INTRACRANIAL ANEURYSMS.

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My colleague, Dr. Fearnsides [1] in an article upon "Intracranial Aneurysms," published recently in this Journal, has honoured me by references to my paper [2] on "Alterations in Arterial Structure, and their Relation to Syphilis." Dr. Fearnsides, on pages 236 to 238, has attempted, by analysis of the statistics scattered throughout my paper, to show the relative frequency with which aneurysms within the cranium and within the body had been found in the Pathological Institute of the London Hospital. The result has demonstrated to me many defects in my writing. In the hope of removing these defects, as far as possible, I have obtained permission from Dr. Fearnsides to revise the analysis.

A comparative analysis of all the lesions tabulated in my paper was rendered impossible by my having in places analysed the records for 1908 to 1913, inclusive, and in places the records for 1907 to 1913, and by my having omitted the total number of examinations in the years 1908 to 1913. In order to draw up a comparative table it is necessary either to add to some of my statistics missing numbers for 1907, for instance twenty-six cases of syphilitic aortitis, or to deduct from others the numbers belonging to 1907. The latter form of correction gives the most accurate figures, because during the year 1907 our methods of controlling examinations and records were in process of evolution. When this correction is chosen, it is obvious that the total number of examinations during the years 1908 to 1913 must be supplied. Both Dr. Fearnsides and I have adopted as a definition of true aneurysm all dilatations of the whole arterial wall. I shall, therefore, first draw up a table, on similar lines to that of Dr. Fearnsides, so as to include all aneurysms which fell within this definition. I shall then, however, discuss the disadvantages attendant upon a comparative table in which this definition is employed, and the criticisms which I have received in respect of this definition. Finally, I shall attempt to remove these disadvantages, and meet these criticisms, by a second table based upon a modified definition of "aneurysm."

During the years 1908 to 1913, inclusive, in a total of 6,829 necropsies, there were 6,751 examinations of the body, and 4,547 examinations of the head; in 78 of these examinations of the head, examination of the body was not permitted.

TABLE I.

True Aneurysms of Aorta :—	
Due to syphilitic inflammation	175 cases
,, tuberculous inflammation, 1 example in	1 case
,, non-granulomatous inflammations, 4 examples in	4 cases
,, medial degeneration (focal, 18 cases ; diffuse, 4 cases +) ..	22 + cases
	202 + cases
Extensive Dissecting Aneurysms of Aorta :—	
Due to medial degeneration	5 cases
,, developmental deficiency..	2 ,,
	7 cases
True Aneurysms of Other Large Elastic Arteries :—	
Due to syphilitic inflammation, 42 examples (all in cases included in above 175 cases of syphilitic aortitis) in	29 cases
,, medial degeneration (diffuse aneurysm)	+ ,,
	29 + cases
True Aneurysms of Small Elastic Arteries :—	
Due to tuberculous inflammation, 10 examples (all aneurysms in pulmonary vomicæ ; many such aneurysms had doubtless escaped detection) in	10 cases
True Aneurysms of Muscular Arteries (excluding the Cerebral Arteries) :—	
Due to syphilitic inflammation, 6 examples (all in cases included in above 175 cases of syphilitic aortitis) in	5 cases
,, infective embolism, 16 examples in	14 ,,
,, spread of non-granulomatous inflammation from without, 3 examples in	3 ,,
,, medial degeneration (focal, saccular, 12 examples in 7 cases ; focal associated with atheroma + ; diffuse +)	7 ++ cases
	29 ++ cases
True Aneurysms of Cerebral Arteries :—	
Due to infective embolism, 15 examples in	13 cases
,, medial degeneration following congenital developmental deficiency, 33 examples in	29 ,,
,, ,, ,, (diffuse + ; focal associated with atheroma +)	++
	42 ++ cases

This revised list gives, I believe, a true comparative analysis of our records. It is, however, of no service for exact comparison, because I am unable to give under every specified cause either the exact number of examples of aneurysm or the exact number of cases in which aneurysms

