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Intracranial Arterial
ANEURYSMS

BY

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PREFACE

Intracranial arterial aneurysms, always considered rare and almost impossible both of diagnosis and of treatment, are now added to the lengthening line of lesions that are curable by surgery. This is another example of the results that can be attained from intensive cultivation of a seemingly barren field. The accumulating pathological studies first called attention to the existence of the lesion. Many years passed without apparent diagnostic or therapeutic possibilities; gradually an increasing number of cases in the hands of neurologists suggested potential diagnostic criteria, and actual diagnoses were, after a time, occasionally made and confirmed at necropsy. In course of time, as neurosurgery became concentrated in the hands of a few, lesions were now and then found at operation, at first only to be regarded as curiosities, but eventually with such increasing frequency that the problem demanded solution. Largely through trial and error, methods of attack have unfolded, and the forthcoming pages present a group of twenty cures. With increasing confidence in diagnosis and with the correlated surgical follow-through, they are now known to be quite common, and many at least to be amenable to cure with a surprisingly low mortality.

W. E. D.

Baltimore

CONTENTS

I

INTRODUCTION

Surgery's Recent Aids to Diagnosis

Arterial aneurysms of the brain were probably first recognized at necropsy by Morgagni (1761). Brinton (1851) collected 40 pathologically verified intracranial aneurysms; Gull (1859), 51; Lebert (1866), 86; and Beadles (1907), 555. In 1939 McDonald and Korb found in the literature and analyzed 1125 lesions of this type.

Virchow (1851) first described the common small aneurysms, to which the well-known designation "miliary" was subsequently applied by Charcot; the more recent term, "berry aneurysm," used by Richardson and Hyland (1941), is more descriptive of their usual size and shape.

Although Lebert (1866) stated that a positive diagnosis of aneurysms could be made with approximate certainty in a group of cases, Hutchinson was probably the first to diagnose an intracranial aneurysm during life and to substantiate it by necropsy. His report on the postmortem findings appeared in 1875, eleven years after the diagnosis had been made. Bartholow (1872), a physician in Cincinnati, after studying a group of postmortem specimens, made an appeal for the recognition of this lesion during life, but he was much too far in advance of his time.

The diagnosis of aneurysms during life received its greatest stimulus from Symonds' publication (1923). He made a positive diagnosis of intracranial aneurysm in five cases; in three of them the lesion was subsequently found at necropsy; the other two were not verified. Several excellent publications have since appeared—Strauss, Globus, and Ginsburg (1932), Sands (1938), Evans and Courville (1939), Irish (1940), Walsh and King (1942), and Richardson and Hyland (1941). The two last named have made a most comprehensive clinical study. But for a lesion seemingly so hopeless in a therapeutic role, these enthusiastic efforts brought scant results. The solution of the diagnostic problem awaited the operative disclosure of the suspected

lesions and efforts directed toward their cure; and this has been forthcoming in the past decade. It is true that occasional presumed aneurysms of the carotid artery have had carotid ligations for over half a century, but they were hit-or-miss performances and advanced the field but little. With the recent intensive cultivation of this operative field, the actual disclosures of intracranial aneurysms have now become quite common, and these in turn have stimulated renewed interest in the diagnostic efforts. From a seemingly barren field, aneurysms in a steadily increasing number have been permanently cured. Moreover, the operative risk entailed is surprisingly small. In addition, a very important group of hitherto unrecognized aneurysms of the intracranial carotid before it branches has been disclosed and offers one of the most favorable types for surgical treatment.

The present report is based on a study of 108 patients with 133 aneurysms which have been verified by either necropsy or operation or both. Multiple aneurysms, therefore, occur in approximately 15 per cent of these cases. Since several aneurysms (as many as five) have been found in one patient, the percentage would be reduced if expressed in terms of patients, but since those cases which have been operated upon would surely have revealed additional aneurysms if pathological examinations had been possible, the actual percentage of multiple aneurysms would probably run about the same, that is, 15 per cent.

Where more than one vessel has been the seat of aneurysms, the case has been entered under each branch of the circle of Willis that is involved. This has been done in order to arrive at an estimate of the relative frequency of aneurysms on each branch. This raises the number of cases to 120; sixteen cases, therefore, have aneurysms on more than one vascular trunk, and there are 13 additional aneurysms on these trunks. The multiple aneurysms on the same trunk are confined

Distribution of Intracranial Aneurysms (Figure A)

	Vessels Involved	*Number of Aneurysms*	*Per cent of Aneurysms*
A. Internal carotid in carotid canal	11	12	9. %
B. Internal carotid intracranially	39	47	35.4 "
C. Anterior cerebral and anterior communicating	25	25	18.8 "
D. Middle cerebral	22	26	19.5 "
E. Posterior communicating	—	—	——
F. Posterior cerebral	2	2	1.5 "
G. Basilar and vertebral	21	21	15.8 "
Total	120	133	100 "

to the internal carotid and the middle cerebral. If all the operative cases had had complete examinations of the brain, such as necropsy

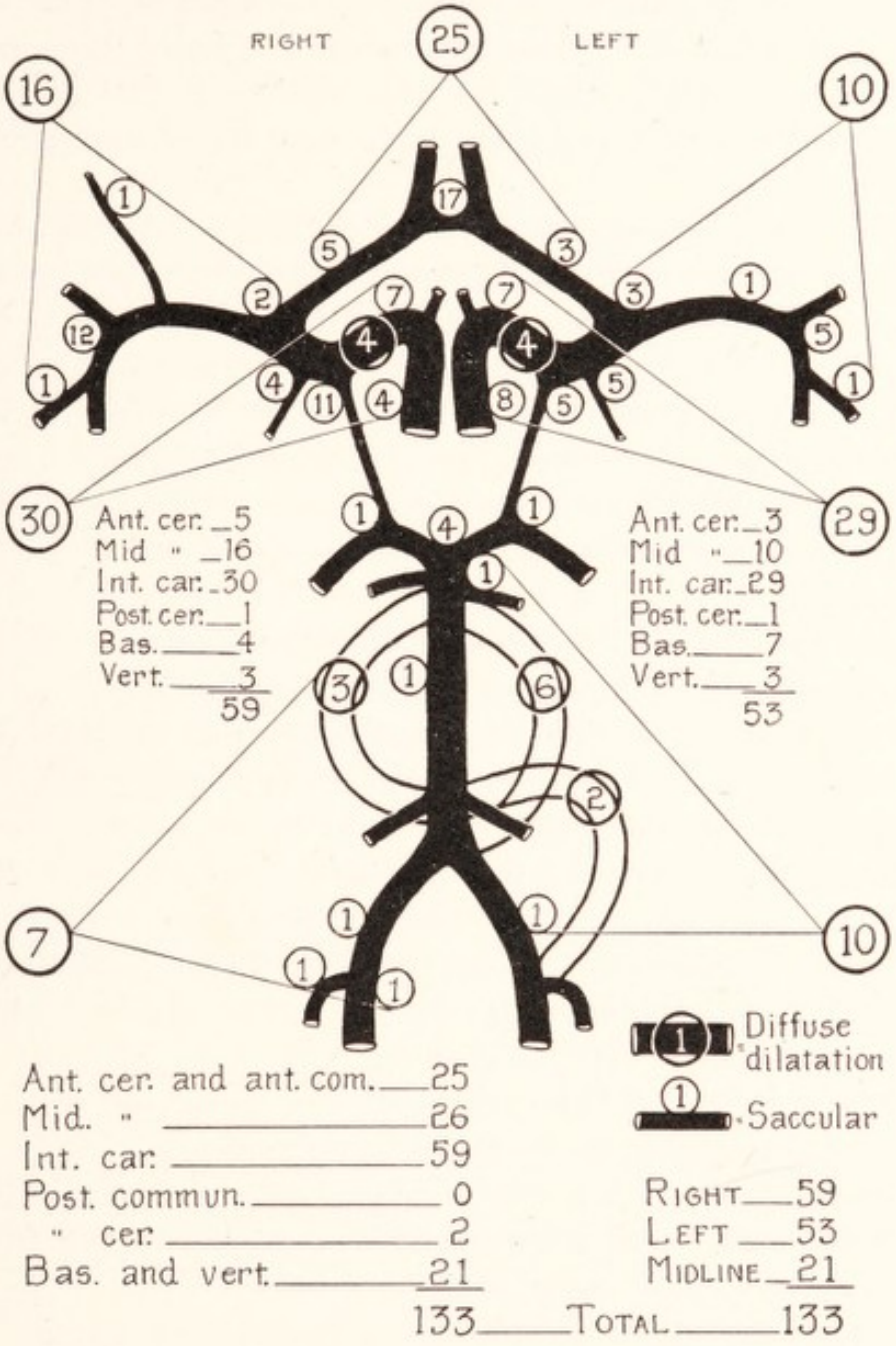


FIG. A.—Distribution as to the arteries involved in the 133 aneurysms of this series. There were 108 patients, but in 16 of these the aneurysms were multiple. The total number of aneurysms for each main area is shown in each of the large circles near the outer borders.

affords, the number of multiple aneurysms would be considerably increased.

From this series, therefore, it appears that aneurysms of the internal carotid are much more frequent than those of any other trunk,

and if those in the carotid canal are added to those on the intracranial portion, they are about twice as common (44.4 per cent) as those of the anterior cerebral, middle cerebral, or basilar (including the vertebral), on all of which aneurysms occur with essentially the same frequency. This has an important bearing on the surgical treatment, because carotid aneurysms offer much the most hopeful prospects of a surgical cure.

Statistical studies of this nature from the literature show marked differences. However, that of McDonald and Korb (1939) is much the most valuable, because most of the 1125 cases reported could be included. Their figures, in 1023 cases differ from those in this report. Their total on the internal carotid artery was 19 per cent, mine 44.0 per cent; theirs on the anterior cerebral and anterior communicating was 23 per cent, mine 19 per cent; on the middle cerebral 29 per cent, mine 20 per cent; on the basilar and vertebral arteries 21 per cent; mine 16 per cent.

Gull (1859) found 40 per cent on the basilar and vertebral arteries, and 24 per cent on the middle cerebral arteries. Richardson and Hyland (1941) reported 53 necropsies of intracranial aneurysms with a fairly even distribution on the major trunks; the highest incidence (30 per cent) was on the middle cerebral; for the anterior cerebral and communicating arteries it was 25 per cent, and for the internal carotid, 25 per cent. Berger (1923) reported 25 per cent of 21 aneurysms from the basilar and vertebral arteries. Lebert (1866), in 53 cases, reported the middle cerebral artery to be involved in much the highest proportion (40 per cent).

The present series is strikingly defective in aneurysms of the posterior communicating arteries, there being none. In nearly every other series from 2 to 10 per cent of the aneurysms are from this vessel (McDonald and Korb, 4 per cent). The explanation of this difference is, I think, quite clear. Most of these aneurysms arise at or very near the junction of these two trunks and they usually lie on the posterior communicating artery, frequently concealing it and giving the superficial impression that the sac arises from it, but if the orifice is carefully examined it will usually be found to enter the carotid.

The higher incidence of basilar and vertebral aneurysms in some of the other reports should also be noted (Gull, 40 per cent; Berger, 25 per cent). When it is considered that 11 of our 21 cases were disclosed during operations for trigeminal neuralgia and Meniere's

disease, the relative scarcity in this series is even more striking; from necropsy examinations alone there would have been only nine aneurysms of these arteries in this series.

Incidence (Sex, Age, and Race)

Intracranial aneurysms in this series are slightly more common in females—61 females and 47 males. The comprehensive statistical report of McDonald and Korb (1939) gives essentially the same relative frequency—574 females, 519 males.

The incidence of these aneurysms according to age is also strikingly similar to that reported by McDonald and Korb. Only one patient in our series was under 10 (age 8) at the time of operation or necropsy. Seven cases occurred in the second decade, and 15 in the third. In the next three decades the numbers were almost the same i.e., 21, 21, and 27. Between the ages of 60 and 70 there were 12 cases, and 7 cases after the age of 70.

In all reports from the literature there are only a few cases under 10, and not so many under 20, but thereafter they are of fairly uniform frequency in succeeding decades until the age of 70, when they decline, although they still remain as frequent as between 10 and 30. The oldest case in this series was 81.

The relatively higher incidence of aneurysms, on the basis of population, in the Negro race is shown in the tables.

Types (Mycotic, Arteriosclerotic, Congenital)

There are three types of intracranial aneurysms—(1) mycotic, (2) arteriosclerotic, and (3) congenital. Perhaps a fourth or syphilitic group should be included, but in none of our cases could this diagnosis be substantiated, although there were several positive Wassermanns. At any rate it is an exceedingly uncommon cause, despite the well known views to the contrary.

(1) *Mycotic*

There are six (4.5 per cent) mycotic aneurysms, five on the middle cerebral artery; and one of these had a second aneurysm of similar

type on the posterior cerebral artery. In every patient, therefore, with a mycotic aneurysm it was on the middle cerebral artery, which would appear to be the most common site for its development. All had ruptured. At times there is a purulent exudate about the ruptured vessel. Nearly always there is a long history of vegetative endocarditis, which is the primary source of the cerebral affection, the cerebral involvement being terminal.

(2) *Arteriosclerotic*

From the postmortem studies arteriosclerosis has been assumed to be the cause in 22 instances (16.2 per cent). At times some doubt exists as to the actual cause of an aneurysm, and an arteriosclerotic basis has been regarded as probable if the contiguous artery is markedly thickened from a similar process. All of these aneurysms are located on the basilar, vertebral, and internal carotid arteries, that is, upon the larger arterial trunks, which bear the major effects of the arteriosclerotic process. Most of the basilar aneurysms have been found at operations in the posterior fossa for trigeminal neuralgia, and the carotid aneurysms at operations for loss of vision when the internal carotids have been involved. The youngest patient in this series with an arteriosclerotic aneurysm was 52; another was 55. Arteriosclerosis is regarded by some writers as a much more common cause of intracranial aneurysms than our studies lead us to believe. From the 1125 cases collected by McDonald and Korb, one-half were presumed to be due to arteriosclerosis, but this analysis is necessarily colored by the impressions of those who reported the cases. Strauss, Globus, and Ginsburg consider it the most common cause. In 13 of the arteriosclerotic aneurysms in which the blood pressure is recorded in our series, it was normal in three patients, all of whom were in the fifties. The blood pressure was 160 in ten patients over the age of 60; 170 in two; 190 in two; and over 200 in five.

(3) *Congenital*

That most of the intracranial aneurysms are of congenital origin is now generally accepted. They could not, as Eppinger (1887) first suggested, have any other derivation. Most of them are in young or relatively young individuals whose arteries show no signs of arteriosclerosis, and their saccular formation with a narrow neck would preclude an arteriosclerotic process.

How many, if any, aneurysms are actually present at birth is not

known. I know of none that have been reported. The youngest case in the literature was reported by Dial and Maurer; the child was two years old at the time of rupture (and death) of a vertebral aneurysm. One of our cases (Table A) had a ruptured aneurysm at the age of two; it subsequently calcified but persisted and was operated at the age of 24. In Hermann and Macgregor's (1940) case the patient was 4½ years old at the time of rupture and death; two aneurysms were present. Church in 1870 collected 13 cases under 20 years of age. The importance of this negative evidence is reduced by the fact that no large series of newborn babies has been subjected to such a study, and most of the reported aneurysms have been found because death was due to their rupture in later years. Although the walls of all aneurysms are clearly defective, they are usually strong enough to hold for many years, death resulting when the weakness has reached the limit of endurance; at times they outlast a long life. Whether the aneurysms are potential or actual at birth cannot, therefore, be stated, but at least there must be a primary vascular defect at that time. My guess would be that they are probably tiny protrusions at birth and that they slowly increase with age. The walls of the aneurysms have been studied histologically by several observers. Turnbull (1918), Forbus (1930), Voncken (1931), and Nattros (1933) described defects in the media, the muscle being absent or markedly defective, and Tuthill (1933) found defects of the elastic tissue. That there must be microscopic defects in the walls at the site of the aneurysm is, of course, obvious. And that these defects must go back to embryonic life is equally clear. Forbus concluded from his studies that the walls are always defective at *the point of bifurcation* of an artery, i.e., at one of its branches, and that an aneurysm of congenital type "*cannot develop elsewhere.*" Several writers on this subject have accepted his view. This conclusion is at once shown to be incorrect by the fact that many of the aneurysms of the internal carotid within the cranial chamber arise where there are no branches. Diagram B in Figure 28 shows the location of a series of aneurysms, in none of which is there an arterial branch at the site of the aneurysm. Moreover, most of the aneurysms upon which his conclusions are based are included in this series, and there is no gross evidence that even they arise at a bifurcation. In most of the cases the aneurysm covers sufficient territory to include one or more branches, but an assumption that they arise at the branch would be perfectly gratuitous, though this does not mean that aneurysms may not and do not arise at a branch.

It seems to me that there can be but one explanation of aneurysms of the congenital type. It is known that the arterial trunks are derived from a capillary network which is gradually reduced, and that little "nubbins" remain on the arterial trunks where the smaller vessels are absorbed. It is probable that from these "nubbins" the little miliary (berry) aneurysms develop: and defective walls are to be expected in vestigial structures. The studies by Miss Hager also lead to the belief that aneurysms are a part in the congenital maldevelopment of the vascular system of the brain, especially involving the circle of Willis. The explanation of aneurysms on the intracranial division of the internal carotid artery—which in the adult is without branches between the ophthalmic and posterior communicating arteries—is probably that they are early embryologic branches that subsequently atrophy and are normally lost. The development of an aneurysmal sac with a narrow neck would appear to be the result of an incomplete disappearance of one of these vessels. At times the continuity of even the normal trunks is broken by this changing and absorbing process in the capillary bed. An excellent example is seen in two aneurysms of the internal carotid artery (Jacques, 1926; Slany, 1938) and two near the termination of the basilar artery (Slany, 1938; McDonald and Korb, 1939) all occurring where the posterior communicating artery should have been present but was absent. Certainly all aneurysms now recognized as congenital have their origin in defects incident to the resolution of the early vascular bed.

Ruptured and Unruptured Aneurysms

Ruptured aneurysms were present in 64, and unruptured in 44 cases. If the number of aneurysms instead of cases is considered, the ruptured number 64 and the unruptured 69—relative percentages of 48 and 52. Over half of the patients with ruptured aneurysms died within 48 hours, and half of these within 24 hours. The immediate result of each rupture depends largely, of course, upon the size of the arterial perforation and, to a considerable extent, upon whether or not tissues are in apposition with the artery at the point of rupture. If the hemorrhage passes directly into the subarachnoid space, the rupture will usually cause death. If the hemorrhage passes into the cerebral substance, the adjacent tissues may stop the hemorrhage and

prevent immediate mortality; a false sac will then be superimposed upon that of the aneurysm and, gradually expanding, behave like a space-occupying lesion, such as a brain tumor. Such patients may live for months or even years. Several of the aneurysms of the internal carotid artery have ruptured alongside the artery itself or onto the third nerve and the adjacent floor of the skull, and adherence to these structures has closed the opening and prevented immediate death. Finally, as in two of the cases, the internal carotid artery may rupture into the cavernous sinus and produce an arteriovenous aneurysm (pulsating exophthalmos). Life is then spared, but an unsightly and devastating deformity results—one, however, that can now be cured with little risk.

Anatomical evidence of spontaneous cure of an aneurysm by complete filling of the sac with a thrombus has been observed only once (Case XIX, Table B, Fig. 15) in the pathological material at our disposal. In this patient the aneurysm of the internal carotid was known to have ruptured four years earlier because paresis of the third nerve had occurred and persisted. Death resulted from rupture of a cortical (congenital) arteriovenous aneurysm in the occipital lobe of the opposite side. The aneurysm of the carotid, as large as a hazel nut, was found at necropsy to be entirely filled with thrombus; but it was still adherent to the third nerve. This is by no means even an approximate estimate of the number of cases that are spontaneously cured, or at least remain symptomatically well for many years. It would hardly be expected from material of this kind—where most of the patients have died as the result of aneurysms—that adequate statistical information concerning healed aneurysms would be obtainable. On the other hand, spontaneously and completely healed aneurysms have rarely been found in the general run of postmortem examinations. Several aneurysms in the literature and at least two in the present series have almost filled with thrombus, but have eventually ruptured from the small remaining lumen.

Evidence of cured aneurysms from the time elapsing after a subarachnoid hemorrhage is also none too certain. Many cases of cerebral hemorrhage with blood in the cerebrospinal fluid are encountered in medical practice and recover, and doubtless many, perhaps most, but certainly not all, were due to intracranial aneurysms. Subsequent ruptures after apparent healing are exceedingly common and may occur ten years or more after the initial break. One must, therefore, look upon so-called cures with considerable skepticism. It

is impossible to do more than guess at the percentage of seemingly certain cerebral hemorrhages of this origin that are permanently cured (by thrombosis). My guess would be that it is not more than 15 or 20 per cent. The only known figures on the subject are those of Richardson and Hyland, who estimate a 48 per cent recovery. Of 118 cases of subarachnoid hemorrhage studied by them, 57 survived and 61 were fatal. Of the survivors 37 were known to be living from one to ten years later, but in some of these cases, of course, the underlying cause of the hemorrhage may not have been aneurysms. Moreover, several of our patients have survived longer than this and the aneurysms have subsequently ruptured.

Repeated Hemorrhages

Then, too, the great frequency of repeated intracranial hemorrhages from aneurysms must be emphasized. For example, 39 of the 108 cases in this series (nearly 40 per cent) are known to have had one or more hemorrhages a few days to a few weeks, or even months or years, after the original bleeding. One patient had four hemorrhages in two months and survived with a large subdural hematoma. In Richardson and Hyland's report, five cases had recurrence of subarachnoid hemorrhage, and in two of these the patient died. This high mortality from subarachnoid hemorrhage has an important bearing on the possibility of better results by surgical treatment of certain aneurysms when instituted promptly. Recently I saw a patient in coma who had had a subarachnoid hemorrhage eleven years ago and made a complete recovery. She then had another hemorrhage, with little clinical effect, and again seemed to be well; two weeks later she suddenly became comatose and died; the aneurysm was curable.

Symptoms and Signs

The cases in this series have been reviewed with the primary purpose of evolving clinical data that might improve the diagnosis and localization in subsequent cases. This hope has met with only limited success. The suspicion of an aneurysm from the sudden onset of intracranial symptoms of any kind is perhaps the most important part in the diagnosis, but all too frequently sudden coma is the first evidence of the existence of an aneurysm.

Extraocular Palsies. Perhaps the most important evidence of an

aneurysm is the sudden, repeated, sharp pains in the eye, or in the frontal or temporal region, frequently followed by ptosis of the upper eyelid and extraocular palsies on the same side. These manifestations are almost pathognomonic of carotid or nearby aneurysms, and fortunately they are the most favorable ones for surgical treatment.

In 31 patients (30 per cent) a third-nerve palsy was present; this effect has been produced by aneurysms in every arterial division, but 27 were related to aneurysms of the internal carotid. Moreover, in all cases of aneurysms of this vessel in the carotid canal and intracranially, before the first branch is given off, an oculomotor palsy was present. Although not pathognomonic, it is therefore a sign of the greatest significance in localizing the aneurysm to this vessel. In eleven cases paralysis of the abducens nerve (N. VI), and in five paralysis of the trochlear nerve (N. IV) was present; in six of these the third, fourth, and sixth nerves were affected. Paralysis of two or all three of these nerves is very suggestive, though not absolutely certain evidence that the aneurysm is in the carotid canal. Bilateral abducens paralysis was present in only one case—a basilar aneurysm.

Denervation phenomena of third nerve referable to an intracranial aneurysm. Dr. Frank Walsh, who has been very much interested in the ocular studies of these intracranial aneurysms and has seen most of them, is responsible for noting two very interesting and bizarre phenomena referable to degeneration of the third nerve and associated especially with aneurysms, although tumors producing paralysis of the third nerve may also cause them.

A patient who for many years had had a total third-nerve paralysis from an aneurysm had noticed that during periods of fright or loss of temper, or when he was interested in the lures of the opposite sex, the affected lid would surge widely open. Bathing in cold water had the same effect. Also, when a solution of mecholyl chloride was dropped into the conjunctival sac of each eye, the wide, inert, paralyzed pupil contracted promptly, whereas that of the normal side was not affected. These phenomena are examples of Cannon's law of degeneration. They have been duplicated in animals by Bender—after section of the third nerve—by inducing anger or fright and also by injecting acetylcholine hydrochloride protected by physostigmine salicylate.

Anomalous movements of the eyeball and upper lid are also observed at times when the functions of the third nerve are in the process of regeneration. These movements are probably due, as Ford and

Woodhall have shown, to misdirection of the returning nerves. They may consist of a strong elevation of the upper lid and a strong inward movement of the eyeball and contraction of the pupil. These findings have been observed by Walsh in two cases from this series. Walsh has also called attention to a regeneration phenomenon that is at times manifest in an Argyll-Robertson pupil. There may be (1) wide dilatation of the pupil and inactivity both to light and on accommodation convergence, or (2) loss of reaction to light but contraction at any time whether the movement be convergence or conjugate movement, or (3) the pupil—usually larger than its fellow but occasionally small—reacts sluggishly to light. The interesting feature of the Argyll-Robertson type of pupil as a result of third-nerve regeneration is, according to Walsh and Ford, that apparently somatic fibers can form effective synapses with postganglionic autonomic fibers; and that when these phenomena are present (after cure of an aneurysm) they persist indefinitely.

Motor signs. Hemiplegia partial or complete can occur with the rupture of aneurysms located intracranially anywhere anterior to the tentorium, but it is far more common with aneurysms of the middle cerebral. It can occur directly from aneurysms within the carotid canal only when the aneurysm has broken through the dural covering of the cavernous sinus and the hemorrhage has spread up the Sylvian fissure. Partial or complete hemiplegia occurred in 14 cases—eight middle cerebral, three anterior cerebral, and three internal carotid. If the hemiplegia is complete and does not improve quickly, the site of the aneurysm is most likely the middle cerebral; if partial and transient it will probably be from the other vessels, the motor area then being compressed by the hematoma and resulting oedema. It is noteworthy that a hemiparesis or hemiplegia can occur from rupture of an aneurysm of the internal carotid into the Sylvian fissure, the hematoma being in sufficient amount to compress the motor area. Although a hemiplegia strongly suggests a middle cerebral hemorrhage, it is not sufficiently positive to preclude the diagnosis of the more favorable types of aneurysms. A monoplegia also occurs at times and is fair evidence that the lesion is on the anterior cerebral artery, because the intracranial hemorrhage can pick out a selected portion rather than the entire motor area. Bilateral motor loss, when due to an aneurysm, indicates the site of the aneurysm to be the basilar or vertebral arteries. Hemiplegia and hemiparesis also result from direct compression of the motor area by expanding aneurysms; the

paralysis is then steadily progressive. Finally, a sudden hemiplegia may result from an embolus given off by an aneurysm of the carotid or middle cerebral, or by direct extension of a thrombus from these sites.

A positive Babinski is one of the most frequent signs associated with intracranial aneurysms, but does not indicate its position. It is almost as frequently bilateral as unilateral, and when bilateral its localizing value is lost. When unilateral or predominantly so it is an important sign of localization to the side of the aneurysm. Very frequently the Babinski is positive when there is no evidence of motor weakness.

Rigidity of the neck is nearly always present in the acute stage of hemorrhage and is due to the collection of blood in the posterior cranial fossa, where the subarachnoid hemorrhage must necessarily collect in the cisterna magna. When the neck is rigid after a sudden episode referable to the brain it is almost as certain evidence of bleeding as the findings by lumbar puncture. It has no significance whatever in the localization of the aneurysm.

A positive Kernig is commonly present during and for several days after an acute hemorrhage. It is nearly always bilateral and is due to the collection of blood in the posterior cranial fossa; it has no localizing value.

Convulsions were noted in 22 cases (20 per cent); in three, however, the convulsions were not related to the aneurysm but were due to other congenital malformations. It is important, therefore, in evaluating convulsions, to know, if possible, whether they are related or concomitant. Convulsions caused by aneurysms mean cerebral damage which may be due to previous rupture of the aneurysm or to its actual size and position in the brain. Only aneurysms in the cerebral hemispheres cause clonic convulsions: those in the posterior cranial fossa produce tonic attacks. Convulsions may be focal or general, depending upon whether or not the motor area of the brain is directly involved. Focal convulsions are more common with anterior cerebral (3) and middle cerebral (2) aneurysms because the hemorrhage is, or has been, in the neighborhood of the motor area. Clonic convulsions do not result from hemorrhage in the subarachnoid space, per se, but can follow when the brain has been damaged by the progress of the hemorrhage up the Sylvian fissure. Attacks of petit mal may occur alone for years before the onset of generalized convulsions. They indicate damage to a silent area of the brain. The analysis of convulsions, therefore, may be of some assistance in the localization of

the aneurysm. However, the seizures are precisely like those from any other lesion of the brain and do not in any way indicate the diagnosis of an aneurysm. Merritt (1930) concluded that convulsions indicated an intracerebral rather than extracerebral aneurysm. This is true, of course, if the aneurysm itself is causing the attacks. But clinically one cannot determine whether the seizures are due to the aneurysm or to prior effects of a rupture. Convulsions occurred in 14 per cent of his cases, and in 9 per cent of those reported by Richardson and Hyland.

Migraine. The relationship of consistently unilateral migraine to aneurysms of the internal carotid artery has been stressed by Goldflam (1923), who reported five instances in eleven cases of intracranial aneurysms, and by Adie (1930), who had seven additional cases. In none of these cases, however, was there a postmortem examination, and consequently the actual proof of this relationship was missing. Adie says that one of his cases was proven to be a congenital aneurysm at necropsy, but his publication does not disclose it. Symonds (1924) stated that one of his cases with proven subarachnoid hemorrhage had migraine, but here again there was no autopsy. Adie also commented upon the development of permanent hemianopsia with the migrainous attacks. Richardson and Hyland's series contained four cases of migraine. Dassen (1931) presented a postmortem specimen of an aneurysm in the posterior part of the circle of Willis; he stressed its relationship to migraine. Only one of our cases (Case V, Table B) had a clear-cut migraine of five years' duration, beginning at the age of 42. It was the only symptom until rupture of the carotid aneurysm on the same side. The aneurysm must have been the cause of the migrainous attacks and was probably due to involvement of the sympathetic fibers in the arterial wall. Another aneurysm in our series of exactly this same type did not cause true migraine, but gave periodic unilateral headache, and, curiously, none of the arteriosclerotic aneurysms involving the entire carotid trunk caused migrainous attacks. Even though the actual percentage of aneurysms with migraine is small, it is certainly a symptom that should make one think of an aneurysm of the carotid as a possibility, and perhaps even more so when heminanopsia is a sequel. That migraine is not necessarily related only to aneurysms of the internal carotid is suggested by Dassen's case, in which the aneurysm was posterior to the carotid (on the posterior communicating artery).

Headache and pain. Always there is severe pain in the head at the

time of rupture of the aneurysm; this is terrific when the rupture is large. The fact that the pain is so sudden must mean that the actual rupture of the vessel (probably due to injury to the sympathetic fibers) is responsible rather than the intracranial pressure. This sudden pain is to be distinguished from the headache which follows and persists for some time when consciousness returns and which is due to intracranial pressure from the bleeding. The most important pains from a localizing standpoint occur with carotid aneurysms when the rupture is small, or when sudden expansions of the aneurysmal wall occur in stages. This pain is in the corresponding eye and the contiguous frontal region and side of the face, and has occurred in a high percentage of the sacculated aneurysms of the internal carotid. Such recurring pains, especially when severe, are very strongly suggestive of an aneurysm and almost pathognomonic of its location on the internal carotid within the cranial chamber, or at times in the carotid canal.

Headache, as distinguished from pain, is, of course, always present after a rupture, and, when the hemorrhage is into the subarachnoid space, it is usually most pronounced in the suboccipital region. At times the pains and aches extend down the back even as far as the lumbar region. Headaches following rupture into the cerebral hemispheres may be localized to one side of the head, and—regardless of the location of the hemorrhage—may be frontal, temporal or occipital; in this general way they are important in indicating the side of the aneurysm, but not its exact location. However, headache may, as in brain tumors, be quite misleading in this respect. In one case the headache was on the contralateral side, and for no obvious reason that could be disclosed by necropsy.

Papilloedema and hemorrhage. Papilloedema must be regarded as an indication of intracranial pressure. Hemorrhage into the retina results from bleeding along the subarachnoid sheath of the optic nerve. When papilloedema and hemorrhage co-exist, intracranial pressure is the cause of both.

One type of retinal hemorrhage, the large, round, so-called subhyaloid hemorrhage, either single or multiple, and occurring without papilloedema, is thought to be pathognomonic of subarachnoid bleeding and is on the side of the aneurysm. In three of our cases the clinical diagnosis was made by this sign. In one case the hemorrhage was found less than ten minutes after an ophthalmoscopic examination had been negative; and in another case a tremendous hemorrhage was

disclosed an hour after a most careful observer had found nothing.

When papilloedema is present, with or without hemorrhage, intracranial pressure is the cause. This is due either to the size of the aneurysm or the size of the hemorrhage. It usually indicates a chronic pressure and is rarely evident in the acute hemorrhages until 48 hours later, and frequently not at all. Hemorrhages without papilloedema occurred in five cases. Papilloedema was present in 12 cases (10 per cent), and only occasionally without concomitant hemorrhage. The subject of papilloedema and retinal hemorrhages has been studied by Riddoch and Goulden (1925), Wagener and Foster (1935), Griffith, Jeffers, and Fry (1938), and Richardson and Hyland (1941).

Roentgenography

Calcified shadows. In six cases (6 per cent) there were calcified shadows from which the clinical diagnosis of an aneurysm was almost certain, either because of the shape of the calcification or because of its position. The percentage of positive calcifications in this series is abnormally low if all cases are included; however, only about one-third of the patients had x-rays of the head. A fair estimate of calcified shadows, if all cases were studied by roentgenography, would probably be about 15 per cent. Calcifications, of course, mean depositions in defective walls, which are almost necessarily present in aneurysms, and they also indicate the long duration of these defects. They occur in small as well as large aneurysms. They may be small circular shadows (Case XIII, Table C); irregular masses (Case IV, Table B); diffuse scattered areas above the sella or in the carotid canal on one or both sides (Cases II and IV, Table B), or linear shadows, usually curved (Case I, Table A). The most striking shadows (Fig. 1) were in Case I, Table A—huge bilateral aneurysms of the internal carotid artery arising in the carotid canal and bulging far upward into the brain on both sides; some of these curved linear shadows were three or four centimeters in length. Their long, linear, and curved character makes a vascular shadow almost certain. This case, reported by Heuer and myself in 1916, was probably the first instance of x-ray shadows of an aneurysm.

Because of their position the shadows of arteriosclerotic aneurysms of the internal carotid arteries are almost but not quite pathognomonic. In Case IV, Table B, there were numerous shadows, almost

confluent, extending directly upward into the cranial chamber from both anterior clinoid processes, and confined to a rather narrow, almost tubular course. They could only mean calcification of the carotid artery. In Case II, Table B, similar findings were on one side only, but there was also a tubular horizontal shadow in the corresponding carotid canal.

In Case XIII, Table C, a small, dense, round shadow was checked

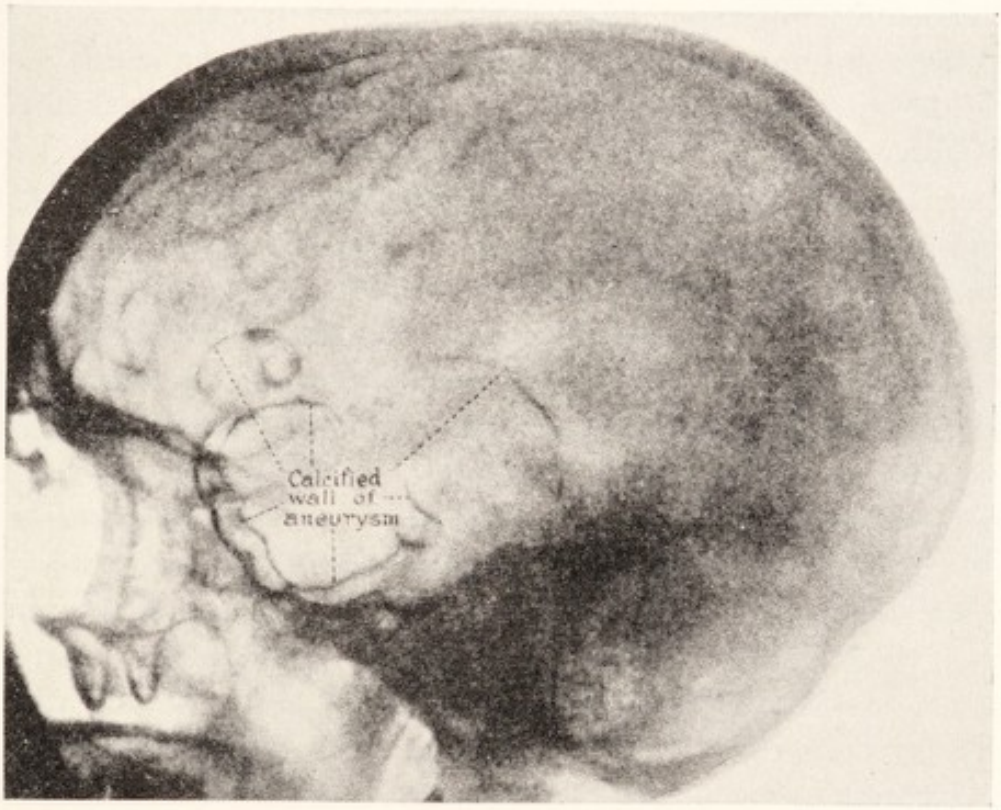


FIG. 1.—Case 1, Table A. Extensive calcification in bilateral aneurysms of the carotid artery at the base of the brain. Note the curved linear shadows of the calcified wall above the sella, which has been completely destroyed. The aneurysms arose in the carotid canals.

by operation to be in the tip of a small, dangling, club-shaped aneurysm of the anterior cerebral artery. Its significance caused some speculation, since this patient had epilepsy; however, the cause of the convulsions was found to be atrophy of the motor region; the aneurysm, therefore, was a coincidental finding and not related to the convulsions. In Case V, Table A, a small, dense shadow in the carotid canal was also found at operation. This aneurysm was known to have ruptured 22 years earlier, when the patient was two years old.

One aneurysm of the middle cerebral artery (Case VIII, Table D) showed a group of small, "fluffy" shadows in the Sylvian fissure and

a tumor was suspected. In none of the x-rays of the basilar and vertebral aneurysms was a calcification detected. However, in these aneurysms the roentgenographic view is not so clear, because of the interposed petrous processes and the mastoid bones. In this connection it is worthy of note that calcifications from tumors in the posterior fossa are seen only occasionally.

Schüller (1918) detected calcifications which were subsequently traced by necropsy and found to be in the walls of an aneurysm. Spiess and Pfeiffer (1924) reported a case diagnosed by calcified shadows in the x-ray. Zollinger and Cutler (1933) found curved linear shadows and destruction of the sella in a case that was presumed to be a hypophyseal tumor; an aneurysm was disclosed at operation and necropsy. Dyke (1936) reported calcifications in seven of eight intracranial aneurysms.

Sosman and Vogt (1926) made an excellent summary of the x-ray findings of aneurysms. Dyke noted an enlargement and destruction of the sella, erosion of the carotid canal, and enlargement of the carotid canal and sphenoidal fissure; these changes were due to a very large aneurysm. Sosman and Vogt and also Dyke emphasized the curved linear shadows which are probably the most emphatic evidence of aneurysms. Bozzoli (1937) found calcifications on both sides of the sella in bilateral aneurysms in the carotid canal. Recently Hamby (1942) included another bilateral aneurysm at this site, with large, curvilinear shadows resembling our first case.

Destructive changes in the skull. The only destructive changes that have been—or possibly can be—found in the x-rays of aneurysms are at or near the sella turcica, including the margins of the sphenoid fissure and the carotid canal. In four cases the sella was entirely destroyed and in a fifth partially destroyed. A sixth case with bilateral arteriosclerotic aneurysms of the internal carotid (Case VI, Table B) also had destruction of the sella, but from a concomitant hypophyseal tumor; there was no reason to suspect an aneurysm in this case and it had nothing to do with the shadow. There is, of course, nothing distinctive about the sellar destruction from an aneurysm; under the law of probability the destruction is more indicative of a tumor, but an aneurysm must always be considered. In one of our cases (Table A, Case I) there was extensive destruction of the floor of the middle fossa; this could be seen only in basal plates. McKinney, Acree, and Soltz (1936) found enlargement of the carotid canal in two instances.

Ventriculography

Ventriculography plays a minor role in the diagnosis and localization of intracranial aneurysms. It is only when the aneurysms give signs and symptoms suggesting a tumor that the localization with air injections is indicated. In two cases (IV and VI, Group C) the localization of a space-occupying lesion was made by a defect in the anterior part of the third ventricle. In one of these cases the aneurysm was found at operation; in the other case the hematoma from the ruptured aneurysm was evacuated, but the aneurysm was not actually seen. In another case (I, Table G) ventriculography localized the lesion—presumed to be a tumor—in the posterior cranial fossa, but operation exposed a large aneurysm of the left vertebral artery.

It is possible that aneurysms of the circle of Willis may at times be demonstrated by spinal injections of air. It is conceivable that small filling defects in the air shadows of the cisterna interpeduncularis and chiasmatis might be found to establish the diagnosis. I know of none that have been found by this method, and I would not be willing to use a spinal injection when an aneurysm was suspected.

Angiography

Since Moniz introduced arterial encephalography in 1927, it has assumed an ever-increasing though greatly overdone role in vascular lesions of the brain. His first aneurysm was diagnosed by this method in 1933. Since then many aneurysms have been graphically demonstrated, notably by Dott (1937), Jefferson (1938), Russel-Brain and Northfield (1936), Tönnis (1936), Sjöqvist (1938), Hill (1938), Fincher (1939), and Krayenbühl (1941). The opaque shadow may indicate the exact size of an aneurysm or only a fraction of it, the result depending upon the amount of thrombosis that obtains on its interior. Then, too, the amount of thorotrast that enters the cavity is dependent upon chance, due to the rapidity of the circulation.

