THE

OCCURRENCE OF COMA IN SUDDEN SPONTANEOUS BRAIN LESIONS.

BY

WM. BROWNING, M D.,

OF BROOKLYN,

LECTURER ON ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM AT THE LONG ISLAND COLLEGE HOSPITAL.

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THIS symptom is one of the most striking and more frequent of those attending intracranial accidents. The absence of coma in some cases even of large cerebral hemorrhage, its occurrence in many cases of limited lesion, and often, but inconstantly, in cases of cerebral embolism are trite facts. No acceptable theory has been proposed to explain or harmonize these apparent discrepancies, and consequently the symptom has not acquired any definite clinical value. It is certainly permissible to attack such a problem in any way that presents, and whilst a complete explanation is not possible, yet it is believed that the symptom can be made clinically available for some classes of cases. Any study of the different forms and degrees of coma will not be attempted. For a brief definition of the term we may cite Gowers. "The prominent feature of apoplexy is loss of consciousness without obvious failure

of the heart's action." In some cases consciousness is simply lost or clouded for a few moments; this can hardly be called coma. Often, unfortunately, our knowledge of the loss or retention of consciousness rests on very unreliable statements. Even careful observers may differ in what they call coma. It should be remembered that the symptom is a general ("diffuse") one, and may be produced by lesions on either side of the brain. "An apoplectic attack, coma, and also delirium, may occur without anything abnormal being found in the brain (or other part of the body) at the autopsy." Lesions of the cord, below the pyramids of the medulla, do not cause coma, though, if in the cervical portion, sudden death may result.

Suddenness of the lesion and increased intracranial pressure are acknowledged factors. Personal observation, and a study of the subject indicate that the seat of lesion is also an important factor. Evidently this view has never met with favor, though general opinions, for and against it, might be quoted. To determine its truth will be one of our objects, especially in view of the opposite opinion so positively adopted by such a recent writer as Wernicke. He assumes that a slower arterial supply, and freer venous discharge explain the slighter mental disturbance in hemorrhage into the mantel-zone. This factor of suddenness is, on the other hand, most prominent in embolism; hence, coma should follow embolism affecting this region, if his attempted explanation were correct.

There are three classes of cases that might come

up for consideration, viz., hemorrhage, embolism, and thrombosis. Thrombosis and spontaneous softenning may occasion loss of consciousness, but are of little value in this discussion. The rapidity with which the former develops, is variable and uncertain. Moreover, it often results from causes that affect the circulation in other parts of the brain, *e. g.*, atheroma, syphilis, neoplasms, and general exhaustive diseases. In many cases of (so-called idiopathic) softening, the rest of the brain is not entirely healthy; the softening represents what has become irreparable.

It is better to begin with the study of embolisms; because,

1. In them, one factor, that of increased pressure, is absent.

2. Their onset is more typically sudden.

3. The portions of the brain most frequently affected by them interest us first.

Embolism.

The effect of embolism on consciousness is considered less subject to rule than that of hemorrhage. The study of these accidents proves to us, however, that increased brain-pressure is not an essential or the only factor in the causation of coma.

There are a variety of matters which render it difficult to determine the effect on consciousness of an embolic insult to a given part of the brain.

a. The large amount of brain-tissue directly menaced in most cases. Even where softening fol-

lows, it does not often include the whole district at first deprived totally or partially of arterial blood.

b. It is questionable whether in all cases the artery is at once completely occluded. Wernicke thinks it admissible to assume this for most cases. However, this is not always true, and, when not, the shock or suddenness of the attack would be mitigated.

c. Many emboli soften and break up, thus either entirely disappearing, or only remaining in part in the shape of smaller emboli in terminal branches of the originally occluded vessel. When this happens early enough, softening is prevented, and we may have no permanent symptoms (no matter how severe the onset), and little or nothing to demonstrate, should the subject come to autopsy. Or, again, softening may have set in before the embolus was dislodged, when there would be nothing to show as the cause.

d. Another possible element of uncertainty lies in the usual co-lesion of the meninges, or at least some extent of the pia mater. The effect on consciousness of injury to this structure is doubtless slight.

e. In a goodly proportion of cases, embolism occurs in persons whose brain-circulation has been greatly weakened by preëxisting intrathoracic disease. In such, evidently, a much smaller shock might cause loss of consciousness. In reality it then appears very similar to syncope. The subject falls, if upright, but on being thus obliged to lie down, the circulation in a few minutes reëstablishes

its equilibrium sufficiently to support consciousness, and the person revives. Such transitory loss of consciousness can hardly be called genuine coma.

