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ON THE EFFECT OF CHRONIC DISEASE OF THE VALVES OF THE HEART UPON THE SOUND ORIFICES, THE CAVITIES, AND THE WALLS.

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Introductory.

The relationship of the condition of the walls and cavities of the heart to that of its apertures altered by disease, is admittedly of the greatest prognostic importance. The doctrines current on the subject, however, are somewhat loose and untrustworthy. Thus the various degrees of incompetence and constriction of the aortic are roughly held to occasion more or less hypertrophy of the left ventricle, but whether one form of lesion occasions it more than another—whether, for example, simple incompetence, incompetence with constriction, or incompetence with dilatation, &c., is attended with the greatest amount of hypertrophy—and what the mechanism is by which the difference, if any, is to be accounted for, seem to be insufficiently understood The effects of a lesion of one orifice upon the size and competence of the others, is also a matter which might well be placed in a clearer light. The mechanism of cardiac dilatation, moreover, appears to be worthy of further investigation.

This paper has been written chiefly with the object of supplying some of the facts which have hitherto been needed to place the pathology of hypertrophy and dilatation upon a firmer footing. For the last thirteen years I have kept an accurate record of the size of the apertures, the thickness of the walls, the length of the ventricular cavities, the weight of the organ, &c., in nearly every *cadaver* which has passed under my notice as pathologist to the Edinburgh and Aberdeen Royal Infirmaries. The facts derived therefrom have mainly served as the basis of the conclusions I have drawn in the sequel.

The measurements of the orifices were all made by carefully-graduated box-wood cones. These are undoubtedly much the

most reliable means of ascertaining the size of any aperture in the body, provided they be judiciously used. The cone should be introduced into the aperture in the same direction as that in which the blood naturally passes through it. It should be pushed through just until it catches, but no violence should be employed to drive it beyond this. It will be found that a graduation to decimals of an inch diameter is a sufficiently fine subdivision. The weights were taken in ounces avoirdupois.

The estimation of the size of the ventricles was arrived at by measuring *the distance from the apex of the cavity up to the base of the nearest sigmoid segment.* I am well aware that this method of ascertaining the size of the ventricle might be held to be open to objection. As a matter of experience, however, it will be found that it gives a much more accurate estimate of the total capacity of the cavity than might at first be supposed, and when combined with a description of the general appearance of the organ, must be held to be infinitely preferable to an unsupported and general statement.

The measurement of the walls was always taken at their thinnest and at their thickest parts; the sub-pericardial fat was not included, and the parts selected were those intervening between the musculi papillares.

The figures which are quoted in the paper are, of course, the *averages* of the cases observed under any particular class.

Weights and Measurements of the Normal Heart.

In estimating the weight and dimensions of any organ, it is necessary to draw conclusions only *from absolutely healthy individuals, who have met their death suddenly by accident.* In no organ must this be more rigidly enforced than in the case of the heart. In order to be exact, we must also have some general notion of the bulk of the individual as derived from the height and girth at the shoulders, and, of course, sex must be taken into the reckoning. It is also essential to be informed of how much of the vessels in the case of the heart was left attached, and whether the heart was weighed before or after emptying it.[1]

[1] The discrepancies to be found in the figures given by Peacock, Glendinning, Bizot, Rosenstein, and others are probably owing to failure in the observance of these precautions.

The following statistics were derived from the examination of 27 males and of 4 females, all over 19 years of age, who were in perfect health at the time of death, and who were accidentally killed. The heart was excised in all cases by cutting through the middle of the arch of the aorta and through the pulmonary artery close to the bifurcation. The attached parts of the vessels were included in the weights, and the organ was invariably opened and washed out before the weight was ascertained.

My results show briefly that the commonest *weight* of the male heart, excised as before mentioned, was from 12 to 13 oz., and that it ranged between 10 and 16 oz. They also demonstrate that the heaviest average of hearts was in the tallest individuals, although there were exceptions to this rule, and that the heart, although generally, was not invariably of low weight in persons of small stature.

In the female the organ usually weighed between 10 and 11 oz. It ranged from 7 to 15½ oz.

The *diameters of the various orifices* were found to be as stated in the accompanying table:—

Diams. of Orifices—Male.

	Greatest.	Least.	Average.
Aortic,	1·3 in.	·9 in.	1 in.
Mitral,	1·8 ,,	1·1 ,,	1·4 ,,
Pulm. Art.,	1·5 ,,	1 ,,	1·2 ,,
Tricuspid,	2·2 ,,	1·3 ,,	1·8 ,,

Diams. of Orifices—Female.

	Greatest.	Least.	Average.
Aortic,	1 in.	·8 in.	·9 in.
Mitral,	1·5 ,,	1 ,,	1·2 ,,
Pulm. Art.,	1·3 ,,	1 ,,	1·1 ,,
Tricuspid,	1·7 ,,	1·4 ,,	1·5 ,,

My statistics also show that the taller the subject the larger the orifices, but that there were exceptions to this rule. When one of the orifices was large, the others were usually found to be correspondingly so.

The measurement of the *ventricular cavities* in the foregoing healthy hearts, estimated from the fixed points before

mentioned, showed that in the male, the lowest for the left ventricle was $2\frac{1}{2}$ in., the highest $3\frac{3}{4}$ in., and the average $3\frac{1}{4}$ in.; while in the female, the lowest was $2\frac{1}{4}$ in., the highest $3\frac{1}{2}$ in., with an average of 3 in.

That of the right ventricle in the male ranged between 3 and 4 in., with an average of $3\frac{3}{8}$ in.; while in the female it lay between 3 and $3\frac{1}{4}$ in., with an average of $3\frac{1}{16}$ in.

The *thickness of the wall of the left ventricle* was, as a rule, found to be about $\frac{1}{4}$ in. at its thinnest and $\frac{1}{2}$ in. at its thickest, both in the male and female; while that of *the right ventricle* was on an average $\frac{1}{8}$ in. all over in both sexes. Parts of it occasionally measured as much as $\frac{1}{4}$ in. or as little as $\frac{1}{16}$ in.

As the statistics which I give of diseased hearts include both males and females, it will be necessary to frame an approximate statement of the weights and measurements for comparison. The following is founded on the different averages just given for both sexes.

Table of Average Weight and Measurements of the Normal Heart.

Weight 10 to 13 oz.