Four aneurysms in this series have had carotid injections of thorotrast and all were done elsewhere, one by Dr. Francis Grant of Philadelphia, one by Dr. Arthur King of Baltimore, another by Dr. W. Gayle Crutchfield of the University of Virginia, and the fourth by

Dr. Irwin Levy of St. Louis, and in each the aneurysm showed beautifully (Fig. 2). There is no doubt whatever of the excellent demonstrations of aneurysms by this method; it is unquestionably the most

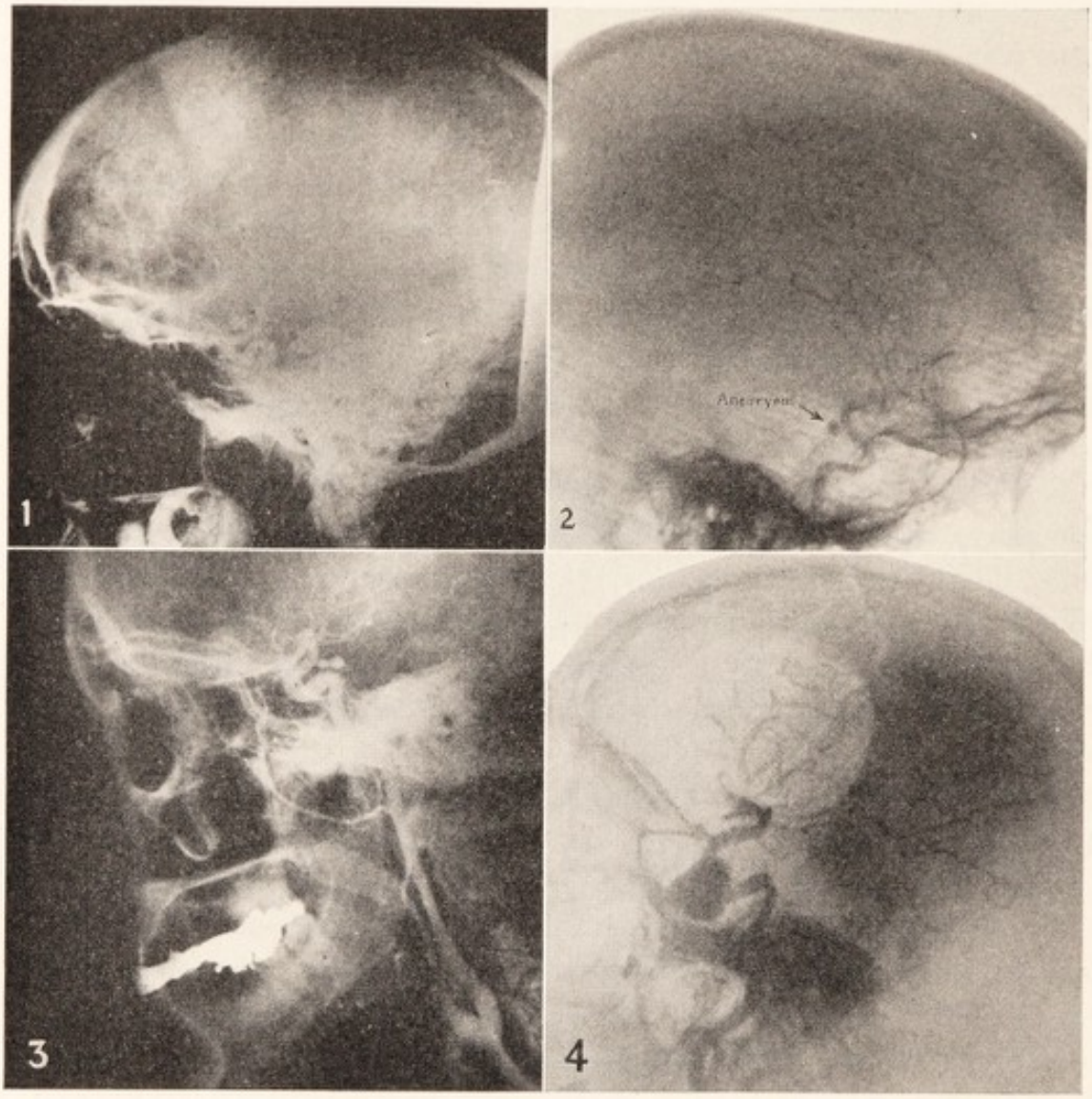


FIG. 2.—Angiograms of (1) internal carotid, made by Dr. W. Gayle Crutchfield of University, Va.; (2) aneurysm of the carotid, made by Dr. Francis Grant of Philadelphia, Pa.; (3) aneurysm of the carotid, made by Dr. Irwin Levy of St. Louis, Mo.; (4) aneurysm in the carotid canal (kindness of Dr. J. M. Sanchez-Pérez of Mexico City.

important, if not the only, function that this procedure serves. And yet I have been reluctant to use it because of the fear of cerebral thromboses. A very disturbing report by Ekström and Lindgren (1938) disclosed cerebral thromboses in 60 per cent of the brains coming to necropsy after intracarotid injections of thorotrast. This sequela of this method unquestionably occurs, though few reports

are available. On the other hand it has been used frequently with no apparent after effects. However, as a good general rule, I feel that the less done to patients the better. In none of the aneurysms causing a third-nerve palsy or paralysis is there need for this injection, the clinical diagnosis being almost assured without it. Moreover, this is the one big group that, thus far at least, offers help by surgery. Where localization of a suspected aneurysm cannot be established otherwise, and the patient's condition and symptoms warrant, it should perhaps be used, and doubtless cases will be found that can be treated by operative means. However, if it is possible to obtain any clinical data that will indicate the side of the circle of Willis that is involved, I should prefer to expose the circle of Willis on that side by operation. It is probably worth while to comment on the late effects of these injections of thorotrast at the site of the injection into the internal carotid artery. On two occasions I have had to expose the internal carotid at the site of the injection a few weeks subsequently, and in each instance the tissues surrounding the carotid were so densely matted that isolation of the vessel was impossible; fortunately it was possible to expose the artery higher in the neck for the ligation. One cannot escape the conviction that thorotrast is a highly irritating substance and one to be used only with a most important objective and as a last resort. And yet it is even injected by many, freely and in large amounts, into the cerebral ventricles and spinal canal.

All of the injections of thorotrast or diatrast have been into the internal carotid arteries and these will not fill the basilar and vertebral arteries which harbor an important group of aneurysms. The vertebral artery was recently injected by Takahashi (1940) and by King (1941) with a resulting excellent visualization of the vertebral and basilar arteries, but without the disclosure of an aneurysm. The posterior communicating and posterior cerebral arteries are also better demonstrated by this procedure than by carotid injections. In disclosing intracranial aneurysms by angiography, therefore, it may be necessary to inject both internal carotids and a vertebral artery before the sac can be demonstrated—a formidable series of injections to anticipate, and all might still be negative!

Important information is unquestionably obtained from angiography. The exact site of the aneurysm and frequently its size is known beforehand. Also, at times, the exact relationship to the posterior communicating and anterior cerebral arteries will be disclosed if these vessels should be filled with the injection material. Much of

this information, however, is usually obtainable when the aneurysm is isolated at operation, but in the larger ones the knowledge beforehand of the exact origin is helpful and may make dissection and possible rupture of the aneurysm unnecessary. I cannot therefore dismiss this method as entirely inadvisable. It is quite possible—if the freedom from accident is proved to be minimal—that I may yet come to use it.

Electroencephalography

Woodhall has just presented a paper (not yet published) at the Southern Medical Society indicating the value of electroencephalography in determining the side of the aneurysm after its rupture. In three verified cases the amplitude of the waves was reduced, the frequency was unstable, and the form was irregular on the side of the hemorrhage. He assumes the reduced blood supply to the side of the ruptured aneurysm to be responsible for the electroencephalographic change. Also, after ligation of the carotid, the postoperative changes in the electroencephalograms were much the same as those following rupture of the aneurysm. Asenjo (1938) demonstrated the same electrographic changes experimentally in cats after ligating the internal carotid.

II

ANEURYSMS BY LOCATION

A. Internal Carotid in the Carotid Canal (Table A)

In this group there are 11 patients with 12 aneurysms (9 per cent of the total). One was bilateral (Fig. 3). Five of the patients were females and six were males. With two exceptions (ages 52 and 53 respectively) they were less than 40 years of age; five were in the twenties and one had symptoms of rupture and nerve paralyses at the age of two.

From the literature 30 additional cases have been found, five being bilateral. The total number of cases, therefore, is 41, and the total number of aneurysms 47. The five additional bilateral aneurysms in the literature were reported by Blane (1800), Jefferson (1938), Di Poli and Zucha (1940), Bozzoli (1941) and Hamby (1942). When bilateral they may be symmetrical (Blane); nearly symmetrical (Bozzoli and Hamby) or markedly asymmetrical (Case I of this report and Jefferson's case).

Jefferson (1938), in an excellent paper, reported 17 cases, but only six of these (including two in one patient) were authenticated; the remaining cases were assumed to be aneurysms in the carotid canal because of signs and symptoms. But, important as are the subjective and objective disturbances, they are not pathognomonic either of aneurysms or of the exact location of the lesion, i.e., whether located in the carotid canal or intracranially. Two of his cases, diagnosed by angiograms, are clearly intracranial because they extend to the circle of Willis.

There are three methods by which the diagnosis of an aneurysm and its exact position can be established, namely, (1) necropsy, (2) operation, and (3) angiograms. A fourth method should probably be included, i.e., x-ray of wire inserted into the aneurysm (Werner, Blakemore, and King's case). There are occasional calcifications in the x-ray, but most of the reports leave a doubt whether the lesion

was a tumor or an aneurysm. It is possible that two of the calcified aneurysms reported by McKinney, Acree, and Soltz may be in the carotid canal, but, since these observers do not differentiate between the intracavernous and the intracranial subdivisions of the carotid, it is impossible to make the determination from their description; moreover, these writers place them in the cranial chamber.

The first description of the curved, linear, intracranial roentgeno-

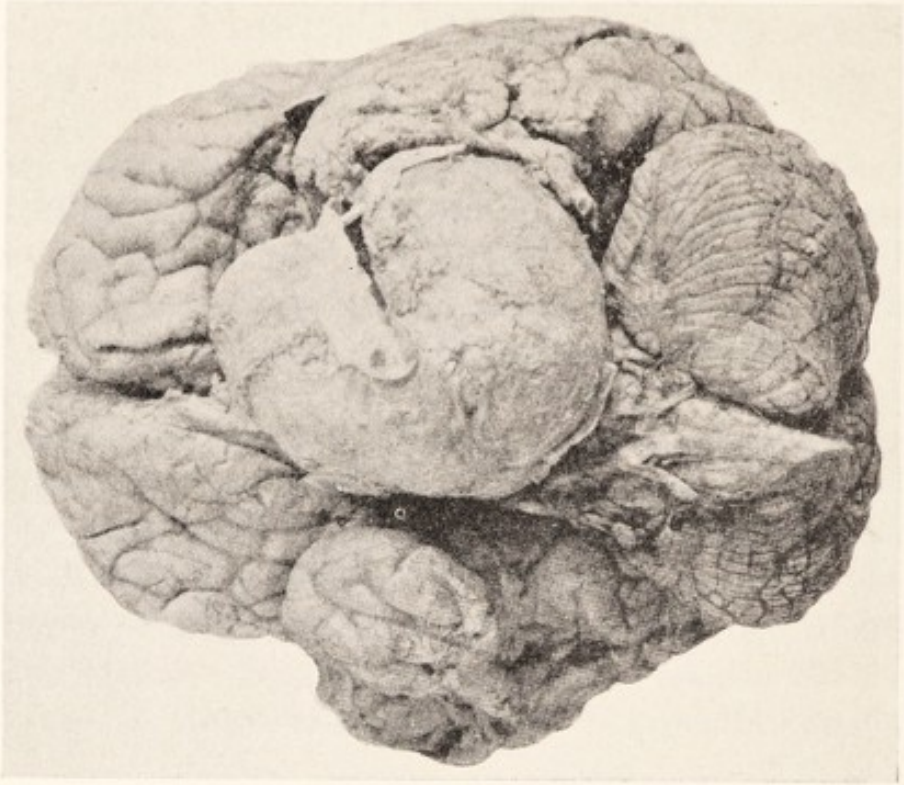


FIG. 3.—Bilateral aneurysms of the internal carotid arising in the carotid canal and projecting upward into the brain.

graphic shadows that are now known to be almost pathognomonic of the calcified walls of aneurysms was reported by Heuer and myself in 1916 (Case I, Fig. 3). Although neither of us was aware of their significance at the time, their import was suggested by Dr. H. M. Thomas, then our professor of neurology, and it is so recorded in the history. In addition, the posterior clinoids were totally destroyed and the floor of the sella was pushed far downward into the sphenoid sinus. Shadows of similar character have since been published by Sossman and Vogt (1926), McKinney, Acree, and Soltz (1936) and Hamby (1942). In Bozzoli's case there were bilateral shadows alongside the sella. In one other case (VI) in my series a small, dense, irregular calcification in the carotid canal could leave no doubt concerning

the diagnosis, especially when correlated with the history. From this series of cases, therefore, evidence by x-rays of aneurysms in the carotid canal occurred in 27 per cent of the cases.

Since the introduction of angiography by Moniz in 1933, intra-arterial injections of thorotrast have demonstrated many intracranial aneurysms. Beautiful examples of those in the carotid canal have been reported by Fincher (1938), Reichert (1939), Krayenbühl (1941), and Kosic (1941). A second aneurysm of Krayenbühl failed to show in an arteriogram and was disclosed at operation. The demonstratives of aneurysms of this type at operation have been made by Cushing (1918, reported by Viets), Magnus (1927), Jefferson (four cases, 1938), Hamby (1942), and myself (10 cases). There can be no uncertainty about the diagnosis at operation, for the aneurysms pulsate and blood can be aspirated in case of doubt. Magnus opened an aneurysm of this type when operating for trigeminal neuralgia, but the patient survived after the aneurysm was packed and the internal carotid artery ligated in the neck. Cushing exposed one through a decompression opening. In one of Jefferson's cases the aneurysm was found during a trigeminal operation and the patient died 14 years later. In his three other cases aneurysms were found when the clinically localized lesions were explored. All of our 10 operative cases were operated upon with the impression that the lesions were aneurysms and with surgical attack in mind.

Aneurysms in the carotid canal vary in size, from the so-called "berry" aneurysm to rounded protrusions the size of an orange (Fig. 5). Some are localized projections from the wall of the artery; others are dilatations of the whole arterial trunk. Six of the aneurysms in this series burst through the dura alongside the internal carotid artery as a tongue-like protrusion. Cases VIII and IX are examples of the enormous aneurysms. Both extended far upward into the brain, pushing the temporal lobe aside and shifting the entire brain to the contralateral side. The bilateral case (I) caused the same cerebral dislocation both upward and backward.

Signs and Symptoms

The outstanding symptom of onset is pain, sudden, severe, often excruciating, in the eye and frontal region of the corresponding side. After a few hours or days the pain usually subsides, but recurs. Pain was the symptom of onset in eight of our ten cases. This pain doubt-

less corresponds with and is due to the rupture—or, at least, sudden expansion—of the aneurysm. In two of these cases (II and V), coma (indicating cerebral hemorrhage) developed soon after the onset of pain; both recovered. It is also interesting to note that a third-nerve palsy appeared within a few hours after the onset of sudden pain in Cases III and V, thereby indicating also a sudden expansion or rupture of the sac. In Case II pains continued intermittently for three

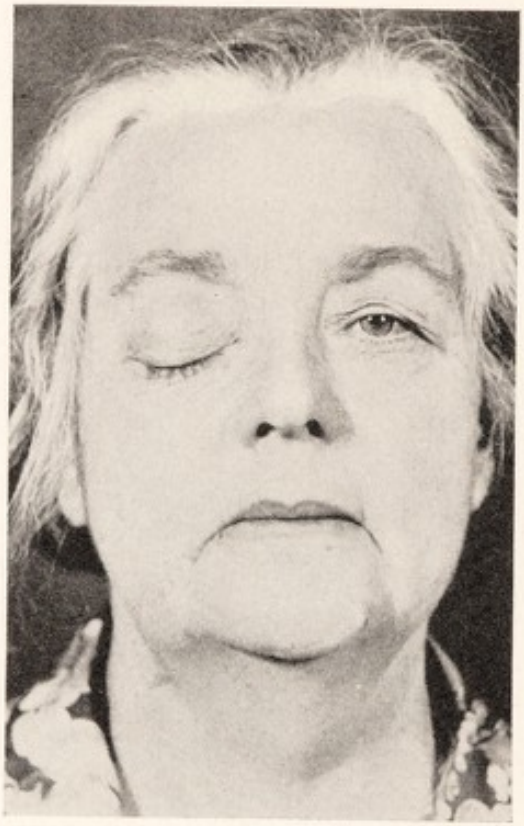


FIG. 4.—Showing unilateral ptosis resulting from third-nerve paralysis due to an aneurysm in the carotid canal.

months, and in Case VI for one month before ptosis and diplopia appeared. The other signs and symptoms of these aneurysms fall into three groups, namely, (1) those giving palsies of the extraocular muscles, (2) those giving trigeminal neuralgia and corresponding sensory loss, and (3) those in which Groups 1 and 2 are combined. By far the most important manifestation of aneurysm in this region is a palsy or paralysis of the third nerve (Fig. 4), and periodic, severe pain in the affected eye or frontal region. These disturbances are also present with intracranial aneurysms of the internal carotid or even of the posterior communicating artery. But when to this pain and paralysis is added involvement, whether subjective or objective,

or both, of the first and second branches of the trigeminal nerve (eventually all three branches), the diagnosis of an aneurysm in the carotid canal is almost absolute.

Quite frequently the fourth and sixth nerves are also paralyzed: in Czermak's case both the abducens (N. VI) palsy and anesthesia of the cornea (N. V) antedated that of the third nerve by six years. Dural tumors of the lesser wing of the sphenoid and neuritis of the

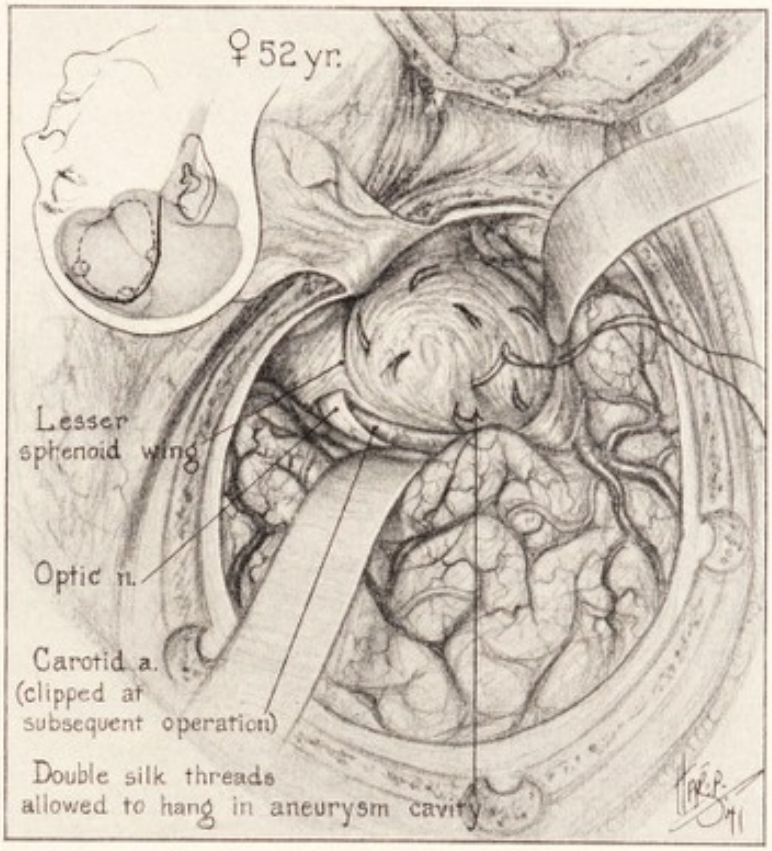


FIG. 5.—Operative sketch of one of the tremendous aneurysms of the carotid canal bulging upward into the temporal lobe and filling the middle fossa. The internal carotid artery is pushed forward by the aneurysm.

third nerve may also give identical objective findings, but in both of these conditions the severe *attacks of pain* in the eye and frontal region *are absent*. Pain of this character and location has been perhaps the most consistent subjective complaint in all of the recorded cases. A sudden severe pain is always suggestive of a vascular expansion or rupture. In six of the 11 cases there was paralysis of the fourth nerve, and in five cases paralysis of the sixth nerve. Case V is interesting in that the third-nerve paralysis dated back 22 years to the age of two, when the aneurysm ruptured. The ptosis and oculomotor paralysis have persisted since that time. The aneurysm was

still patent at the time of operation, blood being aspirated from it.

Since these aneurysms lie upon the Gasserian ganglion, trigeminal neuralgia is to be expected, usually more or less continuous, plus severe attacks and eventually sensory loss. Actually, however, in 10 cases from the literature, these subjective and objective disturbances were absent or were not noted, and in only five cases in this series was the trigeminus affected. Their absence, therefore, does not militate against the diagnosis of an aneurysm in the carotid canal, but their presence is all-important. Romberg's case (1853), one of the smallest aneurysms of the series, had only trigeminal neuralgia; those of Magnus (1927) and Jefferson (1937) had neuralgia of this type plus an abducens palsy.

Jefferson has proposed subdivision of aneurysms in the carotid canal into three groups, (1) posterior, (2) middle, and (3) anterior, i.e., corresponding with the three positions of the lesions in the carotid canal. In the posterior group he places those with involvement of all three branches of the trigeminus and palsies of all the extraocular muscles; in the middle group the third branch of the trigeminus is spared; in the anterior group only the first branch of the trigeminus is involved. However, there appears to be little point in this finer classification. The degree of involvement of the nerves is a better index of the size of the aneurysm.

Loss of vision is an important and common subjective and objective disturbance. It is due to direct pressure of the bulging aneurysm on the optic tracts or upon an optic nerve through the interposed carotid artery, as in one of our cases (VIII), in which there was only a central scotoma. Eventually there may be blindness in the eye on the affected side. Occasionally, as in Cases I and XI, the aneurysm may enlarge sufficiently to attack the optic chiasm and produce blindness in one eye and hemianopsia in the other. Considering the enormous size of many of these aneurysms, it is surprising that vision is retained so long. It is the tightly attached dura at the anterior part of the middle fossa that prevents the actual contact with the optic nerve until the size of the aneurysm grows excessive.

At times papilloedema results from intracranial pressure as the aneurysm grows upward into the cranial chamber. Both eyegrounds may be similarly affected, or there may be primary optic atrophy on the affected side and papilloedema on the other (Case I).

Convulsions were present in only one patient (Case V), but in this

instance they could not have been caused by the aneurysm, since it did not affect the brain.

Exophthalmos was noted in 11 cases (seven cases from the literature and three in this report). Exophthalmos can occur only with the very large aneurysms that rode the walls of the sphenoidal fissure and can then push the orbital contents forward. Pulsation of the eyeball has been noted but rarely, in spite of the violent pulsation of the aneurysm; doubtless it has been missed in some instances. Dempsey's case is noteworthy in this respect because there was an aneurysm of the ophthalmic artery in addition to the diffuse enlargement of the internal carotid artery (diameter ¾ inch) in the canal.

Bruit was noted only in the case reported by Werner, Blakemore, and Kind. Hutchinson was very skeptical of reports of bruit in the intracranial aneurysms and concluded that a bruit was only present in the arteriovenous variety—then termed aneurysm by anastomosis. In none of our cases was a bruit detected. It can occur, therefore, but it is hardly worth consideration in differential diagnosis.

With respect to the ages of patients at the time of operation, death, or disclosure of the aneurysm, the numbers are fairly even according to decades. Only two were under 20, although several of those in the succeeding decade had their origin before 20. In one of our cases (VI) the aneurysm ruptured at the age of two. Nine aneurysms occurred in the third and sixth decades, six in the seventh, five in the fourth, and three in the fifth. The oldest patient was 72 (Jefferson). Eighteen patients were males and 18 were females. Twenty-one of the aneurysms were of the very large type, which protrudes upwards into the cranial chamber; many of these caused intracranial pressure.

The duration of symptoms varies considerably; 20 (50 per cent) had symptoms over three years; nine had symptoms nine years or more; three lived 18, 20, and 22 years respectively, and one (our Case VI) is still living after 25 years. In several cases, however, the course was more fulminating, lasting a few weeks or months.

The very fact that aneurysms are due to defective arterial walls predisposes them to rupture, and, since a high percentage are of congenital origin, the rupture is frequently in the early years of life. Aneurysms in the carotid canal are less susceptible to rupture than those within the cranial chamber, because they are covered by the firm layer of dura, which expands slowly. This accounts for the long duration of symptoms in so many instances. However, they are not immune

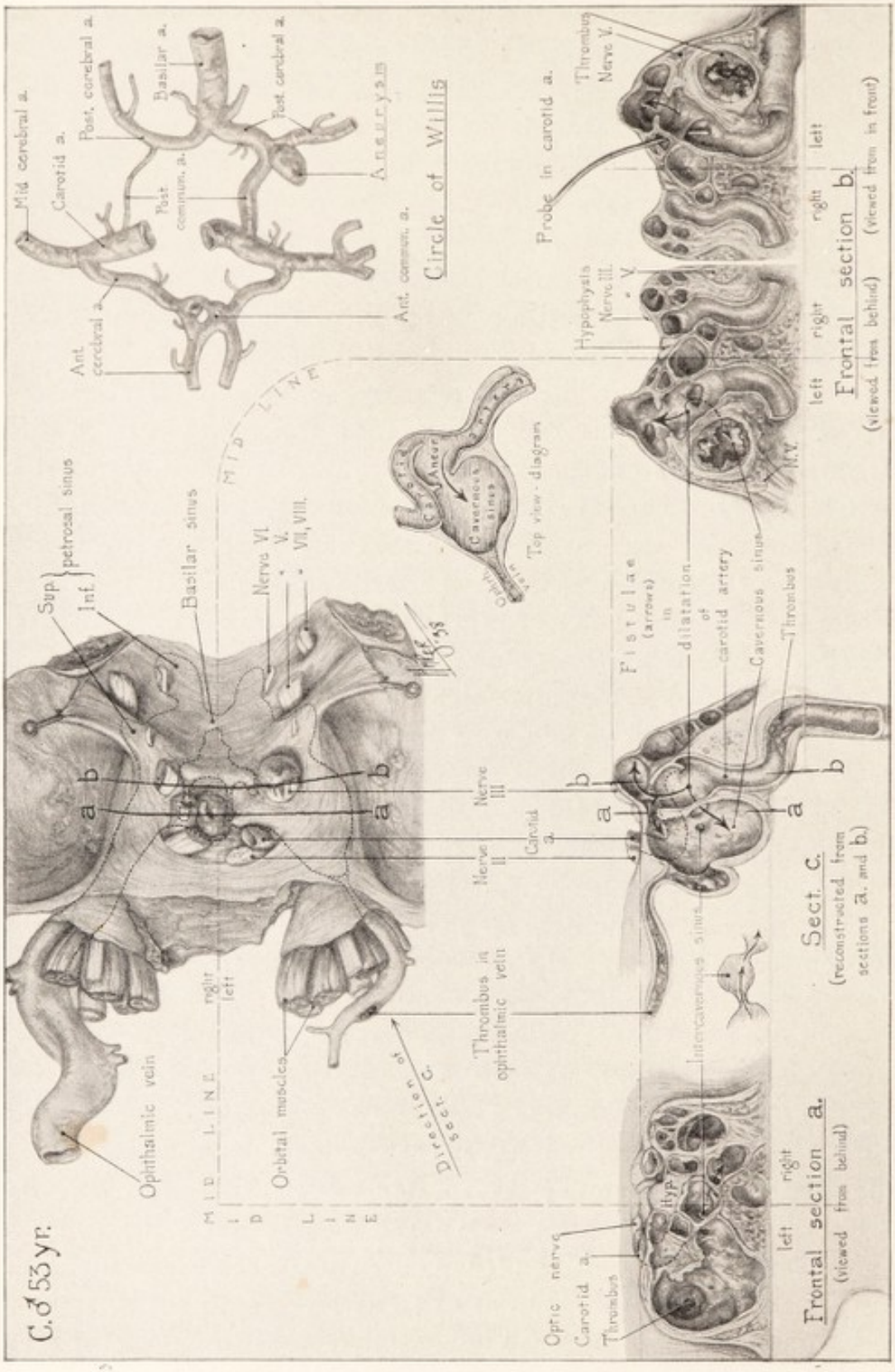


FIG. 6.—Postmortem sketch of aneurysm in the carotid canal. It had ruptured into the cavernous sinus in two places and produced pulsating exophthalmos.

to this termination. Four deaths resulted in this collected series from this cause—the rupture being into the cranial chamber. Three of our operative cases had probably ruptured alongside the carotid (from small intracranial extensions), and the point of rupture had healed owing to the contact with the carotid artery. In one instance (II), two separate small aneurysms had ruptured into the cavernous sinus (Fig. 6) and produced an arteriovenous aneurysm (pulsating exophthalmos, Fig. 7). This is one of the important causes of this remarka-

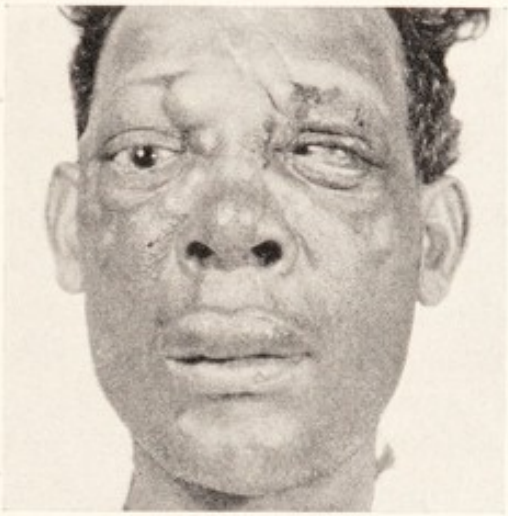


FIG. 7.—Photograph of patient (Fig. 6). Note the extraocular sixth-nerve palsy and the large bed of veins over the forehead. It should be noted that the exophthalmos is on the right, whereas the arteriovenous fistula is on the left. The left eye cannot protrude because the ophthalmic vein has thrombosed.

ble lesion; it may occur spontaneously or be induced by trauma. In this case the existence of the arterial aneurysm as the underlying cause was recognized only at necropsy. In Nettleship's case repeated hemorrhages into the nose finally caused death.

It is doubtful if spontaneous cure of an aneurysm of this type ever occurs. Frequently such an aneurysm is partially filled with a firm old thrombus that is laid down in layers. Hutchinson noted that his aneurysm was "almost entirely" filled with thrombus, but the specimen was not opened; his deduction was made by probing. Jefferson stated that one of his large aneurysms was "almost completely" thrombosed. Many aneurysms are indeed almost completely filled with thrombus, though a central channel usually persists and is in communication with the lumen of the artery; but they still remain active aneurysms. In one of Krayenbühl's cases the internal carotid and the corresponding middle cerebral artery were completely thrombosed (hemiplegia resulting). This may well be a case of complete

thrombosis and cure of the aneurysm, but the patient survived and therefore no pathological report was in evidence. However, occlusion of the internal carotid artery was probably responsible for the cure.

Bilateral Aneurysms

Case I belongs in a class by itself as far as this series is concerned. On each side was a very large sac, the right being as large as a pigeon's egg, and the left perhaps ten times as large. These two masses were in apposition—the left lying above the right. The base of the skull in this region, including the sella, was depressed and largely destroyed; there were no remnants of the sella turcica. The enormous mass protruded far upward into the base of the brain and into the left orbit, producing marked exophthalmos on this side. In the x-rays of the skull there were several curved linear shadows in the calcified walls of the arterial sacs; these shadows covered much of the sellar and suprasellar regions, and extended back to the pineal body and more than halfway through the cranial chamber toward the vault. Although the aneurysm was certainly of congenital origin, the patient had had no symptoms until four and one-half years before death at the age of 26, when vision in the left eye became defective, and three months later was entirely lost. On entering the hospital three years later there was also temporal hemianopsia in the right eye. There was papilloedema of four diopters in the right eye and primary atrophy in the left. Headaches began three months after the initial visual loss and were so severe that the patient spent six months in bed. Thereafter the headaches were only occasional and not severe. Neither a pulsation of the eyeball nor a murmur was detected, although both were sought. All extraocular movements on the left side were absent. Fourteen months before his death a decompression was made to relieve the intracranial pressure.

Differential Diagnosis

Unless there is a concomitant disturbance of the trigeminal nerve or positive roentgenographic findings, there is no way by which an aneurysm of the carotid in the carotid canal can be differentiated from one on the intracranial division of the carotid. The distinguishing sign of both and also of the posterior communicating artery is paresis or paralysis of the third nerve. And even this sign is not

pathognomonic, since aneurysms elsewhere on the circle of Willis may be associated with the same findings. In every case in this group there was weakness of the extraocular movements supplied by the third nerve, and in some cases the fourth and sixth nerves also.

B. Intracranial Portion of the Internal Carotid (Table B)

There are 39 patients with aneurysms of the intracranial portion of the internal carotid artery. Eight of these aneurysms were bilateral or multiple, making a total of 47 aneurysms, or 35.4 per cent of the total number. In one instance (Case XIX) there was an associated congenital arteriovenous aneurysm in the cerebral hemisphere, and in Cases XXIV, XXIX, XXX, and XXXVI there were additional arterial aneurysms that were not on the carotid.

Aneurysms of the intracranial internal carotid are divisible into three distinct groups: (*a*) fusiform dilatations of the carotid trunk—nine of these in six patients, three being bilateral; (*b*) sacculated aneurysms on the arterial wall before the first branch is given off—ten of these; and (*c*) the small, berry-like aneurysms at or near one of the branches of the carotid—24 patients with 29 aneurysms, more frequent than the other two groups combined.

(*a*) *Diffuse Dilatation of the Carotid Trunk* (Cases I–VI)

Four of these six cases (III, IV, V, and VI) were due to arteriosclerosis; the ages were respectively 52, 58, 64, and 67 years. The aneurysm in Case I may also have been due to arteriosclerosis, though I have considered it more probably of congenital origin. Three were bilateral and symmetrical. Three patients were males and three were females. Case V was surely congenital; the patient was 47, a subject of migraine which was on the same side and was probably caused by the aneurysm.

One of these patients (Case III) presumably died of arterial occlusion a few days after an exploration at which the carotid artery was found to be like a large, rigid pipestem. Another died of an extra dural (postoperative) hematoma. Another (Case VI) died after operation for a concomitant hypophyseal tumor. A fourth (Case IV) is still living 3½ years after operative disclosure. Only one of these

aneurysms had ruptured (Case II). Case IV, also exposed at operation, died three days later from a large undiagnosed dural tumor. This patient also had a large S-shaped aneurysm of the basilar and vertebral arteries.

The blood pressures in the arteriosclerotic group were respectively

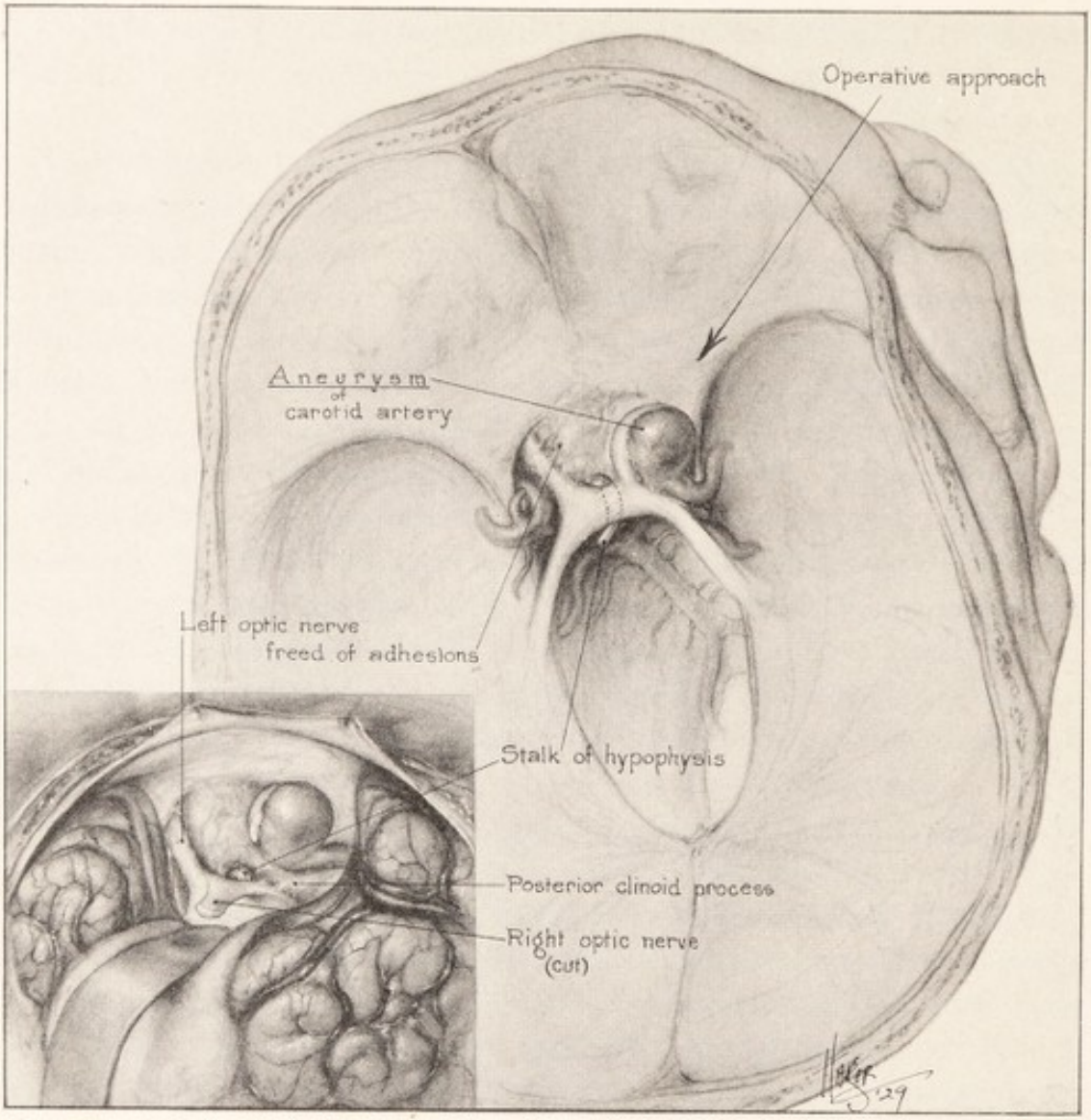


FIG. 8.—Operative sketch of an aneurysm caused by diffuse enlargement of the carotid at the carotid canal. Note compression of the optic nerve.

110/70, 110/70, 120/80, 200/120, and 190/110. An elevated blood pressure therefore may or may not be present even in the arteriosclerotic aneurysms.

The outstanding symptom in this group was loss of vision with primary optic atrophy. This was confined to one eye in Cases I (Fig. 8) and III, and to both eyes in Cases II and IV, where the aneurysms

were bilateral, each optic nerve being compressed by a separate aneurysm. There was no loss of vision in Cases II and VI. One patient (Case III) was nearly blind; in this case the referring physician very properly suspected a suprasellar tumor, but the correct diagnosis was suspected on the disclosure of a faint calcified shadow (x-ray) above the right anterior clinoid process and also of a tubular calcification in the right carotid canal. The duration of visual loss before death or operation was respectively three months, five months, eight months, and (Case I) four years.

In patients over 50 years of age, with primary optic atrophy and blindness in one or both eyes, and without evidence of hemianopsia, an aneurysm of this type should always be considered.

Headache was absent in three patients with arteriosclerotic aneurysms. One patient (Case II, congenital) had typical migrainous headaches on the side of the aneurysm, doubtless caused by it. In Cases V and VI headaches were severe but were due to the concomitant tumors—one recognized, the other not.

Although calcified shadows are important in suggesting the character of the lesion, they are important only when taken in conjunction with the loss of vision. At times one finds calcified arteriosclerotic shadows above the sella and in the carotid canal which cause no symptoms. Calcifications of hypophyseal duct tumors and dural meningiomata may be difficult or impossible to differentiate from those of an aneurysm.

(*b*) *Small Sacculated Aneurysms on the Internal Carotid Before It Branches* (Cases VII–XVI inclusive)

The sacculated character of these aneurysms with the small neck can leave no doubt of their congenital origin. Moreover, with one exception, all were young (aged respectively 20, 31, 31, 34, 39, 42, 43, 44, 47, and 60 years). Four were males and six were females. In only one patient (VIII) was the blood pressure elevated; his age was 60.

In none was there calcification or any other positive finding in the x-ray. A single case (XIII) was a postmortem specimen. She died of intracranial hemorrhage before reaching the hospital.

A third-nerve paralysis was present in every case. Although by no means pathognomonic, a third-nerve palsy or paralysis is by far the most important objective evidence of an aneurysm of the carotid

artery. However, other aneurysms higher up on the internal carotid, or on the posterior communicating and even on the basilar arteries,

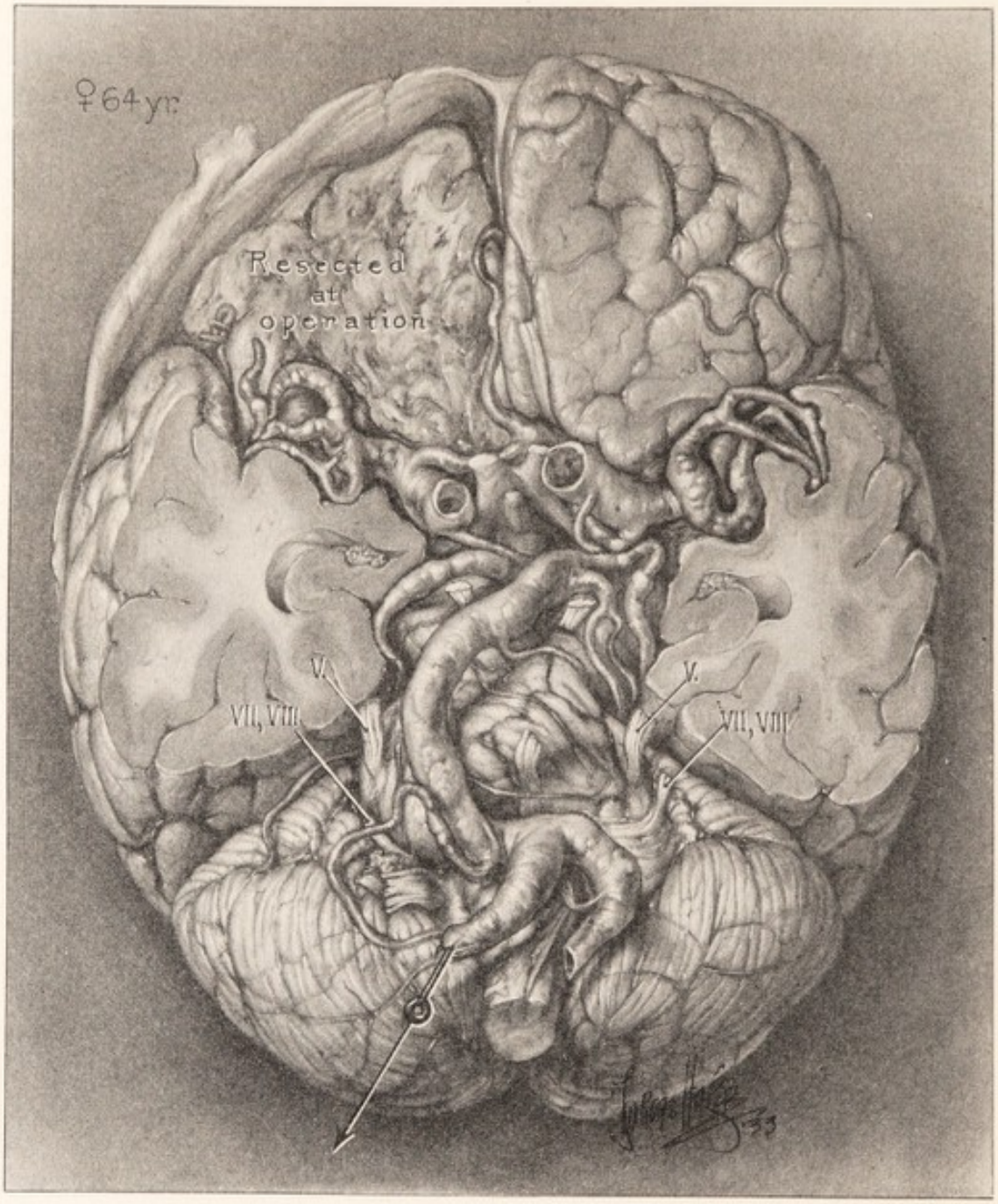


Fig. 9.—Case 5, Table B. Postmortem drawing showing the typical appearance of diffuse bilateral dilatations of both carotids. The vertebral and basilar arteries are similarly affected.

and nearly all of those in the cavernous sinus also produce unilateral third-nerve paralysis. Also, tumors in these environs occasionally give this as the only sign.

The first symptom was always pain in the eye or frontal region of

the corresponding side. Usually the drooping eyelid and diplopia follow in a few hours to a few days, but they may appear months later. Here again it appears very probable that the pain and third-nerve

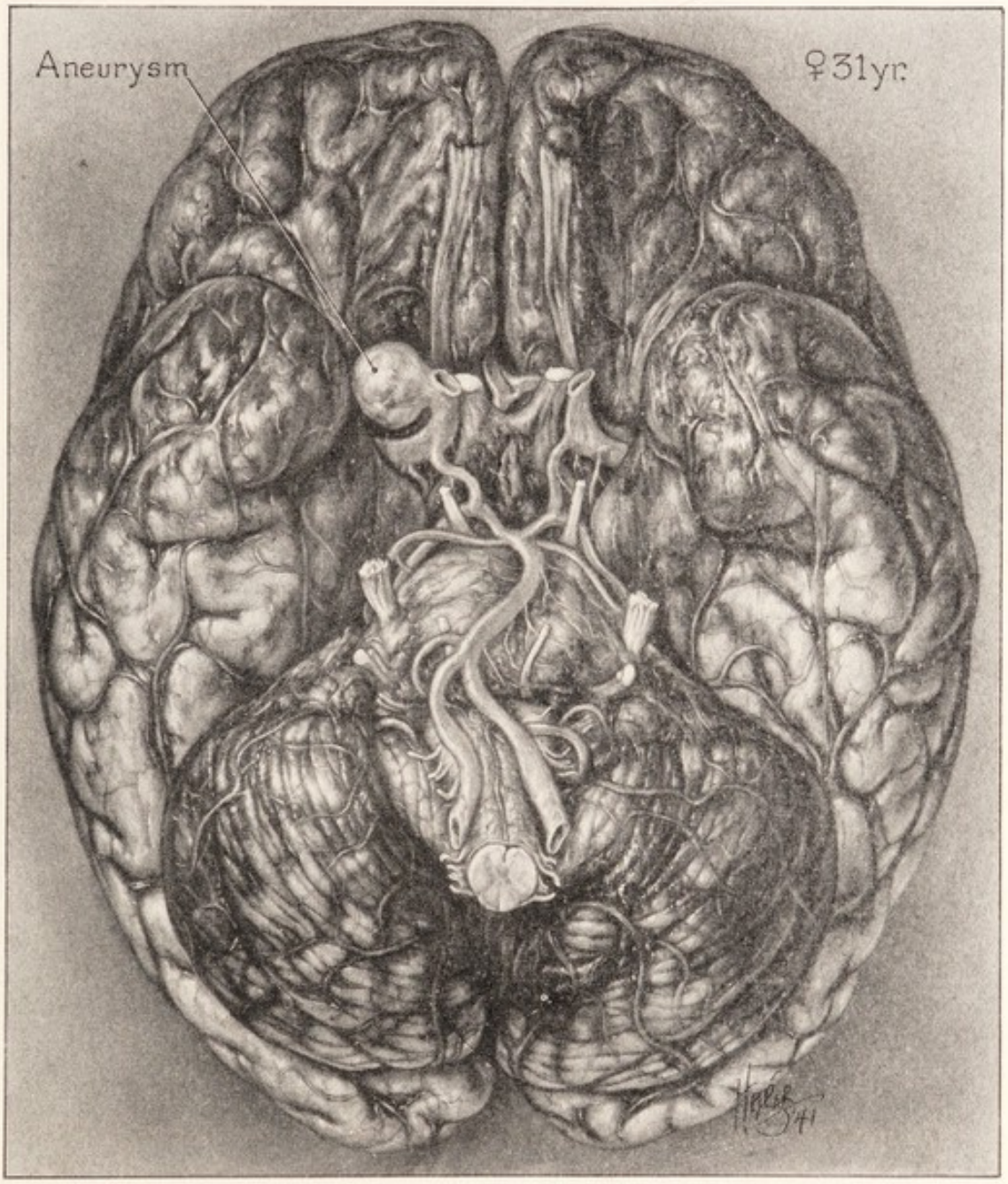


Fig. 10.—Case 13, Table B. Aneurysm of the internal carotid. This is the only specimen in which the aneurysm projected anteriorly; all others have projected posteriorly.

palsy indicate an expansion or actual rupture of the aneurysm. In every case exposed at operation the aneurysm has arisen on the posterior wall of the artery and its tip has been attached to the third nerve. One at necropsy (Case XIII) projected anteriorly (Fig. 10).