A few opinions regarding the present subject may here be introduced, though rather to show the chaos of views that exists than for their real value.

Wilks (*Lectures*, p. 120) says:

"It might be thought that unconsciousness would be less likely to occur in embolism than in sanguineous effusion; but this is not always the case, for although it is true that there is often complete coma in large effusions from rupture of small vessels, as in ingravescent apoplexy, the unconsciousness and mental confusion are often less than in embolism."

Flint (reported in *Boston Medical and Surgical Journal*, 1879, vol. c. p. 668) says:

"From the long continuance of coma, we are justified in considering that the symptoms are probably not due to embolism, but rather to cerebral hemorrhage."

Wernicke (Bd. ii. 136) writes that

"Clinical experience shows unmistakably that isolated embolism of small vessels, up to the calibre of the largest perforating arteries, or the branches to Broca's convolution, does not usually suffice to produce unconsciousness and the fully developed apoplectic insult."

Sachs (*Journal of Nervous and Mental Diseases*, Aug. 1887, p. 507) states that

"It is well known that embolism is far more frequently accompanied by loss of consciousness than hemorrhage. The plugging of even a small cerebral artery is almost invariably followed by loss of consciousness."

*

Nothnagel (*Top. Diagnk.*, 1879, p. 612) holds that

"Observation teaches that in any localization whatever a hemorrhage may set in with loss of consciousness, that, however, it may be absent in any [region], even in the cortex. There is no locality from lesion of which 'the apoplectic insult' must regularly be present or absent."

He adds that large hemorrhages and embolisms with extensive ischæmic districts produce a severe attack, though the converse is far from being as generally true.

Limits in the Selection of Cases.

It is necessary at the start to simplify the investigation as much as possible. In fact any successful study of the subject must largely depend upon the proper exclusion of irrelevant and misleading cases—a principle urged in another connection by Nothnagel in his "Topical Diagnosis."

1. All cases without autopsies are to be excluded.
2. All cases brought on by external shock.
3. All cases attended by convulsions.

Trousseau, *e. g.*, says that convulsions suffice to produce apoplectic stupor, and we have but to note the usual effect of an epileptic attack.

4. Many cases are poorly suited to our purpose, where the person has had a previous apoplectic or apoplectiform attack, is subject to epilepsy, is suffering from other nervous or severe bodily disease, or where there are bilateral or multiple brain lesions.

5. All cases where important particulars are wanting, *e. g.*, where the embolism occurred during sleep.

6. All where the subject was under twenty-one years of age. Children are known to be more subject to convulsions and coma than adults. Many persons certainly attain their development before twenty-one, but to exclude the element of infancy or youth this age has been universally adopted for other purposes and may well be for ours. Perhaps old age ought also be a ground for exclusion, but as senility does not come on with much regularity, such limitation will not be attempted.

7. In making what may be regarded as a primary study of the subject it is better to include only cases where the injury was limited to particular parts or structures. This is necessary before studying the results of co-lesion—even though partial—of two or more structures. As embolism but rarely affects any one of the basal ganglia singly we shall now take up only those cases where the embolic softening was wholly in the extraganglionic portion of the cerebrum (Mantelzone of the Germans).

Embolism of Sylvian (Middle Cerebral) Artery or its Branches.

From the seat of an embolism in this artery no sure conclusion can be drawn as to the exact portion of brain that will consequently soften. That an embolus in its more peripheral portions would rather cause a cortical and sub-cortical softening, and one in its beginning more often softening about the striated body, internal capsule, etc., is probable but in reality uncertain. This fact seems to have been ignored by most who have touched on the subject.

Hughlings Jackson says that in embolism of this artery "there may be no loss of consciousness and the hemiplegia may be transitory, though patients sometimes die in a few days in an apoplectic manner."

S. Mackenzie (*Brain*, 1878, iii.) writes that "most authorities agree that loss of consciousness is unusual in embolism of a Sylvian artery, though Niemeyer is a marked exception. Loss of consciousness may occur and be as complete as in severe hemorrhage, but this is unusual."

