again, it will be well to illustrate by the case of a destructive lesion (a much smaller one than in Loss of Speech D), by softening, destroying but little of Broca's region; I will call this case Defect of Speech D. There is not rarely from such a pathological process a condition essentially like that in some cases of Defect of Speech E. Let us suppose that in the case of Defect of Speech D the patient frequently makes mistakes when he talks, either by using wrong words or by being reduced to very "automatic" utterances, such as "Very well," or that he often uses roundabout expressions such as "It won't come out here" (which, eked out by appropriate pantomime, meant "I can't make water"). And yet, to simplify exposition, I will take the patient's saying "hat" for "carpet" only, that to be a sample of the whole of his condition. Here we see the necessity of distinguishing the two elements, negative and positive. It is certain that the softened brain (really what is brain no longer; there is a hole filled with detritus of nervous elements) cannot be the cause of the patient's saying "hat;" that is the speech left, and is owing to activity of the part of Broca's region which remains perfectly healthy.²² This, his positive condition in speech, is sampled by his saying "hat." His negative condition is alone owing to "disease," and is sampled by his inability to say "carpet."

The negative element in Defect of Speech D is a minor degree of that in Loss of Speech D; the positive element is a major degree of the positive element ("Yes" and "No" only) in Loss of Speech D; in the former the dissolution of Broca's region is shallow and the level of evolution high, whilst in the latter the dissolution is very deep and the level of evolution very low, if there be any in that region. The two cases differ in double degree. In the Defect of Speech D there is much speech left, and there are very many mistakes; in the Loss of Speech D there is very little speech left, and there are no mistakes, the patient correctly assenting to and dissenting from (by "Yes" and "No") all statements made to him.

Now for the physical condition in Defect of Speech D. Answering to the slight negative element, there is loss of but a few complex articulatory movements represented in Broca's region; answering to the positive element there is retention of many other such movements; or we may say, limiting illustration to the samples, there is loss of the complex movements answering to inability to say "carpet," and retention of the complex movement answering to saying "hat." If anyone says that a word (properly a syllable) is not a movement he is agreeing with me; for I am contending that activity of nervous arrangements representing certain complex movements (with, of course, corresponding impressions) of the tongue, etc., is only the anatomico-physiological process going on during the appearance of what are the psychological things—words. I need only say that the softening in Defect of Speech D is the negative lesion equivalent to the exhaustion in Defect of Speech E. The utterances both in Defect of Speech E and D are the outcome of activities of perfectly healthy nervous arrangements. The negative lesion in both is dissolution; in each the speech, however imperfect, as certainly signifies evolution going on on a lower level, as the speech of healthy persons does evolution going on on the normal level.

I have admitted that the patient in Defect of Speech E and D may correct his mistakes—that after saying the wrong word "hat" he may immediately say the right word "carpet,"—and I will here admit that when he says "hat" he knows that the word is not the right one. But this does not invalidate the principle of the explanation. For convenience of exposition I made the arbitrary assumption that there are fixed and unalterable nervous arrangements for particular movements, in this case for complex movements of the tongue, etc., corresponding to syllables. I think the seeming difficulties could be met if, abandoning that assumption, I were to go into detail as to the constitution of nervous centres. At any rate, the attempt is to give a materialistic explanation of the physical condition in aphasia, without confounding the concomitant psychical states with that physical condition. I submit that the explanation is, at any rate, as good as the psychological explanation. If we do invoke the latter so-called explanation, we have not only to account for, to take the samples again, "loss of memory" for "carpet," but also for the "over-memory" for "hat." When the patient cannot say "carpet," why does he utter anything? Why does "hat" come out instead?

And admitting that the "loss of memory" for "carpet" is only temporary, and that next moment the patient can say "hat," we have to account for the temporary defect of "memory" and for its sudden restoration for that case. Moreover, if the patient says "hat" and knows that it is wrong, he can only know it by there being revived in him the word "carpet" at the very same time; certainly these two words must be revived mentally; there is then "verbal diplopia." If it is admitted that the psychological statements as to the aphasic conditions are correct descriptions, those who make them are just as much bound as anyone else to seek the abnormal material conditions of the several phenomena of "amnesic aphasia." And bearing in mind the proof given by Ferrier, Horsley, Schäfer, Beevor, Sherrington, and many others, that the region damaged is motor, a merely morphological account of the physical condition in aphasia will not suffice. If anyone says that he cannot understand how activities of motor nervous arrangements can correspond to words, I would remind him that, except the popular psychologist, no one pretends to understand how any material conditions correspond to any psychical states.

THE GOULSTONIAN LECTURES ON SOME CEREBRAL LESIONS.

Delivered before the Royal College of Physicians of London.

By G. NEWTON PITT, M.A., M.D., F.R.C.P.,

Assistant Physician to, and Demonstrator of Morbid Anatomy at, Guy's Hospital.

LECTURE III.—ON CEREBRAL EMBOLISM AND ANEURYSM.

I PROPOSE considering to-day the cases of cerebral embolism and of cerebral aneurysm, which are for the most part included among them, that have died at Guy's Hospital, between the years 1869 and 1888, a period of twenty years.

In 79, that is, in 0.9 per cent., or nearly 1 per cent., of the inspections, cerebral embolism had occurred. In some of the patients two or more cerebral vessels contained clots; there were ninety-five blocked vessels found in the whole series of cases.