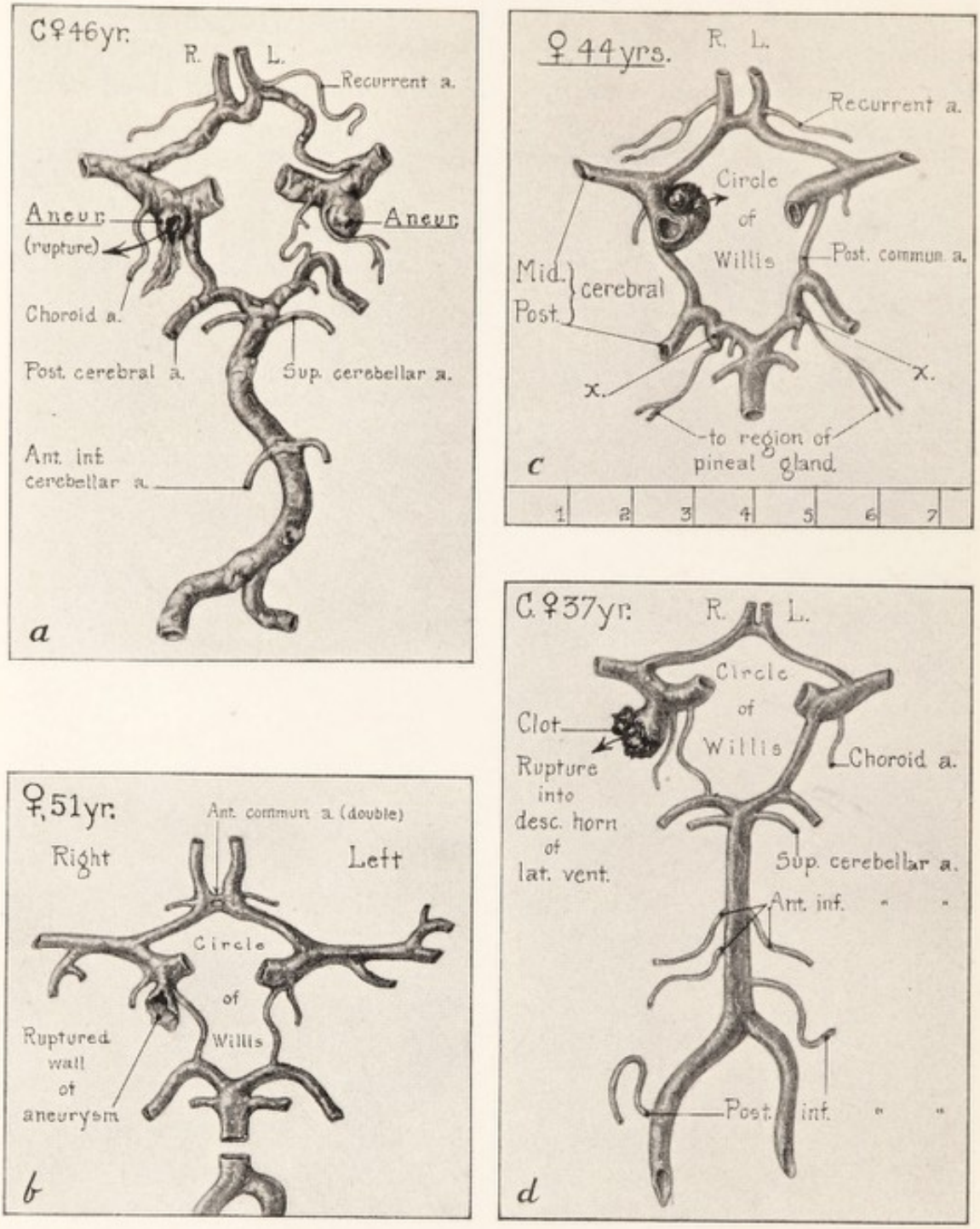


FIG. 11.—Four ruptured aneurysms from Table B: (*a*) Case 22. Showing bilateral aneurysms, one of which has ruptured. The asymmetrical posterior communicating arteries are shown. (*b*) Case 27. Showing rupture of carotid aneurysm at the posterior communicating branch. The top of the aneurysm has blown off with the rupture. (*c*) Case 28. Diffuse ruptured aneurysm at the right carotid and without a neck. The two little swellings on the posterior cerebral arteries, marked *x x*, may be potential aneurysms. These aneurysms bulge to the inside of the circle of Willis, while the others are on the outside. (*d*) Case 34. Aneurysm on the carotid at the choroidal artery. It had ruptured into the descending horn of the lateral ventricle.

This is the only postmortem specimen of one of these aneurysms. It is probable that these aneurysms develop at the site of the primitive

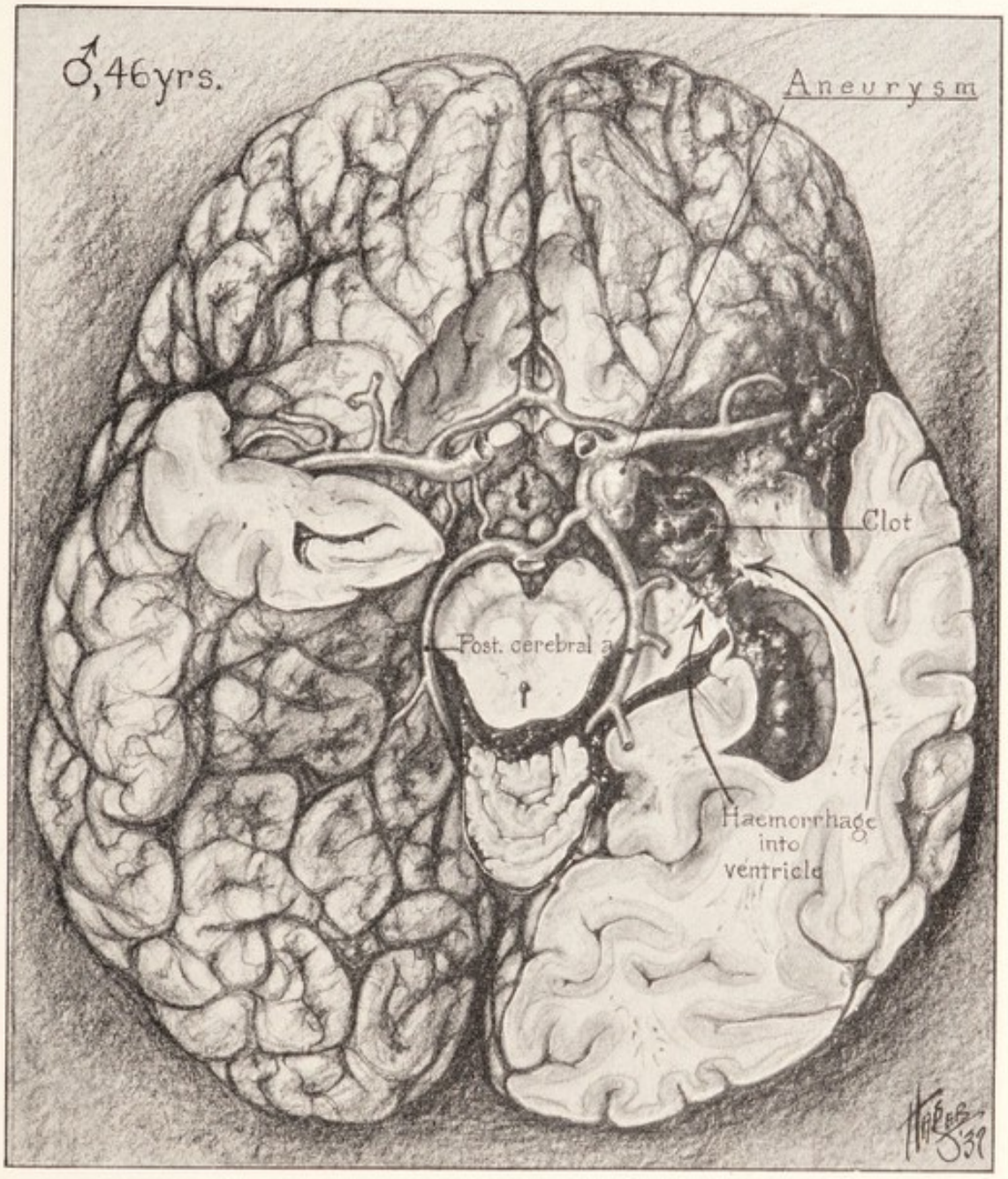


FIG. 12.—Aneurysm of the carotid lying alongside the posterior communicating artery. It had ruptured into the temporal and the descending horn, causing intraventricular hemorrhage.

embryonic ophthalmic artery which later disappears. In Case XV the "stroke" came five days after the onset of ptosis.

In Cases VIII and XI the vision was reduced to 20/40 on the side of the aneurysm. In Case IX the vision was hazy in the affected eye and was reduced to 20/20 as compared to 20/15 on the other side.

Case VII had some dizziness and nystagmus. In Case IX hemiplegia and aphasia followed the severe pain but subsequently cleared completely. At the operation the cause was found to have been a hemorrhage into the Sylvian fissure. One patient (Case XI) survived a period of coma that lasted a month (four months earlier).

Case X had hemorrhages in both discs, but much larger on the

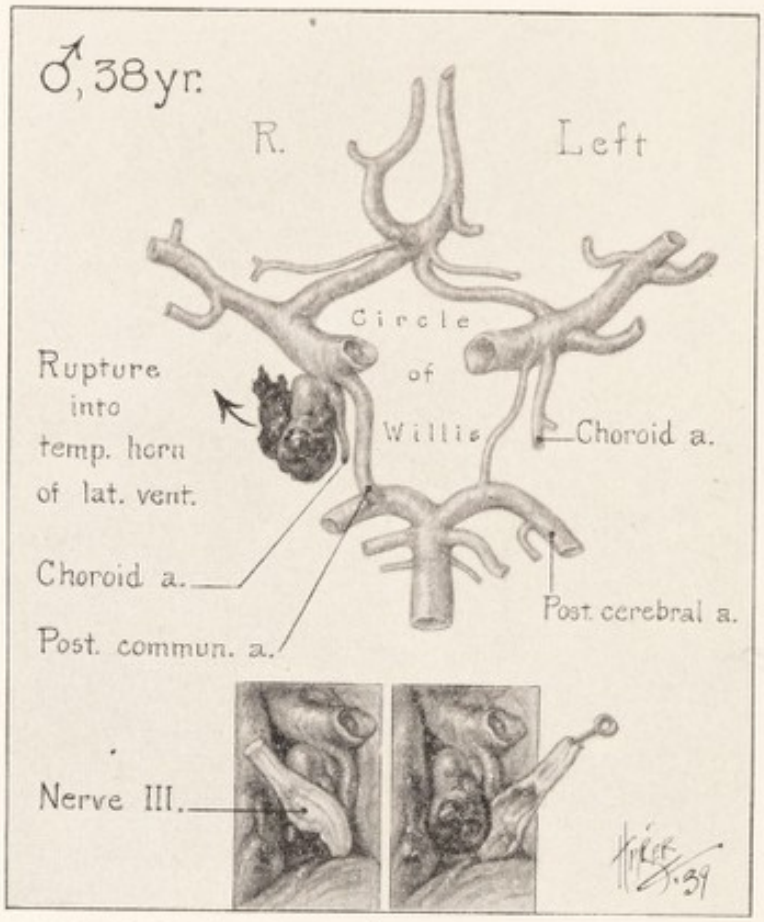


FIG. 13.—Case 33, Table B. Showing rupture of aneurysm into the third nerve, which temporarily sealed it. The nerve was largely destroyed, only a thin shell persisting.

side of the aneurysm. These came one night during the preoperative period in the hospital—his blood pressure was 132/84.

(c) *Aneurysms of the Internal Carotid At or Near Its Branches*

There are 23 cases with 28 aneurysms (21 per cent) in this group —10 males and 13 females. Eleven of the group were colored. This appears to be a striking racial disproportion, and indeed it appears to be true with all intracranial aneurysms. The ages range from 19 to 68 years; the one at 68 was causing no symptoms; ten were in the thirties, six in the forties, four in the fifties, and two in the sixties.

Eleven of these aneurysms arose at the junction with the posterior communicating artery (Fig. 11). One arose at the junction with the middle cerebral; one on the trunk of the artery, 3 mm. above the posterior communicating artery and before the middle cerebral is given off; and one (Case XXVI) sprang from a large anomalous vessel

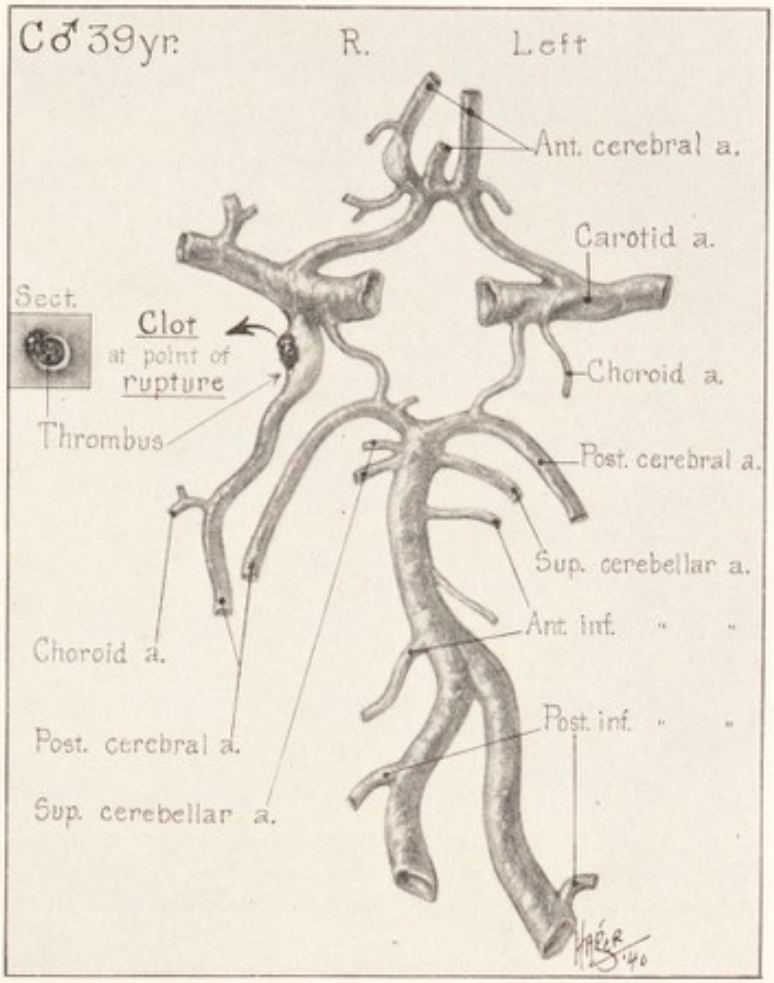


FIG. 14.—Case 26, Table B. Small ruptured aneurysm on a large anomalous vessel corresponding to the choroidal artery; but this vessel extended into the occipital lobe along with the posterior cerebral artery. The choroidal artery arises from this branch.

corresponding to the anterior choroidal artery and situated several millimeters beyond the carotid (Fig. 14). This vessel extended into the occipital lobe and en route gave off the choroidal artery. Another (Case XXIX) arose from the anterior choroidal artery in the choroid plexus (near the foramen of Monro). All are, I think, of congenital derivation.

Three of the aneurysms (Cases XXIV, XXX, and XXXII) in this series were accidentally found at necropsy and had not caused symptoms. One (Case XIX) still had a residual third-nerve palsy,

although the aneurysm had been cured by thrombosis during the four years following its rupture (Fig. 15). Moreover, the vision in the affected eye was only 10/200. In fourteen patients death resulted from rupture of the aneurysm.

The duration of symptom was, on the whole, very short. Four patients died within 24 hours, one in 48 hours, one within a week,

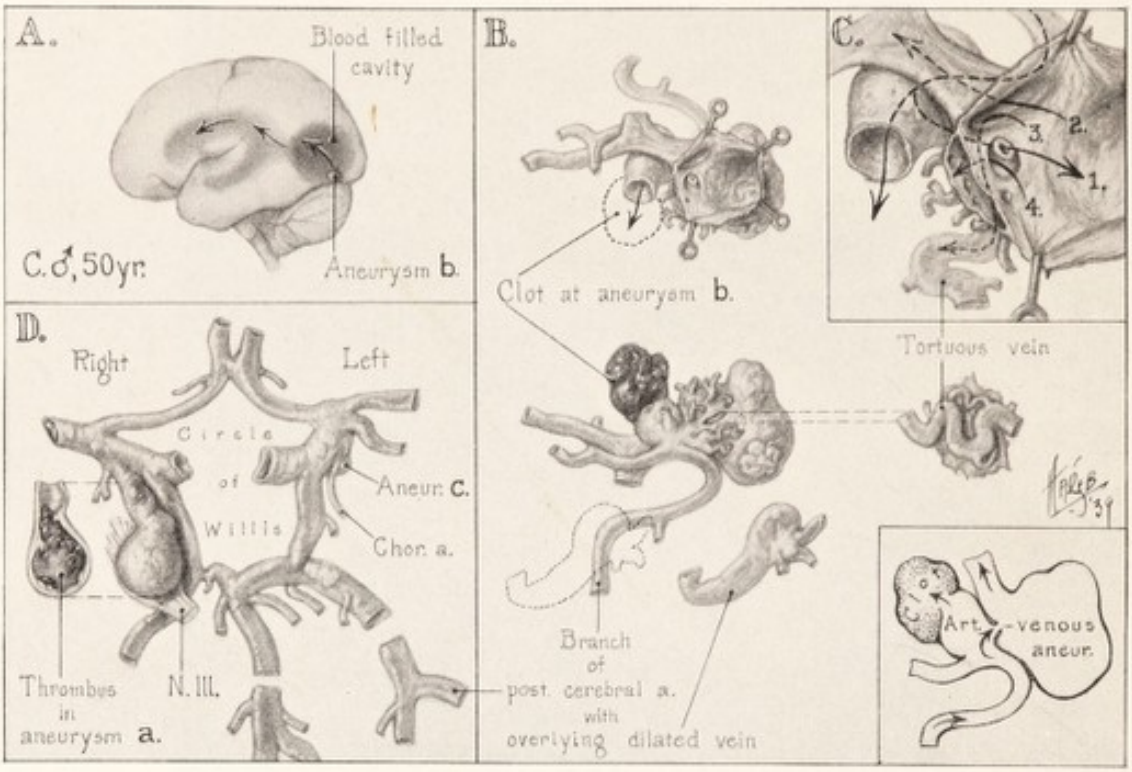


Fig. 15.—Case 19, Table B. This is the only healed aneurysm in the series. It had been imbedded in the third nerve for four years and was filled with a solid clot. The cause of death was an arteriovenous aneurysm in the occipital lobe on the opposite side.

and three within five weeks. Only one had symptoms longer and he (Case XXXIII) had symptoms for seven months.

There were marked variations in the signs and symptoms which might lead to a localization of the lesion: In most instances the diagnosis of an aneurysm was quite probable because of the sudden onset of coma, but this did not localize the lesion.

A third-nerve paralysis was present in ten cases (Fig. 13); pain in one eye in six; unilateral blurring of vision in seven; rigidity of the neck in four; hemiplegia in two, and opisthotonos in two. Convulsions were present in five, although usually these were in the period just preceding death. In Case XXV, however, a single convulsion had antedated the fatal rupture 20 days, and in the interim the patient had been well and at work.

In seven cases there were retinal hemorrhages, four without and three with papilloedema (unilateral in one case and bilateral in two cases). In three of these the hemorrhages were unilateral and on the side of the aneurysm; in four the hemorrhages were bilateral but always much greater on the side of the aneurysm. There was a single, large, round retinal hemorrhage in Case XXV. The presence of retinal hemorrhages without papilloedema always strongly suggests an aneurysm, although it also occurs with rapidly developing tumors. The round subhyaloid hemorrhages are thought to be pathognomonic of an aneurysm, and of one on the same side of the circle of Willis. The rapidity with which the retinal hemorrhages arise may be very striking. I have seen a hemorrhage picked up ten minutes—and in another case one hour—after negative ophthalmoscopic examinations by good observers.

A positive Babinski was present in three instances—bilateral in one and unilateral in two. Bilateral absence of knee-kicks was observed in one case (XVI).

It is worthy of note that one patient in this series (XXI) died while being prepared for a lumbar puncture on another service and before it had actually been started. She appeared to be perfectly well before the necessary manipulation, although hemorrhages in both eyegrounds denoted a high degree of intracranial pressure.

Three of these aneurysms (Cases XXV, XXVII and XXXIV) ruptured into the descending horn of the lateral ventricle (Fig. 12). A third (Case XXIX) arose from the choroid plexus and ruptured into the body of the lateral ventricle.

Literature on Aneurysms of the Intracranial Carotid

The literature contains many examples of aneurysms of the carotid, nearly all of which are duplicated in this series. Most of the aneurysms are included in groups reported from various clinics. This is not an exhaustive review of the bibliography. Epron (1890) had 13 and Berger (1923) had seven aneurysms of the internal carotid among 28 and 21 necropsy specimens respectively. From 22 postmortem examinations of intracranial aneurysms reported by Wichern (1912) two were from the internal carotid. Among 17 intracranial aneurysms Schmidt (1930) had two sufficiently large to fill and destroy the sella. Shaw's case (1901) had a large sacculated aneurysm at the entrance into the cranial chamber and another (ruptured) of

the middle cerebral. Bristow's case (1868) was so large that it included the anterior and middle cerebral and the posterior communicating arteries. In Kirby's (1924) Lodge, Walker and Stewart's (1927), and Zollinger and Cutler's (1933) cases the sella was destroyed and the lesion simulated a pituitary tumor; the aneurysm in the latter case measured 3.5 x 3.5 cm. and was exposed at operation for a presumed tumor. In Fitz's case the aneurysm dislocated the optic tracts. Parker (1926) reported two aneurysms of the carotid. Cookson (1933) reported four aneurysms at the junction with the middle cerebral. McKendree and Doshay (1936) found three at operation. Trevani (1932) aspirated at operation a similar aneurysm alongside the sella.

Among a small series of aneurysms Dial and Maurer (1937) include one with a discrete neck similar to those which have been so satisfactorily treated in Group *b* of this section. This case and Case XIV are the only pathological reports of aneurysms of this particular type that I have been able to find.

Reichert (1939), Sargo (1939), and Krayenbühl (1941) have diagnosed aneurysms of the carotid by intra-arterial injections of thorotrast; Reichert and Krayenbühl exposed the aneurysms at operation.

C. Anterior Cerebral and Anterior Communicating Arteries (Table C)

There are 25 verified aneurysms of these arteries, or 18.8 per cent of the total number. Very frequently the aneurysm is so located that two or all three of these vessels are incorporated in its structure, and it may even be difficult to isolate the point of origin to a single vessel. All are probably of congenital derivation, although a positive Wassermann was found in two; nevertheless, the gross character of the aneurysms was such as to make their congenital origin almost certain. Five of these aneurysms were found in routine postmortem examinations; they had not ruptured and were causing no symptoms. One found at operation was giving no symptoms; 16 had ruptured.

Incidence and Symptoms

Twelve were males and 13 females. Eight of the patients were colored: here again the proportion is high in the colored race. The three youngest were in the third decade, four in the fourth, six in the fifth, eight in the sixth, two in the seventh, and one each in the eighth and ninth decades.

The duration of symptoms after the onset is, on the whole, longer than with the other aneurysms along the circle of Willis. At least five of this group were known to have had two or more (Case XV had four) distinct hemorrhages, each patient dying soon after the last rupture. Only three of the series died within 24 hours. Six died in less than two weeks; five lived for periods respectively of "a few weeks," 68 days, five months, one year, and 8½ years. The onset with rupture was always sudden and the symptoms were quite variable, frequently difficult to describe: "something terrible happened" (Case VII), "something popped in the head" (Case XVI), sudden pain in the eyes, photophobia and loss of vision (Case XII). With two exceptions (Cases XX and XXV) there had been headache and pain. The widely differing location of the headache and pain is disappointing from the viewpoint of localization of the aneurysm. In eight cases the headache was general, in three occipital; one was in the temple of the affected side, and one was in the contralateral frontal region. In Case V the pain was in two places—the back of the head and the lumbar region. Similar bizarre and misleading headaches were also noted in the middle cerebral aneurysms.

Rigidity of the neck was noted in five cases (from hemorrhage). Double vision was present in four, although extraocular palsies were demonstrable in only two; one had a homolateral third-nerve palsy, the other bilateral abducens palsies. Loss of vision was the only complaint in Case XX. Convulsions were present in two, one generalized and one focal; another had generalized rigidity (tonic spasm), but no convulsion, and a fourth had cramps in the contralateral leg but no actual convulsions. Partial hemiplegia was present in five cases, and bilateral Babinski reflexes in a sixth. Blurred vision was a conspicuous symptom in five cases. Severe loss of vision was present in three cases—XII, XXIV and XXV. In Case XXV the vision, the only subject of complaint, was 15/200 right and 20/70 left. In Case XII the vision was reduced to 10/200 left and 30/40 right, and there were bitemporal scotomata. In Case XXIV the visual acuity

was normal but there was hemianopsia for colors and a corresponding quadrantal defect for form. In both of these cases (XII and XXIV) the sella turcica was destroyed. Bilateral papilloedema was found in only three cases, in one of which there were retinal hemorrhages.

It is perhaps more than coincidental that so many of these patients have had an increased blood pressure. It is impossible, however, to tell in how many of them the pressure was elevated before the intra-

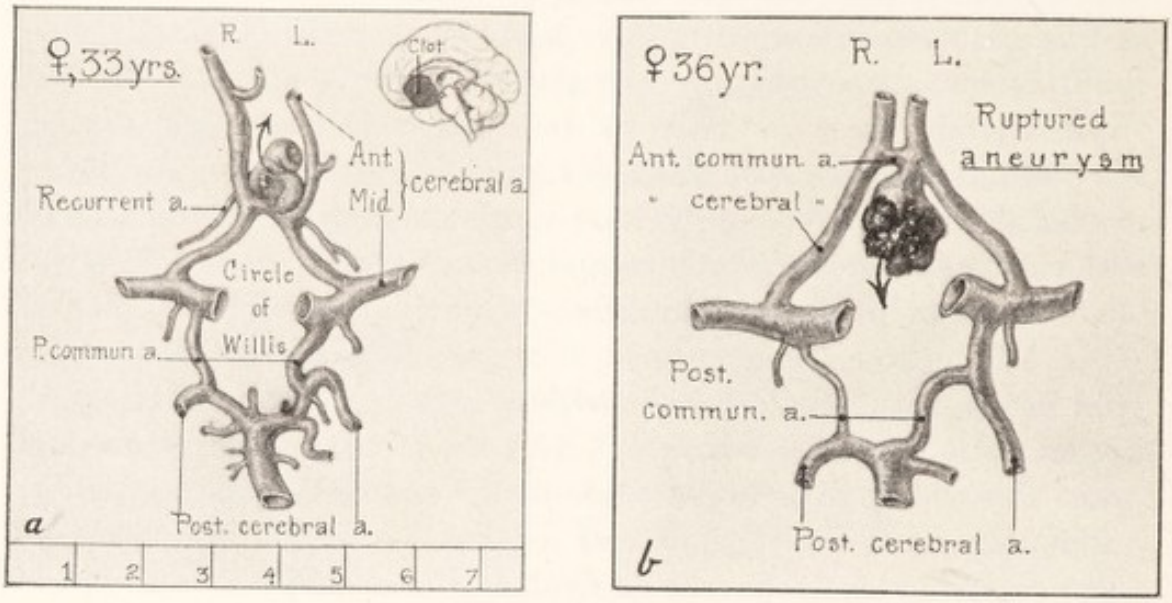


Fig. 16.—Two postmortem specimens of aneurysms of the anterior cerebral and communicating arteries: (*a*) Case 6, Table C. Aneurysm on the anterior communicating artery projecting into the frontal lobes and producing a filling defect in the third ventricle (by ventriculograms). (*b*) Case 10, Table C. Ruptured aneurysm of the left anterior cerebral at the junction of the posterior communicating artery. Note the irregularity in the configuration of the posterior part of the circle of Willis.

cranial hemorrhage, which may be the terminal cause of the increase.

Thirteen increased blood pressures were recorded, nine between 158/104 and 200/120; four between 225/125 and 260/140. Case XIX was known to have had a pressure of over 200 for seven years.

Among 10 aneurysms with measurements, four were fairly large; in Case XIV (Fig. 18) the size was 4 x 4 cm. including the false sac. In Case XX it was 4.5 x 3 x 2.5 cm. In Cases XII (Fig. 17) and XXV (Fig. 52) the size was that of a hickory nut (2.5 x 3 cm.); in Case III (Fig. 19) it was 1.5 x 0.5 cm. In the remaining cases the size ranged from 2 x 2 mm. (Case XI) to 1 x 1 cm. (Cases VI and X, Fig. 16 *a* and *b*). Case XXIV was one of the largest, but could not be measured at operation.

The diagnosis of an aneurysm in Case XII (Fig. 17) was brilliant.

It was made unequivocably only by Dr. Frank Ford, because the patient was known to have coarctation of the aorta with a long standing hypertension (182/104). This combination of coarctation of the aorta and cerebral aneurysm had been noted by Eppinger (1871), Kolisko (1913), Strassman (1922), and Woltman and Shelden (1929). The x-ray showed destruction of the sella with loss of the

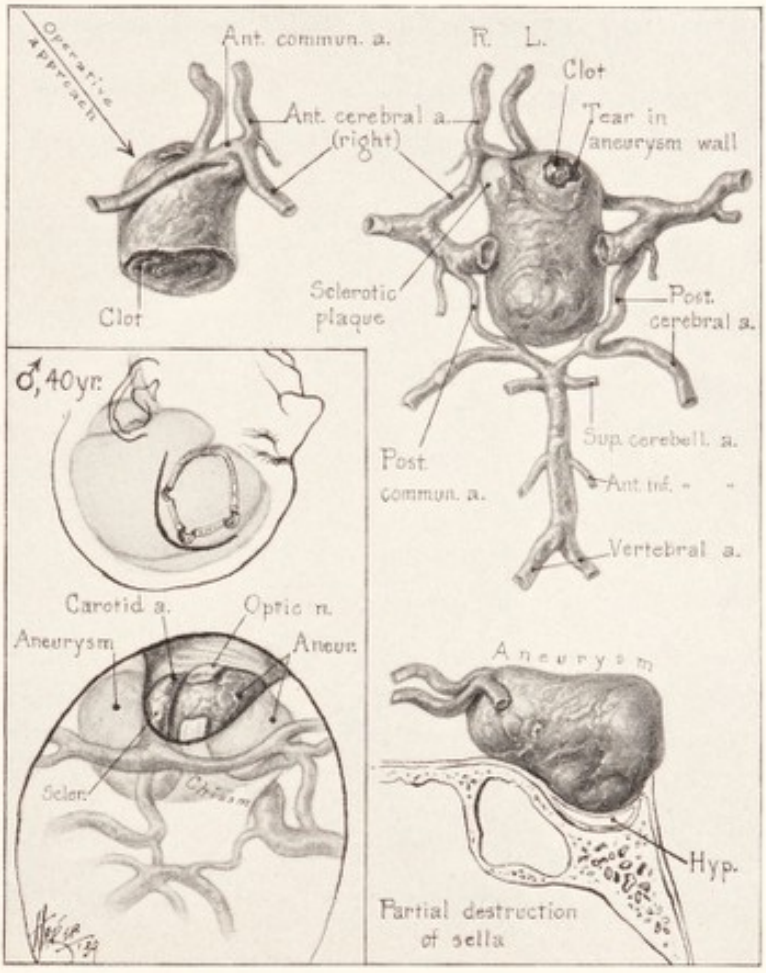


FIG. 17.—Case 12, Table C. Large aneurysm of the anterior communicating artery. It filled the entire circle of Willis. It was exposed at operation and ruptured several months later.

posterior clinoid process (no shadows). There were bitemporal scotomata. A very large aneurysm was found at operation. It was too large to permit dissection in an effort to find the point of origin. An attempt was made to coagulate the interior through a needle carrying a coagulating current. Two months later, when at home, the patient died suddenly. Although it was difficult to be certain from the postmortem examination, I believe the rupture occurred at the point of entry of the needle during the operation. About one-third of the

cavity was filled with lamellae of thrombus, the central cavity persisting.

These aneurysms are prone to rupture into the brain substance and cause intracranial pressure, and at times motor weakness. Figs. 18 and 19 (Cases III and XVIII) are examples.

Literature on Aneurysms of the Anterior Cerebral and Anterior Communicating Arteries

Reports of pathological specimens of aneurysms of the anterior cerebral and communicating arteries are quite numerous. These aneu-

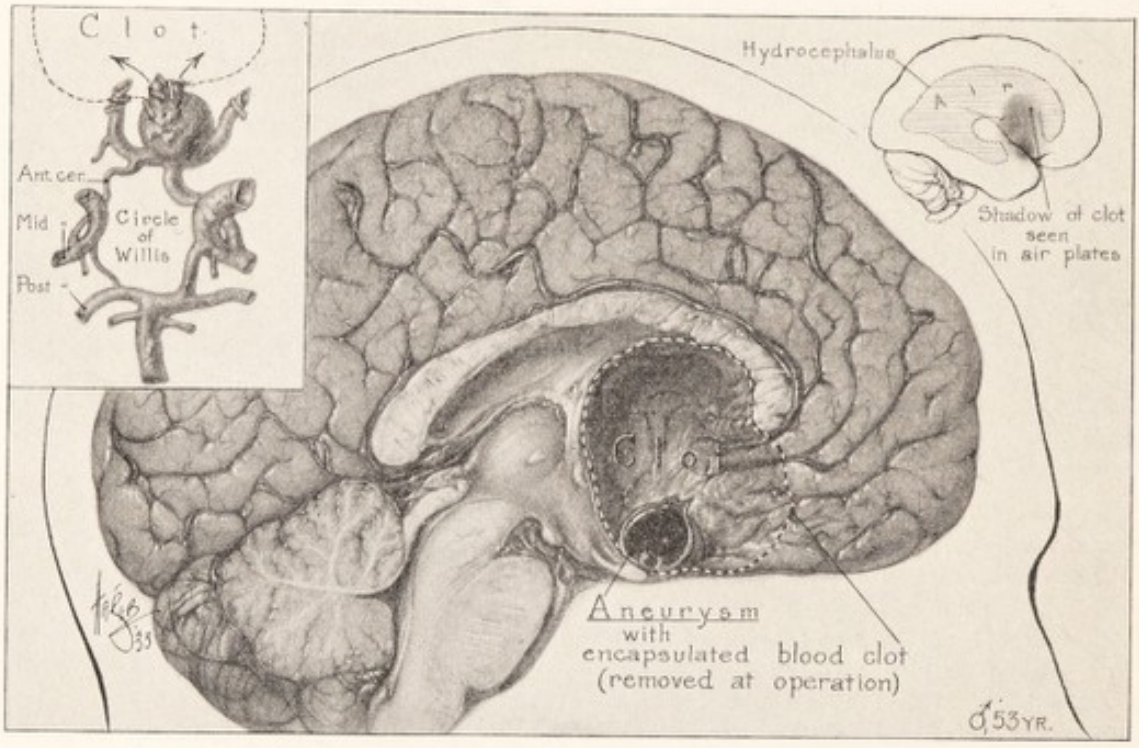


FIG. 18.—Case 14, Table C. Aneurysm of the anterior cerebral and anterior communicating arteries. The mass had produced a filling defect in the anterior part of the third ventricle and was demonstrated by ventriculography.

rysms are more various in character than those of the other cerebral vessels, and more of this type have been found at necropsy. Weir Mitchell (1889) reported a rare specimen of a midline aneurysm into which both carotid arteries entered with a connecting branch between. From his diagnosis one should wonder whether this represented the anterior cerebral and anterior communicating arteries. The patient had bitemporal hemianopsia, and from the necropsy specimen obtained elsewhere he asserted that there was no optic chiasm. Ebstein (1874) reported a long, diffuse aneurysm of an im-

paired anterior cerebral artery, the only one of its kind that I have encountered. Evans and Courville (1939) reported one of the most remarkable cases on record. In a single specimen there was a walnut-sized midline aneurysm of the anterior cerebral artery, an even larger double aneurysm on the left middle cerebral artery, and another smaller one on the right middle cerebral artery—four aneurysms

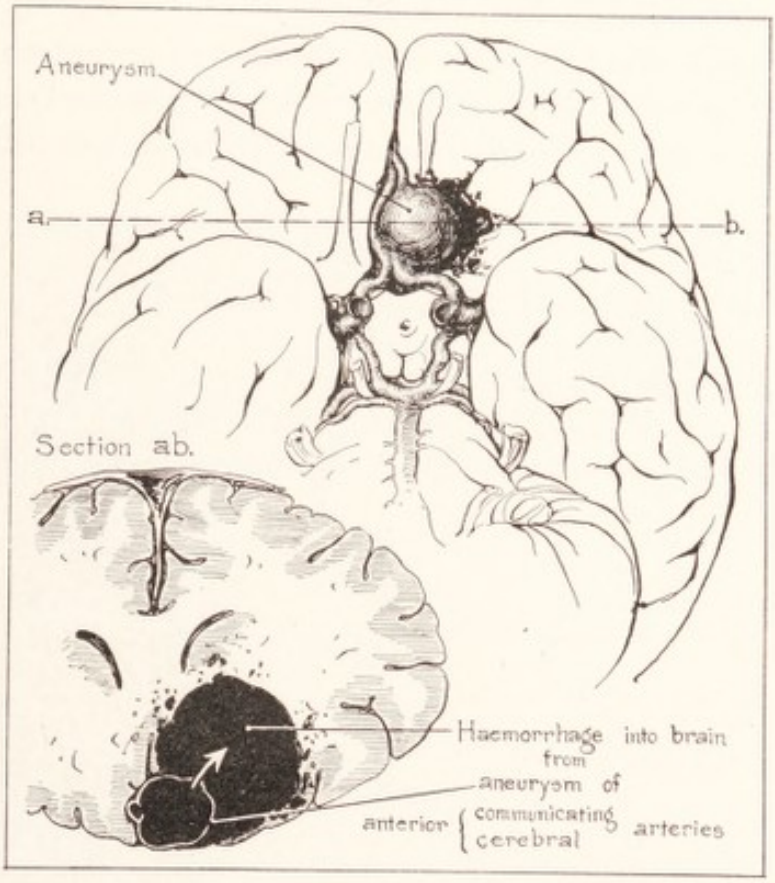


FIG. 19.—Drawing of a postmortem specimen showing aneurysm of the anterior cerebral artery with rupture into the brain substance.

in all! Another aneurysm of unusual size (3.4 x 2.9 x 3.2 cm.) in a woman of 65, was reported by Rice (1904). Roe (1850) found, in a patient only 21 years old, an aneurysm of this vessel which was the size of a hen's egg.

One of the large series (19 cases) of anterior cerebral and communicating arteries is by Courville and Olsen (1938)—all postmortem findings from Cajal's laboratory; they were 40 per cent of his series of 47 intracranial aneurysms. Busse (1921) described a group of 39 anterior cerebral aneurysms. He stressed the abnormalities found in the anterior cerebral and anterior communicating arteries from 400 examinations and concluded that the aneurysms were related to congenital variations. Other examples of aneurysms of

these arteries are found in the publications of MacKalty (1908), Drennan (1921, eight cases), Wallesch (1924, three cases), Booth (1909), Hermann and Macgregor (1940, in a patient only 4½ years old two aneurysms on these arteries, one large and one small), Reid and Glea (1931), Wichern (1912, three cases), Schmidt (1930, three cases), Epron (1890, two cases), Berger (1923, three cases), and Krayenbühl (1941, four cases).

Aneurysms of this type have been exposed at operation by Tönnis (1936), McConnel (1937), Cone (reported by Russel) and myself. These will be considered under the operative treatment of aneurysms.

D. Middle Cerebral Artery (Table D)

There are 26 aneurysms of the middle cerebral artery in 22 cases, 19.5 per cent of the total number of aneurysms. In this group there were also five additional aneurysms on other arteries, making a total

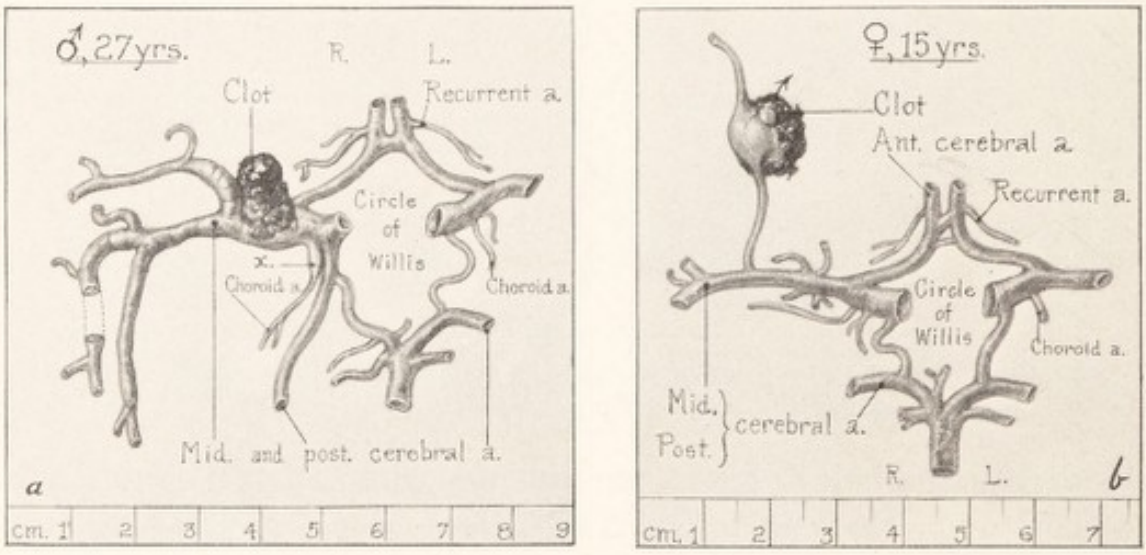


Fig. 20.—Two examples of aneurysms of the middle cerebral artery. (Table D.) (*a*) Case 4. A mycotic aneurysm arising at the origin of the middle cerebral artery. (*b*) Case 10. An aneurysm far out on a small branch of the middle cerebral. This is the only one of this type. Patient aged 15 years.

of 31 aneurysms in the 22 cases. In Case XVI there were three closely adjacent separate aneurysms on the left middle cerebral artery, and in addition, one on the right anterior cerebral artery. In Case XIX (Fig. 21) there was an aneurysm on each middle cerebral artery and two others elsewhere. In Case XXI there were bilateral symmetrical aneurysms at the origin of the middle cerebral artery. Aneurysms of

the middle cerebral artery fall into three groups, namely, (*a*) mycotic (five cases), (*b*) arteriosclerotic (two cases), and (*c*) congenital (15 cases with 19 aneurysms). In four cases (XVI, XVIII, XX and XXI) the aneurysms were found at necropsy and had caused no symptoms.