Nothnagel, holds that "while embolism of the Sylvian artery is, as a rule, attended with symptoms of apoplexy, yet it may occur without them," and Wernicke (ii. 136), that "embolism of the Sylvian artery before its bifurcation, or of one of its two chief branches, regularly causes a developed insult." He thinks that where this does not occur, we are to look for exceptional conditions (partial occlusion, etc.).

The accompanying table gives a nominal total of 32 cases (or 36 presumable embolisms), and includes all so far collected that come within the limitations laid down. No. 15 is insignificant. No. 32 occurred at night, in a patient with high fever. From complications and imperfect descriptions of the onset, several of the other cases are not entirely desirable; Nos. 15 and 32 may, therefore, be excluded, though they present nothing against conclusions drawn from those remaining.

This leaves 30 cases (not counting double ones) involving, one with another, a large part of the

brain-mantle—a number sufficient to justify conclusions. Only in Nos. 7, 9, 18, and 26 was there any question of coma at the onset. The other 26 (resp. 30, separate embolisms) occurred without this symptom. In No. 7 a really comatose state did not develop—delirium from *second* attack. In No. 9, also, at *second* attack there was but momentary loss of consciousness—again not genuine coma. In No. 18 the short loss of consciousness (for ten minutes) is easily accounted for. The patient was in a fever, and one thoracic cavity was quite two-thirds filled by a recent effusion. The heart was slightly displaced, and the pulse weak. Moreover, the softening was beside the striated body—not superficial—and at first that structure may well have been implicated. This leaves but one, No. 26, amongst all the cases, that is exceptional, and even that is not necessarily so. It was presumably, though not certainly, due to embolism. There was bilateral cutaneous anæsthesia—absolute on the right, and nearly so on the left—of five days' duration. Five months later there was, for one day, a return of the various symptoms (at least aphasia, etc., and anæsthesia, though of a different form). It was simply stated by friends that he had rolled his eyes, groaned, and lost consciousness for perhaps an hour. The peculiarities of this case may have been due to multiple emboli or an epileptiform complication.

There is also the possible explanation for any unusual case, that at first the embolus cut off vessels to the central ganglia. Hence, in 30 (resp. 32) cases, or in 32 (resp. 36) embolisms affecting only the

brain-mantle, there is at most but one exception—and this doubtful—to the rule that embolism of this region in the adult does not cause coma. This is a result quite as uniform as other accepted facts in cerebral symptomatology. If, as proposed, all cases not described as embolic, and all not free from important complications, had been excluded, the uniformity would have been complete. But, for reasons already given, exceptions, apparent or real, will occur; it seemed, therefore, better to include the most probable so far found.

These cases do not, it is true, include all parts of the brain-mantle; yet they do cover about all that is often affected by embolism, or of which we have much knowledge, clinical or physiological. They extend over the whole period of adult life, from twenty-one to eighty-three years of age, and include both sexes (males, 20; females, 11; not stated, 1). The list presented makes no pretension to completeness, though cases satisfying the requirements are not plentiful. Bilateral embolism seems commonly, though not always, to cause coma. At least in Stewart's case (*Med. Times*, 1864, *v. Journ. Ment. Sci.*, 1868, p. 66), plugging of both Sylvian arteries, with resulting walnut-sized softening on each side, in a man of twenty-one years, occurred without coma. Though this was presumably thrombotic, yet it indicates that the same might occur in embolism.

In any individual case, embolism limited to this region does not involve a *relatively* very large part of its whole, nor does it cut off tracts to other parts of the cortex (except, of course, association fibres).

Hence it does not deprive of their function parts of the cortex not directly injured, and we have essentially symptoms confined to the part lost (ausfall-symptome of the Germans). If the area destroyed were sufficiently large—how large cannot be stated—there is no question but that coma would result; nor is there any doubt that lesions in this region have some tendency to cause coma. Only, in practice, embolism alone does not suffice.

Taking the cases as they stand, multiple attacks and all, delirium or some obscuration up to temporary loss of consciousness is noted in 5 out of 36 attacks. Lesser disturbances are very common. The present cases do not show whether lesion of different equal areas of cortex has or has not a like effect on consciousness, though destruction of sensory centres seems to be better tolerated.

Conclusions.

1. In adults free from relevant complications, embolism involving directly only extra-ganglionic portions of the cerebrum, does not cause coma.

2. Conversely, where the diagnosis of cerebral embolism is warranted, primary coma indicates implication of the basal ganglia or brain-stem.