Forty-four of the patients were males and 35 females; there was nothing in the histories of the cases to indicate why men were more often affected than the women.

The youngest patient was aged 13, and the oldest 73; the remainder of the cases occurred between the ages of 13 and 66, the distribution being fairly uniform for the decennial periods from 10 to 60. Patients at both extremes of life seem to possess an immunity from this affection.

Cerebral Embolism.

No. of cases	10-19	20-29	30-39	40-49	50-59	60-69	70-79
...	18	15	15	10	15	6	1

In only 6 cases was the source of the emboli not discovered. In 4 of them the vessel had been blocked for a long time (two years, two years, ten, and many months), and it is quite probable that in these, cardiac vegetations or thrombi may have given rise to emboli, and may yet have disappeared and left no trace at the *post-mortem* examination.

In most of the cases there was no difficulty in tracing the source of the emboli; in 68 it was found to be in the heart. The mitral valve was the most common seat, being affected in 46 cases, in 7 of which the aortic valve was also diseased. The vegetations were limited to the aortic orifice in 13; in most (46) of these 68 cases, there was chronic disease of the valves, on which had been grafted a fungating endocarditis, the vegetations were generally friable, and in many ulceration of the endocardium and valves was also present. In 18 of the cases, ulceration had led to rupture of the chordae tendineae. The mitral orifice was markedly stenosed in 26, and was generally associated with recent friable (vi), or fungating granulations; in 5 the valve was free from vegetations, and in 4 of these the embolus had come from *ante-mortem* clotting in the left auricle, while in 1 no clot or granulating surface was found (Case CLVIII). The embolus ori-

²² To give another example, surely it is impossible that when the patient referred to in the text said, "It won't come out here," there was engagement of nervous arrangements which were "diseased" in any way whatever; the "disease" was the cause of his inability to say "I can't make water."

ginated in 5 from an atheromatous aorta, and in 2 from clotting in the left ventricle. The heart-disease was usually due to rheumatism originally, but the cause of the acute fungating disease was, as is generally the case, obscure.

Gonorrhoea was the starting point of the whole disease in the two following cases (Nos. CXLVIII and CXLIX). The first is that of a man aged 21, who came in comatose and died next day. There were red lymph-covered spots, with recent vegetations, on the mitral valves, multiple abscesses in the liver and kidneys, and a general pyamic condition, which may have originated the endocarditis, or have been secondary to it; more probably the former.

The second was that of a girl, aged 18, who had been ill for some time, when she suddenly became hemiplegic and aphasic. She was found to have gonorrhoea, but her heart was healthy, and beyond a thrombosed iliac vein, no source was found for the emboli, lodged in her internal carotid and opposite middle cerebral arteries. A prostatic abscess had, in another patient, set up an abscess in the wall of the mitral valve, which had ruptured into the left auricle, and had also caused a malignant endocarditis of the pulmonary artery, as we have on other occasions observed from gonorrhoea and similar causes. The emboli which were found in the right anterior and middle cerebral arteries had come from the abscess on the mitral valve.

Of the 4 cases in which the mischief started from a clot in the left auricle, there were mitral stenosis in 2 of them, fibroid disease of the heart in 1, and bronchitis and emphysema with an incomplete mitral valve in the last. Twice the clot was caught in the carotid, once in the basilar, and in the last case in the middle cerebral artery.

The two cases in which there was thrombosis in the left ventricle were due to chronic Bright's disease and bronchitis respectively, and the hearts were hypertrophied and dilated. The patients were both about 50.

The aorta, the valves, and the cavities on the left side of the heart are all well recognised sources of emboli when they present an atheromatous surface, recent granulations, fungating endocarditis, or *ante-mortem* thrombosis. There remain for consideration 9 cases to be otherwise explained. In 2 of these in which the hemiplegia had lasted for two years the heart was found to be normal at the inspection. In a third case the patient had old mitral stenosis, and during an attack of acute rheumatism ten weeks previous to her death she became hemiplegic from middle cerebral embolism. At the inspection, the heart was free from any vegetations or *ante-mortem* thrombosis.

The explanation of these three cases is, doubtless, that the vegetations or clots in the heart which had given rise to the emboli had become absorbed in the interval (Cases CXXXIX, CXXXVII, CLVIII). Several similar instances are on record. In the two following cases in which thrombosis had occurred in the right ventricle (Cases CXXXV, CLXVIII) there were clots in the pulmonary arteries, in one if not in both of which they were embolic.

The first was a man, aged 38, admitted with chronic Bright's disease. On May 28th he had symptoms of pulmonary embolism. Four days later he had a fit, followed by aphasia and right hemiplegia, and died the next day. There was a firm, tough embolus at the bifurcation of the left Sylvian artery, with *ante-mortem* thrombosis in the right auricle and ventricle. The posterior part of the third frontal and the adjacent convolutions were softened.

The second was a woman with a uterine fibroid and metrorrhagia. She became hemiplegic, and died the next day. The internal carotid was found blocked at its bifurcation, and there was *ante-mortem* thrombosis in the right ventricle.