Diams. of Orifices.		Cavities and Walls.	
Aortic,	·9 to 1 in.	L. Vent.,	3 to $3\frac{1}{4}$ in.
Mitral,	1·2 ,, 1·4 ,,	Wall $\frac{1}{4}$ in. (at thinnest) to $\frac{1}{2}$ in. (at thickest).	
Pulm. Art.,	1·1 ,, 1·2 ,,		
Tricuspid,	1·5 ,, 1·8 ,,	R. Vent.,	$3\frac{1}{16}$ to $3\frac{3}{8}$ in.
		Wall,	$\frac{1}{8}$ in. (all over).

It should be mentioned that the weights and measurements of the diseased hearts were all estimated on the same conditions as in the case of those which were healthy.

Effects of Chronic Disease of One Orifice upon the Size of the Others.

The general impression is that chronic disease of one orifice in course of time has a detrimental effect upon the size and competency of the others. Balfour,[1] for instance, holds that the most frequent cause of serious tricuspid regurgita-

[1] *Diseases of Heart and Aorta*, 1876, p. 181.

tion is mitral stenosis, and that great obstruction at the aortic orifice has a similar effect. The blood is hindered in its passage onwards, and hence tends to regurgitate upon the right side of the heart. On the other hand, he says that aortic regurgitation, although a frequent enough disease, is anticipated in its injurious results on the other orifices by its own peculiar sources of mortality, and hence it is only comparatively rarely that aortic regurgitation gives rise to tricuspid regurgitation of a serious character.

It remains to be seen in how far this notion is borne out by facts.

A. *Pure Aortic.*

(1) Aortic of normal size, but incompetent.

In this case the average size of the various orifices was the following :—

Aortic.	Mitral.	Pulm. Artery.	Tricuspid.
·99 in.	1·3 in.	1·1 in.	1·8 in.

It would thus seem that in simple regurgitant aortic without alteration in the size of the orifice, the effect upon the size of the other orifices is practically *nil.*

(2) Aortic constricted and incompetent.

Aortic.	Mitral.	Pulm Artery.	Tricuspid.
·75 in.	1·3 in.	1·1 in.	1·7 in.

Here the result is essentially as in Class I., the effect upon the tricuspid being even less than in it.

(3) Aortic dilated and incompetent.

Aortic.	Mitral.	Pulm. Artery.	Tricuspid.
1·2 in.	1·7 in.	1·2 in.	2 in.

The result in this case is different from that in the two foregoing. As will be noticed, the effect of the dilated and incompetent aortic has been to *induce a general distension of all the other orifices.* It is to be explained simply by the wide incompetent aortic allowing of a sudden and full regurgitation of the arterial blood upon the left ventricle during diastole, that is to say, while the mitral valve is open. This diastolic

reflux continued for a sufficiently long time, by maintaining the pulmonary artery and the right chambers of the heart in a chronic state of distension, induces a widening of their orifices. The more constricted the aorta becomes, the less, naturally, will this direct regurgitant arterial pressure react upon the sound orifices.[1]

I do not find a single case in my records in which, when the aortic alone was diseased, it was constricted without being incompetent. The incompetency was, however, sometimes slight.

B. *Pure Mitral.*

(1) Mitral constricted and incompetent.

This constitutes by far the commonest deformity of the mitral resulting from old standing endocarditis. Old standing endocarditis causes thickening of all parts of the mitral. Not only does the substance proper of the valve suffer, but where the disease has been at all extensive the chordæ tendineæ will be found almost certainly to participate. This thickening diminishes the size of the intervals between the subdivisions of the chordæ, and ultimately fills these up so completely that the mass comes to resemble a solid fibrous pillar rather than an aggregation of isolated bands. At the same time the chordæ are dragged upwards towards the margin of the valve, and are so shortened that the tip of the musculi papillares and the edge of the valve may actually be in contact. The posterior wall of the ventricle is also pulled upon, and being thus bound to the mitral is retarded in its free action.

It accordingly follows that when the mitral is constricted, it almost to a certainty becomes incapable of accurately closing the orifice, and so allows of more or less regurgitation. The evil influence, however, of the defect upon the size of the other orifices is inappreciable, as the accompanying figures testify:—

Aortic	Mitral.	Pulm. Artery.	Tricuspid.
·97 in.	·86 in.	1·1 in.	1·7 in.

This result is probably to be explained by the constriction in a

[1] The mechanism of the arterial recoil is entered into more fully further on.

manner modifying the dilating tendency of the mitral regurgitance upon the orifices of the right side.

(2) Pure dilatation of the mitral.

A dilatation of the mitral with a competent and otherwise healthy aortic is a form of lesion only very seldom encountered. In the whole of my records of individuals dying from various diseases I can find but seven cases. A congenitally large mitral, with corresponding enlargement of all the other orifices, occurred in eleven cases. In only one case of mitral dilatation was there evidence of endocarditis, and in this single example it was of an acute nature and of septic origin. In none of the cases was it noted that a murmur was audible, although from the necessarily brief record of the history in the *post-mortem* journals, too much reliance should not be placed upon this statement.

Passing over, in the meantime, those which were evidently congenital enlargements, or at least in which the orifices were all equally dilated, let us examine the effect of the dilatation of the mitral upon the other orifices. The figures are the following:—

Aortic.	Mitral.	Pulm. Artery.	Tricuspid.
1 in.	1·68 in.	1·27 in.	1·98 in.

This practically shows that with a mitral of 1·7 in. diameter, the pulmonary artery and tricuspid corresponded respectively to close on 1·3 and to 2 in.

Most of the subjects unfortunately died from lung disease, and hence it is difficult to say in how far the dilatation of the right orifices was due to this cause. It should be mentioned, however, as supporting the belief that the dilated mitral was one of the chief factors in inducing the enlargement, that the dilatation of the right orifices was quite as great in those cases where the lungs were sound, or at any rate in which they were not the seat of disease unconnected with the cardiac condition.

C. *Aortic and Mitral combined.*

(1) Aortic and mitral both constricted; aortic competent, mitral incompetent.

It might have been expected that in this case the orifices on

the right side would have suffered dilatation. Such, however, as will be seen from the accompanying results, was not the case.

Aortic.	Mitral.	Pulm. Artery.	Tricuspid.
·7 in.	1 in.	1·1 in.	1·6 in.

It is probable that the constriction of the mitral, in these as under other circumstances, prevented the deleterious effects of the regurgitance through its orifice being felt.