3. The absence of coma does *not* preclude involvement of the said lower structures.

4. From the fact that more limited embolic injury involving other portions of the brain is frequently attended by coma, it follows that the location (or seat) of a cerebral lesion is one of the important factors in the causation of this symptom.

5. Wernicke's view, that the slighter disturbance of consciousness by hemorrhage into this region—as compared with hemorrhage into other portions of the brain—is owing to circulatory peculiarities, and hence to the absence of the factor of suddenness, is disproven.

6. Of course, thrombosis also, *under like conditions*, would not cause coma.

7. Whether embolism of the Sylvian trunk will or will not cause coma, is a useless question. It depends on the structures supplied by that artery in any given case.

8. It must not be considered that the above conclusions at all disprove the accepted view that the cerebral cortex is the seat of higher consciousness.

From the basis thus gained it will be possible to take up more intelligently the subject of hemorrhage into the same, and even other regions of the brain.

No.	Observer.	Publication.	Sex.	Age.	Embolism.	Softening (extent of).	Remarks.
1	West,	Brit. Med. Journ., 1885, i. p. 1242.	M.	66	Branch of Sylvian.	Left supramarginal and angular gyri.	
2	Bourneville et Bonnaire,	Prog. Méd., 1882, p. 239.	M.	54	Believed to have been embolic.	First and part of second temporal convolutions, also larger part of lobule, du pli courbe and insula.	
3	D'Heilly,	Gaz. Hebd., 1882, p. 54.	F.	24	Fourth branch of Sylvian.	Gray cortex implicating inferior parietal lobule and part of first spheno-occipital convolution.	Phthisis.
4	Bancroft,	Boston Med. and Surg. Journ., 1881, p. 483.	M.	60	Branch of Sylvian.	Anterior portion of middle lobe 3–4 inches in diameter, also insula superficially.	One of repeated attacks, probably of embolism.
5	Goltdammer,	Berl. kl. Wochr., 1879, p. 367, Case III.	F.	36	Pia branches of Sylvian.	Whole of posterior central, part of anterior central and of first frontal, and most anterior supramarginal convolutions.	
6	Magnan,	Brain, 1879, Case III.	M.	42	Considered embolic	Involved temporal and third frontal convolutions.	Complications.
7	Guitéras,	Phil. Med. Times, 1878, ix.	M.	58	1. Embolic. 2. Posterior branch of Sylvian.	1. Gray matter for 1 inch at middle and lower third of anterior central convolution. 2. The transition convolutions with posterior half of angular gyrus and of first temporal.	Two attacks, delirious (semicomatose) in second only. Focus also in cerebellum.
8	Poulin,	Bull. Soc. Anat., 1878, p. 577.	M.	...	Branch of Sylvian.	Superficial of anterior and posterior central convolutions.	
9	Seguin,	Trans. Am. Neurol. Assoc., 1877, Case II.	F.	...	Middle cerebral, half inch from its origin.	Posterior end of third frontal and anterior half of insula. (The previous attacks were represented by softening in opposite third frontal convolution.)	Two previous attacks on other side; only in second was there loss of consciousness for a moment; later epileptic seizures.
10	Prevost and Cotard,	Études, etc., 1886, Obs. 26 (*vide* Wernicke, ii. p. 138).	F.	83	From atheroma.	Plaques on all the right frontal convolutions, on several occipital, and at bottom of central fissure. Basal ganglia intact.	Two attacks, each without coma.
11	Prevost and Cotard.	Obs. 3 (*vide* Wernicke, ii. p. 138).	F.	70	From atheroma.	Part of inferior frontal, of both central, and the two anterior insular convolutions; anterior part of temporal lobe.	First attack.
12	Radcliffe,	London Lancet, 1866, July 28.	M.	27	Sylvian artery at a bifurcation.	One third of middle lobe.	
13	Ogle,	Pathl. Trans., 1867.	M.	45	Branch of Sylvian, at a bifurcation.	Posterior part (¾×¾ inch) of third frontal convolution, also smaller spot beneath end of Sylvian fissure.	