These cases are difficult to explain. They were considered to be examples of emboli by the pathologists who made the examinations; if so, we must conclude that the emboli were secondary to the thrombi in the right ventricle. That it is possible for embolism of a systemic artery to be secondary to a distant venous source is also suggested by Case CXXXVI, a man, aged 64, in whom emboli in the brain and spleen were found, for which no source but a large carcinomatous growth in the liver could be suggested. In the case of the girl with gonorrhoea alluded to above, it appeared that the infecting material must have passed through the pulmonary capillaries without producing any local mischief, and have ultimately lodged in the brain.

These four cases would suggest that very occasionally the only source for emboli is to be found in some distant local trouble beyond the pulmonary circulation. Possibly these set up some

small local patches of mischief in the lung, from the veins in which the clot or emboli are liberated.

Micro-organisms can undoubtedly pass through the pulmonary capillaries, and set up mischief in the systemic arteries, which would explain the case due to gonorrhoea: but in the other cases it seems essential that local thrombosis should exist in the lungs, and that a small clot liberated from the pulmonary vein should be the source of the embolism. At the same time it might not be possible to detect the spot, especially if very localised, at the inspection. In two of the cases there was clotting in the pulmonary arteries. There remain two other cases, in which not even this explanation can be offered (Cases CXLIII and CXL). One was a case of chronic bronchitis and emphysema with a large dilated heart, and an embolus in the left Sylvian artery; and the other a woman of 56, with chronic bronchitis, who developed finally right hemiplegia and aphasia, in whom recent emboli were found in the kidneys with patches of softening in the brain, the heart was healthy, and in neither was any source for the emboli detected. It is always possible that in these cases of patients with feeble hearts, minute clots may have formed in the cavities and have been discharged, leaving no trace behind them in the heart. The presence of emboli in other organs, the situation of the clots and their character, show that in most of the cases the theory that they were thrombotic cannot be adopted.

The explanation of these exceptional cases is very obscure and demands further investigation.

In 75 per cent. of the cases the emboli were found in the middle cerebral vessels. The vessels which were embolised were:

		Right.	Left.	Both.
Middle cerebral	...	32	38	9
Internal carotid	...	7	4	1
Posterior cerebral	...	3	2	1
Anterior cerebral	...	3	0	0
	Total	45	44	11
Basilar	...	5.		

On the whole the two sides were affected with equal frequency. The cases in which the vessels on both sides were blocked are included also among those on each side.

The character of the embolus varied; it was white, decolorised, or yellow in 32, adherent in 18, non-adherent in 6, firm in 4, calcareous in 2, and softening in 3. In the two cases in which the mass was calcareous, the sources were a calcareous mitral and an atheromatous aorta (Cases CXLVI, CXIX).

In twenty cases the emboli could not be found. In all there was undoubted evidence that the patches of softening or the aneurysms were embolic, from the presence of emboli elsewhere, or from the presence of fungating endocarditis.

There appears to be no doubt that an embolus may cause a patch of softening and later on the clot may disappear through absorption, and leave no trace. Again, a clot may break up and be carried further on, into the smaller vessels, where it is almost impossible to detect it.

A clot may soften and break up into *débris*. This seems to be usually the case with septic clots, such as give rise to aneurysms, for an embolus was only found in 5 out of 13 cases. It is possible, also, that in some instances an embolus may be detained in the internal carotid and produce softening; yet it may be overlooked if the base of the skull be not opened up.

It is not unnatural, therefore, that in about a fifth of the cases the emboli are not found. The fact that the clots disappear from the vessels is an additional reason for believing that they may also disappear from the heart and lungs.

Cases of Embolism in the Middle Cerebral Arteries in Seventy-two Patients.—In 51 cases the emboli were found, and in 21 they were not found. Two-thirds of the cases were due to fungating endocarditis, and hence many of the patients were so ill that the symptoms of the embolism were often masked. In 14 of the patients there were no symptoms of embolism during life, and its presence was only discovered in the *post-mortem* room. In many others the symptoms were obscure, and varied much, as the following list, which gives their frequency, will show. Hemiplegia 32, aphasia (which was found only with left-sided lesions) 17, coma 8, convulsion 7, rigidity of limbs 4, stupor 2, hemianæsthesia 2, numbness on one side 3, onset of hemiplegia without unconsciousness 3, headache 2, pyrexia 2, drowsiness, paralysis of both arms and legs, delirium and erratic movements of the arms, 1 each. In 14, or a fifth of the cases, there were no cerebral symptoms to indicate the onset of the plugging.

Death was frequently due to the associated heart condition, or

to some other complication, rather than to the embolism directly; thus 25 patients died from cardiac failure and the sapræmic condition induced by the fungating endocarditis, 25 from the cerebral lesion (including 12 from rupture of a cerebral aneurysm and 7 from coma due to bilateral embolism), 2 died from gangrene of the leg due to embolic occlusion of an artery, 2 from general pyæmia, and in the remaining cases the causes were pulmonary disease 4, chronic Bright's disease 2, tubercle, cancer, drowning, and suppurative mediastinitis 1 each.

Only 40 per cent. of the patients, therefore, died from the cerebral lesion. The duration of life after the onset of the symptoms is also very variable, being in 12 cases less than twenty-four hours, in 17 less than a week. Twenty-one of the patients survived from one to three months, 1 lived five months, while 2 survived one year and 2 two years.