(*a*) *Mycotic*

Mycotic aneurysms are diagnosed as such because, from clinical examinations, endocarditis was known to be present or was demon-

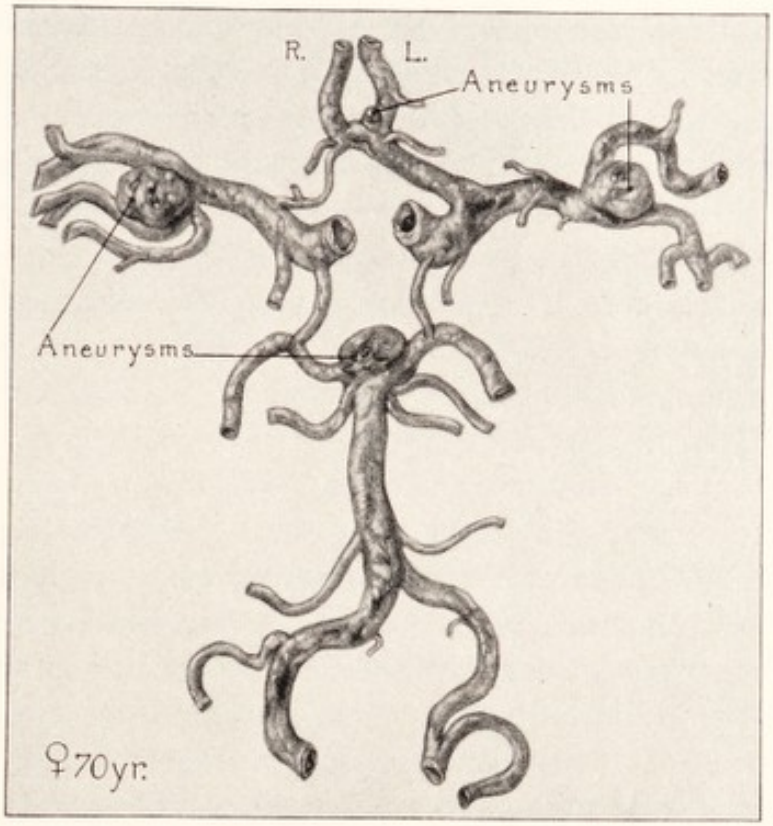


FIG. 21.—Case 19, Table D. Bilateral symmetrical aneurysms on the middle cerebral artery. In addition there was a small aneurysm on the left anterior cerebral and another large one at the anterior tip of the basilar artery.

strated at necropsy. From our series of aneurysms, the middle cerebral artery would appear to be the site of predilection for mycotic aneurysms, although in Case III there was another mycotic aneurysm on the posterior cerebral artery, and both were surrounded by cerebral necrosis and hemorrhage. In each instance the patient had been studied in the hospital for a period varying from several months to three years. The sudden and inevitable onset of hemiplegia from rupture was followed quickly by death in less than 48 hours in four cases,

and five days in a fifth. It is worthy of note that in Case V motor aphasia developed two months earlier and cleared in two weeks. Right hemiplegia and death then occurred within 24 hours. In Case IV there was an antecedent spell of severe headache three days before the onset of right hemiplegia and coma; death followed five days later. Only in Case IV (Fig. 20, A) were there convulsions. Four of the patients were females and one was a male. Three of the patients were in their teens and two were in the twenties. In all cases there was a frail, false, fibrinous, ruptured sac, surrounded by hemorrhage, in the Sylvian fissure and in the contiguous brain substance. Microscopically round and polymorphonuclear leucocytes abounded in the wall of the sac. From the Sylvian fissure the blood reached the subarachnoid spaces. In Case IV there was a purulent exudate about the site of the aneurysm. In each of the five cases streptococcus viridans was grown from the blood stream and isolated from the valvular vegetations at necropsy. The blood pressures in this group were normal or subnormal except in the patient who was unconscious with a large intracerebral hemorrhage (Case IV), and in this case the blood pressure was 140/88.

(*b*) *Arteriosclerotic*

Two aneurysms in this series are probably of arteriosclerotic origin (VI and VII). Both were in colored patients, one in a male of 45 years and the other in a female aged 61; both had ruptured. It is difficult to be certain when arteriosclerosis is the cause of an aneurysm, and, considering the high frequency of congenital aneurysms, the burden of proof lies upon this assumption, which is based upon (1) the existence of widespread arteriosclerosis, particularly in the neighborhood of the aneurysm, and (2) a generalized diffuse enlargement of the entire artery at the site of the aneurysm, or a relatively large opening in the arteriosclerotic wall (Case VI). The blood pressure was 200/115 in Case VI and 165/85 in Case VII.

(*c*) *Congenital*

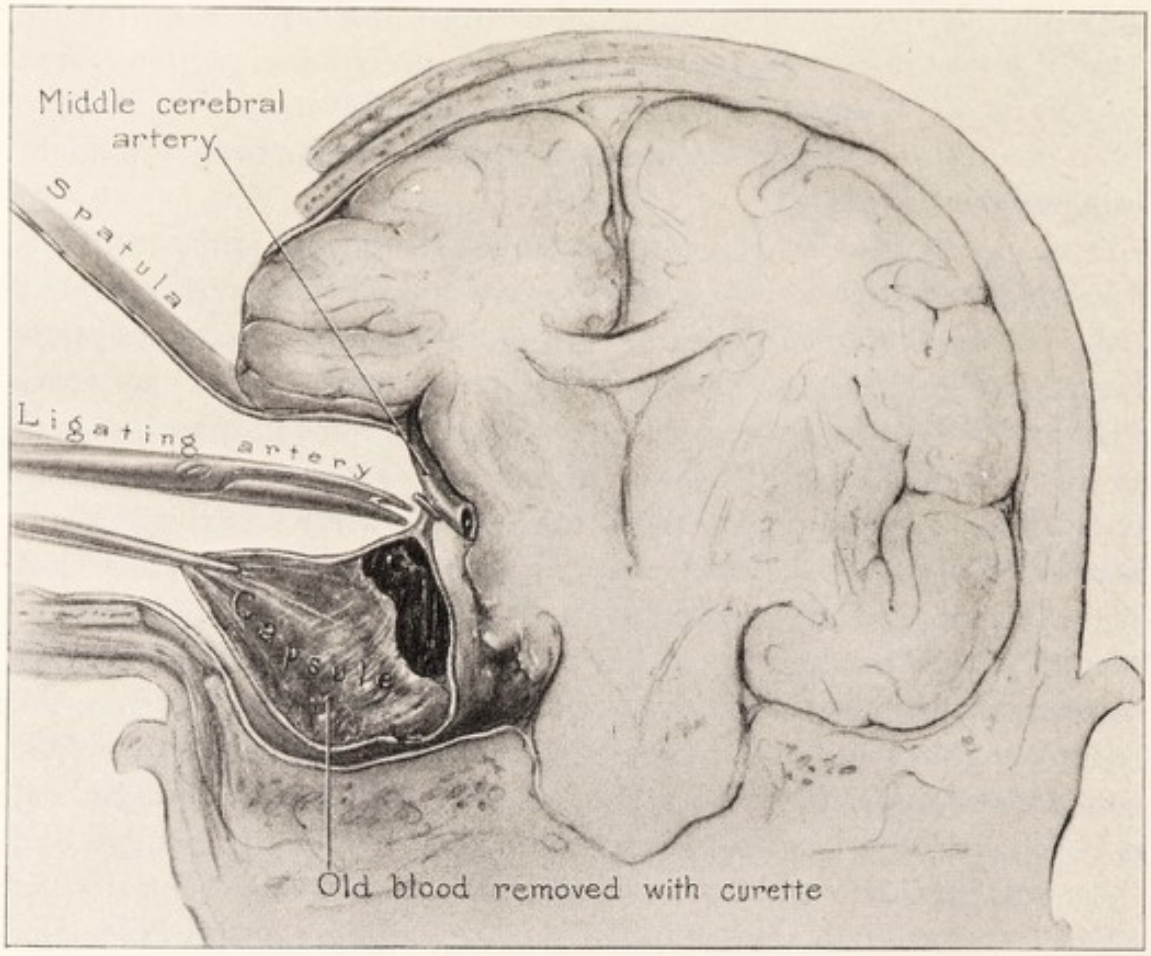
Incidence and symptoms. Of the fifteen cases, seven patients were males and eight were females; two were colored. The youngest was eight years, the next 16 years. After 20 the relation to ages by decades was fairly uniform to the sixtieth year, after which there was

only one (70 years). The duration of life after the first symptoms was 12 hours in two cases; one, two, and three days in three cases; and 12 days, 10 weeks, one year, and 15 years in the others. In Case IX a second hemorrhage occurred one year after the first, and death followed five days later.

Since four of the fourteen aneurysms in this group were symptomless and five patients were comatose very quickly after the rupture, there remain but few from whom symptoms could be elicited. Always there were severe headaches, usually generalized, but in two cases (IX and XIV) the pain, curiously, was in the back and neck as well as the back of the head. In Case XIX the headache was bifrontal. In Case VIII "something snapped in the head" (15 years earlier), headaches and petit mal attacks of epilepsy had been present since that time. The most common objective findings were as follows: (1) Hemiplegia, partial or complete in five cases (out of nine examined). (2) Major convulsions in four and petit mal in one; in only one were the convulsions known to have been unilateral, but it is highly probable that focal manifestations would have been found in all had careful observations been possible. (3) In four patients, and probably more, there was cervical rigidity resulting from subarachnoid hemorrhage. (4) The eyegrounds showed unilateral hemorrhages in three cases; in Case XIII large, round, subhyaloid hemorrhages also appeared in the contralateral eye fifteen minutes before death; in only one (Case XI) was papilloedema observed and it was bilateral. (5) A calcified shadow (not linear) was present in only a single instance (Case VIII) and was the only sign of localization of what was assumed to be a tumor.

Pathology. The aneurysms in this group show a striking similarity in the gross pathological picture. With few exceptions they are small berry-like bodies budding from the wall of the artery at some point in the Sylvian fissure. In Case XVI there were three quite similar unruptured aneurysms lying side by side on the left middle cerebral artery. The aneurysms in this group are roughly spherical or oval in shape, varying in size from 3 mm. to 12 or 15 mm. or even more in diameter, and are precisely like those situated elsewhere along the circle of Willis. The largest of the series was the size of a hen's egg (Fig. 22). In Case X the aneurysm arose far out in the brain substance from a small branch of the middle cerebral, and ruptured into the frontal lobe (Fig. 20, B).

The ultimate size and shape of these aneurysms may be greatly modified when they rupture into the cerebral substance. After cessa-



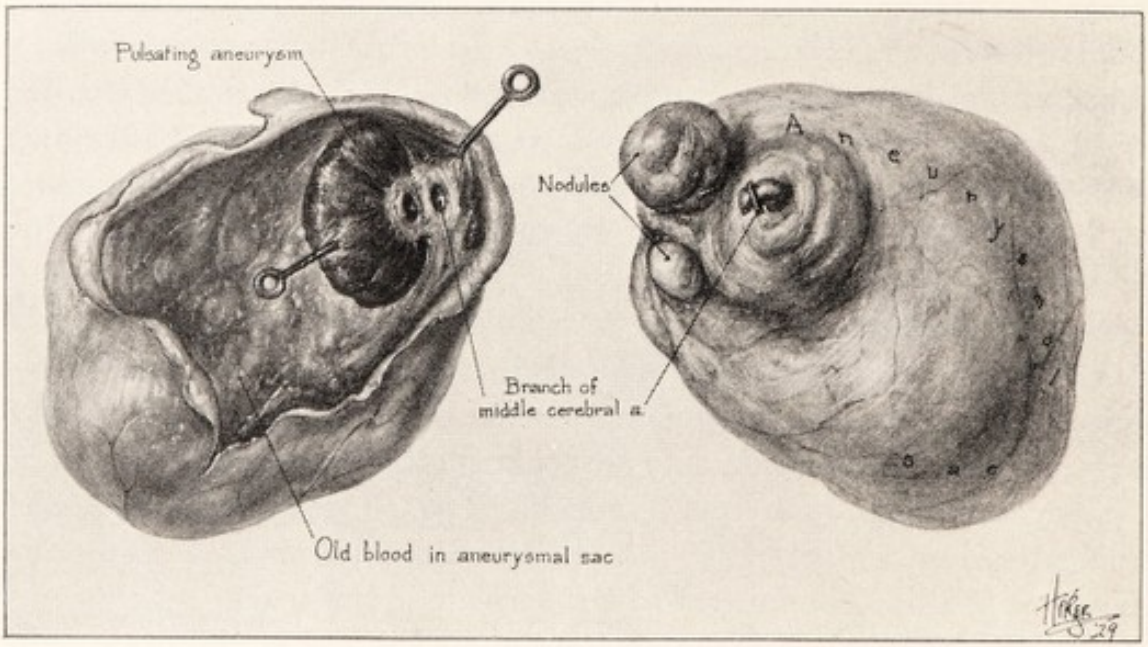


FIG. 22.—Case 8, Table D. Huge aneurysm of the middle cerebral artery encountered at operation. The enormous size of the aneurysm was due to the false sac. A solid blood clot lay upon the opening into the aneurysm and apparently sealed it.

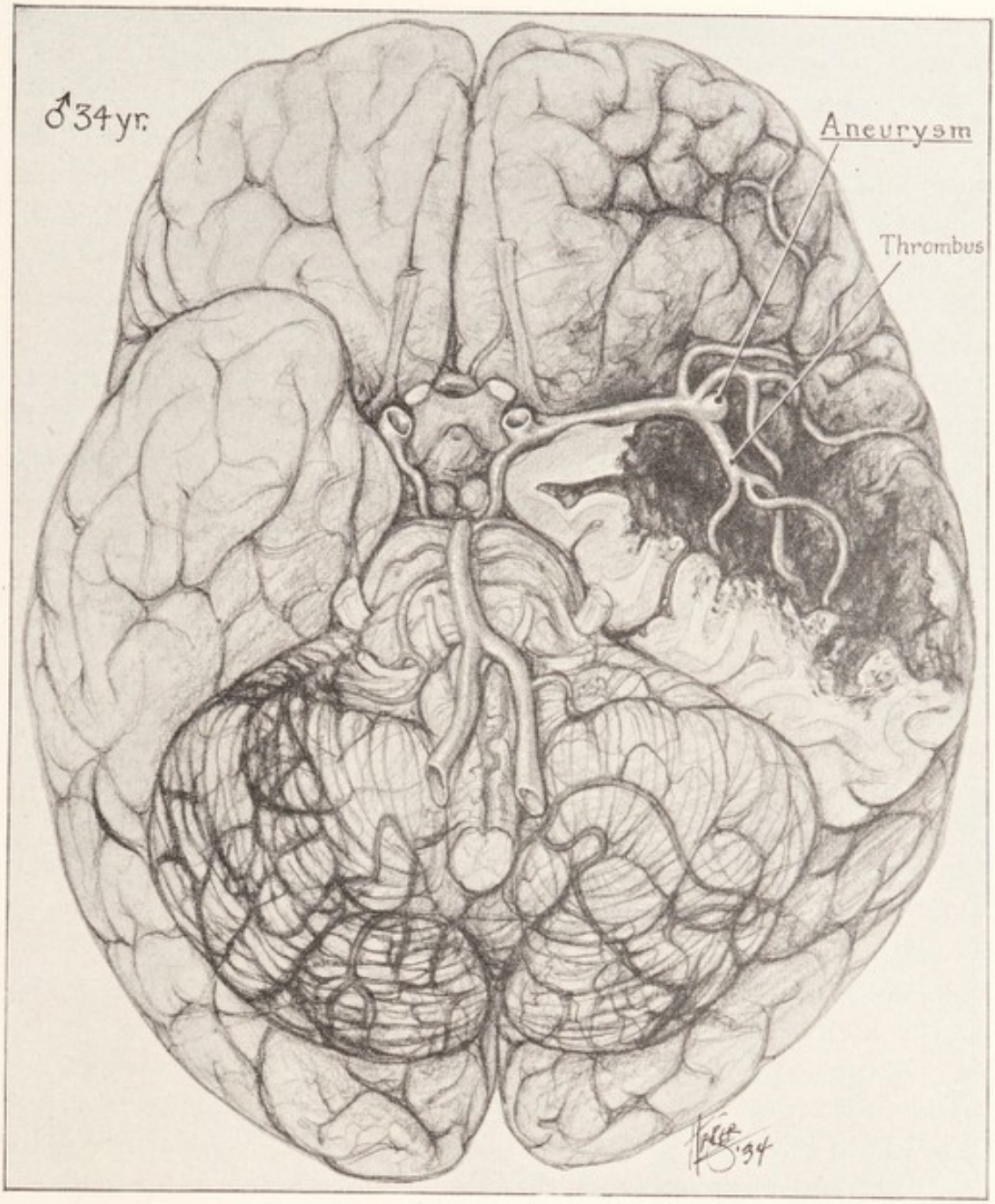


FIG. 23.—Case 9, Table D. Extensive hemorrhage of the brain arising from rupture of a small aneurysm on the middle cerebral artery. The hemorrhage had also ruptured into the lateral ventricle.

tion of the bleeding a large false sac may develop from the hematoma and become continuous with the original aneurysmal wall; Cases VIII and IX are examples of this. The development of a false sac can hardly occur except in the brain substance, where contiguous tissues restrain the bleeding and support the newly developing wall (Fig. 22). Such aneurysms are at times found at operation in patients who have general intracranial pressure and possibly signs of localization

that suggest a brain tumor. A careful history disclosing an abrupt onset of symptoms may yield the diagnostic clue to an aneurysm.

Aneurysms of the middle cerebral artery may rupture (1) into the Sylvian fissure, from which the blood passes into the cisterna chiasmatis and thence into the subarachnoid space, or (2) into the brain substance, whereupon an encapsulated hematoma may result if the patient survives (Fig. 22). The resultant destruction of the softened white matter of the brain may soon let the hematoma break into a lateral ventricle, with rapid death as the result (Figs. 23 and 24).

In six cases the hemorrhage occurred into the temporal lobe, and in five into the Sylvian fissure and subarachnoid space. In Case XVIII a small subarachnoid hemorrhage was found at the base of the brain, from an undetermined source. The aneurysm was at a distance from the hematoma and had not ruptured. In Case IX mild generalized headaches for a year attested the existence of an intracranial lesion. Five weeks before entrance to the hospital there were sudden knife-like pains in the "small of the back and neck," dizziness, diplopia, and ringing in one ear. One month later hemiplegia, aphasia, bilateral papilloedema with retinal hemorrhages, and coma indicated the high grade of intracranial pressure. At operation a large hemorrhage (black fluid blood) was encountered in the temporal lobe and most of this lobe and much of the subcortex of the Rolandic and post-Rolandic areas were necrotic from pressure of the hematoma on the softened white matter. The destruction of subcortex covered an enormous area of the brain. Since there had been no fresh bleeding nothing more was done, but death occurred two days later; the patient was in coma when the operation was performed.

One of the most interesting aneurysms of the entire series was from Case VIII (Fig. 22). Fifteen years earlier "something snapped in his head"; he had since had headache and petit mal attacks. A small area of calcification in the temporal lobe was the only sign of localization. A large, smooth-walled, false sac about 5 cm. in diameter filled the temporal lobe, just reaching the surface. It was entirely filled with lamellae of dense fibrous tissue and did not pulsate. Layer after layer was removed until the neck of the aneurysm was reached; it measured only 4 mm. in diameter. At this point the character of the clot changed; it was blacker and apparently fresher than the more distal clot and appeared discrete and separate from that overlying it. Like a trap door with rounded edges it filled the lumen of the sac. On one side the fusion to the wall was less firm; there was a suggestion of a

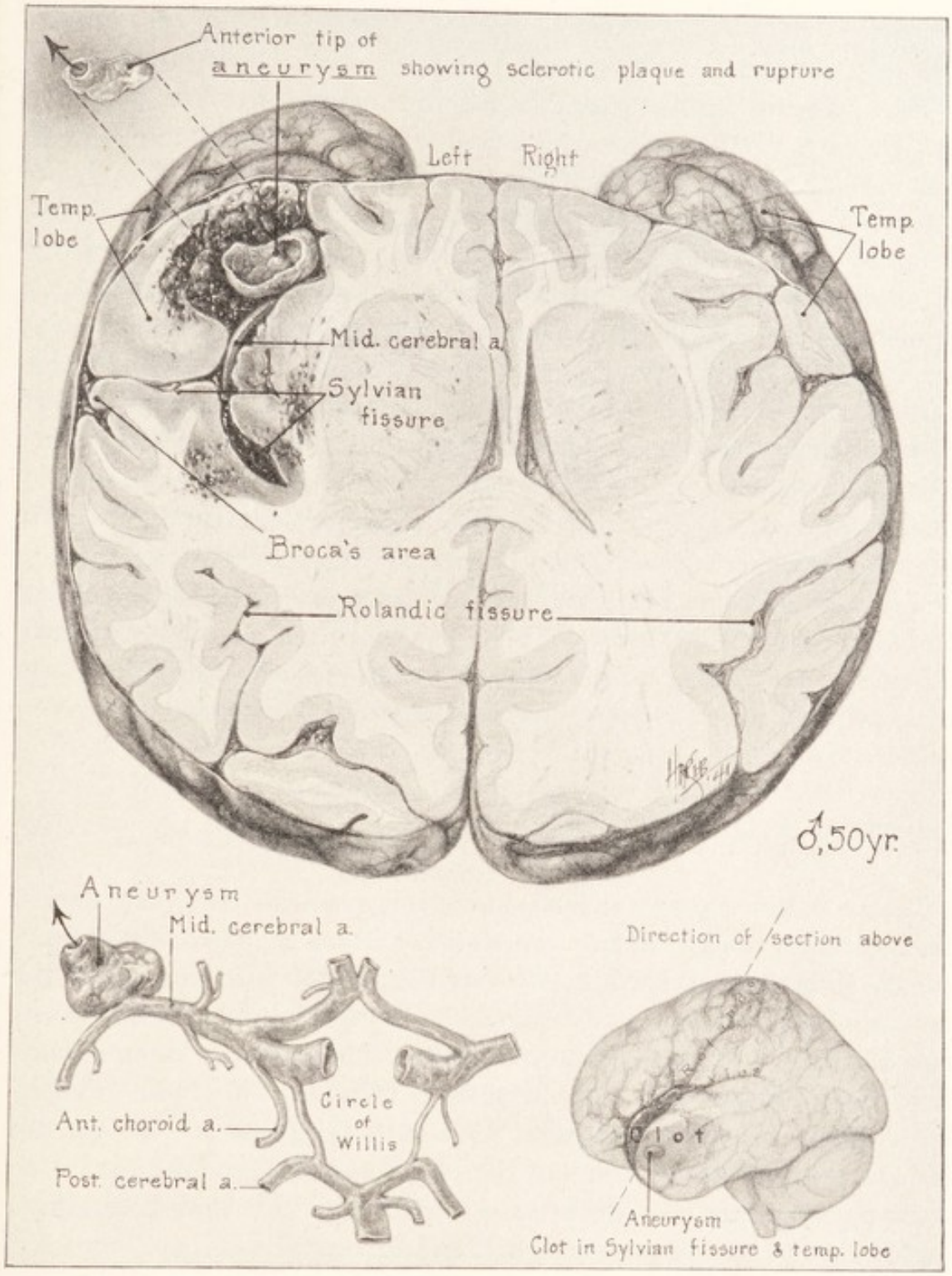


FIG. 24.—Case 17, Table D. Calcified aneurysm of the middle cerebral artery with rupture into the temporal lobe and descending horn of the ventricle. It will be noted that the aneurysm was located on the segment of the artery that contains no branches.

crevice. This was gently explored with the forceps and a burst of blood ensued. Bleeding was immediately controlled with the finger, and the neck of the sac, previously dissected free, was ligated with a silver clip. Death occurred 11 days later.

Literature on Aneurysms of the Middle Cerebral Artery

Berry aneurysms of the middle cerebral artery have been reported by Wichern (1912, eight cases, four of which were thought to be embolic), Elsberg (1918), Shaw (1901), Loewenhart (1923, six cases), Wallesch (1924), Parker (1926), Schmidt (1930, five cases), Lemmel (1931), and Krayenbühl (1941). Larger aneurysms of this vessel are reported by Church (1869) in two cases, in one of which the hemisphere was filled with a hematoma much as in our operative case; by Anderson (1885), two inches in diameter; by Pitt (1890), as large as a walnut; by Chase (1932), 3.2 cm. in diameter; and by Reichert (1939), 3 cm. in diameter. Multiple aneurysms on the middle cerebral have been reported by Bastian (1869), with three aneurysms (one of them ruptured); and by Pitt (1890). Aneurysms on the middle cerebral plus another intracranial artery were noted by Kersley (1934) and Garvey (1934). The most remarkable specimen was that of Evans and Courville (1939), with two large aneurysmal masses lying side by side on the left middle cerebral, a small one on the right middle cerebral, and another large mass on the anterior cerebral artery.

E. Posterior Communicating Artery (Table E)

There is not a single aneurysm on this vessel in the entire series. In most collections of aneurysms they are not infrequent. The reason for the difference is, I think, dependent upon the care with which the mouth of the aneurysm is dissected to determine its relation to the vascular tree. Although most of them lie on the posterior communicating artery, the sac actually enters the carotid and should be grouped with this vessel. The symptoms and signs produced by the sac are indistinguishable from those of the carotid because the sac lies in the same region. They soon drop upon the third, fourth, and sixth nerves and induce palsies or paralyses of these nerves. They do not, however, involve the fifth nerve and can therefore be differentiated in some instances from carotid aneurysms in the carvernous sinus.

An early report of these aneurysms is included in the series of Gull (1852), who found four in a group of 62. Rebert (1866) found seven in 86 cases. Epron (1890) had four in 28 aneurysms. Others have been reported by Hey (1898), Reinhardt (1913), Symonds (1923),

Berger (1923, two cases), Hassin (1927), Shore (1929), Schmidt (1930, two cases), Sands (1929), Dassen (1931, producing ophthalmic migraine), Kersley (1934), and Krayenbühl (1941).

F. Posterior Cerebral Artery (Table F)

In this series there are only two cases of aneurysm of the posterior cerebral artery, both of them necropsy findings. In the one case the aneurysm was mycotic, with another of similar character on the middle cerebral. In the other case the aneurysm was located on the circle of Willis, near the junction with the posterior communicating artery. Those on the circle of Willis are usually small and can scarcely be localized clinically or found at operation. They may give paralyses of the third, fourth and sixth nerves, precisely as do those on the posterior communicating artery. The aneurysms of the posterior cerebral in the brain substance are usually quite large and are indistinguishable from neoplasms in the same region. They may or may not give a contralateral homonymous hemianopsia, and if of sufficient size or if ruptured may cause visual aphasia (if located on the left side), or even sensory and motor changes from pressure upon the cerebral centers for these functions. When silent they may require ventriculography for localization.

An aneurysm of the posterior cerebral artery was reported by Van der Byl in 1855; it was as large as a hen's egg. Another unusual specimen, reported by Squire in 1857, was of the very large variety, measuring $4 \times 3\frac{1}{4}$ inches, and the patient was only 16 years old. Gull (1859) includes three such aneurysms in his series of 62 cases. Lebert (1866) found three of these among 86 aneurysms. Brownwell (1887) described one of the enormous aneurysms of the posterior cerebral; the walls were calcified and the interior was filled with a firm clot; because of the patient's obesity and the nearness of the lesion to the sella, he—at that early date—suspected that pressure upon the hypophysis might have been the cause. Other cases have since been reported by Wichern (1912), Reinhardt (1913, two cases, one aged 18), Berger (1923, two cases), Menninger and Dixon (1933), and Strauss, Globus and Ginsburg (1932). In the last two cases the aneurysm extended into the third ventricle. German (1938) resected one of these aneurysms with the occipital lobe, apparently with good results.

G. Basilar and Vertebral Arteries (Table G)

There are 21 aneurysms of the basilar and vertebral arteries, 15.8 per cent of the total number of intracranial aneurysms. Of these, 16 are from the basilar artery, five from the vertebrals, and one from the posterior inferior cerebellar artery (Case VII). In Case XIII the greatly dilated left vertebral artery was continuous with the basilar

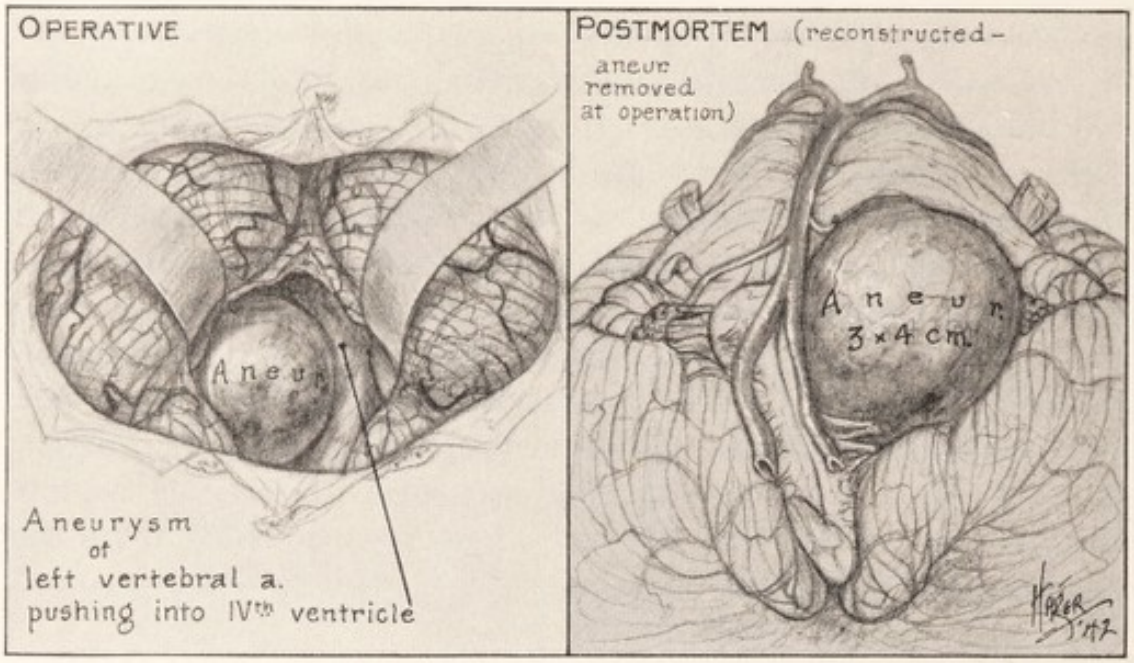


FIG. 25.—Case 1, Table G. Sketch of large sacculated aneurysm filling the posterior part of the cerebellar fossa and pushing into the fourth ventricle. It arose from the left vertebral artery.

aneurysm, and in Case XIV both vertebrals were included with the basilar. Doubtless many of these exposed at operation involve the vertebral artery also. There is no symptom or sign by which the vertebral artery could be suspected clinically instead of the basilar artery.

There are three distinct types of aneurysm in the posterior cranial fossa, as follows:

(1) Large sacculations, giving signs and symptoms of intracranial pressure plus those of localization to the brain stem. We have two cases, I (Fig. 25) and II.

(2) Smaller sacculations, similar to many of those on the circle of Willis (Fig. 21), and causing subarachnoid bleeding. We have eight cases, III, IV, V, VI, VII, VIII, XIX, and XX.

(3) S-shaped elongation of the arteriosclerotic basilar and/or vertebral arteries, with consequent lateral bulging that compresses

either the fifth or the eighth nerve or both. We have 11 cases, IX–XVIII inclusive and XXI (Figs. 9 and 26).

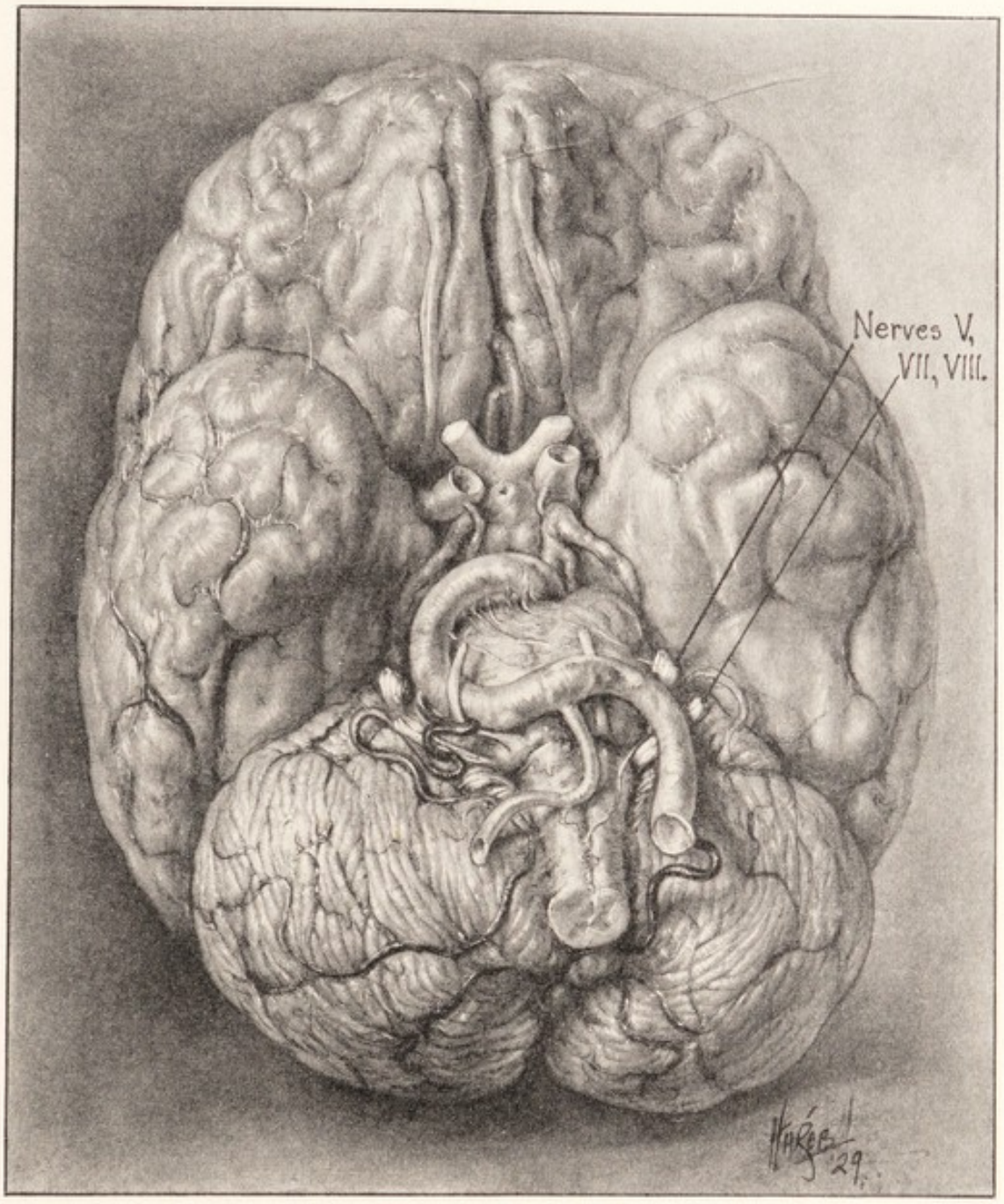


Fig. 26.—Case 13, Table G. S-shaped arteriosclerotic aneurysm of the basilar and left vertebral arteries.

Cases XIV and XX had no symptoms and were found at necropsy. It is worthy of note that 11 of the S-shaped aneurysms were operative findings. Were it not for this, the aneurysms of the basilar and vertebral arteries would be about 50 per cent less.

(1) *Large Sacculations*

The two large sacculated aneurysms could hardly be differentiated from tumors of the posterior cranial fossa. Neither had bled into the subarachnoid space at any time, but their steady expansion produced not only high pressure within the posterior fossa but blocked the aqueduct of Sylvius and the fourth ventricle, causing hydrocephalus. In Case I, 100 cc. of air was required to fill the ventricular system, which, in the ventriculograms, was occluded at the aqueduct of Sylvius. Case II was operated upon before the advent of ventriculography, but bilateral ventricular taps at operation disclosed very large lateral ventricles, and at necropsy hydrocephalus of high grade was disclosed. Bilateral papilloedema was present in Case II, but, despite the extreme grade of intracranial pressure, it was absent in Case I. It is of course well appreciated now that the absence of papilloedema means nothing and should never be emphasized in excluding a space occupying lesion of the brain. Far too many tumors are missed by this still too prevalent attitude. And frequently the resultant delay in diagnosis makes hopeless an operatively curable lesion.

Both of these aneurysmal masses induced definite cerebellar signs, but more important were the marked signs of bilateral involvement of the motor tracts, because they were deeply embedded in the ventral surface of the brain stem. If there could be any possible clue to the preoperative differential diagnosis of an aneurysm from a tumor in the posterior cranial fossa it would be this severe indication of involvement of the under side of the brain stem—even much more than the invasive tumors of the brain stem. Dysphagia and dysarthria are further indications of a lesion of the brain stem, but are so commonly present with tumors that they could hardly be indicative of an aneurysm.

It is noteworthy that both of these patients were young—the two youngest (ages 18 and 27) in the entire group of aneurysms in the posterior fossa. They were fairly well past the age (childhood) of extreme frequency of the medulloblastomata that invade the brain stem. This point might lead one in forthcoming cases to the diagnosis of an aneurysm rather than a tumor.

(2) *Smaller Sacculations*

The smaller sacculated aneurysms can hardly be differentiated from similar nubbins elsewhere along the circle of Willis. Three of these were at the anterior tip of the basilar artery, where it bifurcates to form the two posterior cerebral arteries (Fig. 21). In a fourth (Case III) there was a slit in the lateral wall of the basilar artery at the pons; but alongside the slit was a short, narrow, false sac into which a probe could be passed to the point of rupture.

Recently Kernohan and Woltman reported three fairly similar cases with focal necrosis of arteries in the posterior fossa—two of the posterior inferior cerebellar artery and one of the vertebral—all dying of subarachnoid hemorrhage. They had no explanation to offer concerning the cause. No similar lesions had been found in other cerebral arteries.

Case VI had a 5-mm. sac on the right vertebral artery. Case VII had a similar sac which was on the posterior inferior cerebellar artery and blocked the spinal canal. The ages of the patients (38, 45, 50, 52, 53, 64, and 70) were, on the whole, intermediate between those of the preceding and succeeding groups. Cases IV, V, VI, and VII were unquestionably of congenital derivation. The cause in Case III is uncertain. There was no preformed pouch, but only a longitudinal slit in an otherwise intact basilar artery. This aneurysm was in a male. Of the six congenital aneurysms five were in females, and two of the patients were colored.

The location of the aneurysms at the anterior tip of the basilar artery could never be suspected by any clinical analysis. Arteriography, performed by injecting a vertebral artery, as Takahashi (1940) and King (1941) have recently demonstrated, would undoubtedly disclose these and other basilar and vertebral aneurysms, but the site of the aneurysm must be suspected beforehand, for carotid injections would not disclose them. However, even if such localizations were possible, they could not be exposed at the tip of the basilar artery by any surgical attack now in use. It would perhaps be possible to resect a temporal lobe and thus gain access to such an aneurysm, for the anterior tip of this vessel is usually in full view when performing a third ventriculostomy for strictures at the Sylvian aqueduct. Such a view, however, is possible in hydrocephalus because of the increased exposure that results from tapping a lateral ventricle. To gain such an exposure without hydrocephalus would be possible only

by resecting a temporal lobe. It is not inconceivable that such an aneurysm might be thrombosed with the cautery, or perhaps its neck might be straddled with a silver clip.

All three of these patients died within two weeks, death resulting from subarachnoid hemorrhage. In Case IV the aneurysm was a sac, 2 cm. in diameter, indenting the anterior portion of the pons, and it compressed both the third and sixth nerves, giving paralysis of their functions. This and Case I were the only aneurysms of this group to produce a third-nerve palsy. Such findings with aneurysms in this location are not unlike those of aneurysms of the internal carotid and posterior communicating arteries, in which the same extraocular palsies are the most important signs of localization. It is quite possible that life might have been saved and the aneurysm cured in Case VII. At the time I saw the patient she was in deep coma, had a positive Queckenstedt test from closure of the spinal canal, highly xanthochromic fluid in the spinal canal, and fresh blood in the cerebral spaces. She died 24 hours later.

(3) *S-shaped Elongations*

The elongated S-shaped aneurysms of the basilar artery (11 cases) are a most interesting group and are in most part operative disclosures. Only two were under 60—Case XI, 55, and Case XVIII, 58; two were in the seventies and seven were in the sixties. All are unquestionably of arteriosclerotic origin (Figs. 9 and 26). With the exception of Case XI, blood presure 144/88, and Case XVII, blood pressure 150/76, there were marked signs of generalized arteriosclerosis, with severe hypertension, thickened radial arteries, and enlargement of the heart. Although only two of these cases came to necropsy, there can be no doubt that the pathological picture is similar in all. With advancing age and hardening of the vascular walls, the basilar artery widens and elongates, eventually assuming roughly the shape of an S. The lateral bend in the artery may come to rest upon either the trigeminal (10 cases) or the auditory nerve (Case XIII), or on both, either typical trigeminal neuralgia or Meniere's disease being the result. Six of these patients were males and five were females. Most surprising is the fact that in all except two of the 11 cases the lateral bulge of this aneurysm has been to the left. This can hardly be a coincidence, and yet the explanation is not clear. In Case XIII the left vertebral artery bulged upward and to the left, com-

pressing the auditory nerve, and even reached the trigeminus, but the big curve of the basilar artery was to the right (necropsy specimen).

There are several references in the literature to the greater tendency of the basilar artery to shift to the left.

With one exception nothing has been attempted toward improvement of the aneurysm, which, in no case, has produced any symptoms except the neuralgia or dizzy spells. Moreover, in view of the marked hypertension and generalized arteriosclerosis, it has not seemed that the tenure on life could be long. However, one patient, aged 68 at the time of operation (trigeminal) is still alive and well five years later; another lived two years, and another lived only three months. In two patients who have since died the cause of death is not known.

Literature on Aneurysms of the Basilar and Vertebral Arteries

The literature contains many examples of aneurysms of the basilar and vertebral arteries and their branches. Cruveilhier (1835) probably reported the first vertebral aneurysm; he records it in a beautifully colored drawing. The greatest number of reported aneurysms of these vessels are of the larger type, doubtless because they are more remarkable in appearance. An excellent example is given by Greenfield (1878); it projected forward, filling the whole region of the circle of Willis. Carpenter (1881) reports one on each vertebral artery at junction with the basilar; the patient had syphilis of the central nervous system and the aneurysms were presumed to be of this origin; he was only 30 years old, however, and the location of the aneurysms would make this explanation doubtful at least—and probably mistaken. Schultze (1875) reported a large vertebral aneurysm causing facial paralysis. Brinet's (1910) case was an oval aneurysm of the vertebral measuring 2×1.5 cm. That of Morrow (1921) was 3×2.5 cm., and was also on the vertebral. Krabbe and Backer (1922) show a basilar aneurysm as large as a hen's egg. Smith (1924) reports one of similar size, almost obliterating the fourth ventricle by its upward pressure; clinically the patient was thought to have opium poisoning. In Wells's (1922) case, the aneurysm was 3.5×3.5 cm.; the patient complained of dizzy spells and shortly before death had dysphagia. Other good examples of these aneurysms, ruptured or unruptured, are by Duguid (1925), whose patient was only 17 at the time of death; by Guillain, Schnite, and Bertrand (1930), and Wichern (1912)—aneurysms of the anterior inferior cerebellar artery.

Small sacculated aneurysms have been reported by Semple (1869), von Hofman (1894, 10 cases), Weidman (1915), Rhein (1914), Green (1928, two aneurysms, one at junction of the basilar and posterior communicating, and the other on the vertebral artery), and Bassoe (1939, two cases). Shore's (1929) case had an aneurysm $1 \times 1 \times 1$ cm. at the anterior tip of the basilar—a not uncommon location. Green (1928) presented a case with four aneurysms, one of which was at the anterior tip of the basilar artery; another was a fusiform dilatation of one vertebral artery; the patient was only 21.

The diffuse S-shaped enlargements of the vertebral and basilar arteries have been reported by Möser (1884) and Ruston and Southard (1906, both vertebrals and numerous miliary aneurysms on other branches of the circle of Willis). Shore's (1928) case had two fusiform dilatations of the basilar and one of each internal carotid.

III

THE CIRCLE OF WILLIS

Its Embryology and Anatomy

By Dorcas Hager Padget

§ 1. EMBRYOLOGY

No study of the congenital aneurysms of the cerebral arteries—or the arterial anomalies which occur frequently in association with such aneurysms—would be complete without a consideration of the embryology of these vessels. Leading textbooks on anatomy and embryology do not give a comprehensive picture of the development of the arteries at the base of the brain. No revision of the 1912 edition of the Keibel and Mall *Manual* has appeared, and the account of the cranial arteries in Evans' chapter on the vascular system is based largely on the early work of Mall (1905) and others. Streeter's classic article (1918) is chiefly concerned with the venous system. Congdon (1922), in an exhaustive study of the aortic arch system, described the significant features of the origin of carotid, basilar, and vertebral arteries. To supplement the available information and gain a clearer understanding of the developmental story, the writer, through the courtesy of Dr. George W. Corner, undertook a study of the incomparable material of the Carnegie Embryological Collection. Over 40 graphic reconstructions were made of 17 embryos from 3 mm. to 43 mm. in crown rump length, approximately 3–8 weeks old, and the following (summarized) account of the arterial configuration at various stages is based on this material.