14	Moxon's service at Guys,	London Lancet, 1868.	F.	58	Sylvian at its first branching.	Corresponding gray and some of white substance, œdematous, etc.	
15	Berkley,	MED. NEWS, 1882, ii. p. 61.	M.	73	Minute artery.	Nodule half thickness of cortex in anterior central convolution.	
16	Begbie,	Edinburg Medical Journal, 1866, p. 122.	F.	22	Sylvian beyond a second considerable branch.	White substance external to posterior half of striated body.	Complications.
17	Fritsch,	*Vide* Starr from Wilband's Hemianopsie.	...	...	Parieto-sphenoidal branch of Sylvian.	Entire lower parietal lobule and part of cortex of second occipital convolution.	
18	Vallin,	Gaz. Hebd., 1870, No. 5, p. 77.	M.	21	Two branches of Sylvian.	Near corpus striatum and at posterior extremity of this patch was a cavity filled with débris.	At first unconscious, but recovered in ten minutes (*vide* infra).
19	Greenlees,	Amer. Journ. Ins., April, 1887, Case IV.	M.	32	Greenlees says evidently from embolism of Sylvian.	Lower frontal, both central and anterior part of first two temporal convolutions; whole of insula to striated body.	In an imbecile.
20	Estorc,	Mont. Med., 1882, (*vide* Charcot and Pitres, Rev. de Méd., 1883, Ob. 55.	F.	32	Evidently from embolism.	Cyst in white substance of central region.	Prolonged onset.
21	R. Atkins,	Brit. Med. Journ., 1878, May 11, Case vi.	F.	68	Middle division of Sylvian was obliterated (anterior parietal of Duret).	Lower part of posterior central, extending into centrum ovale. Opto-striated bodies free.	Without doubt embolic, he says.
22	Rosenthal,	Wien. Med. Halle, Mai 18, 1862, iii. (*vide* Schmidt's Jrbch., Bd. 117, Case 6.)	F.	27	Sylvian artery.	Nut-sized focus in white substance.	
23	Tassi,	Rev. sper. di freniat. Journ., 1880, p. 193 (*vide* Charcot and Pitres, Rev. de Méd., Oct., 1883, p. 864–5).	M.	37	Sylvian at its ramification.	Posterior upper part of frontal lobe.	Onset somewhat gradual; epileptiform attacks some days later.
24[1]	R. McDonnell,	Brit. Med. Journ., 1877, ii. p. 49.	M.	64	Branch of Sylvian to Broca's convolution was completely blocked.	Left third frontal convolution.	Onset not directly stated.
25[1]	Beaumanoir,	Arch. Gén. de Méd. Navale, 1879 (*vide* Charcot et Pitres, Rev. de Med., 1883, Obs. 56.)	M.	36	Plug in Sylvian, extending into three superficial branches.	Superficial; central masses intact.	Phthisis.
26[1]	Nothnagel,	Topische Diagnostik, 1879, p. 427.	M.	28	Only mentions embolisms in other parts of body.	Cortical alteration involving somewhat the anterior central, second and third frontal, and third, fourth, and fifth insular convolutions. Cortex adherent to pia, forming a hard plate.	Loss of consciousness for an hour (*vide* infra).
27[1]	Webber and Wyman,	Boston Med. and Surg. Journ. 1880, vol. ciii. p. 109.	F.	59	"Apparently from plugging of a vessel" (atheroma).	Left insula much softened.	Other and older cerebral lesions.
28	Seguin,	Journ. Nerv. and Ment. Dis., Jan. 1886, Case 45.	M.	46	Occipital artery (3d branch of posterior cerebral).	Cuneus, extending into temporal gyri and hippocampus.	
29	Wilbrand,	Deut. med. Wchr., 1885.	M.	60	Described as an embolism.	White substance of occipital lobe shrunken, etc.	Thalamic and other foci from a later attack.
30	Haab,	Klin. Montbl. f. Augld., 1882 (*vide* Jour. Nerv. and Ment. Dis., 1886, pp. 18 and 22).	M.	68	Evidently embolic.	Cyst of softening in occipital lobe.	Onset not directly described.
31	Curschmann,	Centrlbl. f. Angkld. (*vide* Frerc, 1882, Obs. 60); also Rep. 7th German Cong. in Wien. Med. Wochr., 1887, p. 682.	M.	50	"of corresponding artery."	Cuneus.	
32[2]	Charcot,	Leçons, etc., Paris, 1872–3, pp. 72 and 74.	M.	22	Posterior cerebral artery.	Nearly the whole occipital lobe.	Typhoid fever; whether the embolism caused or only accompanied delirium is not clear.

[1] The cases were not published as embolic.

[2] Cases 28 to 32 inclusive, represent embolism of an occipital artery or branch.

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