In the cases which had lasted two years the source of the emboli was not made out. In cases which had lasted a year and six months respectively the vegetations were calcareous; in cases which had lasted for even so long as five and three months, the emboli were found to be associated with fungating endocarditis, and it would appear that this condition must have lasted, without proving fatal, for that length of time.

The aphasia varied greatly; in some patients there was a misuse of words, in others their conversation was limited to merely "yes" and "no." In numerous instances the softening extended to several convolutions; in one case the third frontal, in another the temporo-sphenoidal, and in a third both of these parts were softened. In one case the softening lay outside the corpus striatum, and in three outside the lenticular nucleus. A clot of blood which had spread beneath the meninges over the third frontal had in one case produced aphasia.

The spot at which the clot became arrested varied. In 16 it remained in the trunk of the middle cerebral vessel, in 3 at its origin, in 18 at its first bifurcation, in 5 at its second division, and in 12 in one of the main branches. In 3 the branch was one of those that supply the white matter of the brain, and in 3 some of the cortical capillaries.

It is not sufficient to determine the locality of the occlusion of a cerebral vessel, as the amount of softening which may ensue is very variable. This is well shown by an examination of the amount of softening which was present in those cases in which an embolus was found at the first division of the middle cerebral artery. In 5 the brain appeared normal, in 6 there was extensive softening, in 2 the softening was limited to the nuclei, in 1 to the cortex, and in 2 there were small patches of softening in both. When death takes place within one or two days of the infarction, no change may be apparent in the brain; the short lapse of time in some of the cases explains partially, but not entirely, the great variation there is in the amount of softening. The relation between the symptoms and the situation of the infarction is rendered still more complex by the fact that there may be marked paralysis, and yet the change in the brain may be very slight; and, on the other hand, some cases gave but little evidence of brain mischief during life, in which *post mortem* there was marked softening. The former is well illustrated by Case CL. A woman, aged 61, died from intrathoracic growth. Two weeks before her death she had complete right hemiplegia, recovering power in her leg partially before death. The termination of the main middle cerebral artery was occluded by a clot; the only change in the brain was a barely appreciable diminution in the consistency of the ascending convolutions and in the tip of the temporo-sphenoidal lobe; yet in the woman's feeble condition this seemed to be sufficient to produce marked paralysis.

Only twice out of the whole series was optic neuritis noticed (Cases CL and CLXIIIa). Patients occasionally complained of headache when softening had taken place. It happened (in one-seventh of the cases) that both middle cerebral arteries were blocked, and sometimes, curiously enough, the clots lodged at corresponding spots on the two sides. Very few such cases are on record.

Nine Cases of Emboli in Both Middle Cerebral Arteries.—A man, aged 18; œdema, cardiac failure, large spleen. October 4th. Headache, delirium, numbness left side. Ingravescens coma, death on 26th. *Post mortem*: On the right vessel, one inch along, a small aneurysm, from which blood oozed; on the left, an aneurysm further on, a blood-stained patch of softening on right side. Fungating endocarditis of mitral valve; ruptured chordæ tendinæ; embolism of brachial artery, with commencing aneurysm; infarcts in spleen, acute nephritis.

A man, aged 32; right hemiplegia and aphasia five weeks before death; he recovered his speech, but became comatose, and had convulsive twitchings the day before his death. *Post mortem*: Both vessels were blocked half an inch from their origin by yellowish softening thrombi; a small patch of softening in the left lenticular nucleus; fungating endocarditis of the aortic valves; infarcts in the kidneys and spleen.

A boy, aged 15; ten weeks before death sudden onset of left hemiplegia; the arm remained paralysed; three days before his death he had a fit, became stertorous, and his limbs were rigid. *Post mortem*: Diffused subarachnoid hæmorrhage over the brain; firm embolus in right vessel, with extensive softening into which hæmorrhage had taken place; only a small patch of softening was found on the left side; fungating endocarditis of mitral valve; infarct in spleen.

A man, aged 28; died shortly after admission. *Post mortem*: At the first bifurcation on the left side was an old embolus, and at the second on the right a recent embolus, at the termination of the basilar artery an old embolus had organised on to the wall; softening in left corpus striatum, and temporo-sphenoidal convolution; convolutions ochreous from old meningeal hæmorrhage; fungating endocarditis of mitral and aortic valves; infarcts in spleen; acute nephritis.

A woman, aged 24; admitted with dyspnoea and cardiac failure; left hemiplegia with unconsciousness; partial recovery; died two weeks later, after forty-eight hours' unconsciousness. *Post mortem*: On both sides the posterior branch was blocked; on the right side the vessel was dilated into a fusiform aneurysm, and round it there was purulent lymph and blood clot; there was extensive softening; on the left side there was only a small patch of softening; fungating endocarditis of mitral valve; infarcts in kidneys and spleen.

A man. *Post mortem*: Both vessels blocked at their bifurcation.

A boy, aged 14; admitted with cardiac failure, vomiting, and headache, April 18th; markedly aphasic, April 23rd; double optic neuritis, left radial and femoral pulses almost occluded, April 30th; intense headache, rigidity, and paralysis of the left side, deviation of eyes to right, May 14th; right facial paralysis, May 17th; right arm rigid; he became more drowsy and died on May 22nd. *Post mortem*: On the right side there was extensive softening of the whole tract supplied by the middle cerebral artery, but no embolus was found; in the softened mass three ounces of blood had been extravasated; emboli were found in the right anterior and in the left middle cerebral; the left frontal lobe was softened; fungating endocarditis of the mitral valve; infarcts in the spleen, kidneys, and right popliteal artery; acute nephritis.

