6. → Mitral and ↖ pulmonic murmurs. 7. ↓ Faint diastolic murmur. 15. Mitral murmur scarcely heard. ↓ Diastolic murmur and double 1st sound at mid-sternum. 29. Very anæmic. 31. Prolonged 1st sound at apex. 33. No distinct diastolic murmur. 60. 1st sound loud and ringing at apex; no clear indication of diastolic murmur.

471—11.
Martha W., 16. No note of former attack. Ill 2 weeks. 42nd day going out.

2. Pain in knees and hands. 5. Pain gone from knees. 8. No pain in limbs.

	TEMP.	P.	R.
2.		100	32
5.	99·	90	24
7.	98·8		
8.	98·3		
10.	98·		

2. → Grave, feeble murmur at apex, towards armpit, and at lower sternum. ↖ Pulmonic murmur; ↑ systolic murmur over carotid 4. Prolonged 1st sound at apex, almost a murmur. 7. Prolonged 1st sound at apex, no murmur. 8. No pain in chest; expression of face natural. 15. Heart-sounds normal, except a slight pulmonic murmur.

474—12.
Jane G., 35. 2nd attack. 1st, when a child, for 3 months. Worse 12th day. 51st day discharged relieved.

1. Pain in many joints. 5 Feet and legs much relieved. 6. Almost free from pain. 7. Excessive stiffness. 18. Pain left hip. 26. Feels almost well.

	TEMP.	P.	R.
1.	100°	100	
2.	98°	104	
3.	101°	100	
5.	100°	104	
9.	100·2		
13.	99·92		
19.	98·9	74	24
26.	98·1	80	

2. Prolonged 1st sound or short murmur at apex and lower sternum. 5. → Grave short murmur. 6. Prolongation of 1st sound or short murmur. 9. Excessive perspiration; severe miliary rash. 37. 1st sound still prolonged at apex. 40. Systolic shock a little prolonged; absolute silence between 1st and 2nd sounds. 48. Heart sounds at apex normal; 1st sound at lower sternum a little prolonged.

475—13.
Mary C., 24. 2nd attack. Ill 8 days. 50th day discbarged relieved.

1. Little pain in joints, hands swollen; got up. 9. Better, only stiff. 11. Pain returned in knee and hands. 18. No pain for 5 days; feels perfectly well. 21. Pain in wrist. 48. Quite well.

	TEMP.	P.	R.
1.	101°	100	
2.	100°	92	37
4.	101°	92	
8.	98·3	84	
9.	97°		
12.	101·1	88	
13.	97°	78	

1. Tightness in chest. 2. Worse; face dusky. 2. Expression heavy, but brighter; no tightness; 1st sound prolonged after shock, almost a murmur at apex. 6. 1st sound less prolonged over ventricle. 13. → Distinct but feeble systolic murmur at apex; faint prolongation of 1st sound. 60. 1st sound loud, faintly prolonged at nipple; 2nd sound loud.

ACUTE RHEUMATISM WITH ENDOCARDITIS. LESSENING OR DOUBTFUL MURMUR ON RECOVERY.

402—1.
Charles H., 16. Ill 6 months. 15th day discharged well.

1. Left hand affected. 5. No pain in hands.

1. Heart's action tumultuous; uneasiness over heart; prolonged 1st sound or slight murmur. 4. → Mitral murmur at apex and towards axilla. 6. Prolonged 1st sound; very loud 2nd sound. 6 a.m. → "Smooth systolic murmur slightly audible at apex and towards sternum; inaudible at base" (Clerk).

405—2.
Charles C., 20. No account of previous attack. Ill 10 days. 22nd day discharged well.

1. Pain in ankles, knees, and wrists. 4. No pain in any joint; knee relieved by leeches.

5, 7, 9. → Mitral murmur diminishing in intensity from apex to sternum. 14. Not an exact murmur, but a prolonged boom with systole.

435—3.
John A., 20. 1st attack. Ill 12 days. 25th day discharged well.

1. Pain in limbs; 7. left legs; 9. only in shoulders.

2. Profuse persp.

TEMP.	R.
3. 102·1	90
4. 100·2	84
7. 99·6	

2. Pain in epigastrium; sharp friction sound, with systole over middle of cardiac region (heard through flannel vest): → systolic murmur at apex. 4. No friction sound; still pain in chest; → very loud mitral murmur. 7. Heart affection has left him; prolonged 1st sound. 17, 22. Grave prolonged 1st sound, almost a murmur at apex; ↖ pulmonic murmur.

Transient Pericarditis; marked Endocarditis; probably no mitral disease left.

452—4. M. A. G., 19. 1st attack. Ill 7 weeks. In bed 5 days. 42nd day discharged well.	1. Severe pain in knees. 6. No pain. 16. Lower limbs affected. 19. Pain in back and shoulder. 35. Up; free from pain.	TEMP. P. R. 1. 99·8 72 25 3. 99·9	2. → Systolic murmur, apex and lower sternum; ↖ scratching pulmonic murmur. 16 Murmurs persistent. 29, 30. → 1st sound prolonged, almost a murmur at apex. 31, 32. → Dull grave systolic murmur at apex and towards axilla. 37. Very soft murmur at apex ("more like prolonged 1st sound," Mr. Salaman).

CASES OF ACUTE RHEUMATISM, WITH ENDOCARDITIS, LEAVING A MURMUR ON RECOVERY.

418—1. John S. 1st attack. Ill 10 days. 20th day discharged well.	Ankles and knees swollen. 6. No pain or swelling of joints.		1. Faint grave murmur at apex. 3. Pain at heart relieved by leeching; musical murmur between nipple and sternum. Expression of face less clouded. 8. Murmur more feeble; feels quite well. 10. Systolic murmur over heart, at parts musical, not loud. Pulmonic murmur.
448—2. Keziah D., 26. 1st attack. Ill 3 weeks. 30th day discharged much better.	1. Pain in arms, shoulders, and wrists. 12. No pain. 13. Up. 14. Knee stiff. 24. Still slight pain.	TEMP. 5. 100·8 7. 100· 8. 99·2	1. Systolic murmur over heart; pulmonic murmur. 3. Pain in chest relieved by leeches; murmur almost gone. 8. Murmur at apex. 12. Less distinct. 17–22. Murmur at apex still present, grave prolonged. (Last mention of murmur.)
457—3. Mary L., 32. 1st attack. 65th day discharged relieved.	1. Pain in all the limbs; joints not much affected. 5. Great pain in right knee. 35. Still pain in right arm. 56. Well, except right hand and arm.	TEMP. P. 1. 100· 95 2. 99· 112 4. 97· 108 5. 97· 100 7. 96·53 106 10. 96·15 11. 98·4 100 15. 98· 100 23. 98· 80 25. 98·2 76	1. Pain over heart; increased on 2nd day; very feverish; breathing difficult. 3. Less feverish; → prolonged smooth systolic murmur below heart. 4. Less pain over heart; is more animated; → mitral bellows murmur; feeble, audible below shoulder blades. 6. Severe pain over heart, increased by breathing. 7. Transient friction sound. 10. Mitral murmur louder towards armpit, scarcely audible below shoulder blades. Prolonged murmur. 17. Change in heart sounds; no pulmonic murmur; prolonged 1st sound at apex instead of murmur. 29. Systolic murmur to left of apex. 31. Looks brighter. 44. Murmur not so loud at apex; not audible over right ventricle; double 2nd sound.

CASES OF ACUTE RHEUMATISM WITH ENDOCARDITIS; VALVE DISEASE OF OLD STANDING.

400—1. John T. 2nd attack. Ill 5 days 25th day discharged convalescent.	1. Pain general; knees swollen, shoulders stiff. 8. Free from pain. 9. Up. 22. Slight pain in knee, from running about wards.	Had dyspnœa after 1st attack. 1. Pain over region of heart. → Loud mitral murmur. Troublesome cough. 15. → Loud smooth prolonged mitral murmur, heard extensively.

410—2.
Charles M., 14. 2nd attack. Ill 4 days. 46th day discharged relieved. 4/5/68 re-admitted.

1. Pain in knees, left ankle. 2. Arms. 11. All pain gone.

	TEMP.	P.
1.	101·2	120
2.	101·5	108
5.	102·9	
9.	100·	
11.	99·7	

1. Impulse of right ventricle extensive. → Loud mitral murmur. 4. Sounds unchanged. 5. Pain over heart, with dyspnœa; ringing 1st sound over the right ventricle. 16. Ringing sound has ceased. 18. ↖ Pulmonic murmur. 32. Pain in region of heart.

420B—3.
Alice S., 15. 2nd attack. Ill 8 days. 35th day discharged much relieved.

Had great pain in joints, all of which had been blistered. 1. Great weakness.

1. Pain over region of heart; → grave mitral murmur at apex (the tone of which afterwards varied). 2. Pale, anxious, though cheeks flushed; impulse of vibration from 3rd cartilage to 6th.

Anxious face; pain over heart; signs rather variable:—therefore Endocarditis.

436—4.
Ellen E., 20. 2nd attack. Ill 2 days. 32nd day discharged well.