(2) Aortic and mitral both constricted and both incompetent.

The measurements are the following:—

Aortic.	Mitral.	Pulm. Artery.	Tricuspid.
·8 in.	·77 in.	1·08 in.	1·6 in.

The only effect upon the orifices of the right side, be it a consequence or a mere coincidence, would appear to have been that of rendering them actually somewhat smaller than those of the average healthy heart, more especially as this class was taken entirely from males.

(3) Aortic of natural size but incompetent, mitral constricted and incompetent.

Nearly 50 per cent. of the cases were females, and, as will be seen, the effect upon the size of the orifices of the right side was inappreciable.

Aortic.	Mitral.	Pulm. Artery.	Tricuspid.
·97 in.	·87 in.	1·1 in.	1·6 in

D. *Tricuspid.*

The cases of tricuspid disease observed by myself were unfortunately all combined with disease of the left orifices, and hence no differential conclusions could be drawn from them.

Summary of the foregoing Results.

The data just enumerated bring out the somewhat remarkable result, that the only lesions of the valves of the left side which are accompanied by any appreciable distension of the otherwise sound orifices are *uncomplicated aortic regurgitance with a wide orifice*, and *uncomplicated dilatation of the mitral*. In the former of these, the other three orifices were

considerably above the average diameter, and in the latter, both the pulmonary artery and tricuspid were distended while the aortic remained of natural size. As regards the other classes, it may be noted that *where incompetence of a valve was accompanied by constriction of the orifice, the remaining apertures were not sensibly affected*, the legitimate conclusion deducible from this being that constriction of an incompetent orifice exerts a salutary influence in preventing distension of the other orifices of the organ.

On referring to the sequel it will be found that a constricted aortic and mitral lesion, where both are incompetent, occasions great hypertrophy of the right ventricle, whereas we have just seen it has but little influence upon the size of the right orifices. At first sight these results seem somewhat discordant, but may probably be explained in the following manner. The constricted aortic and mitral will prevent such a direct recoil of the arterial blood during diastole as would happen were they of natural size or dilated. This will tend to keep up a tonic state of high tension within the pulmonary circuit rather than to subject it to sudden exacerbations of pressure. Such a continuous strain in course of time might induce hypertrophy of the right ventricle, seeing that the ventricle will have to contract more energetically in order to open the pulmonary artery orifice, without the orifices becoming much enlarged. Where, however, the aortic and mitral openings are wide and where the aortic is incompetent, there must, in addition, be an instantaneous recoil of the arterial pressure upon the whole pulmonary system during each diastole, with a sudden tendency to stretch the right orifices, resulting most probably in a permanent distension of the latter and hypertrophy of the wall of the right ventricle.

Hypertrophy and Dilatation from Valvular Disease.

A. *Aortic.*

Although in the majority of aortic lesions hypertrophy of the left ventricle will be found to coexist, yet, as Gairdner[1] remarked, there is a capriciousness as to its presence or absence which is

[1] *Brit. and For. Med. Chir. Rev.*, vol. xxiii., 1853, p. 211.

sometimes difficult to explain. When the aortic is experimentally injured, its reserve energy, which, as shown by Rosenbach,[1] is considerable, is called forth. It contracts more vigorously, and does so until new muscular fibre is generated in sufficient amount to compensate for the defect. In course of time dilatation ensues,[2] and is followed by hypertrophy. Balfour states[3] that such is also the sequence of events in man.

Why does dilatation take place, and what is the increased work performed by the heart which calls forth the hypertrophy of its fibre ?

Let us consider the simplest aortic lesion to begin with, namely,—

(1) Aortic incompetence without constriction or dilatation of the orifice, the mitral being normal.

The blood here, of course, will regurgitate from the aorta during ventricular disatole as a primary and immediate result, and the cavity will thus be filled from two sources instead of from one. Increased strain will be thrown upon the cardiac muscle from the arterial recoil, and there will be a much greater tendency to dilatation than in health.

The blood in a sound heart, at least during the first stage of diastole, flows passively into the ventricle, without receiving much if any impulse from the auricle. The latter contracts as it is becoming emptied and drives the remainder of the blood out of its cavity. The blood must therefore impinge against the ventricular wall with very little force during health, and hence the strain will be comparatively slight. It has even been said by Pettigrew[4] that the dilatation of the ventricle is a vital act.

In simple aortic regurgitation, however, the arterial recoil must be enormous, and it occurs during diastole. Rosenbach[5] found it to be sufficient to produce aneurism of the apex in animals in which the valve had been artificially destroyed.

The cause of the recoil is usually held to be the elasticity of

[1] *Arch. f. exp. Pathologie u. Pharmakologie*, vol. ix., 1878, p. 10.
[2] *Loc cit.*, p. 12.
[3] *Diseases of the Heart and Aorta*, 1876, p. 71.
[4] *Physiology of the Circulation*, 1874, p. 115.
[5] *Loc. cit.*, p. 14.

the arterial walls. It is, however, doubtless erroneous to regard this as the only cause. In estimating the factors which are instrumental in raising the blood-pressure, the arteries are commonly looked upon as if they were a series of exposed tubes. In reality, however, there is no part of the body in which the arteries or capillaries can be said to lie exposed. The nearest approach to it is in the case of the capillaries on the alveolar walls of the lung, but even here they are connected to an eminently elastic tissue and are covered by endothelium. In all other parts they are bound down by tissues more or less resilient (yellow elastic fibre, white fibrous tissue, muscle, &c.), whose interspaces are filled with liquid. This liquid will consequently tend to diffuse the elastic pressure of the tissues, and to bind it to that of the arterial wall.

Even in the abdomen the tissues are all so intimately and mutually applied to each other, that the abdominal contents may be regarded as a solid mass.

The skin and the immediately subjacent tissues are, finally, so elastic that they tend to complete a counterpoise sufficiently strong to resist the expansile efforts of the heart, transmitted throughout the whole body by the hydraulic machinery of the blood-vessels.

Donders[1] seems to have been the first to draw attention to this relationship of the arterial walls to the tissues encompassing them. He says, "We have previously seen that the blood-vessels do not bear the entire blood-pressure. They would become more expanded if they were not supported by the surrounding tissues. Part of the blood-pressure is expended upon the tissues and the nutritive liquids which bathe them."

Some years since I endeavoured[2] to demonstrate the importance of the influence exerted by this elastic reaction of the tissues in the processes of healing and organisation.