A man, aged 27; laid up with cardiac failure and œdema, April 24th; he woke up with left hemiplegia; acute bedsores in three days; on May 20th he was found with right hemiplegia and speechless; he died thirty hours later. *Post mortem*: At corresponding spots in both vessels there were emboli, that on the right being old and adherent; extensive softening on the right side, only a small patch on the left; fungating endocarditis of mitral and aortic valves; mitral stenosis.

All the patients died from the cerebral condition, 6 being comatose, and the remaining one from rupture of a cerebral aneurysm. (Two other cases died before a report was made.)

After the first infarction, 1 patient survived ten weeks; 2, five; 2, three weeks; and 1, two weeks. They survived from one to three days after the second vessel was blocked, except in one case, in which the patient lived twenty days.

The 5 cases in which the posterior cerebral artery was found to contain an embolus do not require much notice; in 2 there were no cerebral symptoms, in 2 others the lesions were multiple, and in the remaining one the patient developed right hemiplegia the day before his death, although at the inspection the only embolus which was found was in the posterior vessel. The 3 cases of embolism of the anterior vessel were complicated, and presented no special symptoms. The 4 cases of embolism of the basilar artery presented no special features but unconsciousness, except in the following case (CLXIIIa):

A girl, aged 24, with severe heart disease, was found unconscious, with paresis of the right limbs and the left side of the face. On the right side the third nerve was paralysed, and she was unable to look to the left or to raise the left eye. The right optic disc was choked. She became listless, partially anæsthetic, unintelligible, and comatose, dying a month later. The basilar artery was found to be plugged at its extremity with a decolourised em-

bolus. On 6 occasions an embolus was found in the right, and on 3 in the left internal carotid artery, besides 1 in which both the vessels were found to be plugged. In 2 there were no definite symptoms, in 5 anæsthesia, in 6 coma, in 8 hemiplegia, in 4 drowsiness, in 3 aphasia, in 2 convulsions, in 2 headache, and in 1 numbness, and in another no unconsciousness. It was also specially noted in 4 that there was a well-marked blush over the face, which persisted for some days, and in some recurred. In 1 there was ecchymosis and gangrene of the ears. In 2 cases there was inability to look to one side. There was softening in all the cases except one. Four patients died within a week, 2 survived only a month, and 3 from six weeks to four months.

Cerebral embolism is in the great majority of cases due to fungating endocarditis, and may be survived for many weeks. The patients are often seriously ill with their heart disease, and often the embolism gives rise to no symptoms at all. They die from their heart condition as frequently as they do from their cerebral lesion; the great majority of these latter cases being due to coma from multiple emboli or from ruptured aneurysm secondary to them. The symptoms are not definitely associated with the amount of softening visible, nor is this latter solely determined by the situation of the embolus. When the internal carotid is occluded there is almost invariably hemiplegia, and in some of the cases there is a well-marked recurrent blush on the face for a few days. Embolism gives rise to ecchymosis and congestion of the cerebral tissue at first, passing on to red and yellow softening, but in those cases which have lasted some weeks there is white softening. Aneurysm and hæmorrhage are not infrequent.

An Analysis of Twenty-three Cases of Cerebral Aneurysm.—Cerebral aneurysm is a very rare lesion, having been found only nineteen times out of 9,000 inspections, that is, in less than $\frac{1}{4}$ per cent. There were besides four cases in which rounded masses of clot were found in the cerebral tissue, which were thought to be aneurysmal, but the vessels with which they were connected were not definitely determined. The hæmorrhages were all associated with fungating endocarditis and occurred in young people, and, as is shown further on, the assumption that they were aneurysmal is not unreasonable. There are therefore 23 cases to be discussed. Of these, 15 were males and 8 females, that is, the lesion is twice as frequent with men as with women, a greater proportion than was found to exist in cases of embolism.

The distribution as to age is as follows: 0 to 9, one; 10 to 19, eight; 20 to 29, four; 30 to 39, three; 40 to 49, two; 50 to 59, two; 60 to 69, one. The youngest patient was only 6, and the aneurysm was an unusual one.

CASE CXIV.—A boy, aged 6, was admitted with acute periostitis of the femur, which ultimately led to the hip being excised. He died a month later with symptoms of tubercular meningitis. At the inspection a small aneurysm, which had ruptured, was found on the right vertebral artery. There were old tubercular lesions in the lungs, intestines, and spleen, and it was thought that the aneurysm might have been tubercular, but there was no evidence microscopically that this was so.

The condition which had led to the formation of the aneurysm was obscure. It was thought that possibly it might have been due to tubercular changes in the wall, though there were no other local tubercular lesions in the brain. Another possible explanation is that it was due to some septic emboli, which had been derived from the acutely-inflamed periosteum a month previously.

The most interesting conclusion as to the age distribution is that nearly half (two-fifths) of the patients were between the ages of 10 and 20; and that of those cases in which the cause was an embolism exactly half were between these ages.