Development of the Permanent Arteries

The essential adult pattern of the circle of Willis may be recognized as early as the second month, before the acquisition of the accessory vessel coats. The source of origin of all the definitive arteries is distinct at this time, although their final direction and relative

sizes are yet to be determined by the further growth of the brain, in particular the gradual predominance of the cerebral hemispheres. The early process of development, as established by embryologists, is characterized not by the outgrowth of definitive vessels as such, but by the elaboration of certain channels and the comparative or total disappearance of others in an all-embracing primitive plexus. This process is in accordance with the contemporary needs and relations of the areas to be supplied.

Figure 27 is an 18-mm. embryo (Carnegie Coll., No. 1390) approximately 6 weeks old, and although some temporary arteries still exist, the adult pattern is clearly foreshadowed. The circle of Willis is not yet definitive, because the region of the anterior communicating artery is still grossly plexiform. Furthermore, owing to the brain curvatures, the circle is confusing in any base-view portrayal until the 30–40 mm. stage is reached. Note, for instance, that in the base view of Figure 28, A, the posterior communicating artery appears to proceed anteriorly from carotid to basilar; the choroidal arteries are also directed anteriorly, and the hypophysis appears to be behind the anterior tip of the basilar artery.

The Cerebral Arteries in Order of Their Appearance

The internal carotid artery first appears at approximately the 3-mm. stage. At 4 mm. one sees its two main divisions: (1) the anterior (primitively the supply of optic and olfactory regions), which later gives rise to anterior and middle cerebral and anterior choroidal arteries, and (2) the posterior division, which remains as the posterior communicating artery and gives rise to the posterior choroidal and posterior cerebral arteries. The posterior extensions of the posterior communicating arteries, reinforced by temporary branches of the primitive internal carotid (which for some time lies in close contact with the hindbrain), form bilateral longitudinal channels, which at the 5–8 mm. stage are consolidated to form the basilar artery. Posteriorly the basilar, for a varying period after formation, is connected with the cardiac source of supply only through small irregular channels leading to the first cervical segmental branches of the dorsal aorta.

In embryos of 7–9 mm., the future anterior and posterior choroidal arteries, directed toward the choroid fissure, are the only definite branches of the anterior and posterior divisions of the internal carotid. The superior cerebellar artery is the sole supply of the develop-

ing metencephalon or future cerebellum. At the end of this period the vertebral artery begins to take form. At the 11–12-mm. stage the middle cerebral branch, although still often plexiform, is definite as the primary supply of the developing cerebral hemisphere.

At the 14-mm. stage the vertebral artery is almost complete. It has formed as a longitudinal anastomosis between the upper cervical seg-

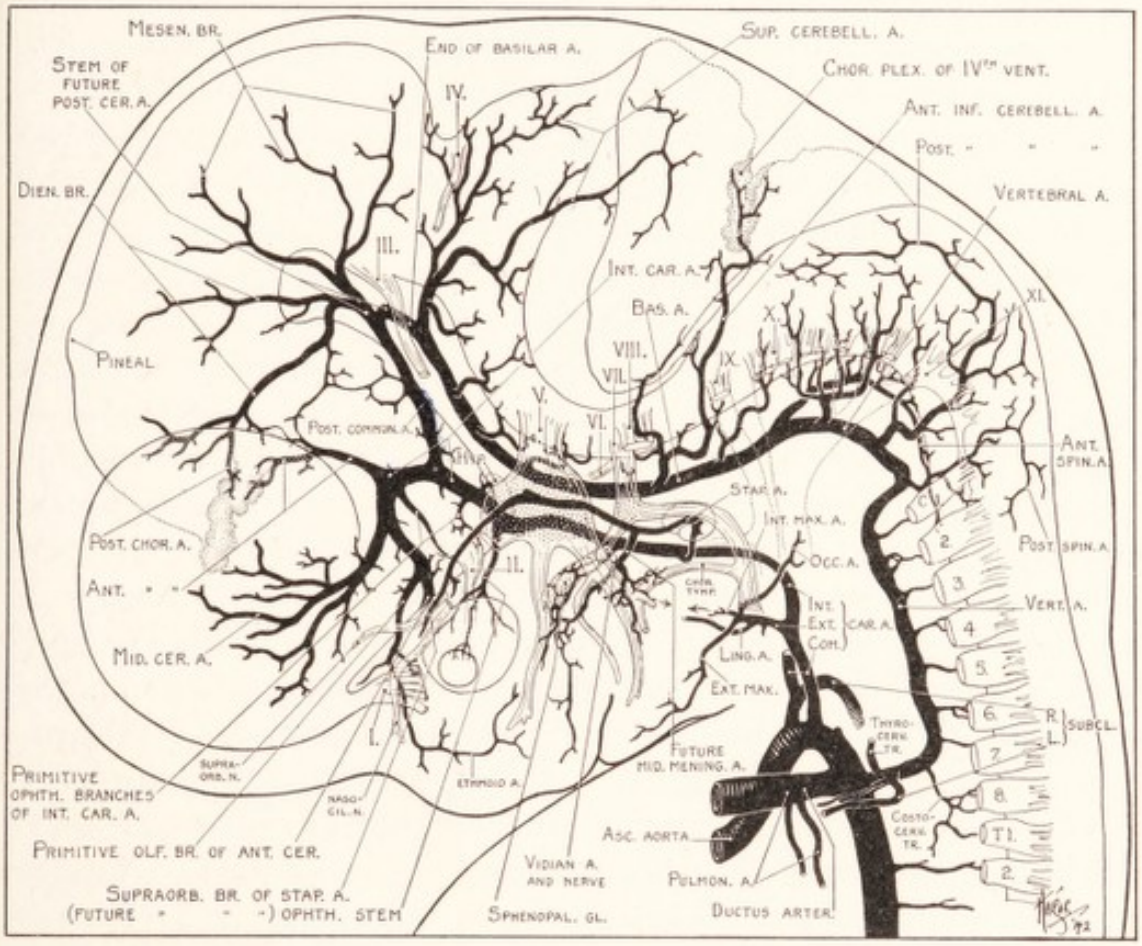


FIG. 27.—Graphic reconstruction of embryonic arteries of an 18-mm. human embryo of approximately 6 weeks. Note the remnants of the primitive ophthalmic branches on the future intracranial portion of the internal carotid, i.e., above the permanent ophthalmic stem.

mental branches of the dorsal aorta, with the subsequent obliteration of all the aortal connections except the seventh segmental, which remains as its subclavian stem of origin. Through the first segmental artery, mentioned above, the vertebral is now connected with the basilar artery. Until this time all the cerebral arteries were supplied primarily by the carotid system. This primitive human arrangement is characteristic of the adult form of a number of lower vertebrates; generally speaking, the vertebral system is an acquisition of higher forms (De Vriese, and others).

At the 18-mm. stage (Fig. 27) the stem of the anterior cerebral artery, which still gives rise to the very prominent olfactory branch, is continued medially towards its fellow of the opposite side. The permanent ophthalmic stem is now recognizable, although it gives rise to only ocular and not yet orbital branches. Both primitive inferior cerebellar arteries have emerged from the elaborate plexus that heretofore supplied the posterior portion of the hindbrain; the anterior terminates in the primordium of the choroid plexus, which has just begun to invaginate from the thin roof membrane of the fourth ventricle.

In embryos of 21–24-mm., the anterior communicating artery has become a definite channel, and the anterior cerebral arteries extend up between the cerebral hemispheres. A smaller, median anterior cerebral artery arises from the anterior communicating artery (Fig. 28, A). This vessel persists normally in many vertebrates and anomalously in man as a single anterior cerebral, accompanied by a relative disappearance of the two lateral anterior cerebral arteries. De Vriese (1905) found this "median artery of the corpus callosum" highly developed in a large percentage of human embryos. The recurrent or Heubner's artery from the anterior cerebral opposite the anterior communicating artery passes laterally to complement branches of the middle cerebral arteries in supplying the basal ganglia. The ophthalmic artery has now acquired its main orbital branches from the anastomosis with the most anterior of the three divisions of the temporary stapedial artery (Fig. 27). The diencephalic branch, which gives rise to the posterior choroidal artery, probably represents a portion of the posterior cerebral, since the posterior choroidal is a branch of the latter in the adult. It is emphasized, however, that the stem of origin of the posterior cerebral, i.e., the divisional branch of the basilar, is usually present from the earliest stages. It was originally the terminal part of the primitive posterior communicating artery, which joined its mate of the opposite side to form the anterior end of the basilar artery.

Therefore it is seen that, with the formation of the anterior communicating artery at about the 22-mm. stage, when the embryo is

FIG. 28 (on opposite page).—A. Composite diagram: The human embryonic arteries at the base of the brain as seen in stages from 12 to 24 mm. The permanent vessels, in roughly their relative sizes, are shown in black; shaded areas are the transitory vessels, or indicate temporary embryonic importance and size. B. The location of reported aneurysms not occurring at a bifurcation. Those on the internal carotid, anterior cerebral, and vertebral arteries occur at the sites of temporary embryonic branches. C. The reported adult anomalies occurring both with and without associated aneurysm. The shading facilitates comparison with the embryo.

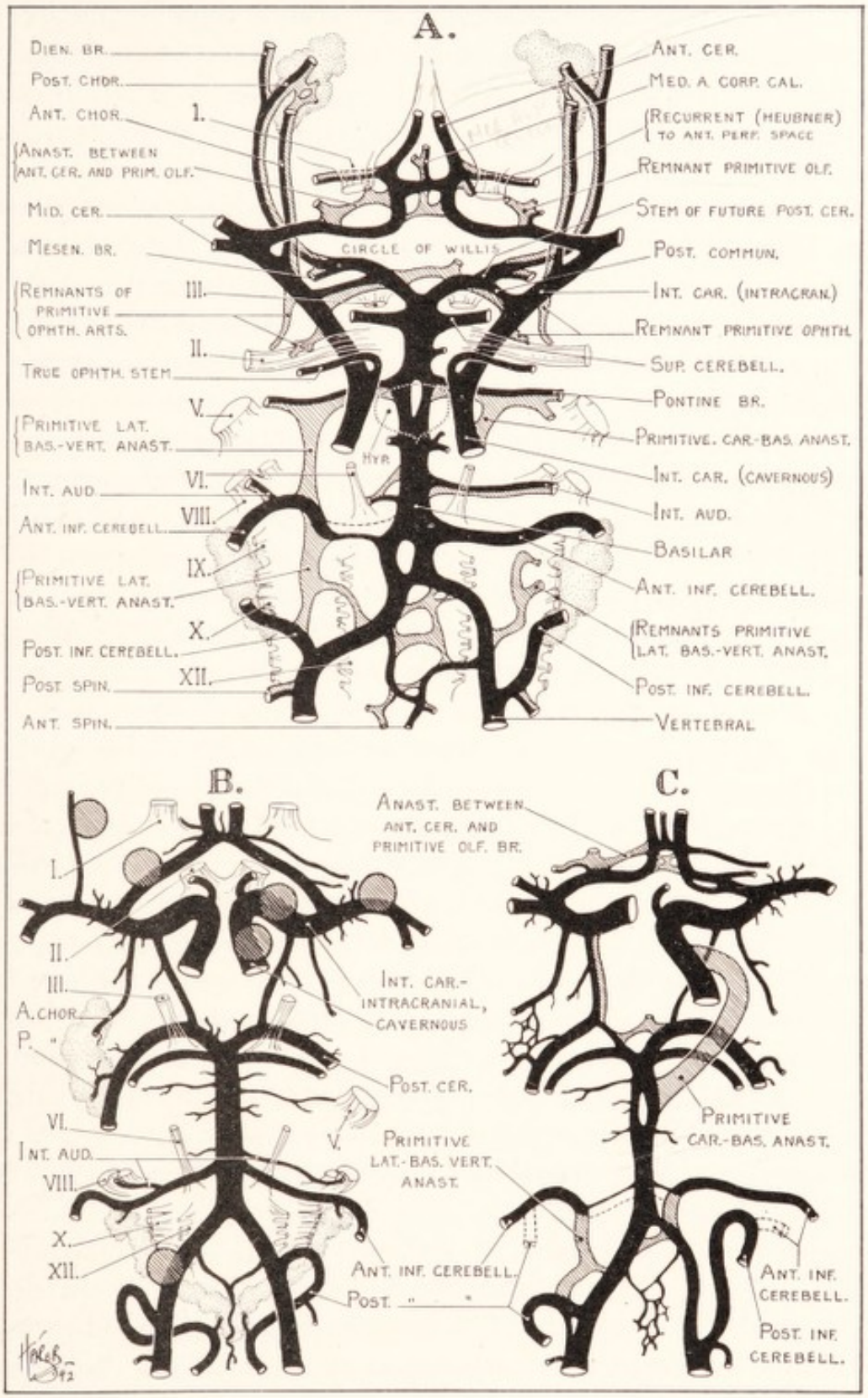


FIG. 28.—See foot of opposite page for interpretation.

only six or seven weeks old, the circle of Willis is complete, includin the permanent origin of all its associated branches.

The Transitory Arteries

The temporary branches and anastomoses of the first two months of development are of especial interest. Some of these are to be noted in Figure 27, at the 18-mm. stage. On the future petrous portion of the internal carotid there is the stapedial artery whose branches, after obliteration of the carotid connection, are taken over by the developing internal maxillary as its middle meningeal branch. The supraorbital branch of the stapedial finally becomes the supraorbital or frontal branch of the ophthalmic artery, and in the adult the embryonic connection between the middle meningeal and ophthalmic may be retained as a source of collateral circulation between the internal and external carotid arteries, or the anterior branch of the meningeal may take the place of an absent ophthalmic artery.

On the future cavernous portion of the carotid there may persist at this stage the carotid-basilar anastomosis (see below and Fig. 28); and on the future intracranial portion, i.e. above the ophthalmic, there are several temporary branches. Note, in Figure 27, a vessel between the ophthalmic and posterior communicating arteries ending in the remnant of the elaborate primitive plexus, which supplied the optic stalk before the permanent ophthalmic stem could be identified. This channel and the carotid branch opposite the anterior choroidal (extending laterally as far as the developing cartilage of the optic foramen) are both remnants of the early arterial supply to the optic stalk and cup when these structures were more closely related to the lateral brain wall from which they were evaginated. Such remnants are therefore labeled "primitive ophthalmic" and were repeatedly seen at various stages in embryos from 7 to 24 mm. in length.

The foregoing and most of the other transitory branches are summarized in a diagram (Fig. 28, A), which represents the stages during which the circle of Willis becomes recognizable. This figure is based on the graphic reconstruction of 10 embryos, 12–24 mm., approximately 5–6 weeks old.*

No persistent embryonic branch is more striking in the adult than the carotid-basilar anastomosis. It has been reported as a

* Carnegie Coll.: Nos. 544, 1171, 940, 390, 144, 1390, 460, 6202, 966, and 632.

gross anomaly by a number of authors (see below), but heretofore its embryonic derivation has not been described. It is first identified at the 3–4 mm. stage as a branch of the primitive internal carotid, contributing markedly to the bilateral channels which later join to form the basilar. It may persist for a varying length of time after formation of the basilar and it may be represented in the normal adult by small branches of the basilar and carotid arteries to the region of the fifth nerve root or ganglion.

The anterior cerebral stem is originally the supply of the primitive olfactory region (see the lateral view of Figure 27). In embryos of 4–12 mm., it courses ventrolaterally, to end either in the olfactory area or at the olfactory pit. About the 14-mm. stage, when the olfactory nerves become definite and the nasal fossae are more medial in position, these olfactory arteries on either side are drawn together towards the mid-line. Meanwhile, on each anterior cerebral stem, a medial branch has appeared which represents the true continuation of the anterior cerebral artery, and anastomoses between the lateral (olfactory) and the medial (anterior cerebral) branches may be seen (Fig. 28, A). At about the time of formation of the anterior communicating artery from the plexus between the medial branches of either side, the primitive olfactory branch has begun to disappear. It may be represented in the normal adult by a very small, inconstant branch of the anterior cerebral artery to the anterior perforated space. The recurrent artery, almost constant in appearance but not size (not mentioned by name in leading textbooks of anatomy but established by Ayer and Aitken, 1909), is probably also a remnant of this early olfactory complex.

The plexiform connection between the early anterior and posterior choroidal arteries may be retained to a greater or lesser degree in the adult, as described by Abbie (1934).

The islands or holes often seen in the basilar are, of course, remnants of the original paired condition. The origin of the superior cerebellar artery, established very early in the embryo, is relatively constant in the adult. The internal auditory artery is a branch either of the basilar or of the anterior inferior cerebellar in both embryo and adult.

In the early embryo most of the basilar and vertebral branches arise in definite relation to the cerebral nerves. Since nerves IX, X, and XI laterally, and XII medially, are closely associated and their root origins together cover a relatively large area on the embryonic

brain stem, there is a rather elaborate arterial branching and anastomosis in the region where the vertebrals join the basilar. This arrangement permits a wide variation in the origin of both inferior cerebellar arteries, and remnants of this lateral basilar-vertebral complex often persist in the adult.

The posterior spinal artery may arise either from the vertebral or the posterior inferior cerebellar artery. The anterior spinal may show evidence that it, like the basilar, was originally paired in the bilateral longitudinal channels that very early extended from the midbrain to the posterior end of the spinal cord.

The form of the temporary branches, as they may occur in adult anomalies, are summarized in a composite diagram (Fig. 28, C).

Possible Relation of Transitory Arteries to the Site of Aneurysm

The sites of all the reported aneurysms not related to an arterial bifurcation are shown in Figure 28, B. There appears to be a rather striking tendency for such aneurysms to occur on the anterior cerebral, the internal carotid, or the vertebral artery, where marked change and obliteration of vessels characterize the course of embryonic development.

The ruptured anterior cerebral aneurysm of the "lead" case in Forbus' well known article was located at such a site (see his Case 2, Fig. 35), and, as he stated, only minute branches were present; these were undoubtedly remnants of the primitive olfactory artery described above.

It is interesting that the youngest instance of aneurysm found in the literature * was in a two-year-old boy reported by Dial and Maurier. The aneurysm was described and illustrated as on the upper vertebral artery "not related to any bifurcation," with an associated "malformation of the vessel wall." A similar aneurysm in our series is shown in Figure 35, Case 20.

§ 2. ANATOMY

The circle of Willis is neither adequately described nor accurately illustrated in many textbooks of anatomy. The following is an attempt to consolidate the available information regarding

* The patient noted as the youngest in the 1125 cases collected by McDonald and Korb (1938) was not 1½ but 9½ years old, according to the original article.

the anatomical basis for a normal collateral circulation at the circle of Willis, and to review the reported deviations from the normal in cases with and without associated aneurysm.

Normal Configuration

First it is necessary to establish some standard for a normal, typical, or average circle (Fig. 29, A). This was determined after a study of the literature plus many personal sketches in a consecutive general autopsy series. That there is a difference in the relative size of the arteries between adult and fetus was suggested by De Vriese (1905), and the diagram (Fig. 29, B) agrees with her detailed description in 75 per cent of 100 injected fetal brains (see also Fig. 33, E). In the fetus as in the embryo, there is apt to be less relative difference in the size of the component arteries than is found in the typical adult. Furthermore, there is some indication that beyond middle age one finds the highest incidence of relatively small arteries. For instance, Fetterman and Moran (1941) found actually "thread-like" posterior communicating arteries in 20 per cent of 200 cases averaging 59 years in age, and all over 38.

The incidence of normal posterior communicating connections in four series of adults (1033 cases) from the literature is summarized in Figure 33 and is less than 50 per cent. The anterior arteries at the circle could not be included in these diagrams but may be considered present, and at least of normal size in about 90 per cent. Though no statement as to size or length of the anterior communicating artery was made by any author, it was apparently considered normal in 68 per cent of 1803 adult brains (Adachi-Hasebe, 83; Blackburn, 220; Busse, 400; De Vriese, 50; Fawcett and Blackford, 700; Stopford, 150; Windle, 200), while various forms of duplication were described in the remainder. The normal form of this artery is probably best described in Cunningham's *Textbook of Anatomy* as "wide but short" (Fig. 29). Busse (1921), whose study in a large series was exclusively concerned with the region of this artery, found it normal in only 43 per cent of 400 specimens, but the remainder were duplicated forms. The anterior cerebral artery was apparently within normal limits in over 90 per cent of 553 cases (Adachi-Hasebe, Blackburn, De Vriese, and Windle) and was absent in only two cases. No statement regarding the size of the anterior cerebrals was made by Fawcett and Blackford (1906) in their

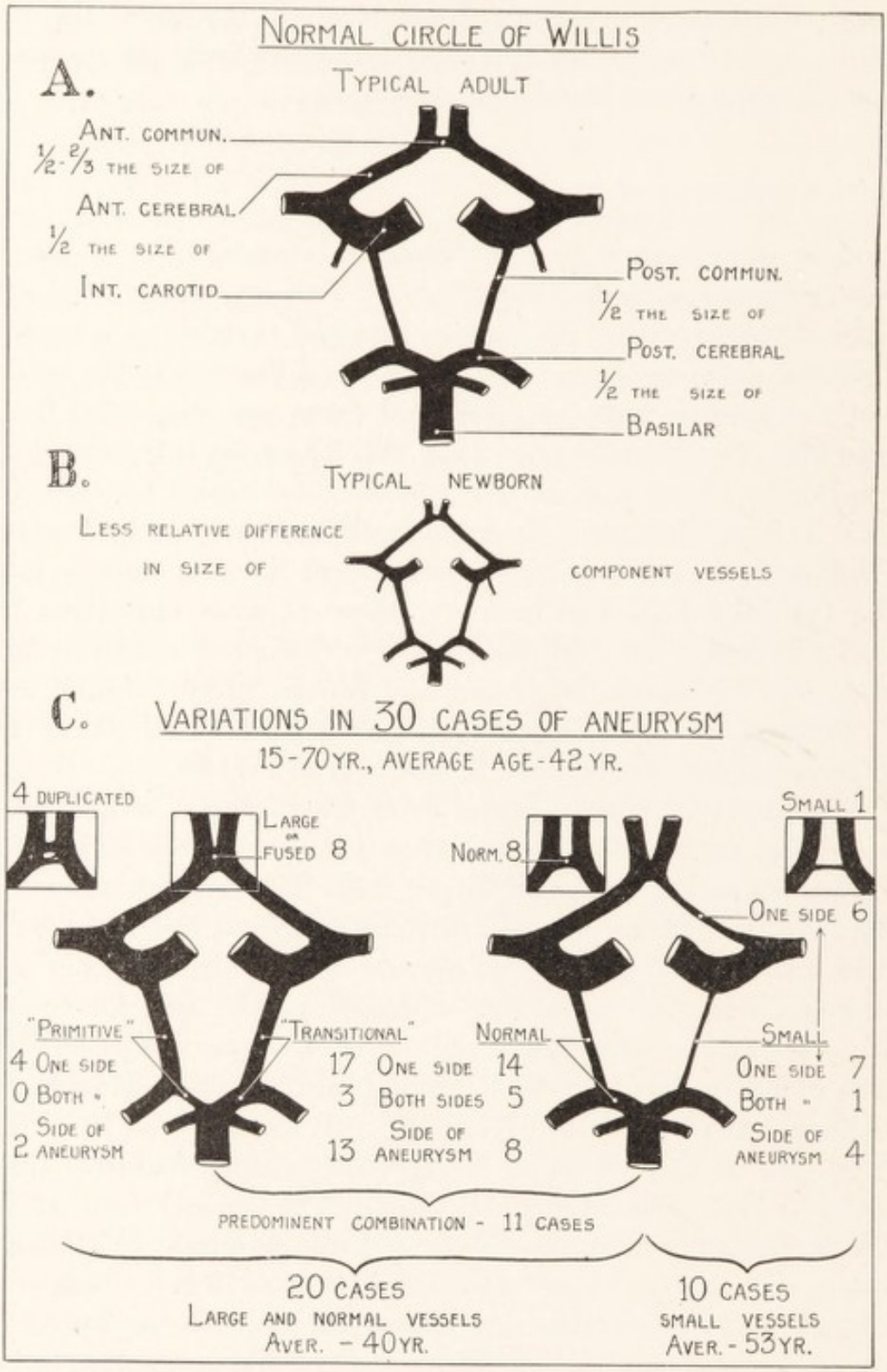


FIG. 29.—A and B. Contrast the typical difference in relative size of the adult and fetal arteries. C shows that, in 67 per cent of the 30 aneurysms of this series available for study, the circle resembled the fetal. The remaining 33 per cent possessed one or more small or impervious vessels and the average age was well above the group possessing normal or large collateral.

700 cases. From the evidence at hand, the anterior part of the circle appears to be more dependable than the posterior, as a source of potential collateral circulation.

Two other diagrams in connection with the normal configura-

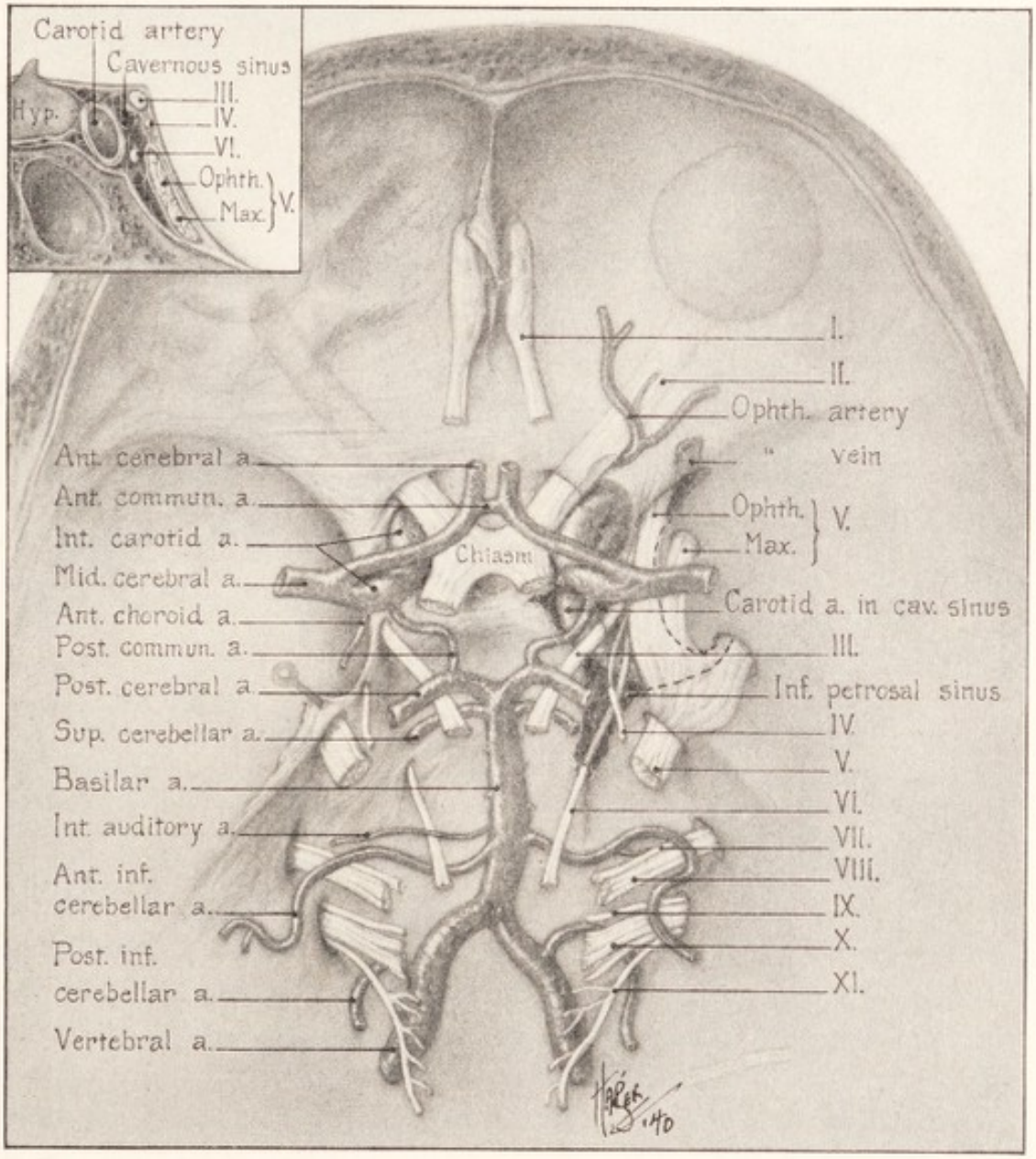


FIG. 30.—The typical relationship of the cranial nerves to the cranial arteries at the base of the brain and in the cavernous sinus. (Courtesy of Dr. Frank Walsh.)

tion are presented. Figure 30 shows the relation of arteries to nerves, as viewed in place at the base of the skull. Figure 31 may aid in the understanding of a rather common source of error revealed in descriptions of the origin of certain aneurysms near the terminus of the internal carotid artery, and in diagrams of the circle of

Willis. As emphasized in the embryo, the internal carotid terminates at its bifurcation into anterior and middle cerebral arteries; the anterior choroidal artery is definitely a branch of the carotid, and the posterior communicating artery arises even more proximally on the carotid stem. The intracranial length of the internal carotid may vary and its branches may arise relatively high or low, but the relation of branches to bifurcation is constant. Statements such as "middle cerebral at the posterior communicating" are incorrect, and yet have been made repeatedly in reports—for

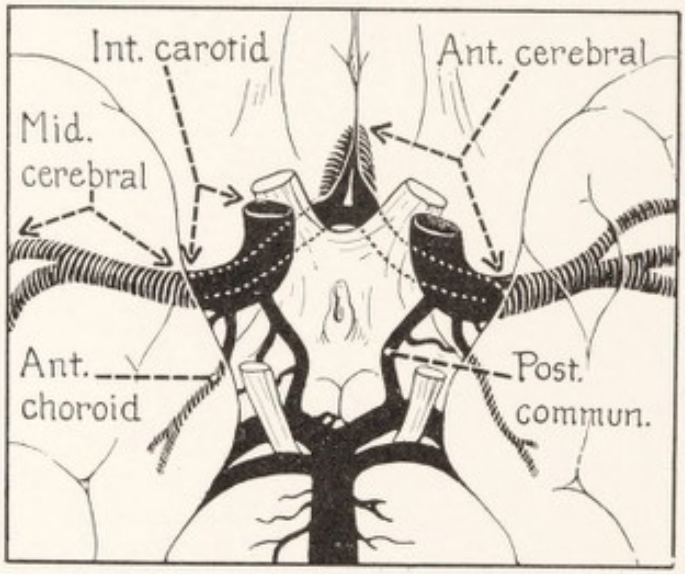


FIG. 31.—The normal circle of Willis in place at the base of the brain. Attention is called to the relationship of the internal carotid and middle and anterior cerebral arteries, and to the fact that the posterior communicating and anterior choroidal are both branches of the internal carotid, not the middle cerebral artery. (See text.)

instance in the 1125 aneurysms collected by McDonald and Korb (1939), who of course quoted the original authors. The fact that such aneurysms actually arose from the internal carotid could usually be confirmed in the detailed descriptions or illustrations of these cases. Aneurysms of the internal carotid must usually be intracisternal and readily seen on inspection of the base of the brain; those of the middle cerebral are usually intracerebral. Consideration of this source of error would alter the high percentage of middle cerebral, as compared to internal carotid aneurysms, in the collected groups of McDonald and Korb and others, and would more nearly approximate our own findings in which the internal carotid was the commonest site of aneurysm (see Fig. A).

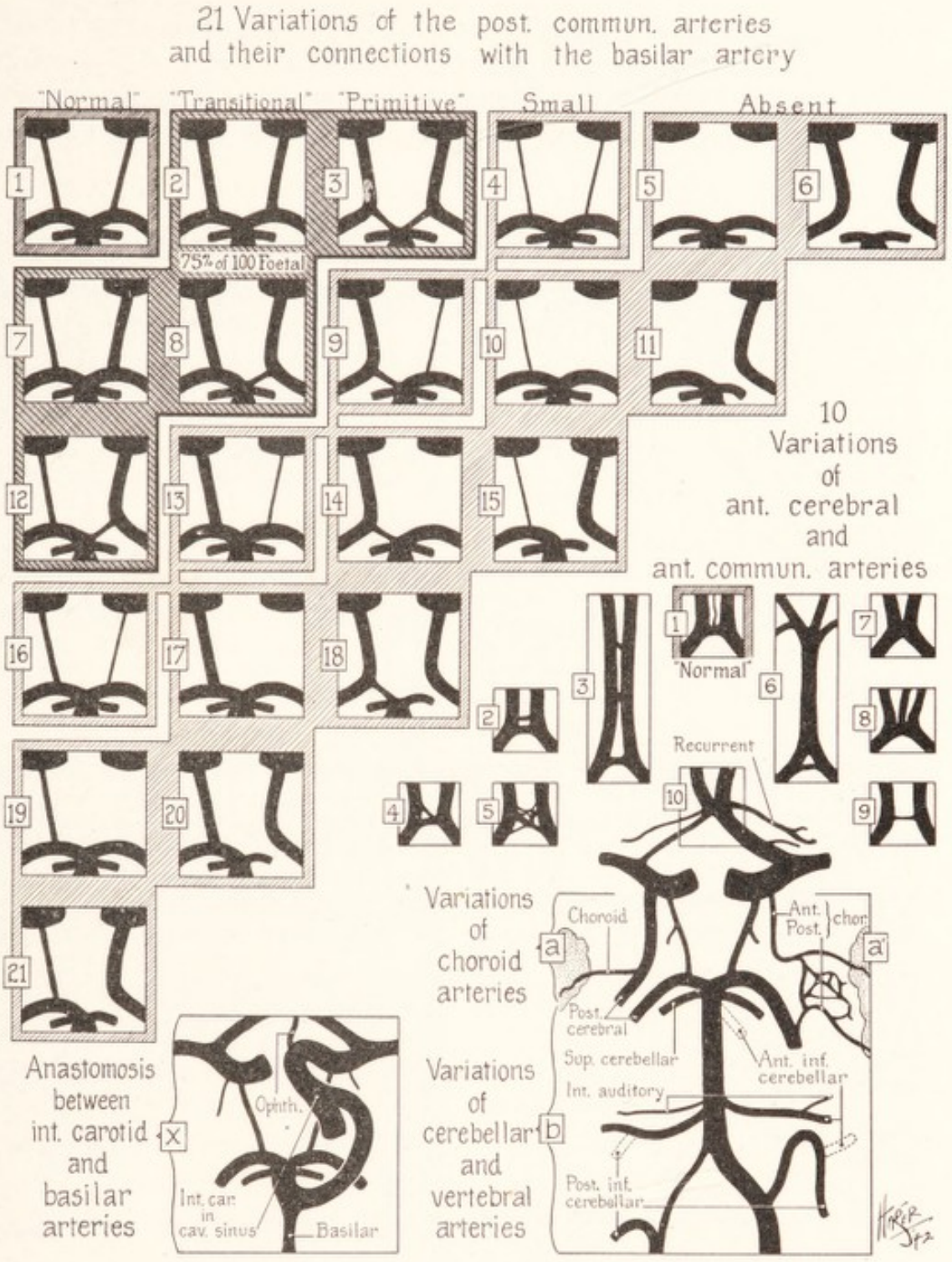


FIG. 32.—Diagram to illustrate most of the recorded variations of the circle of Willis and related branches. The heavily shaded area labeled "75% of 100 Foetal" shows the configuration in the only large reported series of fetal brains (De Vriese). The duplicated types of anterior communicating and anterior cerebral arteries (2, 3, 4, 5, and 8) predominate in the fetus. Figure *x*, at the lower left, illustrates the gross anomaly of a large anastomotic branch between the basilar and the cavernous portion of the internal carotid; in such cases the posterior communicating arteries may or may not be present.

Variations Without Aneurysms

The major variations or abnormalities are shown in Figure 32. It is obvious that more possible combinations are permitted in the

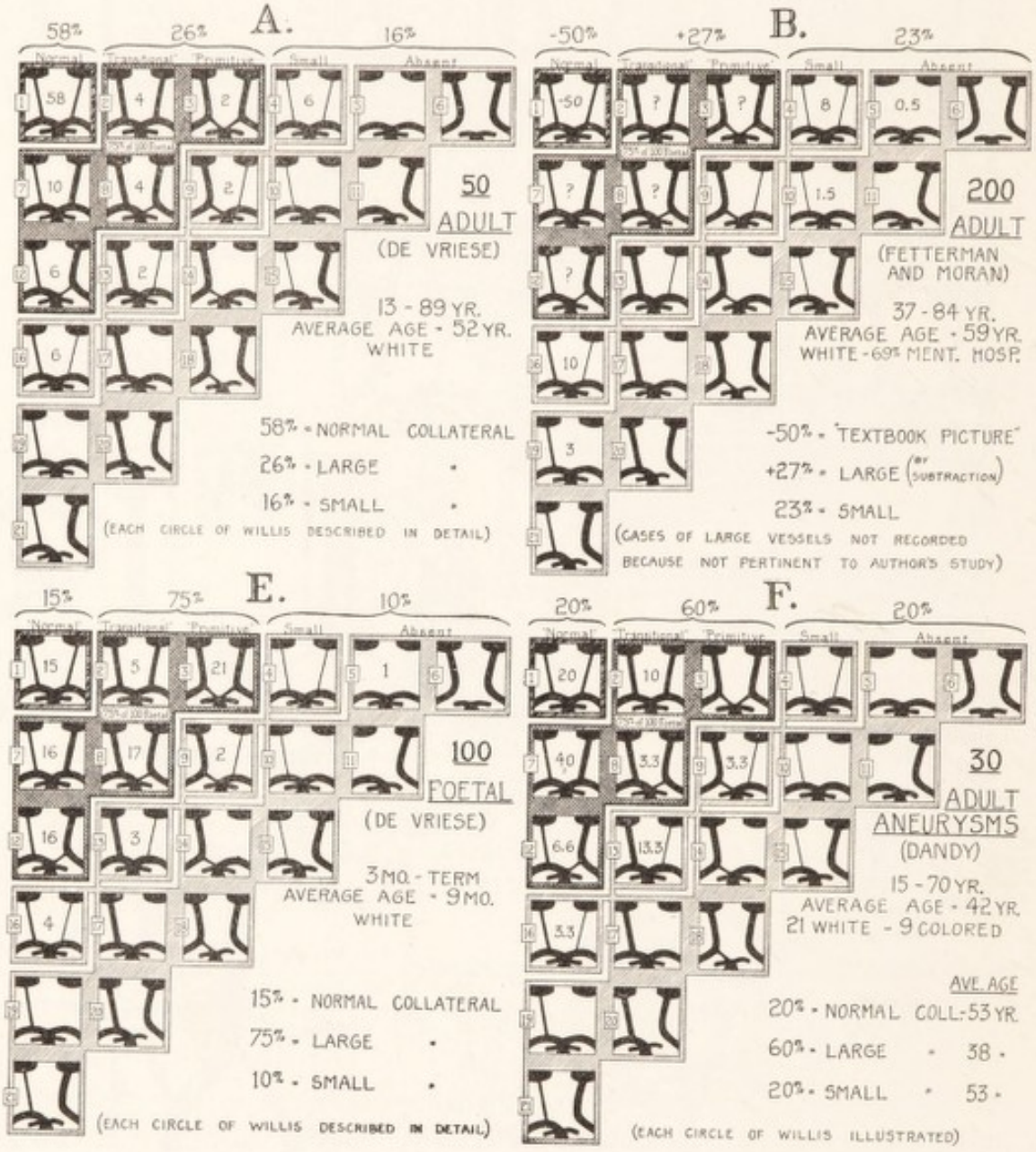
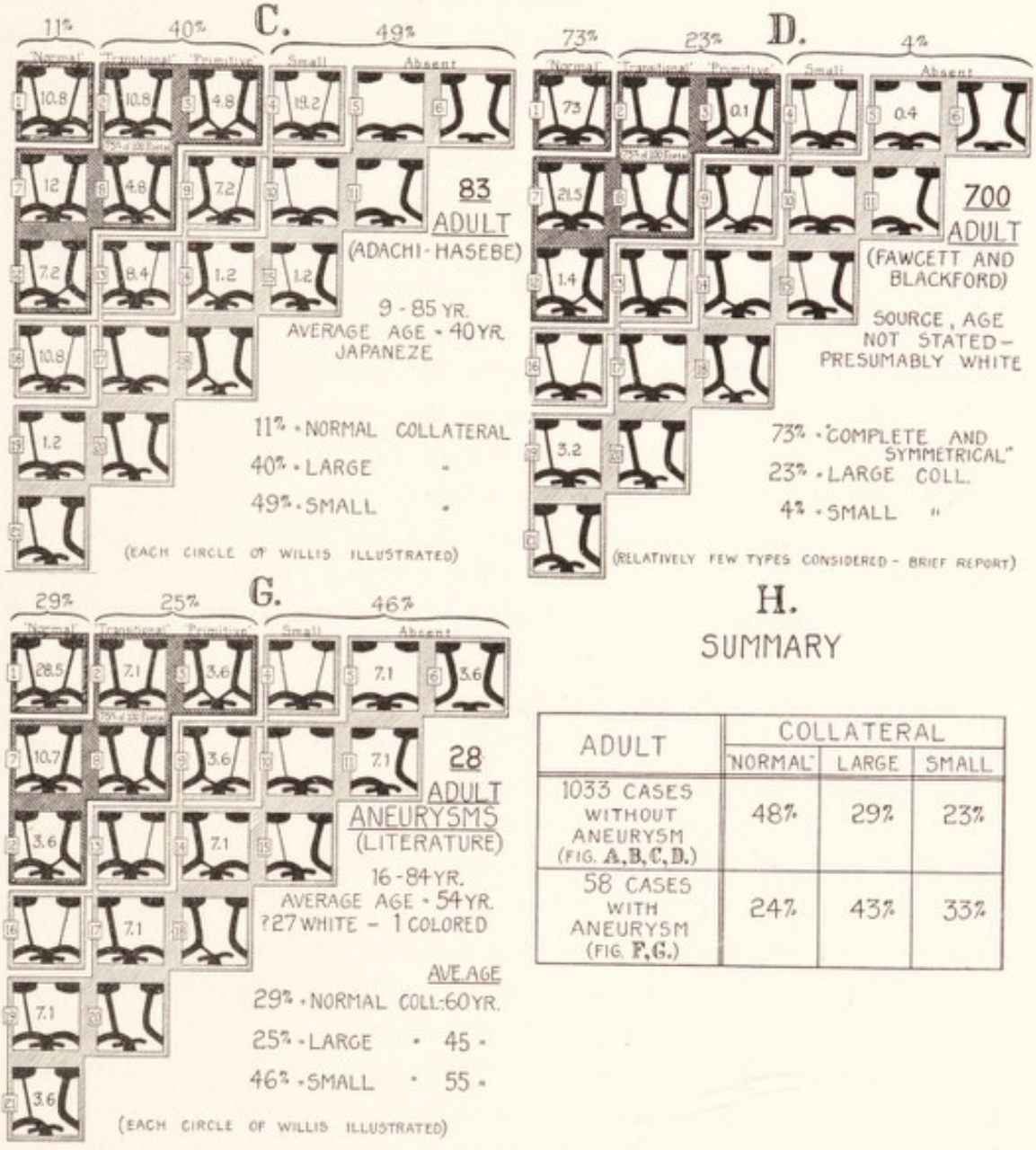


FIG. 33 (in two parts, on this page and the page opposite).—Chart to show the incidence of variation at the posterior portion of the circle of Willis; compiled from available reports in the literature.

region of the posterior communicating arteries than anywhere else on the circle, and only the most significant are here shown. Their varying incidence in four reports from the literature (1033 cases) is shown in Figure 33, A, B, C, D. The "transitional" and "primi-

tive" types, so named by Saphir (1935), are characteristic of many animals (De Vriese) and also, as personally seen, of the human embryo. Combinations of the transitional and primitive and normal types give a potential collateral circulation greater than normal, and



ADULT	COLLATERAL		
	"NORMAL"	LARGE	SMALL
1033 CASES WITHOUT ANEURYSM (FIG. A, B, C, D.)	48%	29%	23%
58 CASES WITH ANEURYSM (FIG. F, G.)	24%	43%	33%

FIG. 33 (second part).—The anterior portion of the circle was apparently within normal limits in over 90 per cent of the cases without aneurysm. The cases with and without aneurysm are summarized under H.

are therefore labeled "large." They occurred in 75 per cent of 100 fetal specimens (Fig. 33, E). As mentioned above, small collateral was most frequent in cases well past middle age (Fig. 33, B). It is unfortunate that the report of Fawcett and Blackford (Fig. 33, D), indicating so little variation in so large a series, cannot be con-

sidered conclusive in the light of other evidence. That there may be a racial as well as age difference is suggested by the report of Adachi-Hasebe (1928) in the very wide deviation from the normal in the Japanese (Fig. 33, C); and also by the report of De Garis (1924) who found almost twice as much variation in the subclavian pattern of the Negro as in the White in 1000 cases of each. In our series of aneurysms, the high incidence in the Negro stock has been mentioned.

The frequently quoted figures of Stopford (1916), Windle (1888), and Blackburn (1907) could not be diagrammed, because of the manner in which the material is presented.* However, by adding their cases to the 1033 of Figure 33, making a total of 1603, the posterior communicating artery was reported absent on one side in 3 per cent, absent on both sides in 3 per cent, and small (i.e. probably often impervious) on one or both sides in 12 per cent. The highest incidence of such deficient collateral circulation reported by any one author was 23 per cent in 200 specimens (Fetterman and Moran).