Landerer[3] has lately gone fully into the subject, and has

[1] *Physiologie d. Menschen.*

[2] *Journal of Anatomy and Physiology*, vol. xiii., 1879, p. 518; *Edinburgh Medical Journal*, vol. xxvii., 1881, p. 385.

[3] *Die Gewebsspannung in ihren Einflusse auf örtliche Blut und Lymphbeengung*, 1884.

estimated the power of recoil of the tissues in various parts and in different animals.

There cannot be much doubt that the subject of the relationship of the elasticity of the tissues, and of their contained liquids to the circulating blood, is one of the most profound in all pathology, and is one which has hitherto been much disregarded. We have been in the habit of attributing variations in the elasticity of the arteries exclusively to modifications of the arterial wall. It must, however, be sufficiently ostensible on reflection, that, as the arterial and capillary coats and the surrounding tissues and liquids may practically be regarded as continuous, and as constituting one texture, any modification in the elasticity of the fibre of the latter, or any difference in the quantity of liquid lying in the interspaces of the tissue, must react upon the blood in the vessels in very much the same manner as the arterial wall itself.

During muscular exertion, moreover, the pressure of these liquids will be increased, and will constitute at least one of the factors which go to raise the pressure in the arteries generally and in the aorta.

In aortic regurgitance all such exacerbations of pressure have to be borne by the ventricular wall, the mitral orifice, the lung, and the right side of the heart; and the larger the aperture of the aorta, as previously pointed out, the more suddenly will the returning gush of blood impinge upon them, and the greater will its influence for evil consequently prove. The mitral orifice and those orifices on the right side, as already demonstrated, will accordingly become unnaturally large, and the cavities of the ventricles will also increase in capacity.

The dilatation of the ventricle would, no doubt, continue to be progressive were means not forthcoming to withstand the undue backward strain brought to bear upon its interior. This is to be sought *in the hypertrophy of the muscular fibre.* The mass of new muscle maintains the tone of the heart, as that of the hypertrophied arteries maintains the arterial tone in cirrhotic Bright's disease or other chronic affection accompanied by a high arterial pressure.

The muscle of the heart, like involuntary muscle surrounding other cavities, has a *twofold* action. *It drives out the*

contents of the cavity, and it prevents over-distension. It is thus engaged, not only in propelling the blood, but also in maintaining the tone of the heart-wall. It is otherwise difficult to explain how the heart, even in health, with the constant filling of its cavities, does not in time become distended. The heart-muscle, in health, seems to possess a tonic function analogous to that of the musculature of the arteries, and this is brought more especially into play when the aortic blood is allowed, through insufficiency of the valve, to recoil upon the interior of the ventricle during diastole.

This tonic function of the heart-muscle is often overlooked, but must be one of very considerable importance.

Gaskell,[1] in summing up the results of his admirable paper on the innervation of the heart, concludes that muscular tissues exhibit three modes of responding to stimulation. Certain muscles possess essentially the power of "tonic contraction," others the power of "rhythmical contraction," and others that of "rapid contraction."

The striated muscle of vertebrates is characterised by—

Rapidity of contraction being most highly developed,
Tonicity rudimentary, and
Rhythmic action still more rudimentary.

Cardiac muscle by—

Rhythmic action being most highly developed,
Rapidity of contraction well marked, and
Tonicity well marked.

Unstriped muscle by—

Tonicity being most highly developed,
Rhythmic action well marked, and
Rapidity of contraction most rudimentary.

It is indeed probable that the muscular fibre which surrounds any hollow viscus in the body subserves the purpose of maintaining the tone of the organ, and of thus preventing over-distension of its cavity. The muscular fibre of the arteries hypertrophies when unduly great distensile strain is put upon their walls, so does that of the bronchi and bladder, and it is only reasonable to suppose that the heart-muscle hypertrophies under like circumstances.

[1] *Journal of Physiology,* vol. iv. pp. 116–118.

The recoil of the arterial blood upon the interior of the ventricle will then be one of the main obstacles which the heart, in this disease, will have to overcome, and this will constitute in the case we are supposing, the chief, if not the only increased work to be done.

The ventricle meets the increased strain thrown upon it while it is filling, probably by being thrown into a series of small contractions before the true systole commences, and hence, possibly, the cause of the jar sometimes communicated to the finger in aortic regurgitant disease.

Marey[1] gives a curious tracing from the carotid artery of a person who suffered from aortic insufficiency, in which numbers of small secondary waves are seen in the diastolic part of the tracing and during the period of rest preceding the ventricular contraction. He interprets these as being caused by irregular contraction of the auricle. The phenomenon, however, might be explained by the aortic blood suddenly injected upon the wall of the ventricle throwing the latter into a series of spasmodic contractions of minor import previous to the occurrence of the true ventricular systole.

Such being one of the sources of overwork to be performed by the heart in the lesion we are at present contemplating, let us next consider what further overwork it has to undertake.

In health the pressure of the blood in the ventricle has to rise superior to that of the blood in the aorta before the aortic valve will open. If the valve is destroyed, and if the orifice be of natural calibre, will the intraventricular pressure have to reach a higher pitch before the blood will leave it to pass into the aorta? Obviously not, for the arterial pressure, there is good reason to believe, remains the same as before. The function of the valve is only called into play during the diastole of the ventricle; it is in abeyance, and might as well be absent during systole.

Rosenbach[2] found that in dogs and rabbits extensive destruction of the aortic, mitral, or tricuspid had no material effect in altering the arterial blood-pressure. In rabbits which were

[1] *La Circulation du Sang a l'état physiologique et dans les maladies*, 1881, p. 679.

[2] *Arch. f. exp. Pathologie u. Pharmakologie*, vol. ix., 1878, p. 1.

allowed to live for different periods after the valves were rendered incompetent, the arterial pressure was still uninfluenced. If, moreover, the orifice of the aorta were narrowed by the introduction of a sound, still no effect followed.

De Jager[1] obtained the same results in dogs after insufficiency of the aortic, mitral, or tricuspid had been artificially established, but in rabbits an absolute fall in the arterial pressure was noticed.

Under any circumstances, therefore, the ventricle will probably not be subjected to overwork in uncomplicated regurgitant aortic from having to overcome increased arterial pressure. It might possibly act at a disadvantage during systole were the cavity dilated, and where, consequently, a greater mass of blood would have to be propelled forwards than in health, but were the cavity not increased in size, the overwork thrown upon its wall would chiefly be that of resisting arterial recoil during diastole and of so maintaining the tone of the organ.