1. Great effusion on knees. 7. Knees better. 9—17. Pains lessened and increased in different joints. 28. Quite free from pain.

	TEMP.	P.
1.	102·5	116
4.	106·	
5.	101·12	
6.	100·8	
11.	98·6	
12.	100·4	108
14.	97·4	96

Has suffered from palpitation since 1st attack, 8 years ago. 1. Severe pain in chest; → loud murmur at the apex. 5. Severe pain over heart. 7. Heart sounds very distant; murmur not audible. 9. → Mitral murmur not so loud; ↖ pulmonic murmur. 24. → Smooth mitral bellows murmur.

From loudness of murmur at first and previous palpitation it was evidently of old standing. The murmur was afterwards suspended by Endocarditis.

455—5.
Mary H., 24. 5th or 6th attack. Ill 12 days. 25th day discharged well.

1. Pains in elbows, hands, knees. 4. Less pain in hands. 8. Knees worse. 9. Almost free from pain. 16. Quite so. 23. Up and dressed.

	TEMP.	P.	R.
1.		105	
8.	100·5	110	
9.	99·5	96	
11.	98·5	96	40

1. Loud prolonged 1st sound at apex, almost a murmur. 9. → Mitral murmur at apex, extending to lower sternum, where it is obscured by normal heart-sounds. 18. → Loud murmur at apex and towards armpit.

460—6.
Emma S., 17. 4th attack. Ill 16 days. 95th day discharged relieved.

Each attack was preceded by chorea; this affected ankles and knees. 1. Pain only in knees. 8. No pain in limbs. 12. Pain knees and ankles. 32. Ankles easier.

	TEMP.	P.	R.
2.		90	34
4.	99·3	92	40
5.	99·6	96	45
9.	98·8	100	36
11.	99	92	36
16.	99	80	

1. Aspect good, eyes watery, cheeks florid. → Loud mitral murmur at apex. 3. Impulse less strong; → murmur less loud. 4. → Murmur louder. 18. Heart's action irregular. 32. Vibrating thrill with apex beat.

470—7.
Sarah B., 18. 2nd attack. Ill 6 days. 34th day discharged relieved.

In bed 4 months from 1st attack. In this attack all the joints were affected, beginning in knees (she had been kneeling on stone floors). 1. Pain and swelling of knees. 6. Hands affected. The joints steadily improve. 11. They are well. 14th. She wishes to get up; feels and looks well. 25. No pain anywhere; walks without discomfort; murmur as before; impulse stronger.

	TEMP.	P.	R.
1.	100°	120	28
2.	99°	120	32
7.	101·3	94	26
11.	97 3	80	24

1. No great look of illness; face flushed; eyes watery; → mitral murmur grave at apex; impulse in 5th space, and strong at cardiac cartilages. 2. Pain below heart (leeches). 7. More flushed; less lively. 8. Less heavy; feels better. 11. → mitral murmur not materially changed.

V.—Cases of Acute Rheumatism in which Endocarditis is probable or doubtful.

394—1.
Mary C., 38. Often had rheumatic pain. Time ill not stated.

1. Pain in shoulders and hands. 2. Easier. 3. Can move hands. 5. Still pain left shoulder. 8, 10. Better. 13. Slight stiffness.

1. → A systolic murmur at apex; the murmur is not again mentioned.

Probably mitral disease of old standing.

447—2.
Sarah S., 15. 2nd attack. Ill 2 weeks. 16th day discharged well.

1. Pain in neck, shoulders, wrists, back, and hands (which are a little puffy). 11. Only complained a little of shoulder.

	TEMP.	P.
1.	102·2	132
4.	102·2	120
5.	99·4	
7.	98°	

1. Is sensitive to percussion over the heart; 1st sound a little prolonged, 2nd accentuated. 14. 1st sound prolonged, almost a murmur.

Probably Endocarditis.

465—3.
Alfred M., 24. 4th attack. Ill 3 months. 37th day discharged well.

1. Has suffered, and still does so, in wrists, ankles, and knees. 6th. Almost free from pain.

	TEMP.	P.	R.
1.	102·2	96	25
2.	99·5	90	
7.	97	48	20

Attack began with pain in chest. 1. → Exquisite musical mitral murmur at apex and over heart. ↕ Double aortic murmur; face clouded. 21. Sounds little changed; face less muddy, lips of better colour.

Endocarditis, probable aortic mitral complication of old standing.

467—4.
Adelaide M., 39. 3rd attack. Ill 5 days. 22nd day discharged well.

1. Various joints affected. 15. Free from pain.

	TEMP.	P.	R.
1.	101·5	104	22
4.	101·3	104	24
5.	101	104	26
6.	100	92	29
9.	98	88	20

1. Feels sinking, weak, and low; 1st sound peculiar, vibrating like *r r r p*. 5. Same sound; dragging pain at apex. 6. Sounds of heart louder; no distinct murmur. 15. No report of murmur; doubling of 2nd sound. 20. Faint systolic murmur at apex.

Endocarditis probable; probably mitral disease of old standing.

VI.—Cases of Acute Rheumatism affected with Pericarditis.

378—1. John B., 18. No account of previous attack. Ill 3 days.	1. Pain in knees, feet and ankles.	1. Short of breath. 2. Pain in left side; great relief from 6 leeches over region of heart. P. 104. R. 72. Cough; phlegm streaked with blood. 6. R. 60 in the minute. 10. P. 101. R. 48. 13. Phlegm still streaked. 16. Diphtheria.	2. Fulness over region of heart. Pericardial dulness extensive and high. Impulse high in 3rd and 4th spaces. Friction sound over region of heart. 3. Friction double, increased by pressure. 4. Pericardial dulness lessened in every direction. Double friction sound more extensive, higher up. 6. Apex beat lowered to 4th space. Double rubbing sound: lowered: not heard above 4th cartilage; more extensive below it. 10. Friction sound not audible unless he holds his breath. 13. No friction sound or murmur.	2. Mitral murmur rather faint. 3. Systolic murmur (mitral) to left of nipple. Murmur not unequivocal at apex. 10. Murmur in neck. 13. Murmur doubtful. No murmur.

Pericarditis and Endocarditis. No Valve-disease on recovery.

406—2. Frederick P., 21. 1st attack. Ill 2 weeks. 66th day discharged relieved.	1. Great pain, wrists, knees, ankles. 3. Less pain in knees, ankles. 8. Arm very painful. 19. Pains in various joints, 38. No pain.	2. Breathing laboured, quick. 8. Aspect not so expressive of dulness. P. 90. R. 30.	1. Distinct pericardial friction sound. 3. Cardiac dulness rather extensive; no friction sound. 6. Pain in region of heart. 9. Tenderness there. 16. Extensive dulness on percussion, and to and fro rubbing sound over the whole pericardial region; impulse high. 19th. Dulness lessened; impulse lower, friction sound less harsh. On 20th the rubbing noise was very extensive and creaking. On the 22nd it had almost gone, and on the 25th the heart-sounds were normal.	1 to 19. A mitral murmur was audible near the apex. which disappeared on the 25th. After this a pulmonic murmur, but on 44th heart-sounds were quite normal.

Pericarditis and Endocarditis. No Valve-disease on recovery.

412—3. Henry W., 20. 1st attack. Ill 1 week. 50th day discharged well.	1. Pain right knee. 5. Increase of pain in arms and back. 10. Pain returned to fingers after using them. 23. Joints well. 35. Left wrist again bad.	1. Pain in chest; skin hot. 2. Breathing quick. 5. More heavy. 6, 7. Pain shooting from breast bone to back. Pulse 84. Resp. 32. 10. More clouded 96—44. 17. Looks oppressed; return of pain.	14. Dulness on percussion, extensive over pericardium and double friction sound; impulse high. 17. Dulness less extensive; friction sound not nearly so loud. 19. Neither rubbing sound nor murmur audible. 21. Heart sounds quite normal.	2. Mitral murmur. 5. Heart sounds normal. 10. 1st sound prolonged. 21, 38. Heart sounds normal.

Pericarditis and Endocarditis. No Valve-disease on recovery.

442—5. James A., 34. No account of previous attack. Time ill not stated. Worse one day. 50th day discharged well.	1. Stiffness and pain in all joints. 2. Skin bathed in perspiration; face anxious; short dry cough; pain in chest. 6. Peculiar prolonged plunging 1st sound. 9. Heart sounds normal.	TEMP. P. R. 1. 101·5 112 30 2. 101·5 112 30 6. 105·1 114 7, 11. 99· 98 12. 98· 112 28	Friction sound at apex. Double noise over heart, somewhat murmur-like, but intensified and made more rubbing by pressure. No friction sound with or without pressure.		

Pericarditis. No Endocarditis.