As noted above, the most common abnormality of the anterior communicating artery is one of the various forms of duplication (Fig. 32); 57 per cent of 100 consecutive fetal and 32 per cent of 1803 adult specimens. The source material does not permit any figures as to its actual size, either in normal or duplicated forms, but absence appears to be rare; at least it was reported missing in only three cases out of a total 1803! In the anterior cerebral arteries, more inequality of size is the most common variation, one often being larger, the other smaller than normal; cases where one artery is small enough to make a practical difference appear to be much less common. Occasionally there is a fusion of the anterior cerebral arteries for a short or longer distance (Fig. 32, 6, 7), resulting in a large or direct anterior communication.

Absence and gross anomalies of the carotid, basilar, and vertebral arteries have been reported. Lowry (1916) found a case of complete absence of the right internal carotid; there was no trace of it on the common carotid stem, and no bony canal in the petrous temperal bone. From the bifurcation of the large basilar, a sizable branch in the position of the posterior communicating artery continued forward as the right middle cerebral and gave off the ophthalmic artery; it supplied a small twig mesially to the right anterior cerebral, which arose, alongside the left, from the normal

* The configuration of each circle of Willis *as a whole* could not be determined.

left internal carotid. Dandy (1929), at operation for a congenital absence of the orbital roof in a 16-year-old girl, found the left internal carotid artery absent.

Fisher (1914) reported the rare anomaly of bilateral absence of the internal carotids in a 39-year-old male, who died of cerebral hemorrhage, without symptoms during life. The enormous basilar divided into rather plexiform posterior cerebral arteries, each giving off an anterior branch which bifurcated into the anterior and middle cerebral arteries of each side. However, these "posterior communicating" branches were almost entirely occluded by fibrosis (the artery, "rupture of which evidently caused death," arose from the anterior end of the basilar and passed into the posterior perforated space), and the author stated that "circulation in the anterior and middle cerebrals was probably carried on through the anastomosis of posterior and middle cerebrals through their temporal branches." The anterior division of each greatly enlarged middle meningeal artery entered the orbit; on the left it took the place of the absent ophthalmic artery (a common condition in vertebrates and characteristic of a stage in the human embryo) and on the right it was accessory to the ophthalmic, which arose from the common trunk of middle and anterior cerebral arteries. It seems probable that collateral circulation from the external carotid compensated for the thrombosed branches of the basilar, and that the blood supply to the left anterior and middle cerebral arteries came from the other side *via* the anterior communicating artery. The left temporal lobe presented a general shrinking, though there was "little arteriosclerosis evident in the basal arteries." This author found no other similar case, but referred to six cases of absence on one internal carotid artery.

Boyd (1933) found absence of the right common carotid artery. The external carotid arose as the cephalic branch of the innominate (i.e., in the position of the common carotid), and the internal carotid was given off, just beyond from the subclavian artery. He described the embryonic origin and quotes reference to six similar cases.

Berry and Anderson (1910) reported a case of nonunion of the vertebrals, resulting in absence of any connection between the vertebral and basilar arteries on one side. The defective right vertebral became narrowed in the neck and ended as the posterior inferior cerebellar artery of this side. These authors found only one com-

parable case in the literature, that of Batujeff (1889), in which the right vertebral terminated as the posterior inferior cerebellar artery, and the left became narrowed and lost after exit from the first vertebra; furthermore, the basilar originated from the internal carotid just before its entrance into the carotid canal, and passed through the hypoglossal foramen. They referred to a case of Cavatorti (1907) in which the two vertebrals ran separately to terminate as the posterior cerebral arteries with no anastomosis between them. In other words, the basilar was absent and this was a retention of the early embryonic configuration. Inequality in the size of the vertebral arteries is frequent, and according to Stopford occurred in 92 per cent of 150 specimens with a marked discrepancy in 72 per cent; the artery was more often larger on the left side.

A dozen or more authors have reported a large anomalous anastomosis between the basilar and internal carotid arteries (Fig. 32). Blackburn (1907), Hasenjäger (1937), Quain (1844), and Smith (1909) present clear illustrations of this gross anomaly and their cases were quite similar. The anastomotic branch was connected with the middle or more anterior portion of the basilar which tended to be considerably narrowed posteriorly. This branch perforated the dura (or the sphenoid bone—Quain) near the posterior clinoid and nerves V or VI and joined the cavernous portion of the internal carotid. The posterior communicating arteries were present and apparently of normal size (Quain, Blackburn, Smith), or were small (De Vriese, 1905), or completely absent (Hasenjäger). Adachi-Hasebe referred to one probable case in connection with descriptions of the circle of Willis and pointed out that such an anomaly could well be overlooked because of its obvious vulnerability on removing the brain from the skull. Batujeff's case (see above) was probably of similar embryonic origin.

There are differing reports as to the clinical significance of anomalies of the circle of Willis. Dorrance (1934) decided that such anomalies are "rarely of sufficient importance to impair its functional integrity." He discussed the various avenues of collateral circulation between the internal and external carotid arteries and quoted half a dozen investigators who believed that anomalies do not adversely affect the blood supply to the brain; for instance Homans (1920), who failed to find in the literature any death following carotid ligation in which an anomaly of the circle was demonstrable. Dorrance's conclusions as to the anatomical integrity

of the circle were based on Fawcett and Blackford (see above) and his broad interpretation of Windle, who remarks that "usually there was a slight anastomosis in the interpeduncular space between small twigs passing from basilar and carotid arteries," in his 13 per cent of absence of one or both posterior communicating arteries.

Saphir (1935) reviewed the literature and suggested that defects in the circle of Willis may be responsible for cerebral softening and hemorrhage. He reported two autopsy cases of softening which, no occlusive lesions being found, he accounted for by the existence of heart failure and cerebral arteriosclerosis plus marked interruptive defects of the posterior communicating branches.

Fetterman and Moran (1941), interested by Saphir's report, examined 200 consecutive brains, 69 per cent of which were mental hospital patients of a high average age (59 years) and found that 23 per cent showed absent or "thread like" posterior communicating arteries on one or both sides. Cerebral softening had occurred in 30 per cent of the total number of specimens, and, "excluding those in which the softening was due to demonstrable occlusive lesions and trauma, there was a definitely higher incidence of softening among the group showing deficiency of the circle (32 per cent) than in those without such anomalies (23 per cent)."

Howe (1903) reported a case of ligation of the common carotid for lymphosarcoma in the neck with a resulting hemiplegia. Autopsy revealed that the anterior communicating artery was absent and that both posterior communicating arteries were reduced to impervious threads.

Variations With Aneurysms

For purposes of comparison it is unfortunate that there are so few illustrated or fully described cases with aneurysms available. Photographs seldom show the true relative size of all the significant arteries. However, 28 from the literature are shown in tracings in Figure 34 and summarized in Figure 33, G. It is obvious from the authors' text that a number of these were illustrated because of the defective circle, and small or absent vessels occurred in almost 50 per cent. Note the absent or impervious posterior communicating connection in the following cases of Figure 34: Cases 5 (Jacques, 1926), 6 (Berger, 1923), 7 (Beadles, 1907), 9 (Chase, 1932), 13 (McDonald and Korb, 1940), 16 and 17 (Dial and

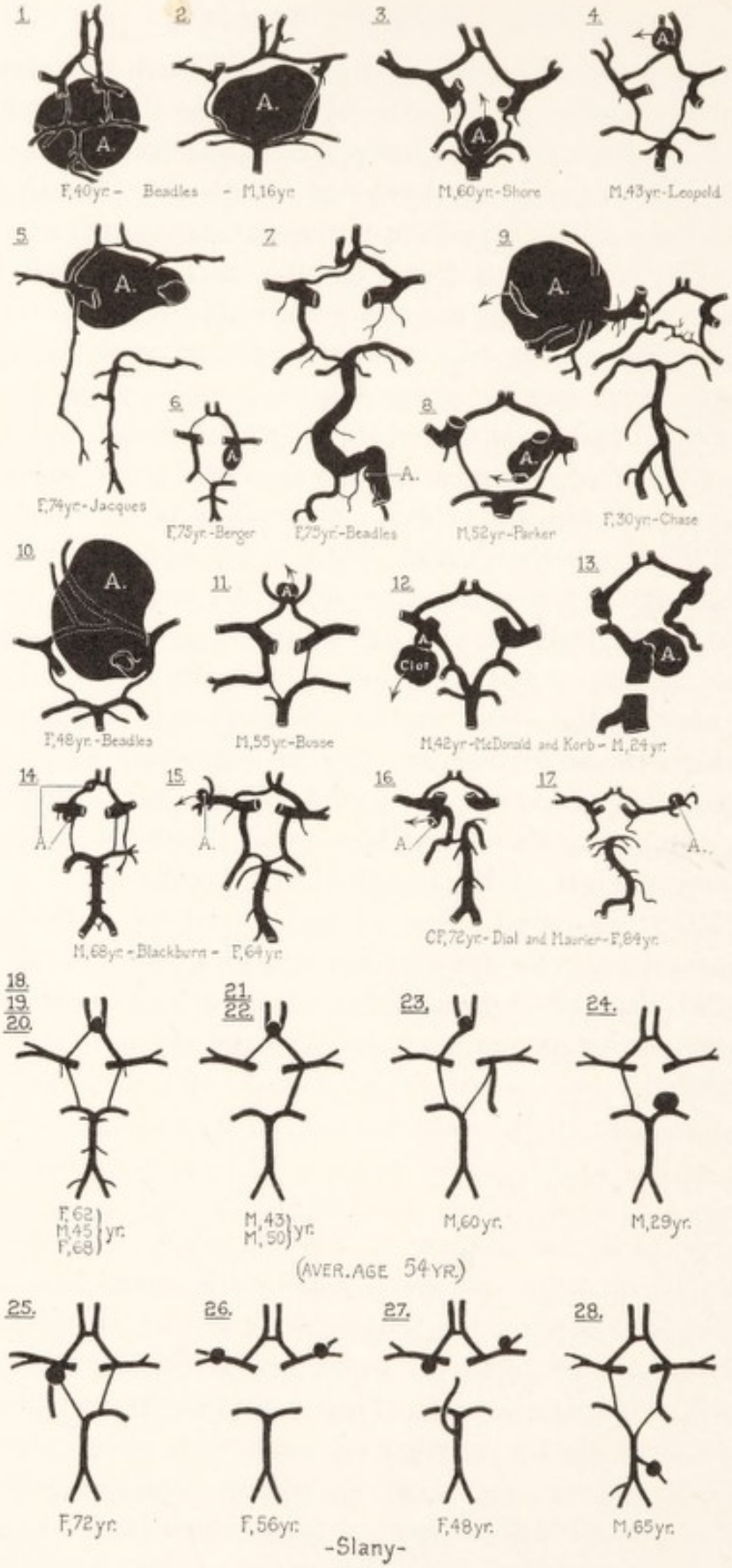


FIG. 34.—Tracings of the illustrations of the circle of Willis in the 28 available cases of aneurysms from the literature. For anatomical analysis see Figure 35, G.

Maurer, 1939), and 21, 22, 24, 26, and 27 (Slany, 1938). The anterior cerebral was absent or very small in the following seven cases: 6 (Berger, 1923), 18, 19, 20, 21, 22, and 23 (Slany, 1938).

Attention is called to the aneurysms occurring at the site of an absent vessel, in the following cases shown in Figure 34: internal carotid at site of absent posterior communicating artery (Cases 5 and 27); divisional branch of basilar (Case 13) and posterior cerebral arteries (Case 24), both at site of absent posterior communicating artery; anterior communicating artery at site of absent anterior cerebral (Case 23).

Smith (1909), in describing the case of a large anomalous branch connecting the basilar and internal carotid (see above), stated that the intracranial portion of the carotid on this side was "expanded in an atheromatous dilatation pressing on and giving complete atrophy to the left optic nerve." This was doubtless a fusiform aneurysm. The anomalous anastomosis between basilar and carotid arteries was also found in Slany's case of bilateral aneurysm in which both posterior communicating arteries were absent (Fig. 34, Case 27). The case of Mitchell (1889) was described as aneurysm of an anomalous artery connecting the internal carotid arteries, apparently just beyond their immergence into the cranial chamber, but unfortunately it was not possible for this author to give a definite picture of the arterial relations in either text or illustrations. Parker (1926) described a case in a 21-year-old male with three aneurysms: (1) fusiform, of the left internal carotid intracranially; (2) sacculated, of the right internal carotid within the sinus; and (3) fusiform of the basilar, with the basal arteries quite abnormal in size and anomalous anastomoses. Ebstein (1874) reported a long fusiform aneurysm of an anomalous unpaired anterior cerebral artery.

Of our own cases of aneurysm, the 30 that were available for illustration since the beginning of this study are shown in Figure 35. These are analyzed with regard to the circle as a whole in Figure 28, C, and 67 per cent showed all vessels of normal or large collateral as opposed to only 33 per cent of small collateral. When analyzed in reference to the posterior part of the circle, 80 per cent (Fig. 33, F) showed normal or large arteries. It is interesting to note that these figures rather closely approximate the fetal configuration. In the anterior part of the circle there were three cases out of 31 with a very small anterior communicating or anterior cere-

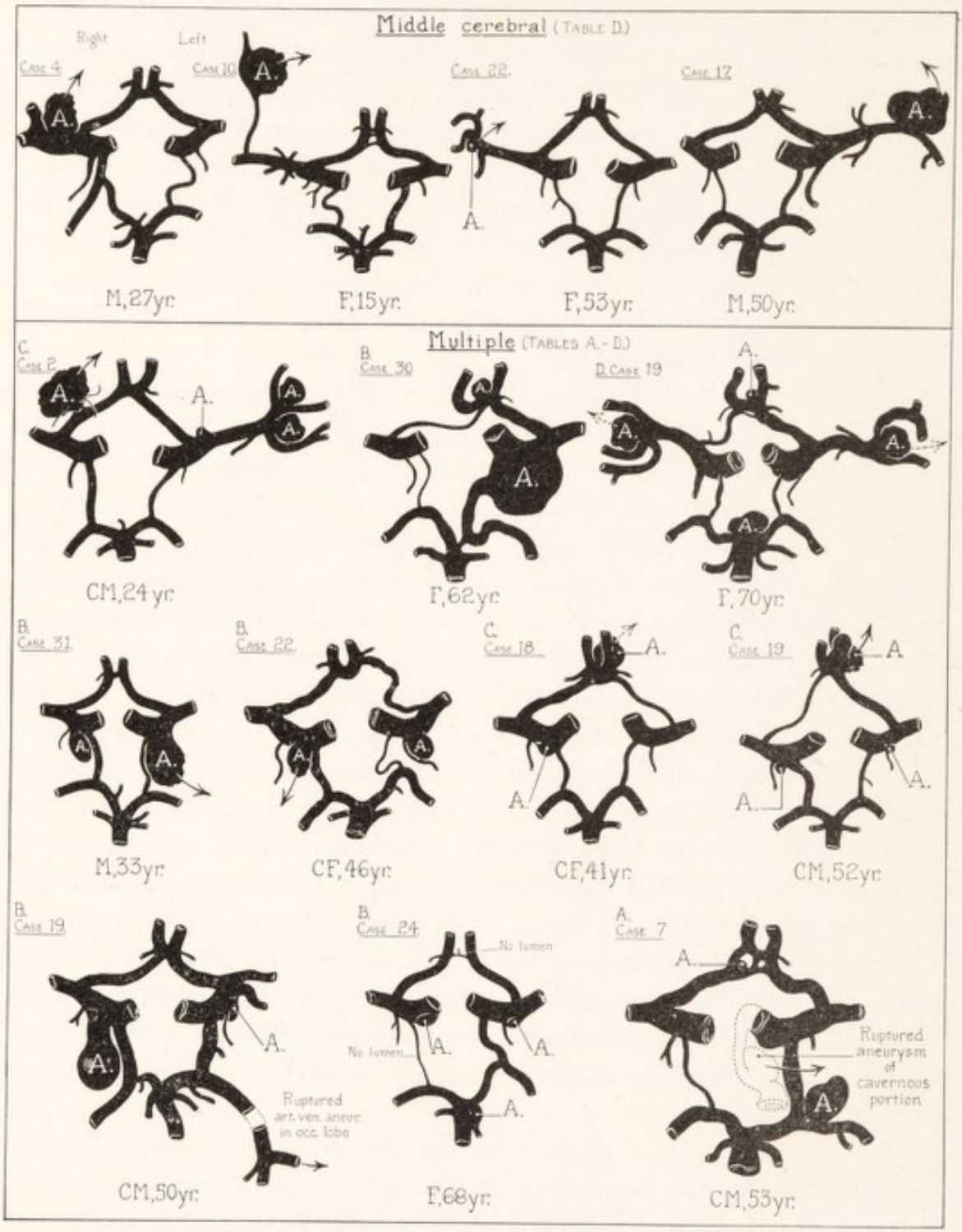


FIG. 35 (in two parts, on this page and the page opposite).—The circle of Willis in the 30 available cases of aneurysms in this series. These cases are analyzed as to their anatomy in Figure 29, C, and Figure 33, F.

bral artery, namely, Cases 24 and 30 of Fig. 35 and Case 18, Table B (that came to autopsy too late to be classified in the diagram) in which the anterior communicating artery was merely an impervious thread.

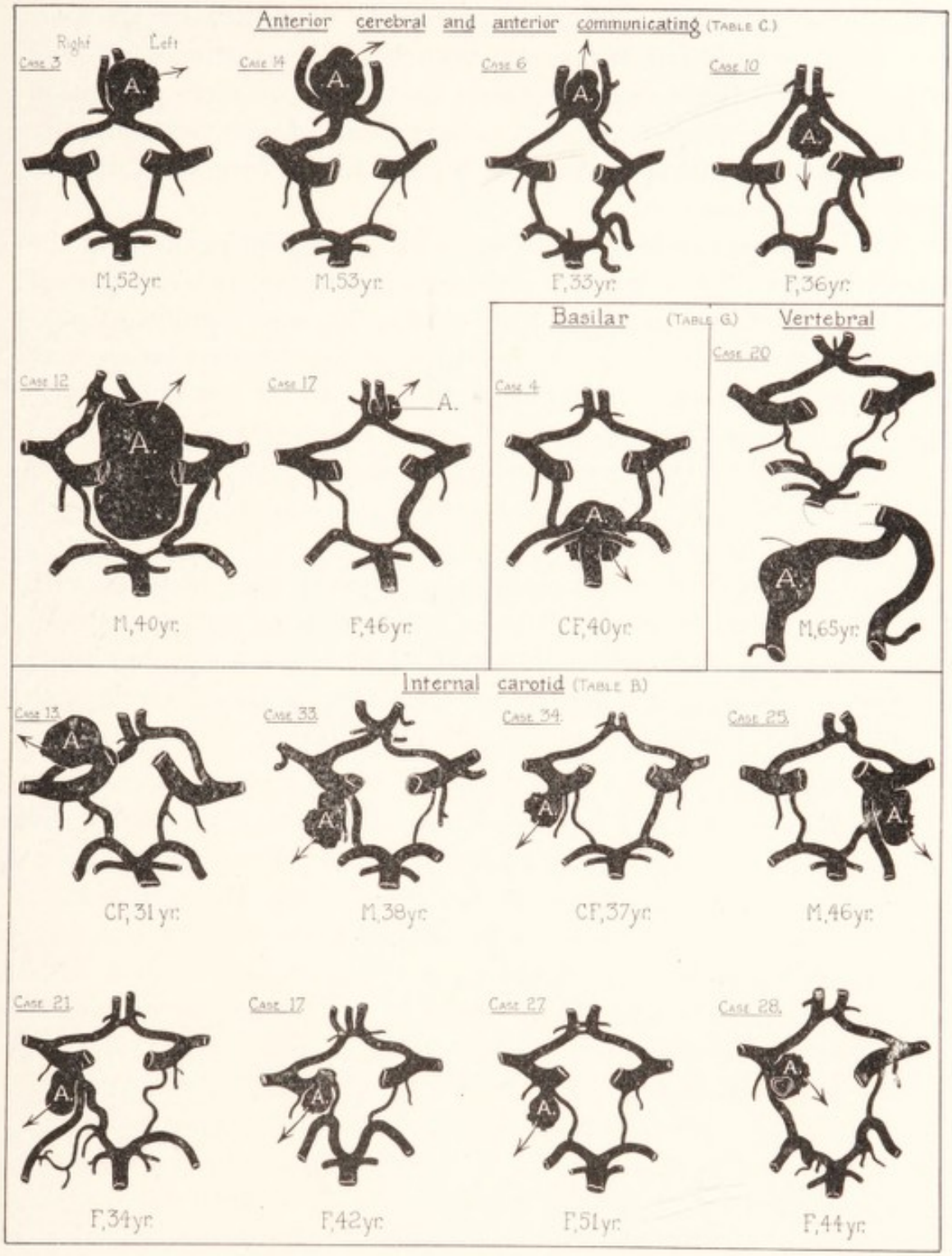


Fig. 35 (second part).—For first part see page opposite.

§ 3. SUMMARY

(1) The early embryonic stages in the development of the cerebral arteries leading to the formation of the normal circle of Willis have been described in outline form. Most of the recorded variations

and anomalies of these arteries are readily explained by the persistence of definite but transitory branches and anastomoses found in the embryo. This study suggests that an incomplete involution or atrophy of these temporary arteries, or of arteries normally present in the adult form, may be related to the formation of congenital aneurysms.

(2) A normal circle of Willis occurs in barely 50 per cent of the reported cases, and variation is more frequent in the posterior part of the circle. However, given healthy vessels, the most significant anatomical factor in reference to potential collateral circulation may be the actual presence rather than the size of the component vessels. In the three largest series of adult cases without aneurysms occurring in the literature (Fetterman and Moran, 200; Fawcett and Blackford, 700; and Windle, 200), an uninterrupted circle was found in 92 per cent.

(3) From limited evidence, it may be said that in cases with aneurysms, variation is almost twice as frequent as in those without aneurysm; there is a higher incidence of absent arteries, but there appears to be an even greater incidence of a closed circle with communications larger than normal (Fig. 33).

IV

PREOPERATIVE PROCEDURES

Ligations of the Internal Carotid Artery, in the Neck and Intracranially

During this initial period of experiment the surgical treatment of intracranial aneurysms has naturally presented unexpected problems. Many of these problems have been solved, although much of this success has been attained through trial and error. Even in the last operation of the series a method was discovered of curing aneurysms which had appeared to be hopeless.

Before any operative attack the determination of the circulatory response by digital compression of the internal carotid in the neck (Matas' test) is of paramount importance. And the fact that many aneurysms are accidentally exposed at operation when tumors have been suspected makes this determination highly advisable as a routine preliminary procedure, particularly for presumed tumors at the chiasm. If preliminary use of the Matas test has not been made and an aneurysm instead of a tumor is discovered, closure of the carotid intracranially is a very hazardous undertaking, regardless of the age of the patient. Should this situation arise, the only safe course is to close the wound and make the test subsequently.

It must be realized at the outset that some individuals, again regardless of age, will tolerate closure of the internal carotid artery with impunity and some will not. The factor determining this variability is the congenital or acquired difference in the collateral circulation at the circle of Willis, i.e., the anterior and posterior communicating arteries. Although statements to the contrary have been made, there is no doubt that age is an important factor, for, with arteriosclerosis of the cerebral vessels, the lumen of these collateral vessels may be reduced by an internal thrombus, even though the circumference of the artery is greater. Under the age of 35 the risk is unquestionably smaller, but, owing to the occasional congenital absence of one or more branches of the circle of Willis, or

to an earlier occlusion of a branch, or to compression by a large aneurysm, one can accept no blanket assurance of a normal circulation. On the other hand, several successful ligations of the carotid have been reported after age 70. Regardless of age, the adequacy or inadequacy of the collateral circulation must be known before operation. If inadequate, it can be made adequate by partially occluding the internal carotid, thus forcing the collateral branches to take up the increased burden and later permit its total ligation without hazard.

In our hands, compression of the internal carotid in the neck for 10 minutes without symptoms such as dizziness, numbness or weakness of the contralateral arm, or loss of vision has never failed to indicate correctly that the internal carotid may be ligated intracranially or in the neck with safety. Compression for a lesser time is not safe. On two occasions this artery was ligated intracranially when the carotid could be compressed without symptoms for only five minutes; once with no untoward effect, the second time with disastrous results. In the latter case a tumor was considered to be the probable lesion at the chiasm, but the test had been made by my assistant, Dr. Troland, who considered the possibility of an aneurysm. Under these circumstances the wound should have been closed and the carotid partially ligated in the neck; a week later the intracranial wound could have been reopened and the carotid clipped with safety. This has since been done and with cure of the aneurysm—a large one in the carotid canal. Taking this chance left the patient with a subtotal hemiplegia and aphasia, both of which will, I fear, be permanent. Various methods of partial occlusion of the internal carotid have been used by different surgeons. Dr. Halsted and Dr. Matas, who were both keenly interested in this problem, used aluminum bands, hoping thereby to avoid cutting the intima. My preference has been for bands of fascia lata or dura if the brain is exposed. They have been used exclusively in this series of cases. Fascia lata has all the advantages of a rigid band, and, being viable tissue and softer, provokes, I think, less destructive effects upon the vascular wall. It too can be removed if cerebral symptoms ensue from anoxemia. Dr. Halsted performed many experiments on animals with fascial bands—especially on the aorta—and gave it up because the fascia disintegrated under the arterial pulsation. This objection is not so serious with the smaller internal carotid, and it can be largely overcome by doubling the

fascial band. Pearse has recently introduced a cellophane band in animal experiments and depends upon the reaction about the foreign body to gradually increase the arterial occlusion. This same reaction, however, is an important factor in the fascial bands. In three or four weeks there is a dense mass of new tissue incorporating the fascial bands, and this induces a gradually progressive reduction in the arterial lumen.

When specimens of *single* layered fascial bands have been removed subsequently (3–8 weeks) a mass of connective tissue completely obscures the fascial transplant; under the microscope it is seen to be largely fragmented, and connective tissue is growing through the fragments. This is in marked contrast to the almost perfect preservation of fascia lata used to cover dural defects. The difference between the perfect preservation in one place and the rapid disintegration in the other can only be due to the arterial pulsation in the latter. The double layer of fascia lata has been used recently, and in one specimen, removed two months later, the gross appearance of the fascia was but little altered, and the microscopic fragmentation was but slightly in evidence. In three specimens removed at operation (two single and one double band) the lumen of the vessel has still been patent though greatly reduced, and the intima has been intact except for a tiny "caruncle" in one specimen. In none was there a thrombus at the site of the band.

Results Following Ligation of the Internal Carotid Artery

It has long been known that ligations of the internal or common carotid arteries are followed by (1) a very high mortality and (2) a high percentage of cerebral complications. Of the latter some survive with residual sequelae such as varying degrees of hemiplegia, aphasia, mental changes, and epilepsy. In the pre-Listerian days the surgical mortality from carotid ligations approached and frequently surpassed 50 per cent. It has since been learned that the mortality rate is less with clean wounds, and that sepsis was responsible for many deaths because of the intravascular spread of an infected thrombus into the cranial chamber. However, precisely the same sequence of events occurs with and without sepsis, the difference being only in the relative frequency. There are two causes

of death or disability, namely, (1) cerebral anemia from inadequate collateral circulation through the circle of Willis, the effects of which appear immediately and may be abrupt or progressive; and (2) cerebral thrombosis and embolism, the effects of which are late in appearing, i.e., 12 hours to several days later, and are usually abrupt, though at times a preceding small attack may warn of the impending event.

Pilz (1868) collected 600 cases of common carotid ligations, the mortality being 38.5 per cent and cerebral complications 32 per cent. Le Fort (1879) reported a mortality of 54.5 per cent. Wyeth (1879) and Ballance and Edmonds (1879) each collected 789 cases with a mortality of 41 per cent (probably by reference to the same material). These three writers give a fair estimate of the risks to life in the days when operations were almost always attended by sepsis.

Zimmermann (1892) collected 65 cases since 1880 with 31 per cent mortality and cerebral complications in 26 per cent, and Siegrist (1899) reported 825 cases collected since 1880 with a mortality of 40 per cent. More recent reports are by Cauchaix (1921) of 150 cases of common carotid ligations with a mortality of 10 per cent, and 13 cases of the internal carotid with 16.7 per cent mortality. Walcker (1924) reported a mortality of 40 per cent in 601 collected cases. A recent report by Watson and Silverstone (1939) with a death rate of 55 per cent and cerebral complications in 70 per cent of their 20 cases should be noted in passing. In all of their cases, however, the ligations were done in conjunction with the removal of large carcinomatous masses (many ulcerated and infected) in the neck. Such statistics could not give a fair appraisal of the risks incurred with simple ligations of the carotid, but they do indicate the dangers that attend the procedure. Matas (1940) in 66 cases of his own (60 common carotid and six internal carotid) had eight deaths (including two reported as due to angina) with a death rate of 12 per cent. The report of Matas brings the risk to the lowest figure heretofore obtained and is representative of the best work of individuals rather than an ensemble of results collected from the literature. I have made no effort to separate ligations of the internal and common carotid arteries. It is frequently stated that ligations of the common carotid are less dangerous than those of the internal carotid because of the collateral circulation through the external carotid in the former. Dorrance (1934) is convinced that ligation of

the common carotid is less dangerous. Theoretically this should appear reasonable and perhaps there may be some difference, but when partial ligations are done and there is inadequate collateral circulation there probably is no difference. With care in the preoperative tests for collateral circulation, and by the proper choice of methods of ligation, the risks of carotid ligations can now be largely eliminated.

I have recently reported a series of 88 cases in which the internal carotid artery has been partially or totally occluded in the neck and "clipped" intracranially for arteriovenous and arterial aneurysms and a few tumors, with the results shown in the table just below:

Method	*Number of Cases*	*Circulatory Disturbances*		*Deaths*
		Immediate	*Late*	
Total ligation in neck	36	0	1 (2.8%)	2 (5.6%)
Partial occlusion in neck	25	3 (12%) *	1 (4%)	1 (4%)
Clipping intracranially	27	1 (3.8%) †	0	1 (3.8%) ‡
Total	88	4 (4.5%)	2 (2.3%)	4 (4.5%)

* One cleared completely and another partially when band was removed.
† Due to large aneurysm compressing the circle of Willis.
‡ Artery torn by clip.

The number of ligations has since been increased to 105 without further deaths, bringing both the mortality and the immediate complications to less than 4 per cent, and the later complications to less than 2 per cent.

The mortality rate and the incidence of complications—both immediate and late—from this series are, I think, the lowest yet obtained. That is due in very large part to the utilization of the Matas test and partial ligations of the carotid when this is indicated.

Partial Occlusions of the Internal Carotid Artery in the Neck

In the above series of cases, fascial bands have been applied for partial occlusion 25 times. In three of the early cases the band was applied too snugly, and within 3–8 hours signs of motor weakness developed as a result. In each of these the band was removed; in one the function was immediately restored and remained unimpaired subsequently. In a second case there was complete return of speech but not of motor power, and in the third no improvement resulted and the patient died 24 hours later from the ruptured aneurysm (it

was known to have been ruptured at the time the band was applied).

Partial occlusions unquestionably carry a hazard that depends largely upon the care of the operator and should be avoidable. At first, I tried to stop the occlusion just short of the "thrill" stage—a warning redolent of Dr. Halsted—but after the above results I have been content to reduce the lumen one-half, or even less, and depend upon the subsequently developing mass of connective tissue about the band to constrict it farther. In all of these cases later total closure of the carotid within the neck or intracranially has produced no aftereffects.

Although partial ligation of the internal carotid is preferred to that of the common carotid, it probably does not make a great deal of difference which vessel is used. At times the internal carotid is quite abruptly reduced in size after leaving the bifurcation and may even be almost funnel-shaped; when this obtains, a partially occluding band may slide down the artery and cause total occlusion. Then, too, the internal carotid is at times redundant and forms an acute bend which a fascial band may intensify. With either of the above conditions, partial closure of the common carotid is preferable and safer. In three cases where the bands were applied to the common carotid, the external carotid has been ligated so that the result is precisely the same as ligating the internal carotid. Ligation of the internal carotid or this equivalent has been used in all cases in this series. In a week or ten days the partially occluded vessel has induced sufficient collateral circulation through the circle of Willis to permit total closure of the artery, either in the neck or the cranial chamber, or both, as the situation demands. In all except one case I have subsequently ligated the artery in the neck as well as intracranially; there then remains no possible doubt concerning the total elimination of flow of blood into the aneurysm from either direction. In one case the neck of the aneurysm was subsequently clipped and further ligation of the carotid was not necessary.

Total Occlusions of the Internal Carotid Artery in the Neck

In 36 cases the internal carotid was totally ligated in the neck, with late appearing cerebral complications and subsequent death in one case. The last five of these were closed with fascial bands inter-

vening between the vessel and the superimposed silk ligatures. In the remaining earlier cases the ligature was applied directly to the wall of the vessel.

Internal Carotid Artery Clipped Intracranially

The internal carotid artery has been clipped intracranially 27 times. One death (after 12 hours) was due to rupture of the enlarged and sclerotic carotid artery when an attempt was made to force a clip that was too small. In another case previously mentioned (Case XIV, Table B) hemiplegia and aphasia developed immediately, although the patient survived. In the other 25 cases the internal carotid occlusion was followed by no aftereffects. Since no *late* effects have appeared after "clipping" of the carotid, I have been led to wonder whether thrombi and emboli might not be of less frequent occurrence after this process than they are after ligations of this vessel in the neck—either partial or total. This idea is suggested by the fact that the intracranial carotid is less than one-fifth the size of this vessel in the neck. Barring accidental injury to the carotid (one case) it has therefore seemed safer to close the carotid intracranially than in the neck. For this reason, when the Matas test is negative and an aneurysm is to be treated by double ligation, I prefer to clip the carotid intracranially *first* and ligate in the neck *later*. Both may be done at the same operation or the second ligation may be made later. The number of cases is too small to prove this point, but at least there are occasional cases with late sequelae following both partial and total ligations in the neck, and as yet, at least, none from the intracranial occlusions (when the Matas test was negative).

Summary of Results of Above Methods

From the above table (p. 95) it will be seen that the immediate cerebral circulatory disturbances are more frequent than those appearing late. However, several of these complications and even the deaths could now be avoided. The greatest number of immediate circulatory disturbances occurred in the group of partial ligations; in each of the three cases the band was placed too tightly. (One of

these recovered completely when the band was removed.) We appear to be more helpless with the late complications after ligations in the neck, but it is probable—though not assured—that ligating the carotid in the neck through an interposed band of fascia may in the future reduce this complication after total ligations. It is hardly possible that the partial occlusion of the carotid was responsible for two deaths in this group, because the aneurysm had already ruptured at the time of operation in one, and in the second the patient was moribund at the time from intracranial hemorrhage. Another death could have been avoided by using a larger silver clip.

Ligations of the Vertebral Arteries

The sites of election for ligation of the vertebral arteries are (1) between the atlas and axis and (2) below the sixth cervical vertebra. Elsewhere along the cervical spine the artery is almost impossible of approach. The transverse process of the atlas protrudes much farther laterally than the axis and this produces a free stretch of artery that is easy of access and ligation. At times the artery can be almost as easily ligated between the atlas and occipital bone on its return to the cranial chamber. Ligation of one vertebral artery at this site has been done perhaps 20 times without any untoward effect. Ligation of the vertebral artery below the sixth cervical vertebra is also easy, but this allows collateral circulation into the artery as it traverses the cervical region. Only once was the second side prepared for ligation in a patient (aged 67) with a known S-shaped aneurysm of the basilar and vertebrals and advanced arteriosclerosis. Before the vessel was ligated it was compressed with the forceps for perhaps two seconds and then released. Death was immediate. Pulse and respiration ceased, and despite release of the compression and artificial respiration there was no return of pulse or breathing. There could be no more speedy death.

Causes of Immediate and of Late Cerebral Disturbance After Carotid Ligations

The *immediate* cerebral complications, of which hemiplegia and aphasia are the outspoken evidence, are clearly explained on the

basis of inadequate circulation to the affected side of the brain. The *late* cerebral involvement is just as certainly explained on the basis of a propagating thrombus in the carotid (probably first reported by Le Fort, 1868), or possibly in some cases an embolus that has broken off from a thrombus and lodged in the cerebral arteries. Usually the late onset is precipitate, but there may be premonitory cerebral attacks with recovery, followed in the succeeding 24–48 hours by complete loss of function.

The pathological reports confirming this view of the late complications are not numerous but are conclusive. Zimmermann (1891) collected five reported cases with postmortem examinations of the carotid artery following ligations; all showed thrombi extending the length of the carotid in the neck. Two of these specimens were examined intracranially, and all three branches of the carotid (in the brain) were filled with a continuation of the thrombus (Verneuil's case, 1871, and Schönborn's case, 1879). In four of his own cases the entire carotid in the neck was closed by a thrombus, and in one of these it was continuous into the ophthalmic and middle cerebral arteries. Stierlin and Meyenburg (1920) reported a specimen obtained after a bullet wound in the neck, but from which the internal carotid was not torn; the thrombus extended from a point 4–5 cm. above the carotid bifurcation into the anterior cerebral and middle cerebral arteries and their branches. In another of his cases (following thyroidectomy) the thrombus extended down the external carotid into and along the internal carotid artery into the middle cerebral artery. Cerebral symptoms appeared suddenly four days after the operation and death was two days later. Perthes (1920) traced a postoperative thrombus (artery tied at the base of the skull) into all three intracranial branches of the internal carotid; paralysis and aphasia appeared eight hours after the ligation and was complete in three hours; death was six days later. The intima and media had been cut by the ligation. Another of Perthes' cases developed hemiplegia during the night following carotid ligation; there was a thrombus at the site of the ligature and an embolus in the terminus of the intracranial portion of the carotid. Apparently the thrombus was not continuous, but a fragment of it was carried up the carotid.

An interesting case is recorded by Esmarck (1857). While the operator was palpating an aneurysm of the carotid in the neck the patient suddenly became hemiplegic and died three days later. Necropsy showed a thrombus formation (from an embolus) in the

internal carotid, extending from the ophthalmic artery into the middle cerebral and anterior cerebral arteries and their branches.

In a personal communication Dr. Cyril Courville, of Los Angeles, has sent two unpublished reports of carefully studied postmortem examinations on this subject. In one a thrombus extended the entire length of the internal carotid and into the anterior and middle cerebral arteries. In another the artery in the neck contained a thrombus that extended part way up the neck, and an embolus therefrom had lodged in the middle cerebral artery, the anterior cerebral being intact.

I recently (1937) reported a necropsy specimen (nontraumatic, noninfective, and nonoperative) from a spontaneously arising thrombus that extended continuously from the bifurcation of the carotid into the middle cerebral artery and its branches and for some distance into the anterior cerebral artery. It was of six weeks' duration and apparently arose from a defective calcified area in the artery within the carotid canal.

Pettermann (1932) reported four cases of sudden cerebral death after carotid ligations, with postmortem studies in three. Softening in the brain was reported in one, a thrombus of the internal carotid extending from the ligature in another. Although he explains the cerebral disturbances by propagation of thrombi (and they undoubtedly are), the pathological studies are not sufficiently complete to use for evidence.

Fetterman and Pritchard (1939) reported the postmortem findings on a patient aged 37, whose internal carotid had been ligated 1½ years earlier; 2½ hours after the operation aphasia and right hemiplegia resulted. The internal carotid (intracranially) was completely obliterated. No note is made concerning the extension of the thrombus into the middle cerebral artery, nor can one determine this from the excellent photographs of the exterior of the cerebral vascular tree. The posterior communicating artery is absent on that side and is very small on the other, but the anterior cerebral appears to be of ample size to take care of the collateral circulation.

In a recent communication Schorstein (1940) has argued at some length to refute this explanation of the cause of cerebral complications after carotid ligations. These aftereffects he explains by anoxemia and without actual occlusion of the vessels. He bases his conclusions on three cases reported by others, in none of which a thrombus was seen at autopsy. However, a lesion of this kind may well have

been missed. He admits that in one of the cases the local pressure of the intracranial aneurysm may have been responsible; this compression would, of course, act precisely like an intravascular occlusion from a thrombus, and in the end the result would therefore be essentially the same.

Schorstein fails to differentiate between the immediate and the late sequelae of carotid ligations. The former, occurring within the first six or eight hours, are unquestionably due to anoxemia, but after this period, in which the cerebral circulation has been tested and proven adequate, subsequent disturbances of the brain can only be due to intracranial occlusions from thrombi or emboli.

The suggestion of an embolus instead of a thrombus can only be conjectural until sufficient pathological material is advanced. Even proof by necropsy may be difficult, because an embolus may be quickly engulfed in an ensuing thrombus. A hiatus between a thrombus in the carotid and another occlusion in the middle or anterior cerebral arteries is the only proof that this occlusion is embolic.

Why does a paralysis so induced sometimes clear in part or completely? Probably because only *branches* of the middle cerebral artery are occluded, or because the anterior and not the middle cerebral artery is closed and the Rolandic area is then affected by the contiguous oedematous reaction. When function does not return it is positive evidence that the main trunk of the middle cerebral artery is filled with a thrombus or embolus.

Possible Prevention of Thrombi and Emboli in Clean Wounds

From a long and carefully studied series of vascular occlusions Dr. Halsted concluded that injury to the intima was responsible for the development of intravascular thrombi. This view has since been amply supported by experimental and clinical data. A silk ligature applied tightly enough to obliterate a large artery always cuts the intima and media and this wound is the starting point for the thrombus. Doubtless the diminished pressure and flow within an occluded large artery is also an important item in thrombus formation. This was long ago stressed by Virchow and later by Senn and by Perthes. A retarded circulation is doubtless also an important item in the formation of venous thrombi and subsequent pulmonary emboli.

Realizing the importance of intimal injury from ligatures as the cause of late cerebral complication, Perthes (1920) suggested two methods of obliterating the artery by which the intima would not be injured, namely (1) by tying the artery with a band of fascia lata, using a knot, and (2) by tying the artery around an interposed bar of fascia lying on one side of the artery. Walcker (1924) approved of this suggestion and used it successfully. Freeman (1921) introduced the method in this country. Kerr (1925) improved this procedure by suturing the fascia (for *partial occlusion*). In recent cases I have placed a band of fascia around the carotid for either partial or complete occlusions; for partial occlusion the artery is subsequently tied with silk around the band of fascia, and for complete occlusion the silk ligature is applied over the fascia (Fig. 37) to make the total ligations initially. I believe that if this had been done in our single case of a late cerebral complication and death, the disaster would not have occurred.