Conclusion.—*The overwork performed by the heart, in aortic regurgitance with a normal sized orifice, is chiefly that of keeping up the ventricular tone. If dilatation of the ventricle coexist, increased propulsive efforts may be required.*

(2) Aortic incompetence with a dilated orifice, mitral normal.

Here the conditions are simply an exaggeration of those in the preceding. From the fact that the wide orifice will permit of a less impeded arterial recoil, the impulse communicated to the interior of the ventricle by the sudden reflux of blood will be more sudden than in the former, and hence the strain during the commencement of diastole will be greater. There will, however, be if anything less difficulty in propelling the blood through the widened orifice during systole than before, and hence the increase of *propelling power* of the ventricle will be called for only where the cavity is much dilated.

As a matter of fact, I find from my average statistics that the cavities of the ventricles in this class of cases are not so large as in that immediately preceding ($3\frac{3}{4}$ inches for each side, as compared with 4 inches for the left, and $4\frac{1}{4}$ inches for the right), but that the ventricular wall is thicker. It may possibly be that

[1] Virchow and Hirsch's *Jahresbericht*, 1883, ii. p. 142.

the wide aortic orifice throws so great a strain upon the ventricle that the muscle hypertrophies before dilatation has reached so high a pitch.

Conclusion.—*The chief overwork performed by the heart, in aortic regurgitation with a wide orifice, is in keeping up the tone of the ventricle.*

(3) Aortic regurgitance with a constricted orifice, mitral normal.

The effect of constriction, in addition to the incompetence, will be two-fold. It will lessen the arterial recoil, or, at any rate, will permit of the blood regurgitating less suddenly than where the orifice is wide, and at the same time it will tend by narrowing the outlet to render the difficulty of propelling the blood forwards during systole greater.

The shock communicated to the interior of the ventricle will, accordingly, be less than where the orifice is wide, and hence, probably, the hypertrophy ought not to be so great as in either of the two preceding forms of disease. As will be seen from the following tabulated account of the effects of aortic disease on the wall, this is borne out in fact. The constriction of the orifice will thus in a manner compensate for the regurgitation and diminish the hypertrophy or dilatation which would otherwise follow. The constriction, on the other hand, will hinder the blood from passing out of the ventricle, and will therefore reflect its influence upon the wall. If the ventricle empties itself, it is clear that pure aortic constriction never could cause a dilatation. If a distended hollow viscus be compressed on all sides from without inwards, it will never become dilated. It is during diastole that dilatation alone can be effected, the pressure then being from within outwards, instead of from without inwards as in systole.

Of course it might be urged that the ventricle empties itself incompletely at each systole, and therefore the blood tends to accumulate within it. This is, however, matter of pure theory, and may well be called in question. If blood tends to accumulate in a chamber of the heart—where does the accumulation end?

I would therefore trace the cause of the hypertrophy in this lesion, firstly, to regurgitation of the arterial blood during

diastole, and, secondly, to the increased efforts required to be put forth by the heart-wall during systole. The effect of the regurgitance, however, will be modified by the constriction, and hence will be less injurious than where the orifice is of full size or dilated. The sum of the effects of both agents might come to be very much the same in regard to the condition of the wall as the single effect of a dilated incompetent orifice or one of natural size.

Conclusion.—*The overwork performed by the heart, where the aortic is incompetent and constricted, is two-fold, namely,* (1) *that of keeping up the tone of the ventricle, and* (2) *that of driving the blood through a narrow orifice.*

Comparative Effects of the foregoing Three Forms of Aortic Disease upon the Ventricular Walls and Cavities.

The above three forms of disease of the aortic constitute by far the largest proportion of pure aortic cases. As previously mentioned, I have failed to find in my records a single instance of a simple constricted aortic without the valve being, after death, incompetent, although sometimes only slightly so. The following statement gives a tabular view of the influence exerted by the three forms of aortic disease referred to upon the walls and cavities of the ventricles:—

	Ventricles.		Walls (Maximum thickness).	
	Left.	Right.	Left.	Right.
1. Aortic normal diameter and incompetent.	4 in.	$4\frac{1}{2}$ in.	$\frac{5}{8}$ in.	$+\frac{3}{16}$ in.
2. Aortic constricted and incompetent.	$3\frac{1}{2}$,,	$3\frac{1}{2}$,,	$\frac{5}{8}$,,	$+\frac{1}{8}$,,
3. Aortic dilated and incompetent.	$3\frac{3}{4}$,,	$3\frac{3}{4}$,,	$+\frac{3}{4}$,,	$+\frac{1}{4}$,,

It would thus appear that where the orifice was of natural size the left ventricle was largest, that it was of medium capacity where the orifice was dilated, and that it was smallest where the orifice was constricted. The explanation of these facts has already been sufficiently entered into.

It will, however, be noticed that the thickness of the wall was

greatest where the orifice was largest, least where it was of natural size or constricted; and this is exactly what should be expected. The greatest arterial recoil takes place with the dilated aortic, the least with the constricted. In the case of the constricted, however, the impediment to the propulsion of the blood constitutes an additional cause of hypertrophy, and hence there is good reason for the wall being as thick as the figures show it to be.

We have previously seen that the dilated regurgitant aortic also exerts the most injurious distensile effect upon the other orifices, and, consequently, there is little doubt that this form of valvular disease is one of the most disastrous to which a heart can be subjected. The more suddenly the defect is brought about, the greater the danger. It is accompanied in larger proportion than any other cardiac lesion by the evils which follow in the train of free regurgitant arterial pressure.

Coexistent Dilatation of Right Ventricle.—It was a good many years ago remarked by Gairdner[1] "that he had never seen an instance of hypertrophy affecting *the left side alone*." Contrary to what less accurate observers frequently affirm, there is an immense deal of truth in this statement, not only as relating to hypertrophy, but also as bearing upon the size of the cavities.

From the foregoing statistics of aortic disease, it will be noticed that the size of the right ventricle had advanced almost *pari passu* with that of the left, and, indeed, in one class of cases had overreached it. There was also a certain correspondence in the thickness of the wall of the right ventricle as compared with that of the left, probably accounted for by the regurgitant pressure indirectly influencing the whole right side of the circulation.