In 1 case there were two aneurysms, one on each middle cerebral artery (Case CLXXXIII); in another there was an aneurysm on one side and hæmorrhage on the other. In 16 out of 21 cases there was fungating endocarditis, that is, in 75 per cent. In 3 there were granular kidneys and atheromatous vessels; and two of these cases were fusiform aneurysms of the internal carotid artery in the cavernous sinus. Putting these two on one side, there remain only three out of the whole series which were not associated with fungating endocarditis. One of these was the case which was thought to be tubercular.

The exact mechanism by which the aneurysms are produced is still under dispute. In the great majority of the cases the embolic plug is not to be found, and yet there can be no doubt, from a consideration of the whole series, that the formation of an aneurysmal dilatation is almost, if not always, preceded by an embolic plugging when the patient is young. Not only must

there be an embolus, but probably it must be septic. This is shown by the association of a fungating endocarditis in all the cases, and in none of them was the source of the embolus an *antemortem* clot, an atheromatous aorta, nor any other non-septic source. The frequency with which the middle cerebral arteries are the seats of aneurysm (this being so in three-quarters of the cases) is in accordance with the liability of these vessels to embolism, and it is to be noted that the usual seat of an aneurysm is close to the bifurcation of a vessel.

Some light is thrown on the mechanism by which an aneurysm is set up by a septic embolism by the examination of their relative sites when the two are found together. This, as pointed out above, is a rare event, having occurred only five times.

In Case CLXXXV, the middle cerebral artery was blocked by an embolus half an inch from its origin. Part of the clot appears to have broken up and passed along the vessel into the capillaries. There were ecchymotic spots in the cortex and fusiform dilations of the vessels filled with puriform clot. It would appear in this instance that there had been local inflammation of the vessels at the point of arrest of the infective material, and dilatation had ensued in consequence.

CASE CLXXX.—In this instance also the artery had dilated at the seat of the blocking by the clot; around this there was some puriform lymph, showing that inflammatory changes had taken place. In other cases of emboli secondary to fungating endocarditis a local meningitis was found around the blocked vessel. There is therefore ample evidence that this cause is sufficient to set up inflammatory changes not infrequently around the wall of the vessel, and *a fortiori* in the wall itself. The reason aneurysm does not occur more frequently is because it is necessary that, not only should inflammatory changes take place in the wall, but the inflamed portion must not be cut off from the pressure of the circulation. In the two cases we have considered the aneurysm formed at the seat of the clot. In the two following the dilatation took place, in the one on the proximal side, and in the other on the distal side of the blockage.

Fig. 1.—Case CLXXXII. No symptoms previous to rupture; death in one hour.

CASE CLXXXII.—A man, aged 50, was found insensible an hour before death. On the third branch of the middle cerebral artery was an aneurysm which had ruptured into the ventricle and on to the surface. An embolism was found at the bifurcation of the vessel just before the third branch was given off. There was an atheromatous aorta, and the man had had syphilis (Case CXCIX).

It has been suggested that the vessel gives way because the embolus is tough and calcareous, yet in the two instances in which they were calcareous this had not occurred, while in some the clot was markedly soft.

It can readily be understood that if the dilatation be due to inflammatory changes, these may sometimes take place at the spot, and sometimes on either side; this will, therefore, explain the differences in the relative sites. The more the clot softens and disappears the more will the inflamed vessel be exposed to the blood stream, and the greater the probability that an aneurysm will form.

We are, therefore, justified in stating that the cause of four-fifths of all cerebral aneurysms is the retention of an embolus from a patch of fungating or granular endocarditis at the bifurcation of a vessel. Inflammatory changes take place in the clot, which in most cases softens and disappears, the wall of the vessel also inflames and dilates; the dilatation may take place at the seat of the embolus occasionally on the distal, and at other times on the proximal side of the obstruction, which may be complete. The more common condition is probably disappearance of the clot,

with softening at the spot. In the great majority of the cases the middle cerebral was the vessel which was affected.

Fig. 2.—Case CLXXXIII. Three weeks' history; found ruptured.

CASE CLXXXIII.—A man, aged 18, was admitted with severe heart disease; he developed headache, delirium, and numbness, with coldness on the left side, dying three weeks later. On the right side there was a ruptured aneurysm of the middle cerebral artery, with a patch of softening in the temporo-sphenoidal lobe containing blood-stained fluid and clot. There was a small aneurysm on the opposite vessel.

The symmetry of the disease and the youth of the patient indicate the embolic nature of such cases. The wall of the sac is in most cases very thin, and it is rare for much thickening from organising clot to take place. In the following case (No. CLXXIX) the wall was thick, and there was a well organised layer of clot.

Fig. 3.—Case CLXXIX.

A man, aged 31, was admitted with advanced heart disease; he had a series of epileptiform attacks, with some unconsciousness, for twenty-four hours, but remained fairly well till his death two weeks later. On the right middle cerebral artery an aneurysmal sac was found pouching towards the brain; the outer part was formed by tough arterial wall, and the inner of blood clot. The whole of the external capsule and the claustrum were ploughed up, and the blood had extended down to the base of the brain, beneath the membranes: fungating endocarditis.

Cerebral Aneurysms.

	Right.	Left.	Both.
Middle cerebral	9	7	1
Posterior cerebral	1	0	0
Anterior cerebral	1	0	0
Internal carotid	2	1	0
Vertebral	1	0	0
	14	8	1

There were six cases of hæmorrhage, which were probably due to embolism, and four of them to aneurysm. The ages were between 12 and 26 (Cases CXCH, and CXCV-CXCIX).