440—4. George G., 35. 5th attack. Ill 6 days. 27th day died.	Was attacked by rigors, followed by severe pain in joints and back. 1. Pain in all joints. 4. Limbs not much swollen. 6. Great pain in back and limbs. 10. Pain confined to right arm. 16. Pain confined to 4th finger.	1. Pain in chest and distress of breathing, preventing sleep. 4. Pain across region of heart, which set in 3 days ago. 10. Looks less anxious. 16. Cough excessively troublesome; phlegm tinted with blood. 25. Alteration for the worse; œdema, purpura; distress and exhaustion increased. On the 27th he died.	1. Loud galloping heart-sounds, with friction sound over the pericardium extending from the first space downwards. 4. Double frottement heard over the whole heart and a great part of the front of the chest, like the rubbing of sand paper. 6 Pericardial dulness lessened; impulse lowered; friction sounds more extensive downwards, reaching to the 8th cartilage. 11. Friction much less in harshness and area; increased by pressure. 15. Friction very feeble; and on 17th scarcely heard.	Had aortic and mitral valve disease when in the hospital 3 months ago. The double aortic and the mitral murmurs were masked by the friction sounds during the attack of pericarditis; and came gradually into increasing play as the rubbing sounds dwindled and disappeared.

Pericarditis, no Endocarditis. Died. Recent pericardial adhesions. Double valve-disease of old standing. Purpura.

476. William F., 28. A Cheesemonger's Assistant. 2nd attack. Ill 3 days. 8th day died.	Ankles, legs, and arms affected. 1. No pain.	Is very stout: covered with sudamina. 1. Face flushed —Pulse 126; Resp. 36. 3. Face of a bright red colour; pupils contracted.—Pulse 120; Resp. 22. On the evening of the 6th day he became violently delirious so as to cause his removal to a separate ward. His symptoms closely resembled those of delirium tremens. He had been a free liver.	3. 1st sound loud and sharp over the ventricles. 3. No murmur. Impulse in 5th space. 3. No pain in the chest. Heart's action excited. 1st sound loud over the ventricles, 2nd sharp. On the morning of the 8th day he died. The heart on its surface and the lining of the pericardiac sac were covered with honeycombed lymph.	

ACUTE GOUT.

380—1 Frederick B., 30. Cabman. No history.	1. Pain and swelling of knees. 3. Pain and swelling much better; elbows very bad. 5. Hands affected 14. No pain in hands. 18—26. Pain returned in arms, knee and hand. 32. No pain 40, 45. Pain returned in toes and ankles. 51. Pain ceased.		1. Heart sounds natural. Cooing noises over the lungs. 51. Prolonged 1st sound.	

381—2. John H., 56. Painter. No account of previous attack. Ill 7 weeks. 30th day discharged well.	Began with pain and swelling of great toe and knee, then in nearly every joint. 1. Great pain in right knee and œdema of both hands. 5. Knees better. 7. Pain nearly gone. 8. Swelling of right fore-finger. 9. Finger less swollen. 17. No pain; getting stronger.	2. Not so excitable. 3. Eyes painful. 15. Eyes nearly well. 29. Complains of nothing.	2. Heart sounds healthy.	1. Iod. of Potass. Belladon. Lin. Colch. Opium.

Joints nearly well in 10 *days.*

383—3. Charles S., 32. Dustman. No account of previous attack. Ill 10 days. 64th day discharged well.	Began with pain and swelling of knees; general fever; pain of loins. 6. Knees better. 9. Pain of hands relieved by injection of morphia. 18. Left knee worse (leeches). 32. No pain in knees. 46. Right hand and left knee affected. 50. Up; better. 53. Stiffness.	TEMP. P. R. 2. 99 96 4. 98 84 96 9. 96·5 84	4. Heart sounds normal. 6. 1st sound prolonged at base. 14. 1st sound a little rough, not prolonged. 18. Heart sounds healthy.	1. Bellad. Lin. 14. Inj. of Mor. under skin. 17, 24. Leeches to knee. 28. Iod. of Pot. 46. T. of M.: Iron. 55. Iod. of Pot.

This man's employment, and the history of the case, point to gout.

384—4. Henry A. Works in laundry. Many former attacks. Ill 4 weeks in chest, 4 days in toes. 25th day discharged well.	Former attacks began in great toe. 1. Both great toes affected. 5. Pain in ankles and loins. 13. Left knee puffy and swollen. 17. No acute pain.	4. Appetite ravenous. 5. Pain in chest. 9. Pain in chest gone.	10. Shock of systole rather prolonged.	1. Iod. of Pot., Iod. of Iron 7, Colch. Liq. Opii. 18. T.F.M.

Joints well in about 20 *days; took iodide of potassium, iodide of iron, colchicum, opium.*

403—5. George H., 47. Butcher. Ill 6 weeks.	Previous pains in great toe. 21st day discharged relieved.	2. Knees swollen, painful; leg œdematous; veins large. 6. Easier.	2. Heart sounds natural.	Iod. of Potass. Lin. Belladonna and Chl. Morph. inj.
404—6. William G., 30. Stationer. 3rd attack. Ill 1 week. 39th day discharged convalescent.	Acute rheumatism, 8 years ago; laid up 3 months. 1. Left knee puffy and swollen. 2. Skin over it shining. 4. Knee relieved by leeches. Œdema over foot. 15. No pain in joints. 22. Left little finger affected.	1. Face pale; prolonged 1st sound at base.	1. Belladonna and Chl. Lin. Leeches. 9. Iod of Pot. 14. T. F. M.	

386—7. Charles B., 31. Labourer. 3rd attack. Ill 4 weeks. 21st day discharged well.	Acute rheumatism 16 years ago. Pain in loins 6 months since. 4 weeks since ankles swollen, and 4 days ago great toes. 5. Pain still in toes and ankles. 10. Pain returned into knees and ankles. 20. No pain about joints.	1. Eyes congested; oppression at chest.	Heart not named.	3. Pot. Iod. 18. T. F. M. Quinia.
408—8. John F., 26. Bootcloser. Several previous attacks. Ill four days. 68th day discharged well.	First attack of acute rheumatism 13 years since; gonorrhœal rheumatism 6 years ago. 1. Pain knees, great toes and right arm. 2. Increased pain in all joints. 6. Leeching gave relief. 9. Less pain and swelling in joints. 18. Relapse. 28. Knees much smaller. 38. Only stiffness in back. 4 days ago pain came in great toe.	TEMP. P. R. 1. 103° 96 2. 103·5 120 4. 101·12 96 5. 103·3 96 6. 101·5 100 8. 98·3 72 19. 104·4 138 16	1. Heart sounds natural. 11. Peculiar rasping systolic sound at apex. 19. Heart sounds normal.	1. Bell. Lin. 20 Pot. Iod. 17. T. F. M. 37 Pot. Iod. Fer.
416—9. Robert W., 33. House-painter. Accustomed to slight pains in joints. Ill 12 days. 38th day discharged well.	1. Pains in knees and shoulders. 7. Pain almost gone. 25. Relapse. 28. Less pain in joints. 31. 2nd relapse.		Prolonged 1st souud. 3—5. Heart sounds healthy.	1. Iod. of Potass.; Bellad. Lin. 25. Morph. Quin. 29. Pot. Iod.
418—10. George W., 25. Barman. No account of previous attack. Ill 3 weeks. 22nd day discharged well.	Has had pain in great toe. 1. Pain knees; 5. and right shoulder. 8. Pain less. 12. Pain gone.		Something like a friction sound. 5. Heart sound healthy.	1. Lin. Bellad. 4. Amm. 21. T. F. M. M. Q.
420—11. William P., 35. Painter. Subject to rheumatism 3 years. Ill 1 month. 28th day well.	Attacked with pain in great toe. 1. Hands swollen; great pain in knees. 6. Left knee has gradually regained its natural size,		Heart not named.	1. Pot. Iod. 7. Lin. Bell.
421—12. Sarah T., 14. 2nd attack 6 months ago; still ailing. 34th day discharged relieved.	Has had pains in great toes. Father had gout. 1. Hands swollen, puffy; pain knees, ankles. 4—27. Various joints affected. 28. No pain.	1. Perspiration prof. 6. Crystals of uric acid obtained by Dr. Garrod's blister test. 12. Goes into the garden. 14. Not so well.		1. Lin. Bel. 9. Pot. Iod. 20. M. Q. 32. Rep. P. I.

423—13.
Maria L., 30. Married. 2nd attack. Ill 3 weeks. 17th day well.

In this and former attacks, great toe was affected. 1. Pain in knees. 7. Feels quite well. only a little stiffness. Got up 8th. 11. Left knee and foot again affected; 12, and great toe. 14. Quite easy; knee stiff.

2. Mitral systolic murmur, musical at beginning. Face not dusky; no indication of internal inflammation. 5. Murmur still perceived. 8. Murmur heard in upright posture, a grave slight noise.

Old mitral disease; relapse from getting up.

425—14.
Jane N., 30. Sempstress. Many attacks. Ill 12 months. 13th day died.

1. Left leg swollen, shiny. 9. Pains all over her.

1. Anæmic. 3. Cough. 5. Still looks very anxious: urine albuminous. 9. Much worn; distressed; breathing hurried. 10. Gasps for breath. 11. Lies in half-unconscious state. 12. *In extremis.*

1. Apex beat strong, far to left. Impulse of right ventricle diffused. → Mitral and ⇕ double aortic murmurs.

Contracted regurgitant mitral orifice; incompetent aortic valves; contracted granular kidneys, with cicatrices.