Cause of Hypertrophy of Musculi Papillares.—This depends upon their functions being overtaxed like that of other parts of the heart-wall. It is most likely that the musculi act as a stay upon the valve to which they are attached, and thus prevent eversion during systole. Pettigrew[2] describes the cusps of the mitral as being floated up during the ventricular diastole. During systole, however, the blood is thrown by the action of the

[1] *Brit. and Foreign Med. Chir. Rev.*, vol. xxiii., 1853, p. 218.

[2] *Physiology of the Circulation*, 1874, pp. 278–280.

ventricle into spiral columns and twists them into each other, while the musculi papillares drag them downwards.

M. Sée[1] says that the papillary muscles contract at the same time as the ventricles. They tighten the chordæ and pull the cusps down. The left cusp of the mitral plays much the more active part in closure, but the right is not unemployed. The musculi might accordingly be expected to hypertrophy where the aortic orifice is constricted, for here the increased effort required to propel the blood through the narrow aortic orifice will react upon the mitral cusps and tend to force them upwards.

Effect of Filling of the Left Ventricle from Two Sources.—As the left ventricle is filled both from the aorta and from the auricle in aortic regurgitation, it follows that there must be a mixing of the blood from the two sources of supply within the chamber. The gross effect will of course be that the amount of blood passing directly from the lungs into the aorta will be less than in health.

The proportion in which the auricular and aortic bloods mix constitutes an interesting and as yet undetermined question. The pressure within the pulmonary artery, as compared with that of the aorta, has been variously estimated as something like 2:5 or as 1:3. The pressure within the pulmonary veins is considerably less, while within the auricle, at least towards the end of the auricular systole, it is again somewhat increased, but still considerably below that of the blood within the aorta. More blood should therefore regurgitate from the aorta than would pass into the ventricle through the auriculo-ventricular opening. The auriculo-ventricular opening, however, is larger than the aortic, and hence there should be less obstacle to the free passage of the auricular blood than to the regurgitation of the systemic.

The pulmonary veins are without valves, and the regurgitation from the aorta occurs during diastole, and hence at a time when the auriculo-ventricular orifice is open. It might *a priori* be expected that if the pressure within the aorta be greater than that in the auricle, injurious influences would be brought to bear directly upon the lung. Such is often the case, but not

[1] *Archives de Physiologie*, vol. i., 1874, p. 552.

always. It is a well-known fact that individuals with aortic disease are frequently unconscious of it until they undergo a medical examination for life insurance or for some other reason. The heart evidently accommodates itself to the altered circumstances, a result which might be accounted for by the pulmonary veins rhythmically closing as the auricle begins to contract. If some provision of this kind were not forthcoming, it is difficult to see how the circulation could continue unimpeded for any length of time.

If the orifices of the pulmonary veins do close, and if the hypertrophied ventricle keeps up the heart's tone, then it is possible to conceive that, with each systole, blood is propelled forwards composed say of two parts aortic and one part auricular. This might naturally be held to occasion such an under-oxygenation of the blood as to materially deteriorate its respiratory qualities, and in certain cases it is so. It must be remembered, however, that if the blood is delayed in its passage through the lung, and if the bronchi and air-vesicles are unobstructed, it becomes hyperoxygenated. Hence, although the fresh blood from the lung circulates less freely, yet its hæmoglobin may be of such a high respiratory capacity as in a manner to compensate for the defect. If, moreover, the auricle were to hypertrophy, the blood would be driven into the dilating ventricle with impetus sufficient to, in a manner, equal or exceed that caused by the arterial recoil.

Landois[1] remarks that hypertrophy of the left auricle occurs as a result of aortic insufficiency, "because the auricle has to overcome the continual aortic pressure within the ventricle." As a matter of fact, however, it will be found that hypertrophy of the wall of the left auricle in this disease is not of common occurrence, but that the cavity, as a rule, is slightly dilated. The dilatation is not so extensive, however, as when the aortic regurgitance is complicated with mitral incompetence and stenosis, for under these circumstances the forces tending to dilate the auricle are of a threefold nature. In the first place, there is the arterial recoil during ventricular diastole; in the second, the hindrance to the free passage of blood from

[1] *Text-book of Human Physiology*, English translation by Stirling, 1885, vol. i. p. 80.

the auricle into the ventricle owing to the constriction of the auriculo-ventricular orifice, also occurring during diastole; and, in the third, the regurgitant systolic impetus conveyed to the auricle during systole. The auricle in this form of disease will be found to be larger than in any other, while in pure mitral disease it is usually of medium capacity.

Hypertrophy does not follow lesions of the orifices with the same facility in the case of the auricles as in that of the ventricles. Hence the abnormal conditions of pressure arising from valvular disease will be found more often to leave their record in auricular dilatation, rather than in an increased thickness of the walls.

B. *Mitral.*

(1) Mitral incompetence with stenosis, aortic normal.

In this, which is the usual form of mitral disease, it might theoretically be expected that the tendency to hypertrophy and dilatation of the left ventricle would be less than in aortic incompetence. There is no arterial rebound upon the ventricular wall during diastole, and from the narrowing of the mitral orifice the blood will be impeded in its flow from the auricle, and hence might not reach the ventricle even with such force as is imparted to it in health.

There is, however, a cause for hypertrophy in this class of cases which is absent in simple aortic disease. The incompetent mitral allows part of the ventricular blood to recoil during systole upon the left auricle, and this will naturally tend to weaken the impulse communicated to the systemic vessels. The ventricle will thus have to work harder in order to keep up the arterial pressure.

Artificial destruction of the mitral in an otherwise healthy animal does not cause a fall in blood-pressure. This is probably to be explained by the ventricle contracting more vigorously, and by the openings of the pulmonary veins closing sufficiently to limit the regurgitant effects to the auricle.

It is very unlikely, however, that the orifices of these vessels would withstand the dilating influence of this regurgitance for long. There is every probability that if the thin wall of the auricle became stretched they would widen, and thus allow of

the influence of the ventricular systole being felt by the whole pulmonary circulation as far back as the pulmonary artery orifice.

The result ought to be, as the following figures show is the case, that this lesion of the mitral induces slight hypertrophy of the wall of the left ventricle.

	Ventricles.		Walls (Maximum thickness).	
	Left.	Right.	Left.	Right.
Mitral constricted and incompetent.	$+3\frac{1}{4}$ in.	$+3\frac{1}{2}$ in.	$\frac{5}{8}$ in.	$\frac{1}{8}$ in.