V

SURGICAL TREATMENT

Aneurysms in the Carotid Canal (Table A)

Of all intracranial aneurysms those which occur in the carotid canal are the easiest and safest to treat surgically. These aneurysms have been treated by "trapping" them between a "clip" that closes the internal carotid artery intracranially (Fig. 42) and a ligature

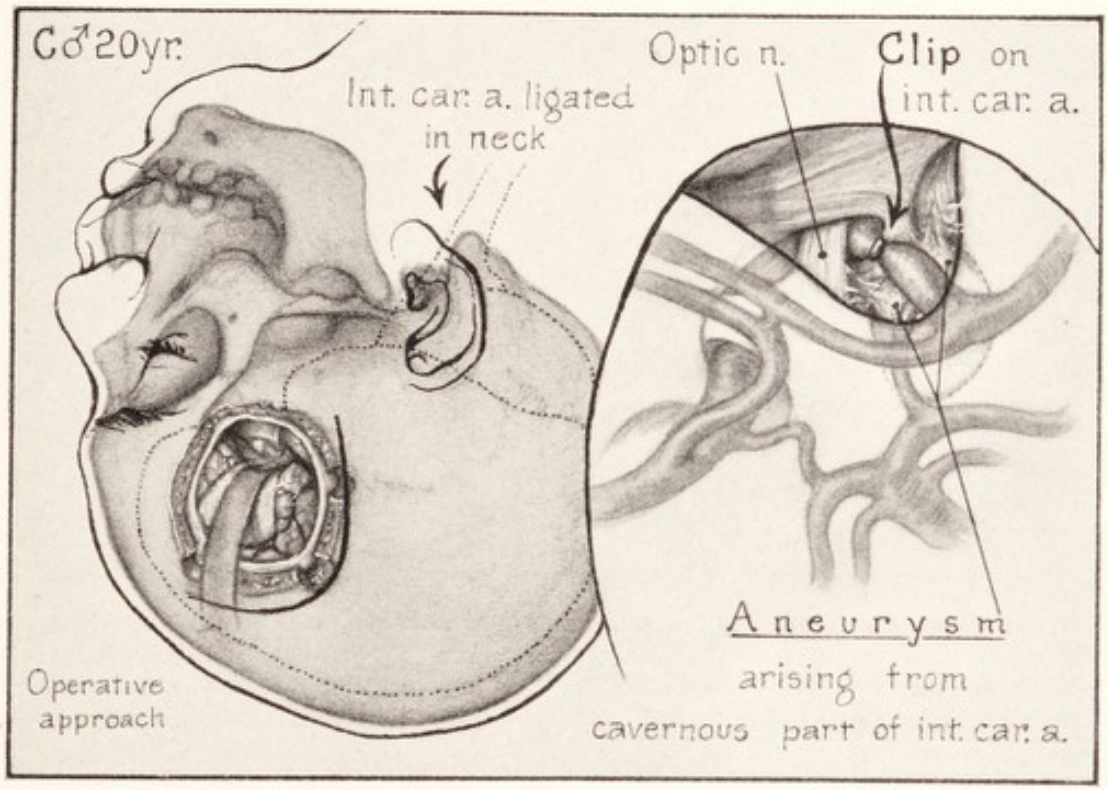


FIG. 36.—The operative approach for all aneurysms of the internal carotid.

on this vessel in the neck (Fig. 37). Both ligations may be done at the same time, or either ligation may be first, and the second closure may follow a few days later. Ten of the 11 cases have been so treated —the eleventh was a postmortem specimen. Nine of these are cured. In six cases both ligations were made at the same operation; in four cases there was an interval between the ligations. It should be em-

phasized that carotid compression (Matas' test) has always been made before operation, so that the operator can be perfectly certain that intracranial closure of the internal carotid will be tolerated. Seven of these patients were young—20–37—and preliminary par-

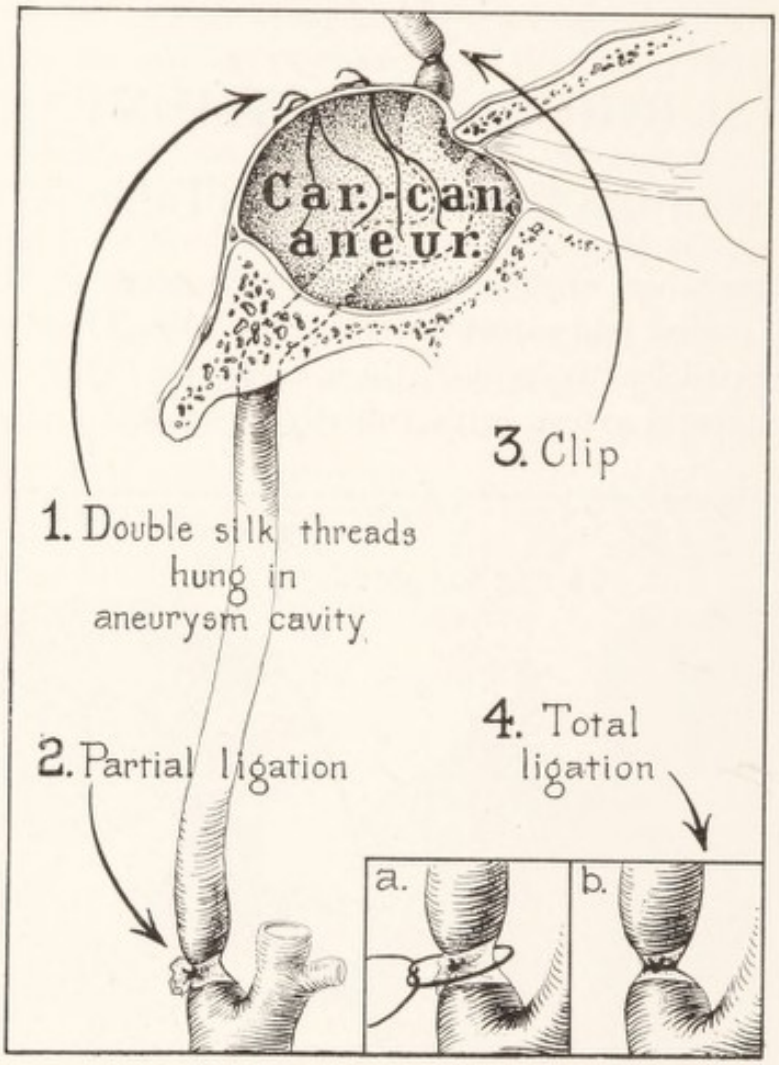


FIG. 37.—Method of trapping the aneurysm in the carotid canal by clipping the internal carotid intracranially and ligating it in the neck. If a partial ligation of the carotid is necessary, a band of fascia partially occludes the carotid and later a ligature for total occlusion is placed over the band (a) and (b). This prevents injury of the intima.

tial closure of the internal carotid was not necessary; it was necessary in Cases VII, VIII and XI, in which the age was 53, 52, and 39 years respectively.

The operative approach for intracranial ligations is similar to that for hypophyseal tumors. The concealed incision is used, and a small bone flap is turned down as far anteriorly as possible without entering the frontal sinus, the size of which is always known from the x-ray (Fig. 36).

Although the smaller aneurysms of this group are easily curable by trapping them between ligatures, I was not convinced until recently that the very large ones could be corrected in any way. I had supposed that the big bulging intracranial sac would compromise collateral circulation through the circle of Willis, and also that the carotid might be hidden from view and its clipping precluded. How-

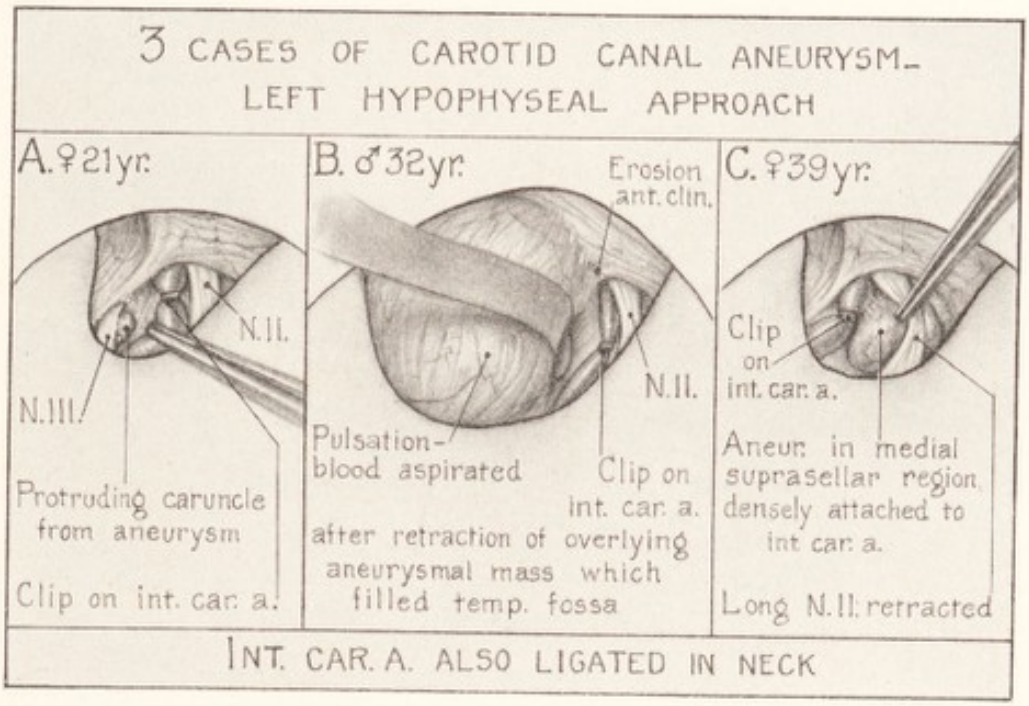


FIG. 38.—Three types of aneurysm arising in the carotid canal: A. Sac protruding through the dura and compressing the third nerve. B. Huge aneurysm filling the temporal fossa and overhanging the carotid artery; the aneurysm is turned aside with a spatula and the internal carotid is clipped. C. Aneurysm bulging from the carotid canal between the carotid artery and the optic nerve and compressing the latter.

ever, in two recent cases the aneurysms were similar to the largest reported, pushing the temporal lobe far upward and causing a high grade of intracranial pressure. In each the carotid was elongated by upward pressure of the mass (Fig. 38, B; also Fig. 5). In one it was easily clipped after a partial closure of the carotid, and the total cervical and intracranial ligations were followed by no bad effects. In the other (Case IX) the artery was entirely hidden by the bulge of the aneurysm, which had to be strongly retracted before it could be seen and clipped. A preliminary partial ligation of the carotid was not necessary in this case. In order to avoid any possible pressure of the aneurysmal mass on the posterior communicating artery with compromise of the cerebral circulation, the two ligations were made almost simultaneously. This precaution may or may not be important.

One death (10 per cent) resulted in the series. This was an arteriovenous aneurysm, caused by the rupture of a pre-existing arterial aneurysm in the carotid canal and was therefore recognized as such only at necropsy. Death resulted from the application of the usual clip to a greatly oversized sclerotic internal carotid artery; the artery

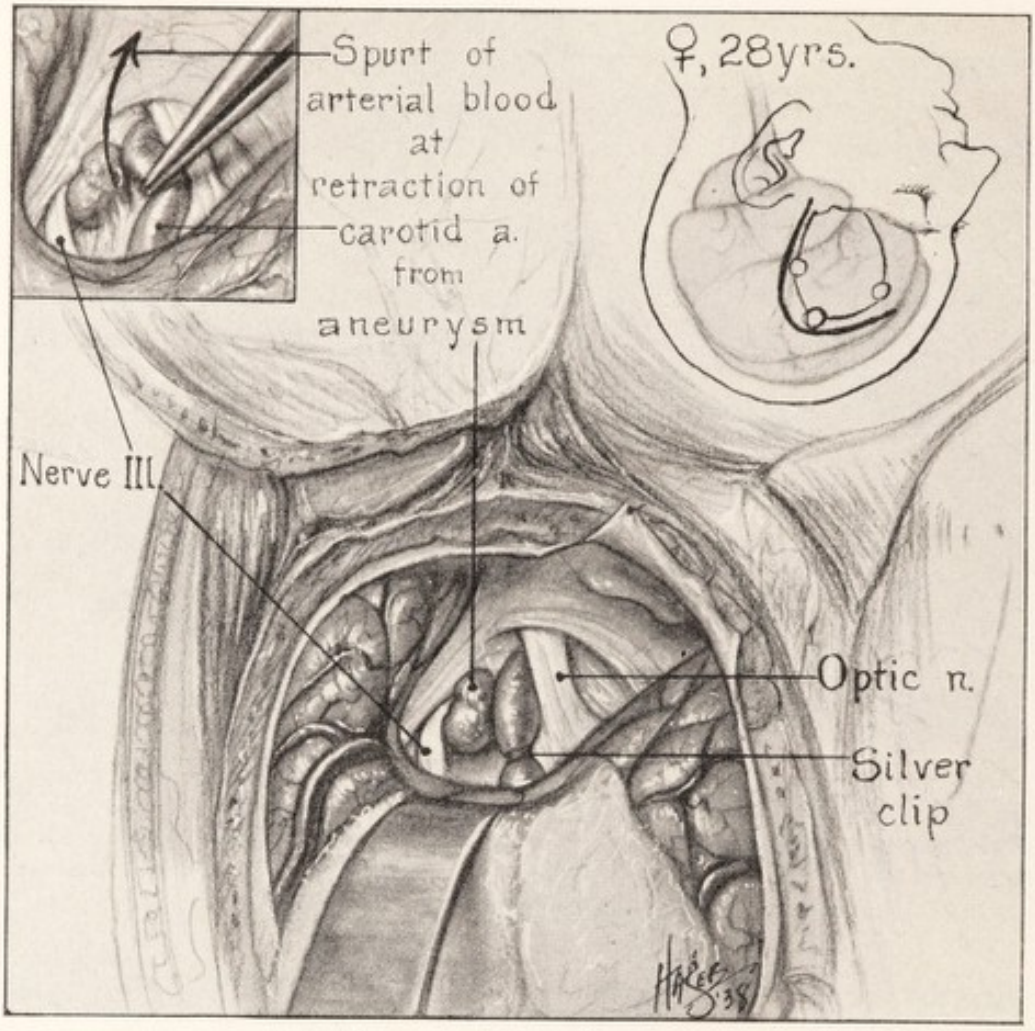


FIG. 39.—Drawing of aneurysm of the carotid artery exposed at operation. A clip has been placed upon the internal carotid artery intracranially. The upper left inset shows the forceps separating the artery and aneurysm and indicates a spurt of blood that immediately followed. The upper right inset indicates the position and relative size of the concealed cutaneous incision and the bone flap.

was torn by trying to force the clip astraddle it, and although the hemorrhage was controlled death followed 24 hours later, probably from the bleeding into the posterior cranial fossa. Death would have been avoided by using a larger clip. In 30 "clippings" of the carotid this accident has occurred twice; there were no ill effects in the second case.

The remaining nine cases are presumably cured: All are free of

symptoms. The longest case has gone 6¼ years; two have gone 4½ years; one four years; one 3½ years; one a single year, and three

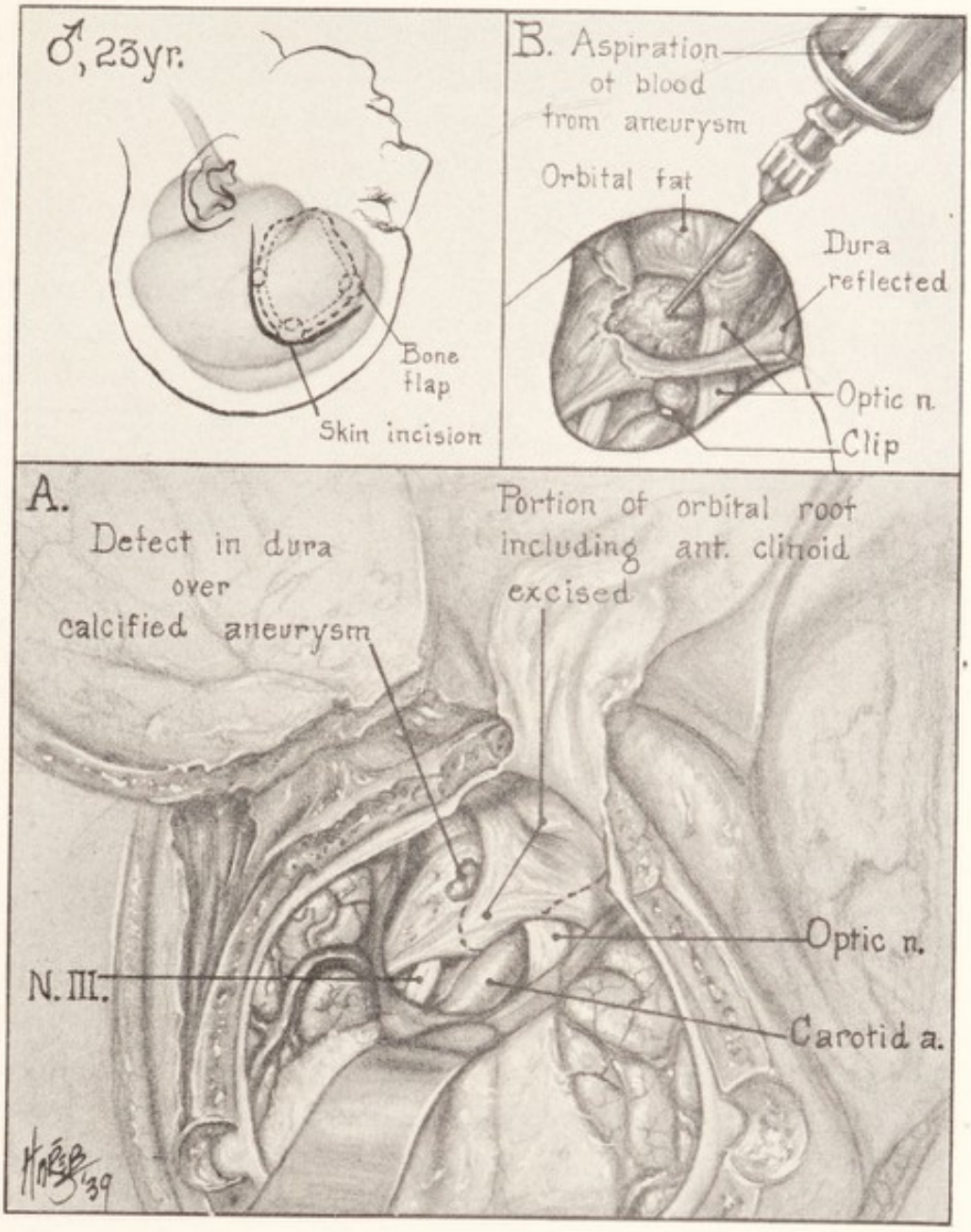


Fig. 40—Case 5, Table A. Operative drawing of aneurysm in carotid canal. It was known to have ruptured at the age of two. It had eroded the wing of the sphenoid and the floor of the middle fossa. Blood could still be aspirated from it. The contiguous findings are shown in Figure 41.

less than a year. There is every reason to believe that all these are cured.

The procedure of "trapping" the aneurysm is identical with that used for carotid cavernous arteriovenous aneurysms. It may be questioned whether the collateral circulation through the ophthalmic ar-

tery, the only sizable branch entering the internal carotid artery between the two ligatures, might not be adequate to maintain the aneurysm, as it undoubtedly does in some of the arteriovenous aneurysms. This suggestion cannot be dogmatically denied, but there has been no evidence that the aneurysms are not cured. Moreover it does not appear probable, because the anatomical relations are entirely different. Arteriovenous aneurysms are notoriously difficult to cure because the arterial flow from collateral circulation passes through the aneurysm, whereas in the arterial variety the arterial blood passes into a closed sac. In Case XI the ophthalmic artery was easily exposed and clipped. Adson has recently done this in an arteriovenous aneurysm, and I have thrombosed this vessel on two occasions for similar lesions. Since the ophthalmic artery is rather difficult to expose for application of a clip, electrocoagulation will probably serve the purpose as well and be more simple of execution.

In Case III a plug of muscle was introduced into the carotid artery in the neck (Brooks's method) and blindness of the eye on this side followed. I interpreted this to mean that the propagating thrombus —rather than the muscle—occluded the ophthalmic artery and extended along its branches. In none of the cases where the arterial or arteriovenous aneurysms have been trapped has there been any disturbance of vision. The fact that vision is not lost by the double carotid ligation proves that there is ample collateral circulation between the ophthalmic artery and branches of the external carotid. Elschnig (1911) injected water into the external carotid and saw it come out of the ophthalmic artery. Walsh and King (1942) have injected the external carotid in cadavers (the carotid being tied intracranially and in the neck), with dyes and radiopaque substances and found the injection material passing out of the ophthalmic artery.

The rapidity and degree of return of function of the third nerve has been variable. In Cases IV and X all subjective or objective evidence of palsy had disappeared within six weeks, and in Case VI recovery was complete in three months. In Case II (Fig. 43) the ptosis cleared completely, but there remained a slight residual palsy of the extraocular movements when last seen three years after the operation. In Case III there has been marked improvement in all functions, but the recovery of function is still incomplete after 4½ years. This function had been lost for 22 years in Case V (Figs. 40 and 41) ; 19 months after the operation there was improvement in the ptosis but none in the extraocular movements. It is open to doubt whether marked im-

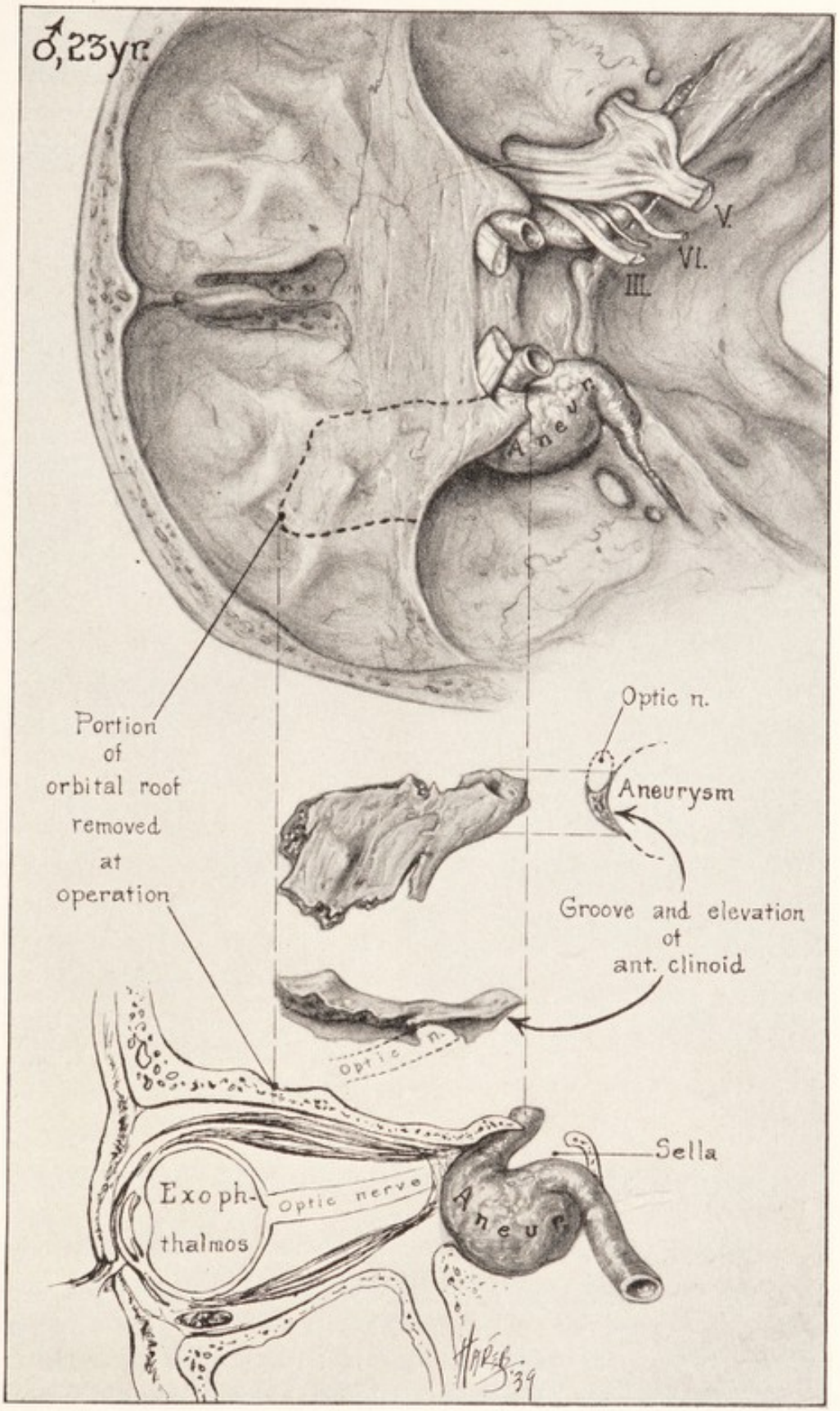


FIG. 41.—Drawing showing reconstruction of the aneurysm and its effects upon the contiguous structures. (Case 5, Table A. See Fig. 40.)

provement can occur after this long interval. The degree and rate of return probably depends upon the extent to which the third nerve is incorporated in the aneurysmal sac, and doubtless, too, upon the duration of the paralysis. In a recent case where the aneurysm protruded into the cranial chamber alongside the third nerve it was

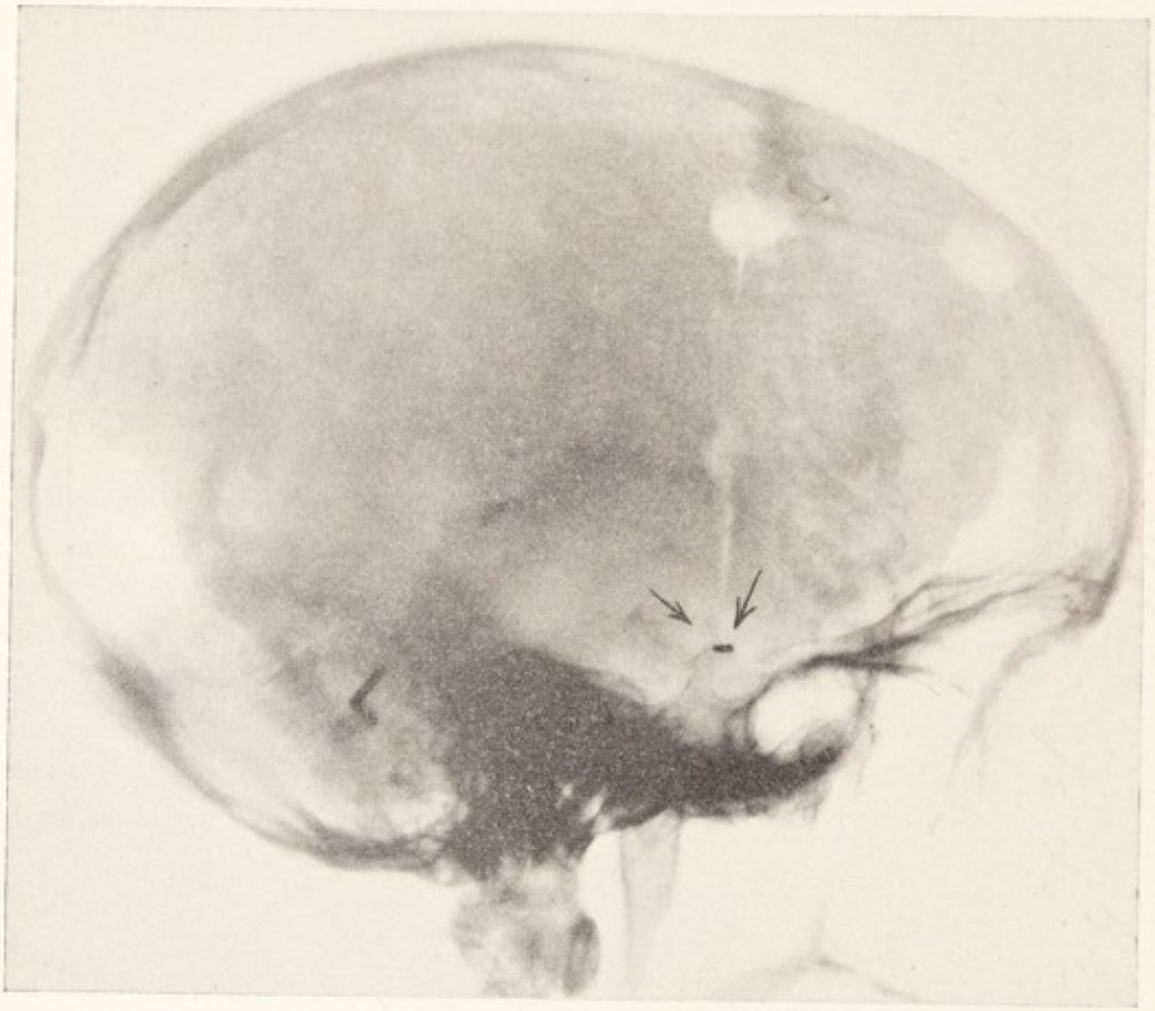


FIG. 42.—Postoperative view of silver clip on the internal carotid. (Lateral stereoscopic plate.)

shrunk by the cautery in order to free the third nerve from its contact (Fig. 38).

It has been suggested that simple ligation of the internal carotid in the neck might be ample treatment. This has been done for over a century in occasional cases where aneurysms have been suspected, but there has been no proof that an aneurysm has been the lesion and that it has been cured. It is quite possible that a propagating thrombus resulting from such a ligature might obliterate the aneurysm, such as at times follows this procedure in the arteriovenous aneurysms, but it would only be a chance occurrence. Walsh and Love

(1937) present the most conclusive report. Since aneurysms are of such vicious character and so prone to rupture, an immediate cure which entails only slight risk and leaves nothing to chance is certainly the safest course. It will be remembered that many of these aneurysms extend into the cranial chamber and that these projections have very thin walls. In many of these patients rupture had already occurred. In two of our cases it was only through apposition of the sac to the wall

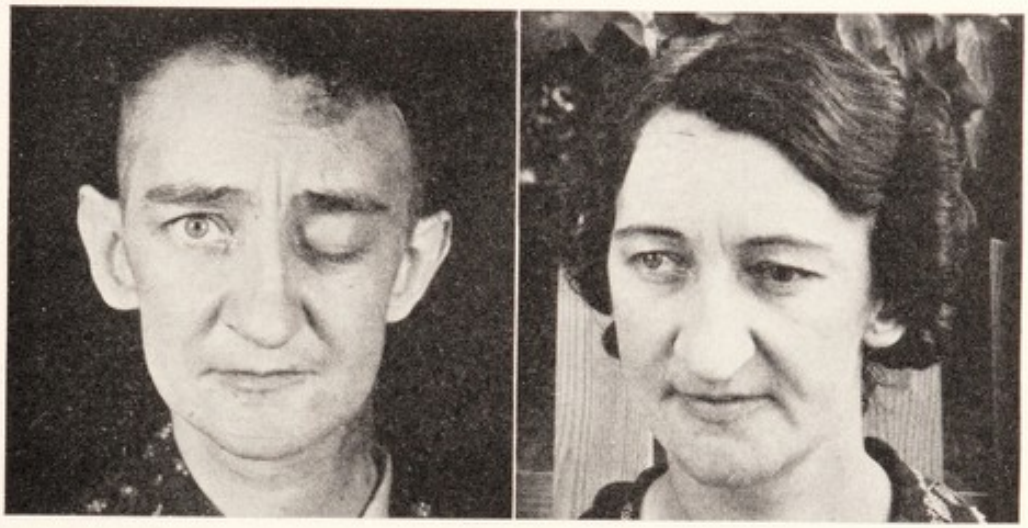


FIG. 43.—Photographs showing (*left*) ptosis before and (*right*) its disappearance after cure of aneurysm in the carotid canal.

of the carotid or third nerve (Fig. 39) that bleeding had been spontaneously suppressed, but with the most insecure and only temporary result. In Case II blood spurted when the sac was barely separated from the carotid. As the sac fell back upon the carotid the bleeding stopped; a slight increase in arterial pressure would surely have caused another and probably fatal rupture.

Treatment of Aneurysms of the Intracranial Internal Carotid (Table B)

Sacculated Aneurysms Before the Carotid Branches (Cases VII–XVI)

Next to aneurysms of the carotid in the carotid canal this group (*b*, p. 35) offers the best prospects of cure by surgery. The aneurysms are small. All have been about the size of a pea or a little larger, and all have had a neck at the point of origin from the arterial trunk.

There are three methods of curing aneurysms of this type surgically: (1) by clipping the neck of the aneurysm, (2) by clipping the internal carotid artery intracranially both above and below the neck of the aneurysm, and (3) by clipping the internal carotid intracranially and ligating this vessel in the neck (Fig. 44).

In four cases (VII, VIII, IX and XVI) the necks of the aneurysms were clipped (Fig. 45) and resulted in cures. This is perhaps the

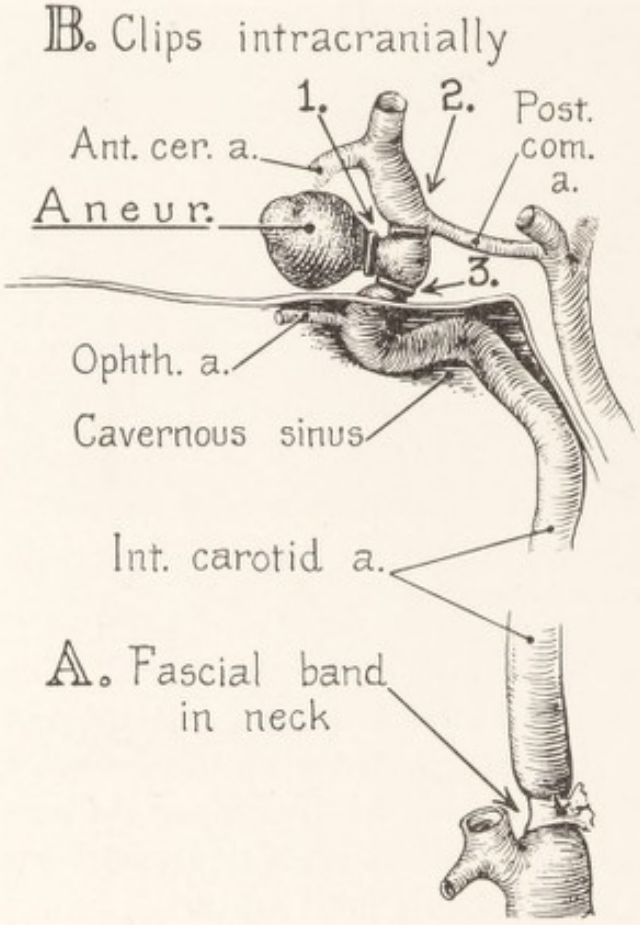


Fig. 44.—Diagram showing location at which clips are applied for the cure of an aneurysm in the internal carotid before its first branch is given off. Clips may be placed on the neck of the sac or on the internal carotid above and below the neck of the sac.

ideal method, since it is not necessary to eliminate the carotid artery, but there is risk because the neck of the aneurysm may be defective and when the clip is applied it may cut through.

The first patient (Case VII, Fig. 46) died 2½ years later of pneumonia in another hospital. Since operation he had had no symptoms whatever referable to his old aneurysm, and his third-nerve palsy had completely disappeared. An autopsy was not obtained.

The second patient (Case VIII, Fig. 47, A) is living and well 2½

years after operation. Since he was 60 years old and would not tolerate carotid compression in the neck (Matas' test), the internal carotid was partially occluded in the neck (with a fascial band) 26 days

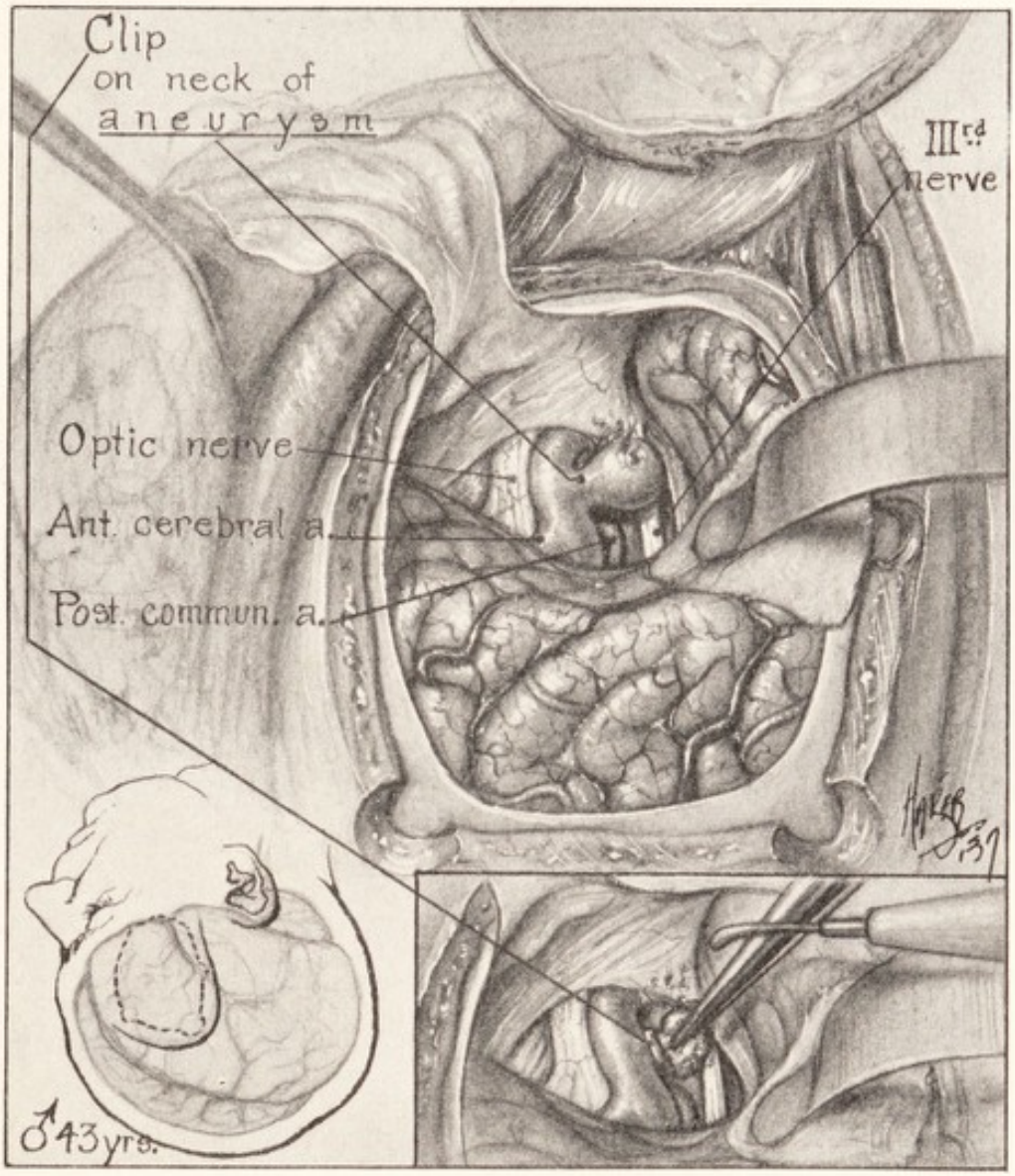


FIG. 45.—Typical aneurysm of the intracranial internal carotid artery, showing the narrow neck of the sac and the bulging aneurysm; also the point of rupture. The inset shows the clip placed upon the neck of the aneurysm, and the aneurysm itself shriveled with the electric cautery.

before the aneurysm was exposed and so treated. The carotid therefore was not totally ligated at any time.

In Case IX the neck of the aneurysm was also clipped, but owing to defective walls the neck ruptured and a severe hemorrhage followed. The bleeding was controlled and the artery was clipped above and below the point of rupture, and the patient made an uneventful recovery.

The aneurysm was dissected from its bed and *removed completely* (Fig. 48).

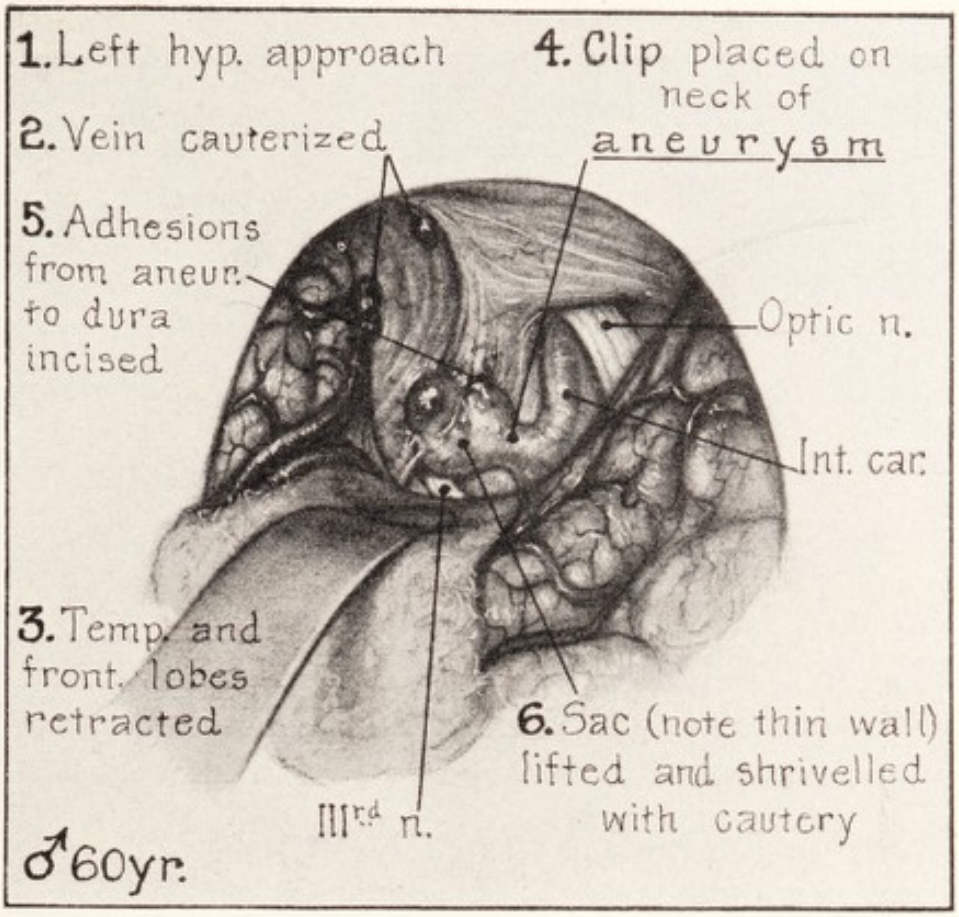
In view of this operative complication—which has since occurred in another case—it might be preferable to use the second method in these cases, i.e., to clip the artery above and below the sac; or, if there should be insufficient room to apply the clip below the aneurysm, to ligate the carotid in the neck (third method). However, this method also is not without its dangers, perhaps even greater than clipping the neck of the sac, for one is rarely certain of the exact position of the posterior communicating artery, and to include this vessel in the clip

FIG. 46.—Case 7, Table B. Showing (*left*) ptosis before operation, (*center*) marked improvement 13 days after operation, and (*right*) complete return of function seven months after operation.

placed above the aneurysm may be no less serious than clipping the aneurysmal neck.

In four cases the carotid was either clipped above and below the aneurysm or clipped above the aneurysm and ligated in the neck. In Case X (Fig. 49) the internal carotid was coagulated instead of being clipped, because the aneurysm ruptured when the neck was being isolated and the bleeding was controlled by the cautery. Forty days later, when at home and apparently well, the patient died of a cerebral hemorrhage, doubtless because the coagulated vessel gave way. Had the artery been ligated in the neck this fatality—the only one in the series—would have been avoided. From the entire group of nine operated cases eight have been cured.

One of the above patients (Case IX) died ten months later of an entirely unrelated and unexplained condition—anemia. At necropsy



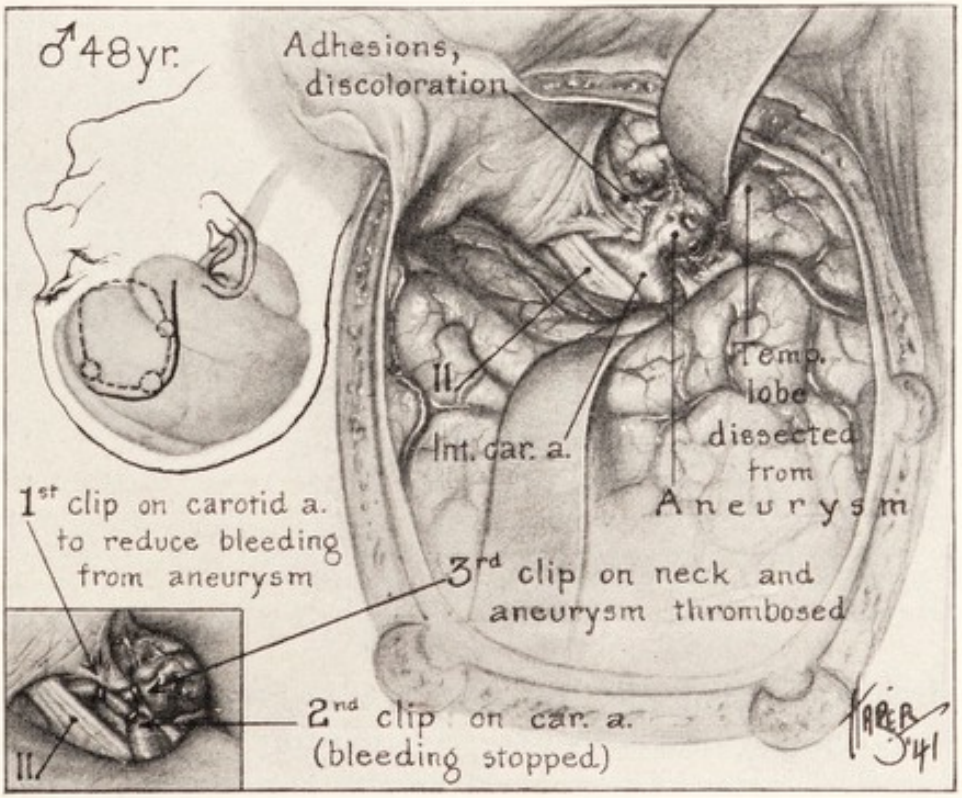


FIG. 47.—Two additional aneurysms of the internal carotid exposed at operation, (*upper*) Case 8, Table B; (*lower*) Case 11, Table B. Note the adhesions in both cases as the result of previous rupture. In Case 11 the neck of the sac tore when being clipped and the carotid artery was then clipped above and below the neck.