445—15.
Alexander R. Baker. No account of previous attack or beginning of this. 15th day discharged well.

1. Severe pain and puffiness in both hands; tenderness of great toe, knees swollen. 4. Puffiness gone from hand, swelling less in knees. 6. Œdema almost gone; feels quite well.

1. Skin hot. Remains of sudamina. 6. Temp. 97·5. Pulse 66. Feels quite well.

1. Heart sounds natural

Iod. of Potass.

455—16.
Richard D., 45. Painter. Dropsy 12 years ago. Colic 6 years ago. Ill 3 months. 21st day discharged well.

1. Pain in back; redness and puffiness of sterno-clavicular joint. Knees affected. 6. Ball of great toe enlarged. 8. Pain in joints lessened. 17. Up; deposits in left extensor tendon.

	TEMP.	P.	R.
1.	100·3	110	24
2.	100·2		30
6.	98		

1. Heart sounds distant. 1. Cough; bubbling râles. 8. Râles almost gone. 20. H. S. normal.

1. Bel. Lin. Iod. of Pot. Tart. of Iron

458—17.
Charles W. Cabman. Many previous attacks. 3 months. 37th day discharged well.

In previous attacks right great toe affected. 1. Deposit in joint of great toe; tender spot below ankle. 29. Has been gradually improving. though he occasionally complains of pain.

1. Heart sounds normal. 7. Giddiness.

Belladonna Lin. Iod. of Potass. 12. Iod. of Pot. Tartr. Iron.

459—18.
James C., 57. Coachman. Many attacks. Laid up 5 weeks. 42nd day discharged relieved.

1st attack at age of 18, affecting great toe. 1. Hands much deformed by old swelling of the knuckles; knees much swollen. 5. Joints more flexible. 10. No pain, except in right elbow. 13. Pain gone. 32. Ankle œdematous when out of bed.

	TEMP.	P.	R.
1.	100	96	22
10.	99·2		
13.	99·8		

1. Appetite good; heart sounds normal; temporal art. tortuous.

1. Bel. Lin.; Iod. of Pot. 7. Iod. of Pot: Tart. Iron.

461—19. Henry C., 44. Letter Carrier. 1st attack. Ill 9 weeks. 62nd day discharged well.	At first off work from sore toe. Then had "rheumatic fever." All joints affected; delirious. 1. Pain in knees and shoulders. 39. Feet œdematous.	Heart sounds natural.		1. Iod. of Potass. 29. Belladonna Lin. 30. Iod. of Potass. Tart. Iron. 50. Dover's Powder; Guaiacum.

Gout or Rheumatic Arthritis.

464—20. William W., '50. Bath Chairman. 2nd attack. 50th day discharged well.	Has had pain in heels, knees, hands, elbows, and in the toes; and at the nipple, shooting to back. 5. Gouty nodule on joints of fore and little finger.	1. Heart sounds distant, feeble; appetite normal. 39. 1st sound loud ringing.		1. Bell. and Chl. Lin. 4. Iod. of Potass.
472—21. John T., 50. Labourer. 2nd attack. 15th day discharged well.	Had rheumatic fever 20 years ago. 1. Knees, ankles and hands affected; expression anxious. 6. Skin corrugated over hands; pain over knees much less severe.	TEMP. 1. 101 8. 98	1st sound very feeble; 2nd sound loud, arteries tortuous. 9. 1st sound less feeble.	4. Iod. of Pot. 13. Iod. of Pot. Tart. Iron.
	Probably Gout.			
473—22. Thomas F. Evidently worker in lead. 2nd attack. In bed 2 weeks. 15th day discharged well.	Had acute rheumatism 5 years since. Attack began in feet, then went to knees and ankles. 1. Now chiefly in shoulders, but cannot move joints. 4. Can move every joint. 7. Up; no pain anywhere. 11. Weak.	7. Pale; traces of blue line on gums.	1. Heart sounds natural. 7. 1st sound prolonged; faint pulmonic murmur.	1. Bel. and Chl. Lin. 7. Iod. of Pot. Tart. Iron.

ACUTE GOUT WITH ENDOCARDITIS; AND TRANSIENT PERICARDITIS.

417—1. George S. Plumber's Labourer. 2nd attack. Ill 15 days. 78th day discharged relieved.	Had acute rheumatism 4 years ago. 1. Pain in all joints. 7. Pain in knees. 28. Relapse. 32. No affection of joints; gets up. 39. Sudden gouty inflammation of great toe.	1, 4. Delirious; low; passed motions involuntarily. 11. Cloudy in mind; still wanders.	Obscure murmur over front of heart. 3. Peculiar plunging 1st sound; distant murmur. 14. Friction sounds heard over heart. 20. Musical murmur; the plunging sound has gone. 22–39. Musical murmur most distinct midway between sternum and nipple. 68. No murmur when he sits up; musical noise when he lies down.	7. M.Q. Fer. 17. Pot. Iod. 20. T. F. M. 61. Pot. Iod.

Gout; Endocarditis; transient Pericarditis.

ACUTE GOUT OR ACUTE RHEUMATISM.

413—1. William S. Horsekeeper. 3rd attack. Ill 3 days. 19th day discharged well.	1. Affection of wrist and left arm and joints of fingers; large joints not affected. 12. Up; walks about.	2. Feels anxious. No heart affection.	2. Iod. of Potass. Belladonna Lin. 25. T. F. M.
415—2. James M. Labourer. 5th attack. Ill 5 weeks. 21st day discharged well.	Acute rheumatism 10 years ago; probably great toe affected in previous attack. Pain in wrists and other joints. 9. Feels well.	Heart sounds natural, *i.e.*, no murmur, but 1st sound very feeble.	1. Lin. Belladon. Pot. Iod.
422—13. Charles B.. 50. No account of previous attack. Ill 5 days. 34th day discharged well.	Pain in knees, ankles, elbows, hands (which are much swollen).	1. No murmur. Looks flushed.	1. Bell. Lin. Quinia.
453—4. James L., 56. Carman. 2nd attack. Ill 7 weeks. 22nd day discharged well.	Had acute rheumatism 4 years ago; has had "gout" in wrists and thumbs since for 2 or 3 days. This attack affected all joints except toes. 1. Knees and wrists affected. 7. Pains quite gone. 9. Some in left knee. 18. None for a week.	TEMP. P. R. 1. 101 120 36	Heart sounds natural, but 1st prolonged at apex; no murmur. 4. Heart sounds feeble; 1st only audible over ventricles.

	Acute Rheumatism.										Acute Gout.	Acute Gout or Acute Rheumatism
			Endocarditis.									
	No Endocarditis.	Threatening of Endocarditis.	No Cardiac Murmur on Recovery.	Lessening Murmur on Recovery.	Apparently Established Murmur on Recovery.	Valve-disease held to be of old standing.	Total with Endocarditis.	Endocarditis, probable or doubtful.	Pericarditis.	Total Acute Rheumatism.		
Sex :												
Males	7	10	2	3	2	2	9	1	6	33	20	4
Females	7	12	12	1	1	5	19	3	0	41	3	0
	14	22	14	4	3	7	28	4	6	74	23	4
Age :												
At or below 20	5	8	7	4	0	5	16	1	2	32	1	0
21 to 25	6	10	5	0	0	1	6	1	1	24	4	2
26 to 30	2	1	1	0	1	0	2	0	1	6	5	0
31 to 40	2	2	1	0	1	0	2	2	2	8	4	0
Above 40	1	1	0	0	0	0	0	0	0	2	7	2
Not named	0	0	0	0	1	1	2	0	0	2	2	0
	14	22	14	4	3	7	28	4	6	74	23	4
Previous attack or not :												
1st attack	7	4	7	3	3	0	13	0	2	26	2	0
No account of previous attack	2	8	1	1	0	0	2	0	2	14	5	1
1 or more previous attacks	5	10	6	0	0	7	13	4	2	34	16	3
	14	22	14	4	3	7	28	4	6	74	23	4
Time ill before admission :												
1 week and under	8	11	4	0	1?	4	9	1	3	32	2	2
Above 1 week and under 2	3	5	8	2	1	2	13	1	1	23	4	0
Above 2 weeks	2	3	2	2	1	1	6	1	0	12	14	2
Time ill not stated	1	3	0	0	0	0	0	1	2	7	3	0
	14	22	14	4	3	7	28	4	6	74	23	4
Time of discharge :												
3 weeks and under	4	8	2	1	1	0	4	2	1 died	19	8	2
From 3 to 5 weeks	6	8	6	2	1	5	14	1	1 died	30	5	2
Above 5 weeks	3	6	5	1	1	2	9	1	4	23	9	0
Time not stated	1	0	1	0	0	0	1	0	0	2	1	0
	14	22	14	4	3	7	28	4	6	74	23	4
Time at which the joints ceased to be affected :												
Within 11 days	5	11	3	3	1	2	9	2?	0	27	5	3
12 to 21 days	5	7	4	0	0	2	6	1	2	21	6	0
Of these had relapse	**2**	**1**	0	0	0	0	0	0	0	**3**	**1**	0
Above 21 days	2	3	7	1	2	3	13	0	1	19	11	0
Of these had relapse	**1**	**2**	**2**	**1**	**1**	**2**	0	0	0	**9**	**7**	0
Doubtful	0	1	0	0	0	0	0	1	1	3	1	1
Died	1	0	0	0	0	0	0	0	2	3	1	0
Passed from Acute Rheumatism to Phlebitis	1	0	0	0	0	0	0	0	0	1	0	0
	14	22	14	4	3	7	28	4	6	74	23	4

TABLE SHOWING THE MEASUREMENT OF THE INFLAMED JOINTS (in inches).