Rosenstein[1] remarks that in this valvular defect the left ventricle is so small, as compared with the dilated right, that it looks almost like an appendage of the latter. The above figures do not entirely support this statement. The difference in the dilatation of the two cavities, as will be noticed, was comparatively slight, and there was no appreciable hypertrophy of the right.

(2) Mitral dilated, aortic normal.

This being a rare condition, and not always accompanied by regurgitation, it is difficult to say what its actual effects might be. The large mitral, of course, if incompetent, would allow a still greater reflux than in the foregoing. The combination of a dilated mitral orifice with any amount of incompetency must, however, be looked upon somewhat in the light of a pathological curiosity.

The following figures may be taken for what they are worth :—

	Ventricles.		Walls (Maximum thickness).	
	Left.	Right.	Left.	Right.
Mitral dilated, but doubtful whether incompetent.	$3\frac{3}{4}$ in.	4 in.	$+\frac{1}{2}$ in.	$\frac{3}{16}$ in.

[1] *Cyclopædia of Practice of Medicine*, edited by von Ziemssen, English translation, vol. vi. p. 126.

(3) Mitral constricted but competent, aortic normal.

This, as previously indicated, is also a rare lesion. The commonest cause of it is the projection into the funnel-shaped valve of vegetation or calcareous masses, tumours, &c. In other instances the margin of the valve is alone contracted without the entire valve being converted into a leather-like structure, a condition which apparently is compatible with the valve closing.

The effects of such a lesion would, of course, be to hinder the blood in its transit from the auricle to the ventricle, unless the auricle compensatorily hypertrophied to drive it onwards with increased force.

C. *Aortic and Mitral combined.*

(1) Aortic and mitral both constricted and both incompetent.

Here the left side of the heart may be regarded as devoid of valves, and with its orifices so constricted that the blood is trammelled in its passage through them. It is a rhythmically-contracting tube unprovided with competent valves at either end. The systemic arteries thus become directly continuous with the pulmonary; they form one set of vessels with this rhythmically-contracting chamber—the mutilated auricle and ventricle—between them. The scheme of the circulation in fact comes to resemble that of the fish. There is in reality one auricle and one ventricle (the right); with a vessel (pulmonary artery) conducting to the lungs; vessels conveying the blood (pulmonary veins) to a pulsatile vessel or chamber (the left auricle and ventricle), thence to the systemic arteries. The ultimate result must be, of course, that during ventricular diastole the pressure within the pulmonary vessels as far back as the nearest obstruction, *i.e.*, the pulmonary artery orifice, will tend to be equalised with that in the systemic arteries. In fact, if the mitral fails to close, and if its aperture, although narrow, remains larger than the constricted aortic, as usually happens, the ventricular blood ought by preference to pass backwards through the mitral instead of forwards into the aorta. The lung would thus have imparted to its vessels actually more of the ventricular energy than is exercised upon the systemic arteries, the only counter-

balancing agent being the pulmonary blood which is being driven in the opposite direction by the right ventricle.

The pulmonary capillaries would thus have to bear at each ventricular systole both the pressure imparted to them by the right ventricle and the regurgitant pressure from the left. They are not even free from injurious over-pressure during ventricular diastole, because they are then subject to the backward recoil of the whole arterial system.

It cannot be a matter of wonder that under these circumstances the delicate-walled capillaries of the lung, entirely unsupported as they are on one side, should become converted into huge nævus-like masses. They in fact have to support from three to five times the pressure they were originally intended to bear, and it is only because they are so distensile that the effects of this do not prove to be more disastrous than they are.

But where does this regurgitant effect cease? Theoretically, it ought not to be felt beyond the next lock, that is to say, beyond the orifice of the pulmonary artery, provided that the valve of this vessel remains competent. The orifice is very rarely altered in size, and the incompetency of the cusps is a thing almost unknown. The sharpness with which they can be heard to click together in valvular disease of the left side is evidence of their timely and often premature closure.

How do the organs on the right side of the heart therefore come to suffer?

We have seen that in the majority of valvular lesions of the left side the tricuspid and pulmonary artery orifices are not altered in diameter. It might, therefore, well be argued that so long as the pulmonary artery orifice is competent, the regurgitant pressure imparted to the lung could not possibly affect the organs further back. Yet pulsation may be felt and seen in the jugulars and in the liver in such cases with every ventricular systole.

The right ventricle will undoubtedly have more to do in such a case than in health, because before the sigmoid segments will relax, the pressure of the blood within the right ventricle will have to rise superior to that in the pulmonary artery. Its muscular fibre will therefore tend to hypertrophy, as the accompanying figures demonstrate:—

	Ventricles.		Walls (Maximum thickness).	
	Left.	Right.	Left.	Right.
Aortic and mitral both constricted and both incompetent.	$3\frac{1}{2}$ in.	$3\frac{1}{2}$ in.	$+\frac{1}{2}$ in.	$+\frac{1}{4}$ in.

When this hypertrophied muscular fibre contracts the blood will press injuriously against the tricuspid valve. This valve at no time is perfectly competent in man, and when increased strain is thus put upon it, it will become much less so than it is naturally. Hence blood will regurgitate and cause the pulsation and other injurious effects so common in such cases. The amount of evil occasioned would thus be proportionate to the extent of the hypertrophy of the right ventricle, and the great diversity noticed in the state of the liver, kidney, and other organs in various forms of valvular heart affection may thus be accounted for. In some instances there is mere congestion of these organs, in others not even this, while in yet others they have suffered extreme atrophy.

An *emphysematous lung* may cause hypertrophy of the right ventricle, and in this case also the same effects are noticed in distant organs. The injury in both cases is inflicted during the systole of the right ventricle. It is while the walls of this chamber are contracting, not while they are in a state of relaxation, that the destructive backward impulse, rendering their valves incompetent, is conveyed to the large veins.

If the ventricle failed to empty itself, and if distension of its cavity was thereby occasioned, the tendency to render the tricuspid incompetent would be increased. We do not know, however, whether this takes place. It must, moreover, be remembered that simple distension of the right ventricle would probably never induce a tricuspid regurgitation, if the pressure within the pulmonary vessels remained normal, because the blood even from a distended ventricle would by preference run through the pulmonary artery rather than backwards through the tricuspid. Where the pressure within the pulmonary artery is above that of health the ventricle must contract more

energetically to lift the pulmonary artery lock, and even after this has been accomplished the blood will continue to be opposed by this over-pressure during the whole time the valve is open; hence the overwork performed by the ventricle will have to be sustained. It is only reasonable to believe that this, in course of time, must react injuriously upon the tricus pid andadd to its natural incompetence.