occurred. To estimate the total number of aneurysms, as defined by Dr. Fearnsides and by me, would, for instance, in most cases of syphilitic aortitis, have been an almost impossible task owing to their great number. Still greater difficulty would have attended an attempt to ascertain and record the exact number of cases exhibiting certain forms of aneurysmal dilatation of which I merely mentioned in my paper the frequent occurrence. Thus, in regard to aneurysmal dilatation due to medial degeneration of the aorta, I recorded eighteen cases in which there were "small pitted or wrinkled areas," and four cases of "very marked, diffuse dilatation." In a preceding paragraph, however, I mentioned that diffuse dilatation of the aorta, especially of the thoracic aorta, was a very frequent sequence of medial degeneration and "is easily recognized in almost every male over the age of 50." I also stated that in the other large elastic arteries, "slight diffuse dilatation frequently results from medial degeneration." Again, in regard to aneurysmal dilatation of the muscular arteries due to medial degeneration, I recorded, in Table VII, seven cases in which there were saccular dilatations of muscular arteries other than the cerebral. I had, however, previously mentioned (pp. 244-5) that diffuse dilatation due to medial degeneration "is frequently found in the muscular arteries," including the cerebral arteries, and that "slight focal dilatation may be present at the sites of atheromatous plaques," this being "almost constant in atheroma of the cerebral arteries."

Even if I was in a position to make the above table complete by the substitution of exact numbers of cases for the sign +, the list would, as I have learned from correspondence, give a false impression to the majority of readers. This is the consequence of the definition of "true aneurysm" which has been adopted by Dr. Fearnsides and by me. I believe that our definition, which includes every dilatation of the whole wall, is both the only scientific and the only really practicable definition. This definition includes, however, a very large number of dilatations which the clinician, at any rate, would never regard as aneurysms. Indeed, one kindly critic wrote that he regretted my "old-fashioned" definition, and would have had me apply the term aneurysm to saccular dilatations alone. In a general discussion on the causation of aneurysm it would obviously be impossible to accept no dilatation as an aneurysm unless it was saccular. The small crateriform pits and narrow sulci, for instance, which are almost constant in syphilitic aortitis, are all potential saccular aneurysms. Areas which show, post mortem, only such minute dilatations may be more widely dilated during life; thus, the aortic commissure may post mortem only show small sulci in an intima con-

spicuously thickened by inflammation, and the aortic valves may be competent to the water test, although clinical observation and the post-mortem appearances of the heart prove that relative incompetence of the valves had been present during life. There are diffuse dilatations which all investigators would call aneurysms, and which give clinical signs and symptoms similar to those caused by saccular aneurysms. Further, I have found it impossible to estimate exactly what is to be regarded as a saccular dilatation. For exact definition of a saccular dilatation some arbitrary proportion between depth and diameter would require to be determined and universally accepted.

I realize, however, the desirability of attempting to distinguish between what might be called "practical" and "academic" aneurysms; I realized this when preparing my paper. Without such a distinction it would appear impossible to compare our figures with those of other observers. The aneurysms in the cerebral arteries tabulated as due to congenital weakness and to infective embolism, and certain aneurysms in the other muscular arteries due to medial degeneration, were unquestionably saccular and would be regarded by all as aneurysms. In the cases, however, of syphilitic inflammation of the aorta, other large elastic arteries and muscular arteries, and in the cases of medial degeneration of the aorta, I was unable to determine a satisfactory distinction between the practical and academic dilatations. I attempted to make a distinction by quoting the number of dilatations which had given rise to clinical manifestations by rupture, by pressure, or, in the case of aortitis, by causing relative incompetence of the aortic valves. A distinction based on such clinical criteria is, however, obviously imperfect. In a fresh attempt to make the distinction, I have made the following modification in our definition. I have only accepted as aneurysms: (1) definitely saccular dilatations; (2) saucer-like dilatations; and (3) sharply defined, focal, fusiform dilatations. For convenience I have referred to the shallow, saucer-like dilatations as "pouches." With this modified definition I have compiled Table II. This table includes, I think, all the dilatations which would have been accepted as aneurysms by all. In the case of syphilitic inflammation of the aorta and other large elastic arteries, and especially in the case of medial degeneration of the aorta, dilatations are also included which would probably not be considered, by the majority, worthy of the term aneurysm. Inasmuch, however, as Dr. Fearnsides compiled his table in order to show the exceptionally large number of cerebral aneurysms in the records of the London Hospital Pathological Institute, it is well that, if the modified definition

should err, it should err by including too many aneurysms of arteries other than the cerebral.

I regret that I am unable even with this modified definition to give the exact number of aneurysms in syphilitic aortitis; in several cases small examples have merely been recorded as "multiple."

TABLE II.