The following conclusions may be drawn from the cases of aneurysm of the middle cerebral arteries: The size was in six described as that of a pea, four were oval sacs from a half to three-

Fig. 4.—Case CLXXXI. Few weeks' history; found unruptured at post mortem.

quarters inch in diameter, one was the size of a walnut, and one was a bilobed sac the size of two nuts. In twelve cases death occurred from rupture of the sac; in six of the patients death had occurred from other causes, and the aneurysmal sacs were found unruptured. Death in these had taken place from heart disease (2), from multiple patches of softening in the brain (2), from amputation, and from suppurative mediastinitis.

CASE CLXXXI.—A girl, aged 16, was admitted with severe heart disease and left hemiplegia of five months' duration. She ultimately died from suppurative mediastinitis. At the bifurcation of the right middle cerebral artery was a bilobed aneurysmal sac, each being the size of a nut. There was fungating endocarditis, with suppuration around the trachea.

The hæmorrhage had these features: the aneurysm was situated on the surface in all but three, and two of these extended to the surface. The clot had extended into the cerebral tissue, and had ploughed it up in several, in five rupturing into the lateral ventricle, in eight on to the surface; in several of the cases it had extended in both directions. In consequence of the blood so readily reaching the surface death frequently took place rapidly. One, three, seven, and nine hours were the most rapid cases. In many of the cases there were no symptoms previous to the rupture, except a headache, which was a feature in some of them. In other cases in which the onset of the embolism could be fixed by the symptoms, the duration of the aneurysm after the blocking was shown to be three weeks in (CLXXXIII and CLXXXVI), two weeks in a third, and five months was the longest (CLXXXI). The onset of the embolism was marked by hemiplegia in six, and in several associated with a dull stupid mental condition, in others by a period of unconsciousness, and in some by fits. The onset of softening distinct from the embolism was well marked in the above case (CLXXXI).

The cases of aneurysm of the anterior and posterior cerebral arteries presented no characteristic symptoms to indicate the vessels affected. Three of the aneurysms were on the internal carotid artery; two of them were fusiform dilatations, occurring in men aged 66 and 56, and they differ entirely in their etiology from the other cases we are considering.

Fig. 5.—Case CLXXXIX. Carotid aneurysm; four months' history; chief symptom, paralysis of third nerve.

CASES CLXXXIX and CXCI.—In the first, the main symptom besides ill health was ptosis, due to the compression of the third nerve by the aneurysm in the cavernous sinus. The man had old kidney disease, and died with suppurative nephritis. The aneurysm was due to diseased vessels secondary to his bad kidneys. The second patient died with meningitis. In both the aneurysm set up suppuration round the pituitary body.

The third case was that of a boy, aged 17, who, six weeks before his death, was seized with right hemiplegia and aphasia, and died with heart disease. At the inspection there was fungating endocarditis, with an embolic plug at the bifurcation of the internal

The main conclusions that may be drawn are:

1. It is most exceptional to find a cerebral aneurysm which is not associated with fungating endocarditis.

2. The lodging of a septic embolus is the starting point of the changes which take place in the vessel.

3. The clot inflames and in most cases disappears, the vessel dilating from the inflammatory changes in its coats.

4. An aneurysm may form and rupture in three weeks.

5. The onset of the embolism may not be marked by any symptoms, and the rupture of the aneurysm may be the first indication that there is danger.

CASE CLXXVII.—A man brought in dead, ill one hour. Aneurysm on middle cerebral had ruptured into lateral ventricle. The clot weighed 4 ounces. Fungating endocarditis.

The aneurysms are usually on the surface of the brain, and when they rupture they are more rapidly fatal than other forms of cerebral hæmorrhage. As the aneurysm increases in size it tends to spread inwards, plunging up the cerebral tissue, and may rupture into the lateral ventricle, although it has originated on or near the surface. Cases of cerebral hæmorrhage in young people will generally be found to have originated in aneurysm.¹

THE QUESTION OF OPERATION UPON FISTULA IN PHTHISIS.²

By HERBERT WM. ALLINGHAM, F.R.C.S.,

Surgeon to the Great Northern Hospital, Assistant-Surgeon to St. Mark's Hospital for Fistula, etc.

IN books of medicine I have scarcely been able to find any real, clear, and useful guidance as to the course which should be pursued in cases which fall under the heading of this paper—namely, patients who suffer from phthisis combined with fistula. Moreover, many of the books to which I have referred appear to me to be sadly contradictory one of the other; some say, "Do not on any account touch a fistula in a patient suffering from phthisis;" others state that it is advisable to operate. By far the greater number of the authorities give barely more than a single paragraph to the treatment of this combination of phthisis and fistula.

I must make exceptions. Dr. Theodore Williams, in his book, mainly alludes to the frequent alternation between the free discharges from the fistula and the activity of the phthisis, and adds that this circumstance has deterred many physicians from counselling and surgeons from performing operations in such cases, from the dread of the eruption of miliary tubercle that sometimes follows on a successful operation on fistula in phthisical subjects.