Conclusions.—*The increased work performed by the left chambers of the heart in this form of disease is in keeping up the arterial pressure. Part of the energy so liberated is expended upon the pulmonary circulation, which, by raising the pressure within the pulmonary vessels, necessitates increased effort on the part of the right ventricle in opening the pulmonary artery valve. This is frequently followed by hypertrophy of the ventricular walls. The unusually energetic contraction also reacts upon the naturally incompetent tricuspid and renders it more incompetent, thus affecting deleteriously the whole venous circulation.*

(2) Aortic of natural size but incompetent, mitral constricted and incompetent.

The conditions here, so far as the effects on the right side of the heart are concerned, are probably more favourable than in the former case. As the aortic is of normal size the blood will pass more easily through its orifice, and hence will not tend to regurgitate so freely through the incompetent mitral. The amount of hypertrophy of the right ventricle ought for this reason to be less than in the foregoing example, because the blood of the right ventricle will experience less opposition in being propelled into the pulmonary artery. The following are the actual average measurements:—

	Ventricles.		Walls (Maximum thickness).	
	Left.	Right.	Left.	Right.
Aortic of natural size but incompetent, mitral constricted and incompetent.	$3\frac{1}{2}$ in.	$+3\frac{1}{2}$ in.	$\frac{1}{2}$ in.	$+\frac{1}{8}$ in.

It might be argued that the wider aortic orifice will allow more blood to regurgitate during diastole; but it is questionable whether this, seeing that the mitral is constricted, would be so injurious in the long run as the regurgitance of the ventricular blood during systole, where in addition to the mitral constriction the aortic is also of narrow calibre.

(3) Aortic and mitral both constricted, aortic competent, mitral incompetent.

The argument based on the figures under the class just considered (No. 2) gains strength from those accompanying this.

	Ventricles.		Walls (Maximum thickness).	
	Left.	Right.	Left.	Right.
Aortic and mitral both constricted, aortic competent, mitral incompetent.	$3\frac{1}{8}$ in.	$3\frac{1}{4}$ in.	$\frac{1}{2}$ in.	$+\frac{1}{8}$ in.

In this case one element of regurgitant pressure is removed as compared with Class 2, namely, the aortic incompetency. There is superadded another, however, the aortic constriction. The figures remain very much as before, showing that *with an incompetent mitral*, aortic constriction has much the same effect upon the thickness of the walls and size of the cavities as aortic regurgitance. Where incompetency of the aortic is combined with constriction of its orifice, however, as in Class 1, the difference in the effects is demonstrated by the greater thickness of the walls, both of the left and of the right ventricles.

D. *Tricuspid Stenosis.*

The cases of this lesion that have come under my personal notice, as before mentioned, have been so complicated with disease of the orifices on the left side, that it is impossible to form from them an accurate idea of what the result on the walls and cavities of the heart would be.

Concluding Remarks on Hypertrophy from Valvular Disease.

It must be remembered that the heart when deprived of its natural locks may still have a certain inherent power of driving the blood onwards. Just as the œsophagus seizes the draught of liquid, and conducts it to the stomach even against gravity, so the heart may be supposed to do the same with the blood.

Pettigrew has described the ventricle as twisting the mass of blood within it in a spiral manner. If the heart-wall has no further hold upon the blood than a mere contractile sac, it is hard to conceive, where perhaps three of the valves are diseased, how the circulation continues to maintain even the desultory course that we know it does.

It should also be borne in mind that an impaired valve need not be a totally useless valve. It may subserve its purpose in an incomplete manner.

The chief redeeming point in valvular disease of the heart probably is that the pulmonary semilunes are seldom incompetent. This in a manner separates the venous from the arterial circulation, and prevents the pressure on both sides from becoming equalised.

Connected Tabular Statement of the Measurements referred to in the Foregoing Paper, along with the Average Weights (Males and Females included).

	Weight in ozs. Avoird.	Orifices.				Cavities.		Walls (Maximum thickness).	
		Aortic.	Mitral.	P. Art.	Tricusp.	L. Ven.	R. Vent.	L. Vent.	R. Vent.
1. Normal heart.	10–13 oz.	0·9–1 in.	1·2–1·4 in.	1·1–1·2 in.	1·5–1·8 in.	3–3¼ in.	3 1/16–3⅜ in.	½ in.	⅛ in.
2. Aortic of normal size but incompetent.	21¾ ,,	0·99 ,,	1·3 ,,	1·1 ,,	1·8 ,,	4 ,,	4½ ,,	⅝ ,,	+3/16 ,,
3. Aortic constricted and incompetent.	18¾ ,,	0·75 ,,	1·3 ,,	1·1 ,,	1·7 ,,	3½ ,,	3½ ,,	⅝ ,,	+⅛ ,,
4. Aortic dilated and incompetent.	22¾ ,,	1·2 ,,	1·7 ,,	1·2 ,,	2 ,,	3¾ ,,	3¾ ,,	+¾ ,,	+¼ ,,
5. Mitral constricted and incompetent.	16 ,,	0·97 ,,	0·86 ,,	1·1 ,,	1·7 ,,	+3¼ ,,	+3½ ,,	⅝ ,,	⅛ ,,
6. Pure dilatation of mitral.	20½ ,,	1 ,,	1·68 ,,	1·27 ,,	1·98 ,,	3¾ ,,	4 ,,	+½ ,,	3/16 ,,
7. Aortic and mitral both constricted, aortic competent, mitral incompetent.	12¾ ,,	0·7 ,,	1 ,,	1·1 ,,	1·6 ,,	3⅛ ,,	3¼ ,,	½ ,,	+⅛ ,,
8. Aortic and mitral both constricted and both incompetent.	20¼ ,,	0·8 ,,	0·77 ,,	1·08 ,,	1·6 ,,	3½ ,,	3½ ,,	+½ ,,	+¼ ,,
9. Aortic of natural size but incompetent, mitral constricted and incompetent.	20½ ,,	0·97 ,,	0·87 ,,	1·1 ,,	1·6 ,,	3½ ,,	+3½ ,,	½ ,,	+⅛ ,,