ACUTE RHEUMATISM.

Name.	Date.	Temp.	Measurement of knee. Right.	Left.
428 (p. 463). James H.	1	103·1	15·7	—
	4	101·8	15·	—
	7	98·6	14·25	—
	10	98·25	14·	—
424 (p. 459). Thomas F.	1	—	Left. 14·5	—
	4	102·2	13·2	—
	5	100·6	13·	—
	7	98·	12·6	—
406 (p. 469). Fred. P.	1	—	Left. 14·3	—
	3	—	13·8	—
	8	—	13·	—
	11	—	12·2	—
401 (p. 460). Martha W.	4	99·8	Right. 13·1	Left. 13·1
	6	98·4	12·1	12·4
	8	—	12·	12·
400 (p. 466). John T.	2nd or 3rd	—	Right. 14·5	Left. 14·
	6	98·6	14·	14·
	8	97·8	13·3	13·7
405 (p. 465). Charles C.	2	—	—	Left. 13·5
	4	—	—	12·9
	7	—	—	12·6
414 (p. 457). Jesse R.	3	—	Right. 14·	—
	6	—	13·8	—
	9	—	13·4	—
	13	—	13·2	—
426 (p. 463). Matilda L.	2	102·1	Right. 12·5	Left. 12·6
	5	—	12·3	12·2
468 (p. 459). Eliza C.	1	—	Right. 13·9	Left. —
	6	—	13·7	14·1
	8	—	13·4	13·7
	9	100·4	—	13·5 Painful.
	13	99·3	—	13·
	17	—	12·6	13·
	23	—	12·25	12·3
411 (p. 457). Henry T.	1	102·4	Right. 15·5	15·2
	4	101·8	15·	15·3
	6	—	14·	14·5
	8	—	13·9	13·9
466 (p. 459). Laura B.	1	103·8	13·8	13·8
	4	102·	14·4	14·2
	9	100·	13·7	13·8
	13	99·2	13·4	13·4
	19	97·	12·6	12·7 No pain.
455 (p. 467). Mary H.	8	100·5	15·4	15·5
	9	99·5	15·	15·
	14	99·7	14·8	14·8
	18	98·	14·75	14·75
444 (p. 464). Jane C.	1	—	15·*	14·7
	4	101·	12·5*	14·
	8	99·1	13·2*	14·3
			Across metacarpus. Rt. wrist.	Lt. wrist.
Same patient	7	99·	10·	9·75
	10	100·	7·5	7·75
			Below styloid process.	
	7	99·	8·9	8·5
	10	100·	6·5	6·75

ACUTE GOUT.

Name.	Date.	Temp.	Right.	Left.
418 (p. 472). George W.	4	98·9	14·5	14·7
	5	—	14·7	14·2
	7	—	14·5	14·
	9	—	14·1	14·
	11	—	13·9	13·9 No pain.
416 (p. 472). Robert W.	1	—	Right. 14·5	Left. 14·5
	4	—	14·	14·5
	8	97·16	14·1	13·6
461 (p. 474). Henry C.	1	—	Right. 12·2	Left. 12·2
	6	—	11·6	11·6
445 (p. 473). Alex. R.	1	—	Right. 14·	Left. 13·9
	4	—	14·	13·9
	6	—	13·5	13·25

* Below patella.

TABLE SHOWING THE DAILY AMOUNT OF URINE.

ACUTE RHEUMATISM.

463 (p. 464). Martha H. — Endocarditis. On admission joints affected.

Day after admission.	Fluid taken in 24 hours.	Urine passed in 24 hours.	Reaction of Urine.	Appearance of Urine.	Specific gravity of Urine.	Temperature (at 1.30 p.m.).
	pints.	oz.				
2	3½	24	not acid			
3	4	24	not acid			
4	4	22	acid	very thick	1037	101·3
5	4	22	...	...	1032	100·2
6	4½	24	...	...	1030	99·1
						joints better
7	4	24	...	...	1020	99·3
8	4	34	...	...	1022	99·1
9	4	34	...	...	...	98·2
10 to 13	4	34	...	...	1020	98·2
					1020	98·
					22	98·3
14 to 21	3½	32	...	...	1020	97·4
					1018	...
					1020	98·
22	4	36	...	...	...	97·2
24 to 26	3, 5	36	...	...	1000	97·
					1016	98·
28	4½	38	...	...	1015	...
29	4	34	...	pale clear	1008	97·2
			Scarlet fever; renewal of stiffness of knee.			
31	4½	34	...	...	1009	100·
33	5	36	neutral	...	1012	100·2
35, 36	4½	34	...	...	...	101·
38	4½	32	...	...	...	100·
44	3½	30	very slightly acid	...	...	99·
45	3½	30	acid	...	...	98·1

474 (p. 465). Jane G. — Endocarditis. On admission joints affected.

Day after admission.	Fluid taken in 24 hours.	Urine passed in 24 hours.	Reaction of Urine.	Appearance of Urine.	Specific gravity of Urine.	Temperature (at 1.30 p.m.).
	pints.	oz.				
6 to 9	4	24 to 22	acid			
12	3½	22	acid	...	1027	
13	3½	24	acid			
15	4	34	scarcely acid			
16	3½	30	acid	...	1021	
21	3½	30				
32	3½	36				
33	4	30	commenced to take Iodide of Potassium			
34	4	26				
35	4	26				
38	3½	22	acid	nearly clear	1030	
39	3½	26	acid	clear	1030	
40	3½	28	acid	clear	1020	
41	3½	30	slightly acid	...	1020	

419—3 (p. 462). Improvement with increase of Urine.
George P., Acute Rheumatism, with threatening of Endocarditis.

Date.	Urine.	Drank of fluid.		
2	32 oz.	5 pints.		Cardiac pain.
3	24 ,,	5 ,,		Cough.
4	34 ,,	4 ,,	Pain in knees.	Better.
5	32 ,,	4 ,,	Less do.	Still cough.
6	38 ,,	4 ,,	Still pain.	
7	40 ,,	5 ,,	No pain.	Cough better.
8	48 ,,	4 ,,		
9	40 ,,	5 ,,		
10	50 ,,	5 ,,		
11	48 ,,	5 ,,		
12	32 ,,	5 ,,		

448—4 (p. 466).

Improvement, with increase of Urine.

Keziah D., Acute Rheumatism. Endocarditis: pains in joints. Drank 4 pints daily.

Date.	Temp.	Urine.	Sp. Gr.	
3	...	20 oz.		
4	...	22 ,,		
5	100·8	24 ,,	1032	
6	...	24 ,,	...	Feels better.
7	100·	22 ,,		
8	99·2	22 ,,	1025	Pains less.
9	...	34 ,,	...	Pains better.
10	...	34 ,,		
11	...	36 ,,		
12	...	...	...	No pain.

467—5 (p. 468).

Improvement, with increase of Urine.

Adelaide M., Acute Rheumatism, probably Endocarditis: pain in joints. Drank daily 3½—4 pints.

Date.	Temp.	Urine.	Sp. Gr.	
2	...	18 oz.		
4	101·3	23 ,,	1033	
5	101·	20 ,,	1030	
6	100·	20 ,,	1032	
9	98·	22 ,,	1030	Deposit: but little pain.
10	...	38 ,,	...	Clear.
12	97·4	38 ,,	1022	

457—6 (p. 466).

Slight if any improvement, with increase of Urine.

Mary L., Acute Rheumatism. Endocarditis: pain in joints. Drank 3½ pints daily.

Date.	Temp.	Urine.	
1	100	...	Pain over heart.
2	99	32 oz.	
3	97	20 ,,	
4	97	20 ,,	Less pain over heart.
5	97	20 ,,	Right knee very painful.
9	...	27 ,,	Little pain, except shoulder.
20	...	26 ,,	Better, brighter.
35	...	34 ,,	Pain, right shoulder.
44	...	34 ,,	Pain, left side.
62	...	38 ,,	General improvement.

456—7 (p. 461).

The Urine did not increase, either when the temperature fell, or when improvement took place.

Fanny R., Acute Rheumatism, threatening of Endocarditis. Drank 3½—4 pints.

Date.	Temp.	Urine.	
2	99	20 oz.	Perspiration at first considerable.
3	100·5′	20 ,,	No decided improvement during
4	99·5	20 ,,	this period; as pain left one joint
5	100	20 ,,	it attacked another.
7	98·5	22 ,,	
10	98	22 ,,	On the 10th looked very anxious.
11	99·5	22 ,,	
12	100	22 ,,	
14	99	20 ,,	
16	98	20 ,,	On the 16th she was cheerful, had
17	...	18 ,,	less pain.
18	...	18 ,,	
19	...	18 ,,	
20	...	18 ,,	On the 21st she was free from pain.
24	...	18 ,,	

436—8 (p. 467).