Dr. Douglas Powell, in the last edition of his book on *Diseases of the Lungs*, writes to the following effect: "Fistula in ano occurs in something less than 5 per cent. of cases of phthisis, and, in my experience, is almost limited to males. Amongst those afflicted with fistula, however, phthisis commonly supervenes or already exists." He then states two circumstances which should be borne in mind:

1. Fistula may arise from various causes, with no necessary connection with tubercle; but, having become chronic, it is very apt to become tubercular, and to lead to secondary tubercle in other organs, especially the lungs. Characteristic bacilli have been found in the indurated walls of most chronic fistulæ. In cases of this kind, timely operative treatment of the fistula may avert secondary tubercular affections of its walls and of other organs.

2. Fistula more commonly occurs in cases of phthisis when the cachexia of that disease is already marked. It is then impossible to say whether the disease was tubercular from the first, but it soon becomes so. In the presence of decided chest disease fistula is of secondary importance, and, unless causing much additional suffering and exhaustion, should not be operated on.

He further remarks that "the observed alternation between the activity of fistula and of chest disease has deterred surgeons from operating with willingness. A more real objection is the risk of operations failing from the morbid character of the surrounding tissues, and the further danger of the activity of the chest mischief supervening on the breaking of the tubercular induration about the fistula, or upon the renewed febrile action attendant

on operations in unhealthy persons." Dr. Douglas Powell adds that bad cases of fistula may require some operative measures.

In his book on *Phthisis*, published a quarter of a century ago, Dr. J. E. Pollock writes as follows on the question of operating on fistula in phthisical cases:

"The result of such cases as we have witnessed is that the operation is not ordinarily successful in healing the fistula; that, if successful for a time, the discharge almost invariably returns; that the phthisical symptoms always increase after the healing of the sinus; and that the discharge itself is a condition favourable to the quiescence of the irritation in the lung. In very frequent instances the aggravation of all the symptoms of phthisis has occurred almost immediately after the suppression of the discharge." (p. 317). On the next page he writes: "When the operation is successful the patient is commonly, as regards the phthisis, in a quiescent state; but generally, within one or two months, the lung disease assumes activity." He then gives an analysis of 31 cases of phthisis with fistula. "Of these, 10 were operated on with the following result: Fistula healed and chest symptoms became worse in 3 cases; phthisis commenced after the operation in 2 cases; the fistula returned in 3 cases; result doubtful in 2 cases. In 21 cases no operation was performed; fistula persistent, chest symptoms remained chronic, in 14 cases; fistula persistent, chest symptoms became worse, in 4 cases; fistula had existed before phthisis, the latter being of a periosteal form, in 3 cases." (pp. 319-320).

Just contrary to some of the above opinions is the following passage from the late Dr. Fuller's book on *Diseases of the Chest* (p. 441): "As long as the discharge from a fistula is insignificant and the patient's mind is not seriously disturbed by its continuance, so long is it advisable to confine our efforts to the treatment of the constitutional malady, and not to disturb the fistula. Any attempt at curing it by operation, under these circumstances, would probably be followed by an immediate increase in the cough and the pectoral symptoms, and therefore would be highly injurious. But I do not hold with those who maintain that a fistula occurring in the course of phthisis ought never to be interfered with. In some instances the discharge is profuse, and constitutes an important source of waste, and the patient is so distressed and alarmed at its continuance that no treatment can be of avail until his nervous apprehensions are overcome. He can neither eat nor sleep for thinking of it, and his whole system is depressed in consequence. In such a case I have known the greatest benefit result from an operation, combined with the formation of an issue in the arm, the use of a proper diet, and the administration of cod-liver oil, quinine, and other appropriate remedies. Not only has the fistula healed, but the general health has improved, the patient has gained in flesh, and the physical signs of pulmonary disease have materially decreased."

In the face of these contradictory opinions I think that, for the guidance both of physicians and surgeons, some fairly definite rules had better be laid down in fuller detail than has hitherto been the case. For what at present is the procedure too often pursued? If the patient comes to a surgeon, the latter perhaps at once says, "there must be an operation," without consulting a physician as to the condition of the lungs. On the other hand, the physician may refuse to allow an operation without consulting a surgeon as to the nature and extent of the fistula. It is absurd for either surgeon or physician to form a decided opinion without conferring one with the other, both as to the nature and extent of the fistula and the condition of the phthisis. Without such consideration, how can it be accurately determined whether the fistula ought to be treated or not, and, if so, in what manner?

Were such prior consultations universally held, it would be found that in patients who suffer from fistula combined with phthisis there are certain peculiarities of aspect to be observed in the fistula itself and in the surrounding parts. Most of these peculiarities were, I think, first recorded by my father; and, as they are of extreme importance from a surgical point of view, I have endeavoured to arrange the following tabulation:

Fistula in a Healthy Subject.—A fistula in the healthy occurs about as frequently in females as in males; the buttocks are plump, and the ischio-rectal fosse well filled with fat; there is an average amount of crisp hair; the sphincters have good contractile power; fistula begins, as a rule, with an acute abscess, attended with great pain; the external opening of the fistula is small and pouting; the skin about the external opening and along the back of the fistula is healthy, or only slightly discoloured; the internal opening is usually small, barely admitting the probe; the sinuses

¹ It would have occupied too much space to publish here even the briefest abstract of all the cases with which these lectures deal. The lectures will be republished with the cases *in extenso*.

² Read before the Medical Society of London.