Focal, Saccular, Saucer-shaped and Fusiform Aneurysms, 1908-1913—*Aorta*:—			
Due to syphilitic inflammation (in 23 cases shallow pouches only)	Examples,	114	+ in 83 cases
,, tuberculous inflammation	,,	1	,, 1 ,,
,, non-granulomatous inflammation	,,	4	,, 4 ,,
,, medial degeneration (all very small or shallow pouches)	,,	4	,, 4 ,,
	Examples,	123	+ in 92 cases
Other Large Elastic Arteries:—			
Due to syphilitic inflammation (all cases are present also in the above 83 cases of syphilitic aneurysms of aorta)	Examples,	6	in 6 cases
Small Elastic Arteries:—			
Due to tuberculous inflammation (in vomicæ) ..	Examples,	10	in 10 cases
Muscular Arteries (excluding Cerebral Arteries):—			
Due to syphilitic inflammation (1 case is present also in the above 83 cases of syphilitic aneurysms of aorta)	Examples,	2	in 2 cases
,, infective embolism	,,	16	,, 14 ,,
,, spread of non-granulomatous inflammation from without	,,	3	,, 3 ,,
,, medial degeneration	,,	12	,, 7 ,,
	Examples,	33	in 26 cases
Cerebral Arteries:—			
Due to infective embolism	Examples,	15	in 13 cases
,, medial degeneration following congenital developmental deficiency	,,	33	,, 29 ,,
	Examples,	48	in 42 cases

In regard to aneurysms caused by syphilitic inflammation of the large elastic arteries other than the aorta, and of the muscular arteries, it must be emphasized that in all the cases recorded in this table and in Table I the syphilitic inflammation involved vessels close to the aorta, and the aorta was affected by similar inflammation. Thus, of the syphilitic aneurysms of the large elastic arteries recorded in both tables the aneurysm farthest from the inflamed aorta lay in the upper two inches of the right common carotid, the lower inch of this artery also

showing evidence of inflammation to the naked eye; of the syphilitic aneurysms of the muscular arteries the aneurysm farthest from the inflamed aorta lay in the centre of the splenic artery. This aneurysm is one of the two recorded in Table II. It was associated with gummata in the peritoneal tissue round the pancreas, in addition to aortitis. In this case the infection had probably arisen independently of the aortitis. In all the remaining cases of syphilitic inflammation of the large elastic and muscular arteries in Table I and Table II the inflammation would appear to have been merely an extension from the aorta.

If Table II is accepted for purposes of comparison, then aneurysms of the aorta occurred in 92 cases or in 1·36 per cent. of examinations of the body. Aneurysms of large elastic arteries other than the aorta occurred in 6 cases; all these aneurysms were due to syphilitic inflammation and were accompanied by aneurysms of the aorta. Aneurysms of muscular arteries, other than the cerebral arteries, occurred in 26 cases, or 0·38 per cent. of examinations of the body; only 2 of these aneurysms were due to syphilitic inflammation; both of these were accompanied by syphilitic aortitis, 1 by an aneurysm of the aorta. Aneurysms of cerebral arteries occurred in 42 cases, or in 0·92 per cent. of examinations of the head; in 13 cases the aneurysms were caused by infective embolism, and 3 of these 13 cases are recorded also in the list of cases of embolic aneurysms in other muscular arteries.

If aneurysms in tuberculous vomicæ and in peptic erosions or ulcers are omitted, the following table compares the number of cases in which aneurysms were found in the arteries of the body with the number of cases in which aneurysms were found in the head, during the years 1908 to 1913:—

Cases with aneurysms of arteries in the body	..	117,	*i.e.*,	1·73	per cent. of examinations.
,, ,, ,, ,, *cranium*	..	42,	,,	0·92	,, ,, ,,

The cases which Dr. Fearnsides quotes, from the literature, of aneurysms of cerebral arteries caused by syphilitic inflammation, remind me of another, serious, omission from my original paper. I should have stated explicitly that I had attempted to give an account of personal observations on alterations in arterial structure, that I had given references to papers to which I was indebted for information, but that I had not examined the literature in order to ascertain to what extent my observations and conclusions corresponded to those of others. My observations were not confined to the cases given in my tables. At the time the paper was written I had seen over 9,000 necropsies in two

general hospitals. It was perhaps presumptuous to decide what conditions were rare on this material alone, although, on the other hand, rare cases are more likely than common to be recorded in the literature. In this connection I may add that a subsequent 1,500 necropsies have not given me an aneurysm due to syphilitic inflammation in a cerebral artery. Immediately after publication I regretted, for another reason, this omission of an explicit statement of the method in which my paper was written. I realized that in its absence I would appear to claim priority for conclusions which other workers had already published. I had, and have, no doubt that the great majority of my observations and conclusions, if correct, could be found in previous publications; there must be very many, besides those quoted by Dr. Fearnsides, whose work I had merely confirmed.

REFERENCES.

[1] Fearnsides, E. G. *Brain*, 1916, vol. xxxix, p. 224.
[2] Turnbull, H. M. *Quart. Journ. Med.*, 1915, vol. viii, p. 201.