The temperature rose and fell, and the pain increased and diminished. The quantity of Urine was fair, and did not vary.

Ellen E., Acute Rheumatism. Endocarditis on admission. Drank daily 3—4 pints.

Date.	Temp.	Urine.	
5	...	20 oz.	Knees better.
6	...	50 ,,	
7	100·8	50 ,,	
8	...	40 ,,	
9	100·7	40 ,,	Joints worse; leeches.
10	...	40 ,,	Sudden pain in heart.
11	98·6	40 ,,	
12	100·4	50 ,,	Hands stiff; feels relieved.

441—9 (p. 466).

Urine scanty. Great increase of it on the 18th day, was not followed by improvement until the 21st and 23rd days.

M. A. C., Acute Rheumatism. Endocarditis; perspiration was not excessive.

Date.	Temp.	Urine.	Fluid taken.	
3	99 4	24 oz.	3½ pints.	Pain in the chest, and tenderness over the heart continued with varying pains in the limbs until the 10th, when she looked brighter, but the shifting pains in the joints continued, and on the 19th she had darting pain in the chest; steady improvement was not established until after the 23rd.
4	...	16 ,,	...	
5	...	12 ,,	...	
6	101	16 ,,	...	
7	100·2	24 ,,	...	
8	101	24 ,,	...	
10	101·8	24 ,,	4 ,,	
17	99·8	20 ,,	2 ,,	
18	...	34 ,,		
19	...	34 ,,		

ACUTE GOUT.

381—10 (p. 471).

John H., Gout: took Iodide of Potassium on alternate days.

Days on which he took the Iodide.			Days on which he did not take it.		
Date.	Urine.	Sp. Gr.	Date.	Urine.	Sp. Gr.
20	52 oz.		21	54 oz.	1010
22	36 ,,	1019	23	50 ,,	1013
24	44 ,,	1011	25	52 ,,	
26	42 ,,	1011	27	51 ,,	1014
28	44 ,,	1020	29	46 ,,	1020
Daily average 43½ oz.			Daily average 50½ oz.		

384—11 (p. 471).

Henry A.

Date.	Urine.	
7	54 oz.	Took Iodide of Potassium.
9	62 ,,	,, ,,
10—13	60 ,,	,, ,,
15	64 ,,	,, ,,
16	56 ,,	,, ,,
17	50 ,,	,, ,,
18	48 ,,	Took Tinct. of M. of Iron.
19	50 ,,	
23	50 ,,	
24	50 ,,	

453—12 (p. 475).

James L., Acute Gout or Acute Rheumatism. The Iodide was followed at first by diminution of Urine.

Date.	Urine.	
2	28 oz.	
3	20 ,,	
4	40 ,,	
5	33 ,,	Took Iodide of Potassium.
6	38 ,,	
7	48 ,,	
8	62 ,,	

417—13 (p. 474).

George S.

Date.	Urine.	
15	32 oz.	Taking Quinine and Iron.
16	30 ,,	
17	34 ,,	Took Iod. of Pot.
18	36 ,,	,, ,,
19	32 ,,	,, ,,
20	32 ,,	Took T. of M. of Iron w. Quin.
21	36 ,,	
22	32 ,,	
23	32 ,,	
24	28 ,,	
25	27 ,,	
26	32 ,,	
27	38 ,,	
29	40 ,,	
30	30 ,,	

No. in Author's List of Cases.	No. of Cases.	ACUTE RHEUMATISM.									GOUT.*				
				ENDOCARDITIS.					PERICARDITIS.						
		No Endocarditis.	Endocarditis threatened, but not developed	No Murmur on Recovery.	Lessening Murmur on Recovery.	Apparently Established Murmur on Recovery.	Valve Disease of old standing.	Endocarditis probable or doubtful.	With Endocarditis.	Without Endocarditis.	No Endocarditis.	Endocarditis or Pericarditis.	Gout or Rheumatism.	Time of Disappearance of Joint Affection.	Duration after Admission of Pericarditis.
1866—		Date.	Date.	Date.											
370	1	5 Oct.	...	...	...	...	...	...	...	...	...	...	...	5 or 6	...
371	2	...	...	30 Oct.	...	...	...	...	...	...	...	...	...	26 or 27	...
376	3	...	5 Oct.	...	...	...	...	...	...	...	...	...	...	8	...
377	4	...	9 Oct.	...	...	...	...	...	...	...	...	...	...	8?	...
378	5	...	...	...	...	...	...	...	12 Dec. on adm.	...	...	...	...	...	10 or 16
379	6	31 Dec.	...	...	...	...	...	...	...	...	...	...	...	...	...
1867—															
380	**7**	...	...	...	...	...	...	...	...	...	**16 April**	...	...	**51**	...
381	**8**	...	...	...	...	...	...	...	...	...	**9 May**	...	...	**17**	...
382	9	17 May	...	...	...	...	...	...	...	...	...	...	...	...	...
383	**10**	...	...	...	...	...	...	...	...	...	**7 June**	...	...	**50, 53**	...
384	**11**	...	...	...	...	...	...	...	...	...	**10 June**	...	...	**17, 20**	...
385	12	28 June	...	...	...	...	...	...	...	...	...	...	...	11	...
387	13	23 July	...	...	...	...	...	...	...	...	...	...	...	21	...
392	14	...	3 May	...	...	...	...	...	...	...	...	...	...	+ 28	...
393	15	...	4 May	...	...	...	...	...	...	...	...	...	...	9	...
348	16	...	19 July	...	...	...	...	...	...	...	...	...	...	12	...
394	17	...	...	...	...	...	...	9 Aug.	...	...	...	...	...	13	...
396	18	...	20 Sept.	...	...	...	...	...	...	...	...	...	...	42	...
397	19	11 Oct.	...	...	...	...	...	...	...	...	...	...	...	10	...
398	20	...	11 Oct.	...	...	...	...	...	...	...	...	...	...	18	...
399	21	...	11 Oct.	...	...	...	...	...	...	...	...	...	...	12	...
400	22	...	...	...	...	...	4 Nov.	...	...	...	...	...	...	8, 22	...
401	23	...	6 Nov.	...	...	...	...	...	...	...	...	...	...	8	...
402	24	...	...	...	13 Dec.	...	...	...	...	...	...	...	...	6	...
386	**25**	...	...	...	...	...	...	...	...	...	**29 June**	...	...	**20**	...
403	**26**	...	...	...	...	..	...	...	...	...	**13 Dec.**	...	...	...	...
404	**27**	...	...	...	...	...	...	...	...	...	**22 July**	...	...	**15**	...
405	28	...	...	...	13 Dec.	...	...	...	16 Dec.	...	...	...	...	5	...
406	29	...	...	...	...	...	...	...	...	...	...	...	...	38	...
1868—															
407	30	...	3 Jan.	...	...	...	...	...	...	...	...	...	...	11	...
408	**31**	...	...	...	...	...	...	...	...	...	**14 Feb.**	...	...	**38**	...
409	32	...	...	17 Feb.	...	...	...	...	...	...	...	...	...	24	...
410	33	...	...	...	...	...	6 June	...	...	...	...	...	...	11	...
411	34	30 Mar.	...	...	...	...	...	...	...	...	...	...	...	7	...
412	35	...	...	...	...	...	...	...	31 Mar.	...	...	...	...	50	14—19
413	36	...	...	...	...	...	...	...	...	...	...	...	17 April	12	...
414	37	22 April	...	...	...	...	...	...	...	...	...	...	...	...	...
415	38	...	...	...	...	...	...	...	...	...	...	...	May	9	...
416	**39**	...	...	...	...	...	...	...	...	...	**12 May**	...	...	**31**	...
417	40	...	...	...	...	...	...	...	...	...	...	**13 May**	...	...	...
418	41	...	...	...	...	14 May	...	...	...	...	...	...	...	6	...
418	**42**	...	...	...	...	...	...	...	...	...	**29 May**	...	...	**12**	...
419	43	...	29 May	...	...	...	...	...	...	...	...	...	...	7	...
420	**44**	...	...	...	...	...	...	...	...	...	**29 May**	...	...	...	...
421	**45**	...	...	...	...	...	...	...	...	...	**1 June**	...	...	**28**	...
422	46	...	...	...	...	...	...	...	...	...	...	...	2 June	...	...
423	**47**	...	...	...	...	...	...	...	...	...	**19 June**	...	...	**14**	...
424	48	...	19 June	...	...	...	...	...	...	...	...	...	...	...	...
425	49	...	19 June	...	...	...	...	...	...	...	...	...	...	7	...

* The Cases of Gout are printed in *Italics*, the figures in Egyptian type (4).

Day of Discharge, dating from Time of Admission.	Nature of Discharge.	No Medicine given on Admission, or early in the case	Belladonna Liniment with or without Chloroform.	Iron, with date after Admission, or when given.	Quinine given separately.	Opium or Morphia given internally.	Morphia injected under Skin.	Leeches. To Region of Heart	Leeches. To Joints.	Bicarbonate of Potash.	Iodide of Potass. with date.		
			†	†	†	†	†	†	†	†	†	†	
8	well	none	Lin. Sap. co.	...	...	2 P.Dov.	...	...	...	...	...	...	
66	well	none	39	11	...	3 L.O.S.	...	...	1	...	...	...	
23	well	none aft. 1st day	...	...	...	...	...	...	...	...	...	...	
19	well	none	...	...	...	...	...	...	...	...	...	M. Amm	
73d	last report	...	10	31	...	3 T. O.	...	2	...	...	...	...	Had phlebitis.
ng.	well	...	...	9	...	...	...	...	...	...	...	...	
58	...	...	...	...	...	...	...	...	...	...	...	...	
30	*well*	...	1	...	...	1	...	...	...	...	1	...	
4	well	none	1	...	...	...	1	...	...	...	...	Imperial	
14	*well*	*none*	1	46	...	...	14	...	...	...	28	...	
25	*well*	...	1	18	...	7	...	...	...	...	1	...	
1	well	...	...	...	...	...	...	...	...	...	...	*Colch.* 7	
2	well	*none*	2	29	...	...	2	...	...	...	...	...	
36	well	none	1	45	15	17	...	...	...	...	...	...	
2	well	none for 2 days	3	3	...	1 Op.	...	...	...	...	...	...	
9	well	none for 2 days	1	...	3	1 Op.	...	...	...	...	...	...	
2	...	none aft. 2nd day	...	...	...	...	...	...	...	1st and 2nd days	...	...	
5	well	none	34	43	...	...	1	...	...	...	...	...	
3	well	none	...	...	...	...	...	...	...	...	...	...	
8	convalescent	none	1	35	12	...	...	...	...	...	...	...	Relapse after getting up.
2	well	none	1	25	...	...	...	...	...	...	...	...	
5	convalescent	none	1	9	...	...	...	...	3	...	...	...	
1	convalescent	none	1	9	...	L.O.S.	...	...	1 June	...	...	...	
5	well	none	1	8	...	...	...	...	...	...	...	...	
21	*well*	...	...	18	...	...	...	...	...	...	...	...	
21	*well*	...	1	...	...	...	1	...	...	...	1	...	
9	*convalescent*	none	1	14	...	...	...	...	1	9	9	...	
2	well	none	1	9	...	...	...	...	...	13	...	...	
6	relieved	none	1	39	20	...	...	?	...	...	...	...	
1	well	...	1	2	...	...	...	...	...	...	2	...	
18	*well*	*none*	1	11	1	19 T.O.	...	...	5	...	20	47	
6	well	...	1	8	...	5	...	...	...	..	...	...	
6	relieved	none	1	13	...	...	...	...	...	...	8	...	
6	well	none	1	22	...	...	...	leeches	...	...	...	...	
0	well	none	1	39									
9	well	...	1	25	...	...	...	...	...	...	2	...	
4	well	none	1	...	10	...	...	...	...	...	...	...	
4	well	none	1	...	...	...	...	...	...	...	1	...	
8	*well*	...	1	...	25	...	...	...	...	...	1	...	
8	*relieved*	*none*	...	20	...	...	...	...	...	...	17	...	
6	well	none	1	15	14	...	...	...	...	...	...	...	
2	*well*	*none*	1	21	...	...	...	...	...	...	...	Amm. 4	
5	well	none	1	6	...	...	...	...	...	...	...	...	
8	*well*	...	7	...	1	...	...	...	...	...	1	...	
4	*relieved*	*none*	1	9	20	...	...	...	...	...	9	...	
4	well	...	1	...	...	...	...	...	...	...	...	...	
7	*well*	...	...	...	...	...	...	...	...	1	...	...	
6	*well*	*none*	1	...	...	...	...	...	...	...	...	...	
8	*well*	*none 2nd day*	1	...	...	...	...	...	...	1st day only	...	...	

† The figures in these columns show the date on which the remedy was used.

No. in Author's List of Cases.	No. of Case.	ACUTE RHEUMATISM.									GOUT.*				
		No Endocarditis.	Endocarditis threatened but not developed.	ENDOCARDITIS.				Endocarditis probable or doubtful.	PERICARDITIS.		No Endocarditis.	Endocarditis or Pericarditis.	Gout or Rheumatism.	Time of Disappearance of Joint Affection.	Duration after Admission of Pericarditis.
				No Murmur on Recovery.	Lessening Murmur on Recovery.	Apparently Established Murmur on Recovery.	Valve Disease of old standing.		With Endocarditis.	Without Endocarditis.					
1868—		Date.	Date.												
425	**50**	...	...	...	...	...	...	...	...	...	**23 June**	...	...	...	...
426	51	...	...	24 June	...	...	...	...	...	...	...	...	...	...	...
426b	52	...	...	...	...	...	10 July	...	...	...	...	...	...	7	...
428	53	...	...	11 July	...	...	...	...	...	...	...	...	...	...	...
429	54	...	14 July	...	...	...	...	...	...	...	...	...	...	14	...
427	55	11 June	11 June	...	...	...	...	...	...	...	...	...	...	18	...
431	56	5 Aug.	...	...	...	...	...	...	...	...	...	...	...	80	...
432	57	...	24 Aug.	...	...	...	...	...	...	...	...	...	...	...	...
433	58	...	...	28 Aug.	...	...	...	...	...	...	...	...	...	22	...
														23	...
434	59	...	15 Sept	..	...	...	...	...	...	...	...	...	...	...	...
435	60	...	...	...	15 Sept.	...	...	...	...	...	...	...	...	8 or 9	...
436	61	...	...	...	...	...	6 Oct.	...	...	...	...	...	...	28	...
437	62	...	6 Oct.	...	...	...	...	...	...	...	...	...	...	...	...
438	63	8 Oct.	...	...	...	...	...	...	...	...	...	...	...	8 or 9	...
439	64	23 Oct.	...	...	...	...	...	...	...	...	...	...	...	11	...
440	65	...	...	...	...	...	...	...	...	9 Nov.	...	...	...	...	16
441	66	...	...	14 Nov.	...	...	...	...	...	...	...	...	...	22	...
442	67	...	...	...	...	...	...	...	...	13 Nov.	...	...	...	...	6
443	68	...	13 Nov.	...	...	...	...	...	...	...	...	...	...	15–17	...
444	69	...	...	14 Nov.	...	...	...	...	...	...	...	...	...	16	...
445	**70**	...	...	...	...	...	...	...	...	...	**18 Nov.**	...	...	**7**	...
446	71	...	...	18 Nov.	...	...	...	...	...	...	...	...	...	9	...
447	72	...	...	...	...	...	...	4 Dec.	...	...	...	...	...	12	...
448	73	...	...	...	.	24 Dec.	...	...	...	...	...	...	...	...	...
450	74	...	...	24 Dec.	...	...	...	...	...	...	...	...	...	...	...
451	75	...	...	24 Dec.	...	...	...	...	...	...	...	...	...	...	...
452	76	...	...	...	30 Dec.	...	...	...	...	...	...	...	...	35	...
1869—															
453	77	...	...	...	...	...	...	...	...	...	15 Jan.	11	...	...	...
454	78	15 Jan.	...	...	...	...	...	...	...	...	...	...	...	...	...
455	79	...	...	...	...	...	15 Jan.	...	....	...	...	...	...	18	...
455	**80**	...	...	...	...	...	...	...	...	...	**16 Jan.**	...	...	**17**	...
456	81	...	19 Jan.	...	...	...	...	...	...	...	...	...	...	21	...
457	82	...	...	...	...	19 Jan.	...	...	...	...	...	...	...	...	...
458	**83**	...	...	...	...	...	...	...	...	...	**5 Feb.**	...	...	...	...
459	**84**	...	...	...	...	...	...	...	...	...	**6 Feb**	...	...	...	...
460	85	...	...	...	...	...	8 Feb.	...	...	...	...	...	...	...	...
461	**86**	...	...	...	...	...	...	...	...	...	**10 Feb.**	...	...	...	...
462	87	...	26 Feb.	...	...	...	...	...	...	...	...	...	...	18	...
463	88	26 Feb.	...	...	...	...	...	...	...	...	...	...	...	...	...
464	**89**	...	...	...	...	...	...	...	...	...	**18 Mar.**	...	...	...	...
466	90	...	19 Mar.	...	...	...	...	...	...	...	...	...	...	16	...
467	91	...	...	...	...	...	...	19 Mar.	...	...	...	...	...	15	...
468	92	...	9 April	...	...	...	...	...	...	...	...	...	...	...	...
469	93	...	...	10 April	...	...	...	...	...	...	...	...	...	...	...
470	94	...	...	...	...	...	14 April	...	...	...	...	...	...	...	...
471	95	...	30	30 April	...	...	...	...	...	...	...	...	...	25	...
472	**96**	...	...	...	...	...	...	...	...	21	**30 April**	...	...	...	...
473	**97**	...	...	...	...	...	...	...	...	22	**30 April**	...	...	...	...
474	98	...	...	21 May	...	...	...	...	...	...	...	...	...	...	...
475	99	...	...	21 May	...	...	...	...	...	...	...	...	...	...	...
476	100	...	...	...	...	...	...	...	...	12 June	...	...	...	...	...
465	101	...	...	...	...	...	...	19 Mar. doubtful.	...	...	...	...	...	...	...

* The Cases of Gout are printed in *Italics*, the figures in Egyptian type (4).

Nature of Discharge.	No Medicine given on Admission, or early in the case.	Belladonna Liniment with or without Chloroform.	Iron.	Quinine given separately.	Opium or Morphia given internally.	Morphia injected under Skin.	Leeches. To Region of Heart	Leeches. To Joints.	Bicarbonate of Potash.	Iodide of Potass, with date.		
		†	†	†	†	†	†	†	†	†	†	
died	...	**1**	**1**	...	...	...	...	**10**	...	...	*M. Amm.*	*Contracted kidney Double valve-disease.*
well	none	1	13	...	...	...	...	...	...	...	...	
much relieved	...	1	1	...	...	...	...	...	...	...	...	
well	none	1	...	...	...	...	...	5	...	...	...	
well	none	1	...	4	...	...	...	...	...	...	...	
...	...	...	...	...	...	...	...	...	...	...	...	
died	...	1	...	...	...	...	...	...	1	...	...	Delirium; large kidneys.
well	none	1	29	24	3	...	...	...	...	...	...	
well	none	19	28	...	25	9	...	...	3rd and 4th days	...	...	
well	none	1	...	...	1	...	...	...	...	...	...	
well	none	19	24	...	1	...	...	...	...	...	...	
well	none	1	15	...	4	...	...	10	...	...	...	
well	none	1	24	...	...	...	8	...	...	...	...	
well	none	1	...	...	...	...	...	...	...	...	3 T. Val.	
well	...	...	...	...	...	...	...	...	...	...	...	[cine.
died	none	1	16	...	...	4	1	...	...	8	...	No record of medi-
well	none	1	22	5	...	...	...	...	...	...	...	Purpura. Aortic &
relieved	none	1	...	35	...	...	4	...	...	...	...	mitral disease of
well	none	1	35	6	...	...	...	...	...	...	...	old standing.
well	none	1	...	8	...	...	...	...	...	...	...	
well	...	...	...	...	...	...	...	...	..	...	...	
well	...	1	13	12	...	...	8	...	...	...	1	
well	none	1	...	...	...	...	...	...	...	...	1 Imper.	
much better	...	...	...	...	...	...	...	...	...	...	...	
well	...	...	...	...	...	...	...	...	...	...	...	
well	none	1	21	...	...	...	...	...	...	...	...	
well	...	1	...	2	...	...	...	...	...	...	...	
well	...	1	4	...	...	...	...	...	...	4	...	
well	...	1	...	...	35	...	...	...	...	...	28 Brom. of Potm.	Subject to attacks of jaundice.
well	none	1	...	...	...	...	...	...	...	...	...	
well	...	**1**	**1**	...	...	...	...	...	...	...	...	
well	none	1	...	...	...	...	...	...	...	**1**	...	
relieved	none	1	52	32	...	...	...	...	...	...	...	
well	...	**1**	**12**	...	...	...	...	...	...	...	...	
relieved	...	**1**	**7**	...	...	...	...	...	...	**1**	...	
relieved	...	1	47	...	...	...	...	...	...	**1**	...	
well	...	**1**	**30**	**50**	...	...	...	...	...	...	78 Cit. of Potash.	
well	none	1	...	21	...	...	...	...	...	**1**	...	
well	none	...	...	...	10	...	...	...	...	...	18 Amm.	
well	...	**1**	...	...	...	...	...	...	...	...	...	Diarrhœa.
well	none	1	...	5	...	...	...	6	...	**1**	...	
well	none	1	...	...	...	...	...	...	...	...	...	
well	none	1	none	39	...	...	2	8	...	...	...	
well	none	1	60	...	...	...	2	...	...	...	...	
relieved	none	1	...	6	8	...	3	...	...	...	...	Took scarlet fever
going out	none	1	...	...	...	...	...	...	...	...	...	when recovering
well	*none*	...	**13**	...	...	...	...	...	...	...	...	from attack.
well	*none*	**1**	**7**	...	...	...	...	...	...	**4**	...	
relieved	...	13	13	1	13	...	...	...	...	**7**	...	
relieved	none	1	...	...	...	...	...	...	...	34	...	
died	none	1	...	...	4	6	...	...	...	6	...	
relieved	none for 4 days.	1	...	...	4	...	...	...	...	...	...	Delirium tremens.
											4 Amm.	

† The figures in these columns show the date on which the remedy was used.

XXVII.

THE FLEXIBLE STETHOSCOPE.

To the Editor of the Medical Gazette.

SIR,

I HAVE been well pleased to see the able communications on the Flexible Stethoscope from Drs. Burne and Bird, in the pages of your journal. I cannot but lament that a correspondence so interesting and valuable to the profession should degenerate into personal attack and mutual recrimination. However, this good effect has been answered: each controversialist has dismantled the other of any lurking pretension he might have to the introduction of the instrument in question, and has attributed it, correctly, to my estimable friend, Dr. Stroud.

In the summer of 1838, I employed a flexible stethoscope, consisting of two feet of the ordinary elastic flexible tubing, and a small funnel-shaped extremity of wood, in some experiments I made to satisfy myself as to the cause of the various sounds occurring during the heart's action. This instrument conducted the sounds more distinctly, and was much cheaper, than the flexible ear trumpet: being devoid of an ear piece, it remained in the ear without being held there; thus leaving one hand at liberty to feel the pulse. Since that time I have constantly employed the flexible in preference to the solid stethoscope. I am informed by Mr.

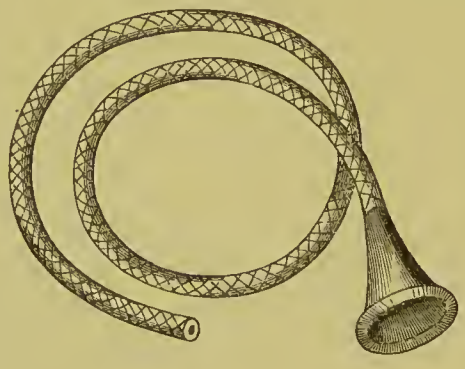
Thompson, instrument-maker in this town, that he has sold about a hundred instruments similar to that described above to medical practitioners residing in this town and neighbourhood.

I beg to corroborate Dr. Bird and Dr. Burne's statements as to the advantages possessed by the flexible stethoscope. In addition I feel desirous of calling the attention of the profession to the perfect ease with which pressure may be modified in using this instrument.

In simple pericarditis, in the early stage, on applying the extremity of the stethoscope lightly, the natural sounds of the heart are heard; on increasing the pressure they become replaced by the friction-sound. Up to a certain point the greater the degree of pressure the louder is the friction-sound. In the more advanced stages of pericarditis, when the pericardial surfaces are much roughened immediately over the apex of the heart and the lower part of the sternum, the friction-sound is heard feebly on light pressure; but at a short distance from these points the natural sounds alone are distinguished; when pressure is exerted and increased, the friction-sounds come into play. When pericarditis and endocarditis coexist, if the inflammation of the lining membrane of the aorta be sufficient to modify the natural sounds, the bellows murmur is heard, on light pressure, over the centre of the sternum: on increasing the pressure the friction-sound replaces it.

In adhesion of the two pericardial surfaces, with general enlargement of the heart, accompanied, as is often the case, by regurgitation from the left ventricle during its systole, through the auriculo-ventricular opening into the corresponding auricle, owing to imperfect closure of the mitral valve, the noise from the shock of the walls of the heart against the parietes of the chest may be heard over the

apex; followed by the dull rumbling sound of muscular contraction, ending in an abrupt loud sound, caused, I conceive, by the sudden withdrawal of the hardened walls of the heart from the thoracic parietes. Near the seventh or eighth dorsal vertebra on the left side a bellows murmur is heard during the first sound, caused by the auriculo-ventricular regurgitation: increase of pressure does not modify this sound. On applying the stethoscope slightly over the region intermediate to this situation and the apex of the heart, the



bellows murmur is heard; on making strong pressure it is replaced by the various sounds heard over the apex. In other cases of diseased heart, varying the pressure modifies the abnormal sounds.

In the various murmurs produced by pressure over the veins in the neck, over the uterus, liver, and abdominal tumours, the ease with which the degree of pressure may be modified by the flexible stethoscope greatly facilitates the production of the sounds.

In those cases of diseased lungs, where the vocal resonance communicated from the thoracic parietes is altered, the flexible stethoscope affords great facility in making a rapid comparative survey of the chest. The vocal reso-

nance is much more extensively heard on light than on firm pressure.

With your permission I hope to follow this short statement of the advantages to be derived from modifying the pressure of the stethoscope in the diagnosis of the diseases of the heart and lungs, by further communications containing a more detailed account, with illustrative cases, of the points which have been mentioned in the foregoing remarks.

I am, sir,

Your obedient servant,

FRANCIS SIBSON.

Resident Surgeon, General Hospital, near Nottingham.

Aug. 13, 1841.

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