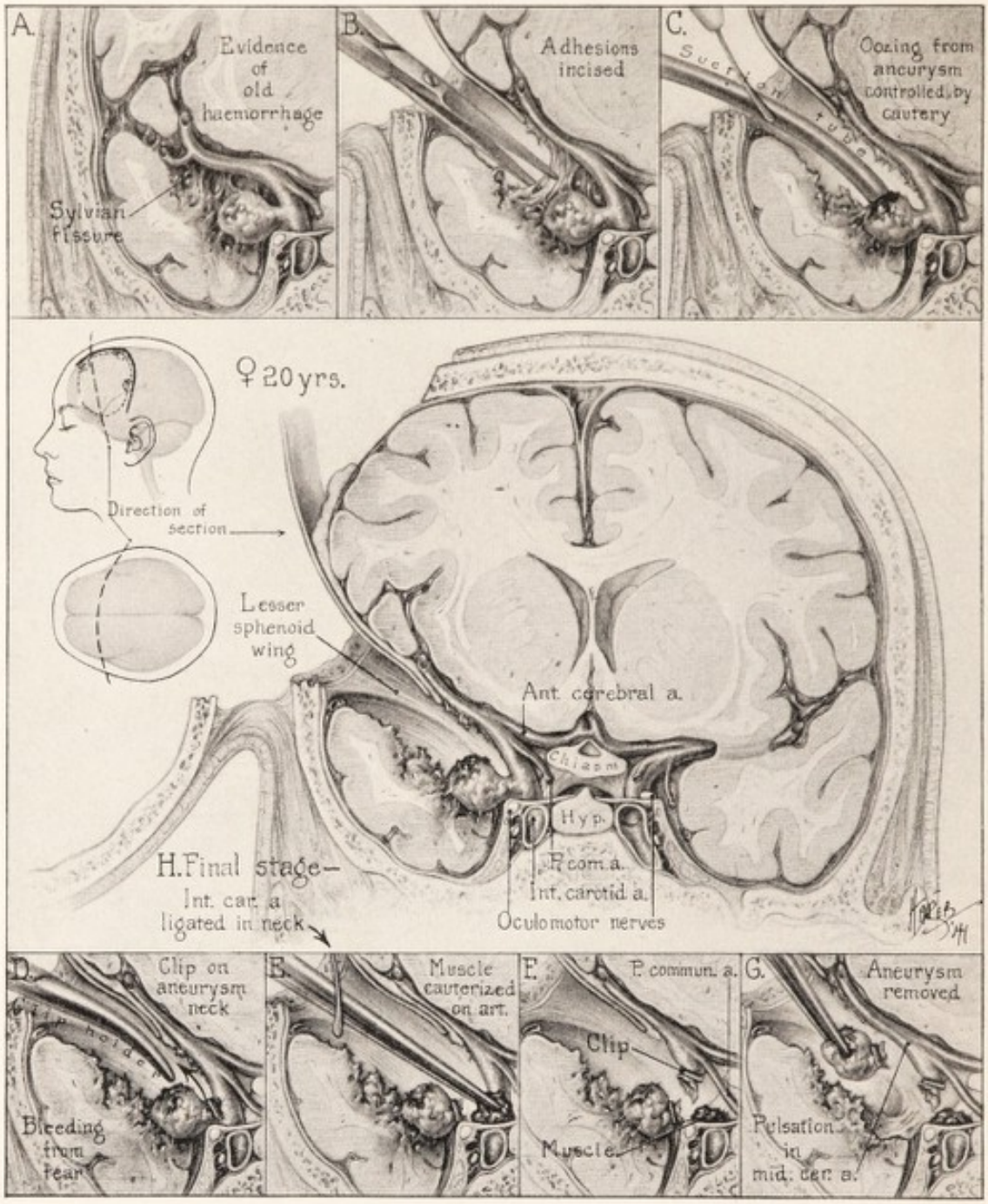


FIG. 48.—Drawing of operative attack upon a sacculated aneurysm of the internal carotid which ruptured when the clip was applied to the neck. The internal carotid was then clipped above and below the neck of the aneurysm and the aneurysm was completely excised. Inset G, the eroded brain tissue; the adhesions about the aneurysm were due to previous rupture into the Sylvian fissure.

the adrenals, thyroid, and hypophysis were greatly atrophied. The brain was normal. Ligation of the carotid could, of course, in no way have been responsible. The cerebral circulation had been normal (since there was no cerebral atrophy) but was carried solely through the anterior cerebral artery; the posterior communicating artery had

been thrombosed at the time the carotid was coagulated by the electrocautery (Fig. 50).

The mortality in this group, therefore, was 11 per cent, and the operative cures were 89 per cent.

A major risk attending operations upon this group of aneurysms occurs when the aneurysm is being dissected in order to determine its

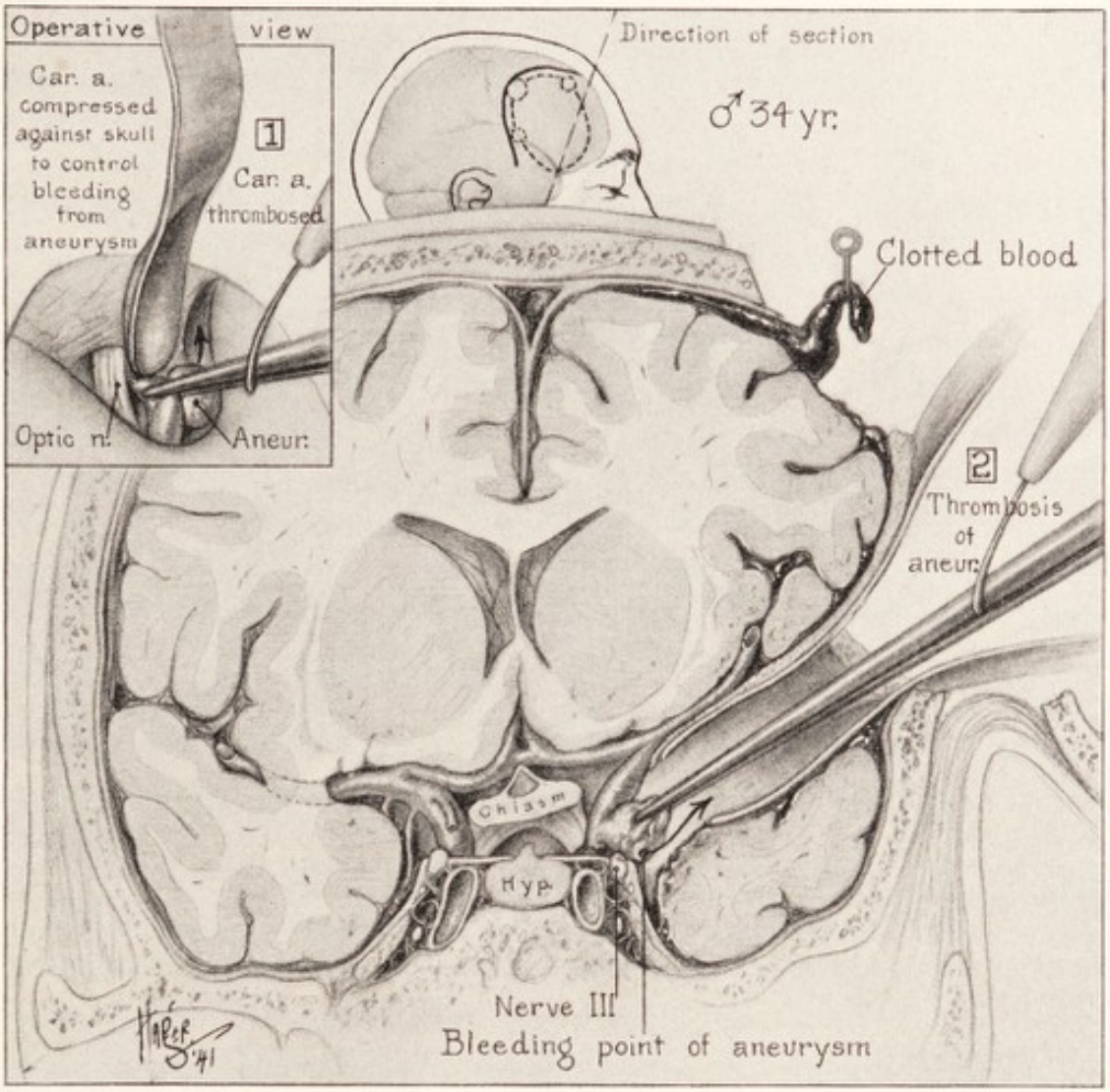


FIG. 49.—Diagrammatic sketch of operative attack upon sacculated aneurysm of the carotid during the acute stage of rupture. There was a subdural hematoma over the hemisphere and blood in the sulci. This aneurysm ruptured during its dissection and the bleeding was controlled with the electric cautery.

point of origin (Fig. 49). Aneurysms in this region, though small, frequently fill all or most of the space between the circle of Willis and the base of the skull, and their point of origin may be from the internal carotid or possibly the posterior communicating artery, or it may be above or below the posterior communicating artery. To de-

termine the line of treatment, if any, it must therefore be known exactly where the neck of the sac arises. In most instances the sac has already ruptured before operation and the sealed opening is a very flimsy covering of fibrin that frequently ruptures when lightly touched in dissection. And a ruptured aneurysm is a hazard requiring the utmost in surgical skill for the control of hemorrhage.

Three of these cases ruptured (Fig. 47, B) during this manipula-

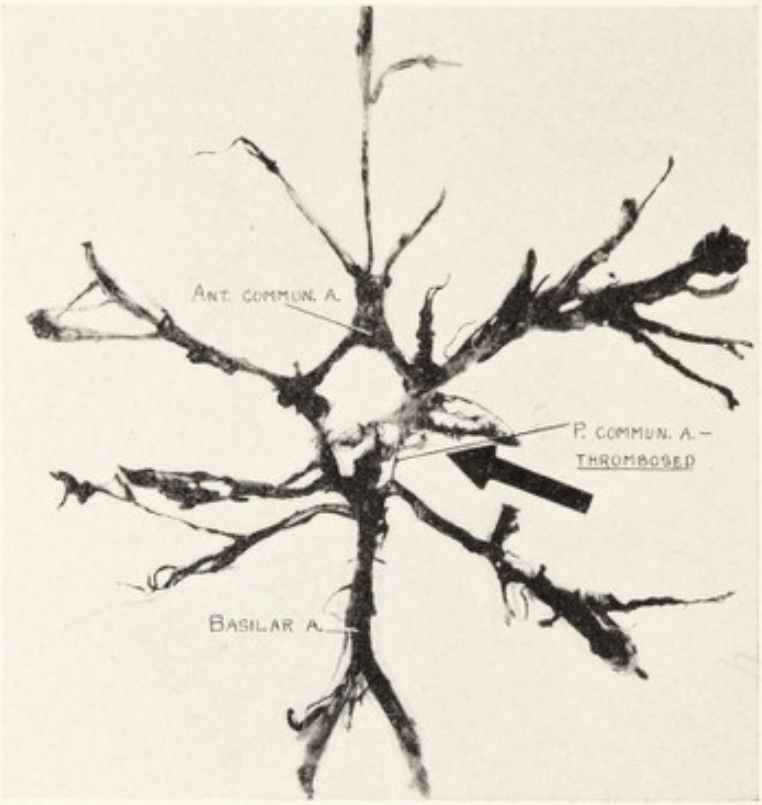


FIG. 50.—Photograph of circle of Willis at necropsy from Case 9, Table B, ten months after operation. (Courtesy of Dr. Francis Grant.) The posterior communicating artery had been destroyed by the cautery at the time the aneurysm ruptured at operation. The circulation was maintained through the large anterior communicating and anterior cerebral arteries.

tion; fortunately all were saved without aftereffects. In one of these the hemorrhage was not violent; as the aneurysm was gently separated from the carotid, a spurt of arterial blood followed, but stopped when the sac fell back upon the vessel to which it had been sealed. In the other two cases the hemorrhage was full blown—quickly filling the wound. It is probable that in the earlier cases the dissection of the sac was carried farther than the surgical indications demanded, because our efforts were then directed solely toward clipping the neck of the sac.

It is probably advisable, after the aneurysm has been isolated by

clips and ligatures, to shrivel the sac with the electrocautery (Fig. 45) in order to pull it away from the third nerve. There is reason to believe from a single case at necropsy (Fig. 13) that the firm attachment of the aneurysm, even when cured, to the third nerve will prevent the return of its full function. A few light applications of the cautery to the surface of the sac will contract it immediately; its base can then be dissected from the third nerve without risk.

Treatment of Diffuse Aneurysms of the Carotid (Cases I–VI)

In this group (*a*, p. 33) are two aneurysms (Cases I and II) that were probably congenital in origin; they involved the entire circumference but not the entire length of the vessel. Case I was disclosed at operation 15 years ago. At that time the cure of an aneurysm was scarcely conceivable; it could now be cured easily by ligating the carotid intracranially and in the neck. Had it been possible to explore the aneurysm in Case VI there were possibilities of a cure, for although the aneurysm was much larger than the preceding one there was room between its upper terminus and the posterior communicating artery to apply a clip. The only symptom, antedating rupture, had been migrainous attacks on the same side.

Our experience has shown that operations upon those in the arteriosclerotic group are very dangerous, for two reasons: First, arterial supply of the brain is usually severely compromised on both sides because of widespread thickening of the arterial walls; closure of the internal carotid in the neck, either partially or completely, must throw a severe burden upon other arteries that are ill equipped to sustain it. Secondly, any trauma, however slight, to the wall of the carotid intracranially may quickly increase a preexisting or produce a new thrombus in the arterial lumen and hasten its closure.

In Case III the left optic nerve was sectioned and liberated from the greatly oversized and rigid artery, which was incompressible and like a pipe stem. It was hoped that by section of this optic nerve the contralateral optic nerve might be freed from its close contact with the other aneurysm. This at least seemed to be the only conceivable operative attack that could offer any relief. Three days later, after an uneventful postoperative period, left-sided hemiplegia developed over a period of a half hour, and without loss of consciousness or headache; death followed two days later, very probably from carotid

occlusion (autopsy not obtained). How much, if any, the operation contributed to the increase of the vascular occlusion can only be conjectured, but the close time relationship suggests the probability that trauma activated a pre-existing thrombus. Moreover, it is noteworthy that the occluded artery was on the operated side, i.e., it was the artery that had been exposed and liberated from the nerve at operation. My guess would be that thrombosis was hastened by the local effect of slight manipulation of the vessel. The optic nerve had been stripped from the adventitia, to which it was quite snugly adherent, by sharp and blunt dissection. The postoperative story in Case IV is not dissimilar except that the patient survived. Eighteen hours after the operation she suddenly became hemiplegic and aphasic, although the only trauma to the internal carotid artery was in freeing the optic nerve from the adherent aneurysm. Here, too, the vascular occlusion (or at least this I presumed to have been the lesion) was on the side of the operation. Function began to return six days later and rapidly improved; three weeks later all function had completely returned, but she has since had occasional convulsions beginning on the right side. She is still alive (3½ years) and well except for the seizures.

Treatment of Terminal Aneurysms of the Internal Carotid (Cases XIV–XXXVIII)

Only six aneurysms of the group (*c*, p. 40) were exposed at operation, the remainder being postmortem findings of patients not subject to operation.

Two of the five (Cases XVIII and XIV) have been cured, but one (XIV) survived with hemiplegia and aphasia, probably permanent in large degree.

Case XXXIX is the last one in the series to be operated upon, and the assumption of a cure is not therefore based upon the verdict of time but upon what is known to have been accomplished. Until this operation I had assumed that aneurysms arising at the branches of the carotid could not be cured. This patient, a female aged 51, had a negative Matas test, so that partial occlusion of the carotid was not necessary. The lesion was known to be an aneurysm because there had been four hemorrhages in the past two months. Its localization was known by a quadrantal right upper homonomous visual

defect. The aneurysm had also been beautifully demonstrated by angiography (by Dr. Crutchfield of the University of Virginia).

At operation there was a subdural hematoma (about 2 cm.) covering most of the left hemisphere, and a firm clot, 3 cm. deep, under the frontal lobe. The aneurysm was quickly located just posterior to the optic nerve and along its entire length. The aneurysm was probably 1.5 cm. long and completely obscured the internal carotid artery, but from its inferior border there was a band of adhesions, perhaps 0.25 cm. long, extending to and attached to the dura over the carotid artery. This was probably at the point of rupture of the aneurysm. At first this was taken to be the carotid, but with a little dissection it became attenuated and finally broken off (without bleeding). The internal carotid could be seen mesial to it. There was just room to apply a clip to this vessel, which was at a somewhat greater depth because of the pressure by the superimposed aneurysm. A defective silver clip cut through the vessel and a furious hemorrhage followed. This was controlled by the finger while my assistant ligated the internal carotid in the neck. The operative field for this exposure had been included before the operation began. There was still quite vigorous bleeding when the finger was removed, but the suction tube could be pressed against it and that stopped the bleeding. Application of the electrocautery to the tube then coagulated the carotid and all bleeding stopped. The spatula then retracted the aneurysm to its neck, which was at the origin of the posterior communicating artery (Fig. 51). The neck of the aneurysm, being defective, also tore at this stage and blood again spurted, but prompt pressure of the spatula stopped this. The cautery was applied through the sucker, and this bleeding point, together with the entire neck of the aneurysm, was coagulated, after which the wound was dry. The aneurysm was then partly shriveled by applying the cautery to its outer wall.

When the operation was concluded the patient was talking and moving her right side, and her postoperative course was smooth and uneventful. It must be admitted that this fortunate outcome was the result of the torn carotid artery. I had intended to do nothing more than clip the carotid, being fearful of dissecting such a large aneurysm, and at the same time it seemed doubtful if one of these aneurysms—so high up on the carotid—could be cured. There can be hope for other cures by this method, i.e., by exposing and sealing

the neck of the aneurysm and shriveling the aneurysm itself with the electrocautery.

Inspection of the angiograms had clearly demonstrated that the orifice of the anterior cerebral artery was well above the posterior

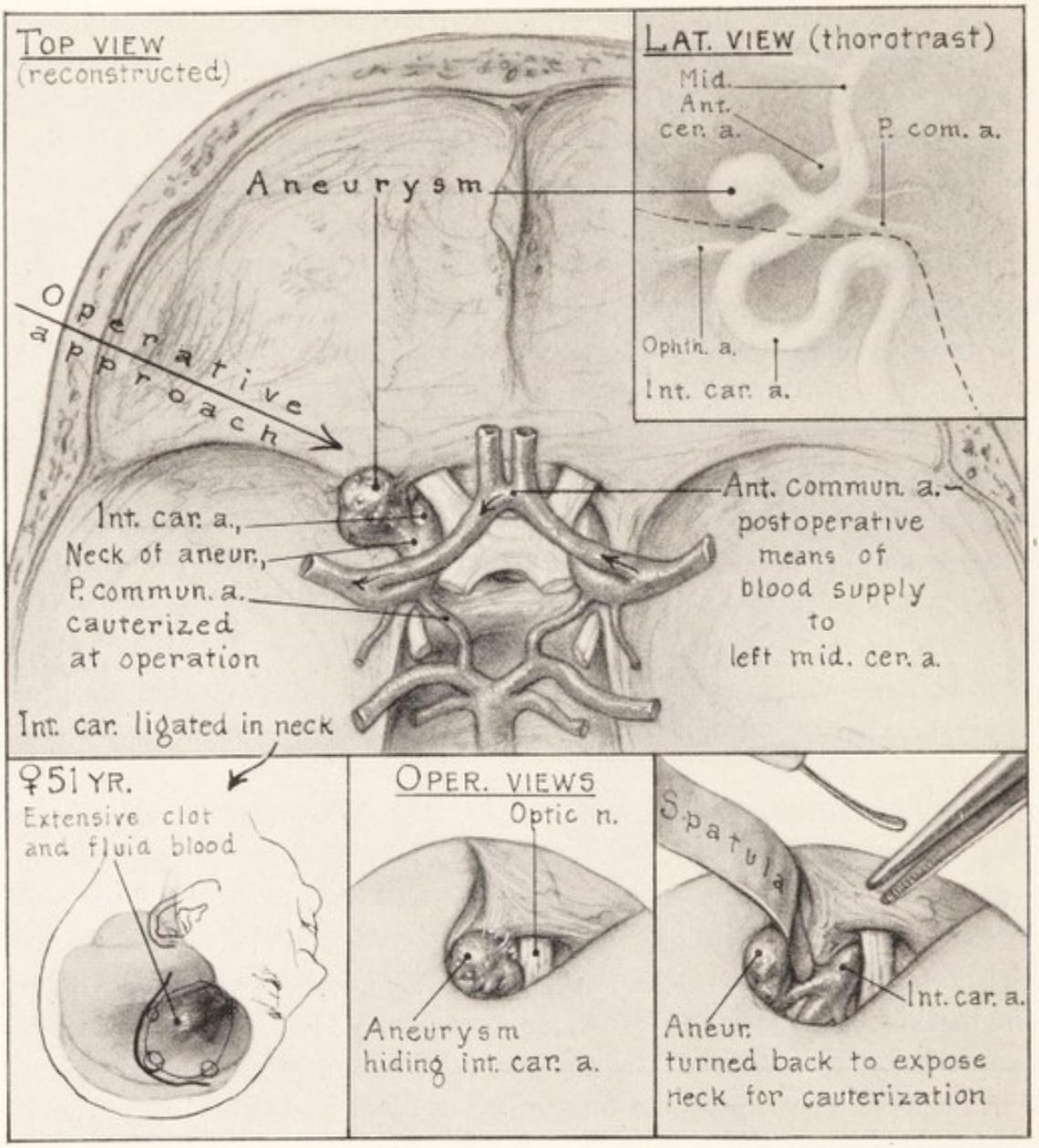


FIG. 51.—Case 39, Table B. Aneurysm of the internal carotid turned back with the spatula, thus exposing the neck, which was thrombosed with the cautery.

communicating artery and the neck of the aneurysm, and there was therefore a fair margin of safety in coagulating both. It did not, however, indicate that the anterior communicating artery was able to take care of the collateral circulation, if the posterior communicating artery had been injured.

Case IX is another example of loss of the carotid and posterior communicating arteries, with perfect preservation of cerebral function over a period of a year (necropsy).

In two cases of this group (XVII and XVIII) the same fortunate

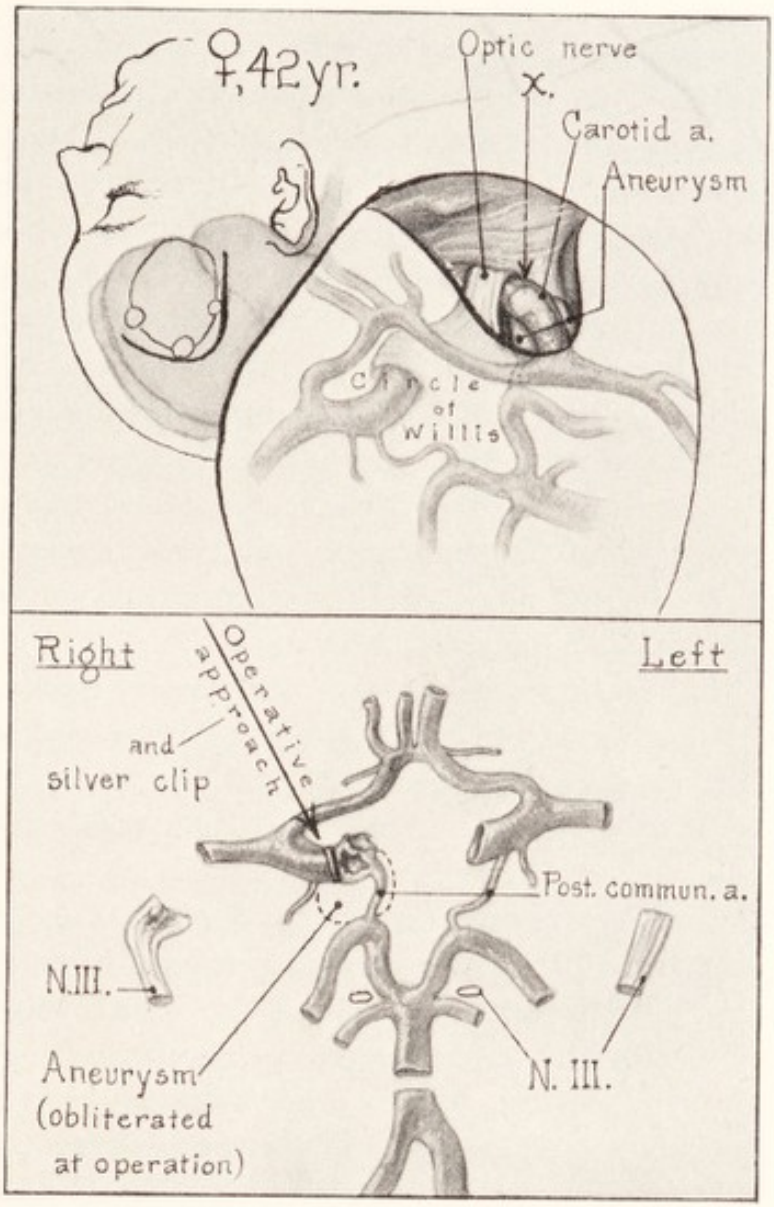


Fig. 52.—Drawing of postmortem specimen in which the posterior communicating artery arose abnormally low and was therefore below the clip that occluded the carotid artery. Despite the fact that the anterior cerebral and communicating arteries appeared to be of adequate size, the patient had hemiplegia immediately after the operation.

outcome did not occur when the posterior communicating artery was sacrificed. In both the posterior communicating came off below the aneurysm, which was thought at the time of operation to be well below this vessel, but this vessel was found at necropsy to be abnormally placed (Fig. 52). Both were hemiplegic when the operation

was completed. In Case XVII the anterior communicating artery was normally present but apparently inadequate; and in Case XVIII it was a mere thread without a lumen. Probably the latter condition would be found in Case XIV of the preceding group (B) if the brain could be examined.

Other aneurysms of similar location and when similarly treated should be cured when the anterior communicating artery is intact. So far there is no test for this vessel's presence, although its actual absence must be more frequent than the literature indicates. However, its actual size must always be regarded as uncertain.

Only rarely does one actually see the posterior communicating artery. When normally placed, it is well above the point at which these ligations were made, but unless this vessel is actually seen one is always taking a chance on its not uncommon congenital misplacement (Fig. 52) and upon the integrity of the anterior communicating artery as well. In each of these cases a clip might have been placed on the neck of the aneurysm with resulting cure. Although the method of clipping above and below an aneurysm appears to be safer in some cases, it may be more hazardous in others. It is not improbable that angiograms may yet perform a greater function by serving to demonstrate the exact position of the neck of the aneurysm and the position and adequacy of the collateral branches of the circle of Willis—serving even better than visual localization of the aneurysm.

This group of aneurysms can offer little, but perhaps something, by simple ligations of the carotid intracranially. One can, in the absence of any better treatment, hope for a favorable result and occasionally obtain one, such as perhaps results at times from ligations in the neck; this should be done when nothing more can be offered. Case XX was so treated and may be such a fortunate result. This was a very large aneurysm filling the region posterior to the internal carotid. This vessel was clipped a few days after sudden hemiplegia from rupture of the aneurysm. The patient is alive and in essentially the same condition 2½ years later.

The aneurysm in Case XIX was so solid that it was presumed to be spontaneously healed, and at necropsy this proved to be true—the only spontaneously cured aneurysm in the series.

Operations on Aneurysms of the Anterior Cerebral and Anterior Communicating Arteries (Table C)

Beyond the internal carotid there is a much more serious hazard in operative attack upon aneurysms. There many are larger, and the secondary feature of the intracerebral hematoma is frequently superimposed.

Six aneurysms in this group have been encountered at operation. One is certainly cured (XXV, Fig. 53) and another almost certainly (XXIV, Fig. 54). In Case XXV the aneurysm—the size of a pigeon's egg—was completely removed from the right anterior cerebral artery. The aneurysm was found when exploring the chiasmal region for loss of vision, the only subjective or objective disturbance. It must be admitted that the extirpation was begun under the impression that the mass was a tumor, and not until there was a sudden burst of hemorrhage was its character appreciated. Fortunately the internal carotid had been exposed and was promptly clipped, and all bleeding ceased at once. It was known beforehand that the Matas test was negative. The patient has been perfectly well during the 13 months since operation. Despite the fact that the upper half of the right optic nerve was resected, the vision in this eye at the time of discharge from the hospital remained unchanged and has since greatly improved (Fig. 53, B). The vision in the left eye returned to normal both in acuity and field vision; before operation the visual acuity was 20/70 and colors were absent except for a small central area for red. Moreover, a central scotoma completely disappeared. Six months after operation the vision in the resected nerve was 20/70 as compared with 15/200 before operation, and the fields for both color and form had returned to normal except for a lower temporal quadrantal defect. The vision in the left eye had improved from 20/20 at the time of discharge to 20/15.

In retrospect, with another aneurysm of this type it would probably be preferable to clip the carotid before undertaking extirpation of the aneurysm.

The second patient (Case XXIV, Fig. 54) that I *think* is cured has also gone 13 months since the operation and has recovered most of the functions that were affected at that time. In this instance the aneurysm was situated just below the optic chiasm and projected

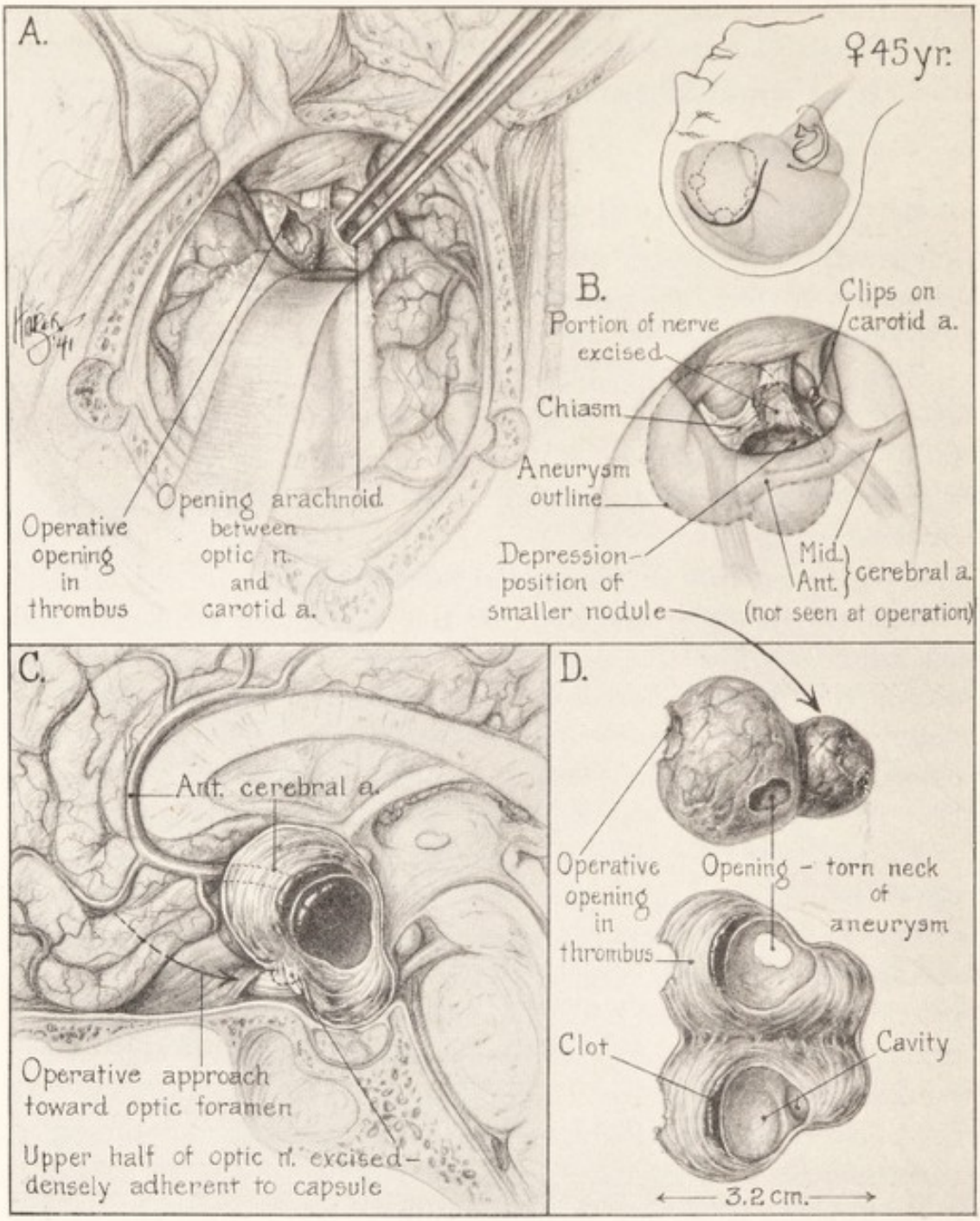


Fig. 53.—Case 25, Table C. Aneurysm of the anterior cerebral artery completely removed at operation. In liberating the aneurysm from the optic nerve it was necessary to excise the upper half of the nerve (B). The location and relative size of the aneurysm are shown in C. The opening into the anterior cerebral artery and the partial filling of the aneurysm with thrombus are shown in D. The aneurysm as drawn showed a length of 3.2 cm.

between the optic nerves. It was too large to permit dissection, completely filling the suprasellar space. It was opened with a narrow-bladed knife which struck blood at a depth of 1 cm. The cavity of the aneurysm was packed with two pieces of muscle, the first per-

haps as large as a hickory nut, the second much larger. The first piece of muscle was quickly lost in the big cavity. The second was coagulated with the cautery for some time before the bleeding ceased. The left internal carotid artery was partially closed with a fascial band seven days later, and totally occluded in another ten days.

Before operation the patient had had severe headaches for two years, marked motor aphasia, weakness of the right arm and leg,

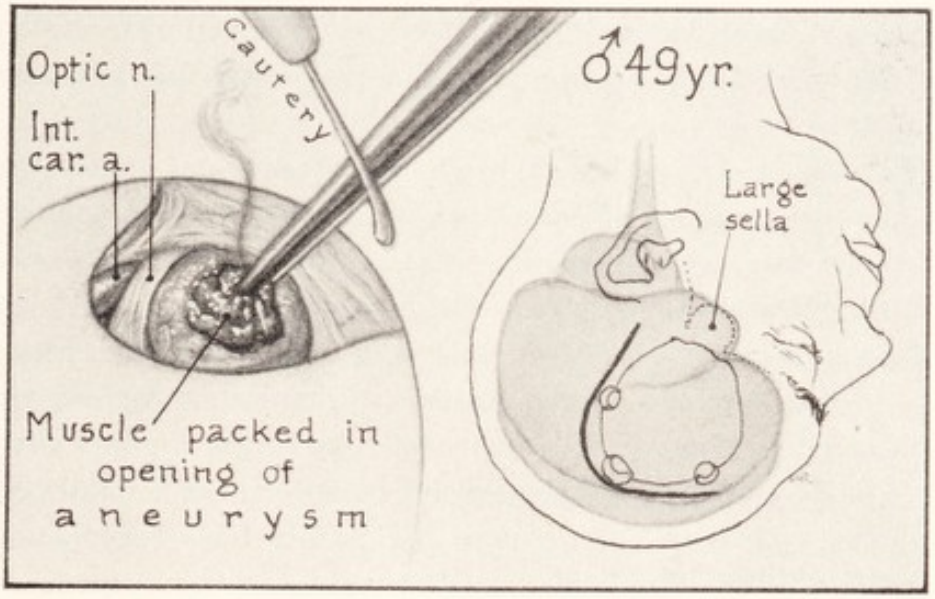


FIG. 54.—Drawing of aneurysm (Case 24, Table C) at operation. Note the insertion of large pieces of muscle into the aneurysmal cavity, after which the aneurysm was coagulated with the cautery.

and a right upper quadrantal defect in the visual fields, as well as marked loss of memory and personality changes. After operation his aphasia and motor weakness, loss of memory, and personality changes disappeared, and the headaches ceased. He has since returned to work (now 13 months after operation).

A third aneurysm of the anterior cerebral artery (Case XIV, Fig. 18) was partly excised 10 years ago, when surgery of this character was hardly adequate to the task and when the cerebral functions of the affected arterial trunks was less well appreciated. The patient died the following day. The aneurysm was exposed after resecting the frontal lobe. It is now known that if both anterior cerebral arteries are lost life cannot be preserved. The anterior cerebral arteries (probably the left, but collateral circulation will maintain either artery if only one is lost) supply that part of the brain which is responsible for consciousness, and when both are lost no motor or mental responses are possible. Since many of these aneu-

rysms are at the anterior communicating artery, and since both of the anterior cerebral arteries lie very closely together, the risk of operative treatment is very great. In this patient both anterior cerebral arteries had been lost. Another important point to remember is that after closure of an anterior cerebral artery the frontal lobe will probably become necrotic and it may later be necessary to extirpate it. This was true in Case XXV. After operation the patient had a series of convulsions and became drowsy. The wound was reopened and the soft necrotic anterior lobe was removed. Her subsequent course was uneventful and no subsequent convulsions have appeared (13 months).

In Case XII (Fig. 17) a huge aneurysm was encountered between the optic nerves. Because of its size and strictly median position there was no chance of isolating the neck of the sac or of determining the vessel from which it arose. It was hoped that cauterization by means of a needle passed into the aneurysm and cauterization of the surface might induce a thrombus within, but four months later the aneurysm ruptured—perhaps at the site of the needle's path, although this could not be determined with certainty.

Two aneurysms in this group (VI and XIV) were localized by ventriculography—the hemorrhage producing the space occupying mass. Both were localized by filling defects to the anterior part of the third and lateral ventricles and tumors were suspected. One (VI) was exposed by the pineal approach and the hemorrhage was evacuated, but the aneurysm was not seen. The patient died shortly afterward. The second case (XIV) was operated by the hypophyseal route, with the findings and results noted above.

The sixth case (XIII) was exposed during an operation for focal epilepsy and was diagnosed beforehand by a small round shadow in the x-rav. It was a small, white, firm, club-shaped structure dangling from the anterior cerebral artery, and was freely movable with the forceps. It was not responsible for the convulsions (the cause of them was found in the motor area), was causing no symptoms, and was not disturbed. Although the patient was only 26 years old his systolic blood pressure was 170.

Surgical Treatment of Aneurysms of the Middle Cerebral Artery (Table D)

Four patients from this series of aneurysms came to operation. The lesion was found in three of them and the hemorrhage—without detection of the aneurysm—in the fourth. There were no cures; all died shortly after operation. There is probably less likelihood of curing a middle cerebral aneurysm, at least in leaving a useful citizen, than in the case of any other aneurysm of the brain. All had ruptured, and as a result there was necrosis of most of the parietal and temporal lobes—in one case of the frontal lobe, where the hemorrhage had occurred. In one case (VIII) the aneurysm, as large as a hen's egg, was dissected down to an entering vessel; that was clipped and the aneurysm was removed (Fig. 22).

In all cases the lesions were assumed to be tumors at the time of operation. Since the aneurysms are always so intimately connected with the main trunk of the middle cerebral artery, it may not be possible to cure them without sacrificing this vessel, and such a result would be worse than death. If, however, a small unruptured aneurysm should by any chance be uncovered at operation it might be thrombosed with the electrocautery.

Treatment of Aneurysms of the Posterior Communicating Artery (Table E)

No aneurysm of the posterior communicating artery has been exposed at operation and none has been found at necropsy. A case not included in this series, because it was not an actual aneurysm, had a large anomalous artery passing from the posterior communicating artery to and through the dura at the entrance of the third nerve, causing paresis of this nerve. This vessel was clipped intracranially and the carotid intracranially and closed in the neck. The patient recovered completely and has been well 2½ years. The carotid was closed because it was thought that an actual aneurysm might be present in the carotid canal.

Treatment of Aneurysms of the Posterior Cerebral Artery (Table F)

There have been no operative attacks on aneurysms of the posterior cerebral artery. Both of the cases were necropsy findings, one a mycotic aneurysm and the other causing no symptoms. From the literature there are cases that are certainly potentially curable. Those within the occipital lobe should be curable by resection of the lobe together with the aneurysm, at which time the inferior portion of the arterial trunk would be ligated or coagulated with the cautery. Such extirpations should be possible with minimal risk. Aneurysms arising on this vessel just after it leaves the basilar artery might also be attacked by resecting the occipital lobe and then clipping or thrombosing the aneurysm on each side of its neck —a not insuperable problem. German (1938), in a report of a single line, states that he has excised an aneurysm of the posterior cerebral artery and that the patient recovered.

Treatment of Aneurysms of the Basilar and Vertebral Arteries and Branches (Table G)

Of the saccular aneurysms only two were operated upon. One (Case I) was found at operation after localization by ventriculography. It was shelled out with almost no bleeding, but the pressure within the posterior cranial fossa was extreme and the patient did not survive. This was seven years ago. An earlier operation on this aneurysm might well have produced a cure, and would almost certainly do so in the present status of intracranial surgery. At the other operation (Case II), 27 years ago, exploration of the cerebellum was negative and the aneurysm was disclosed at necropsy. One patient from the series had an aneurysm (found at necropsy) that could probably have been cured had it been uncovered at operation (Case VII, Table G). It was a small, sacculated aneurysm arising from the posterior inferior cerebellar artery and causing no intracranial pressure. It would have been quite easy to clip the neck of the aneurysm without injuring the arterial trunk. I know of no successful outcome from operative attack upon an aneurysm in the posterior cranial fossa, but for those on the vertebral and posterior

inferior cerebellar arteries, which afford good exposure, cures will certainly come in time. Five years ago I made the diagnosis of a ruptured sacculated aneurysm of the vertebral artery in a man, aged 54, with hypertension of 200. While hunting he had a sudden terrific pain in the occipital region. This cleared during the day but

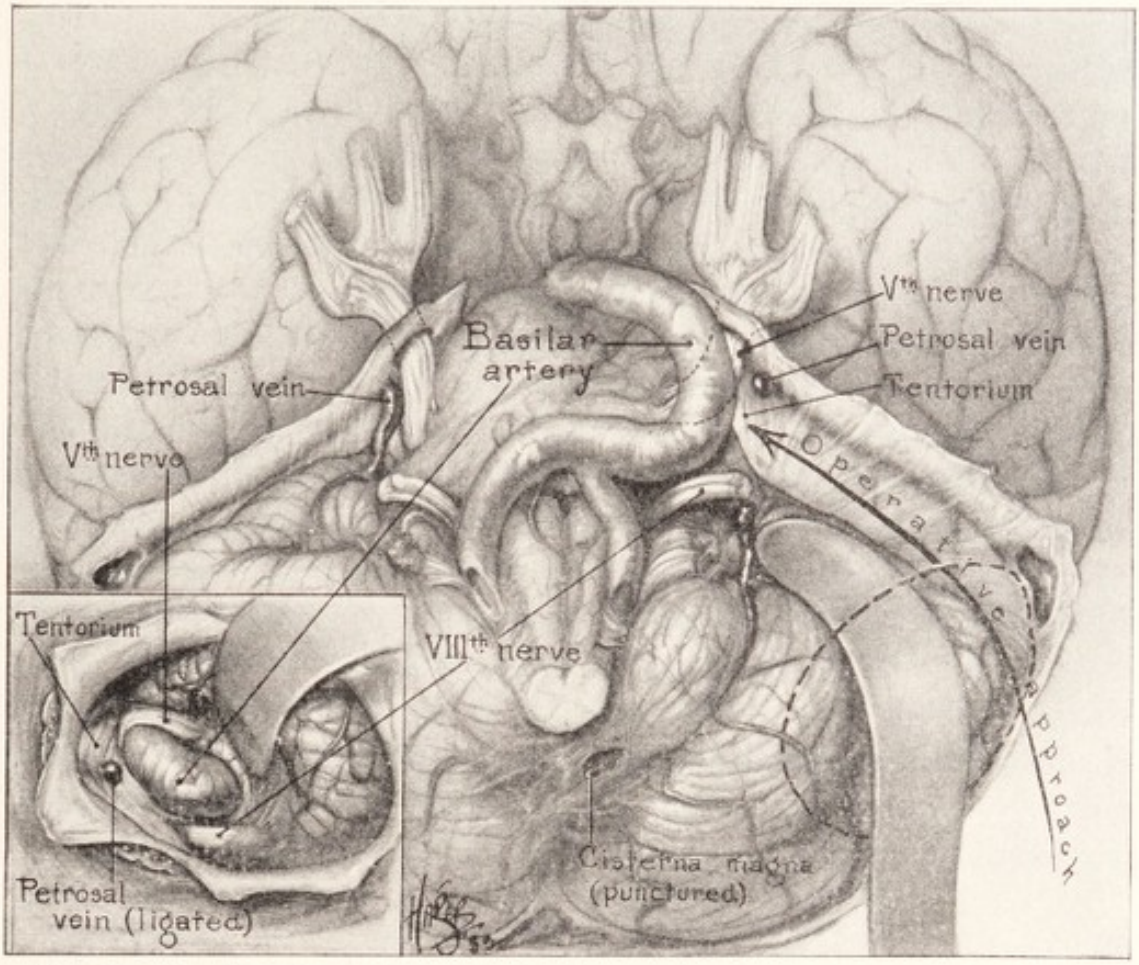


FIG. 55.—Drawing of a postmortem specimen of an arteriosclerotic S-shaped aneurysm involving both the vertebral and basilar arteries. This is a common lesion impinging upon the fifth nerve and causing trigeminal neuralgia. The inset shows the operative approach and disclosure of the aneurysm by the unilateral approach.

recurred in the same form 24 hours later and then radiated into the left temple and frontal region. There was total paralysis of the left hypoglossal nerve and definite weakness of the left spinal accessory functions. These objective findings, plus the sudden onset of terrific occipital pain, made the diagnosis of a ruptured sacculated aneurysm of the left vertebral artery appear certain, but I have no proof. The left vertebral artery was ligated between the atlas and axis. He is still free of all symptoms referable to this lesion.

For the elongated, diffuse, S-shaped aneurysms in the posterior

cranial fossa (of arteriosclerotic origin) there is probably little, if anything, to be done. Their typical appearance at operation is shown in Figure 55 (Case XXI). They probably have but little tendency to rupture and therefore do not require surgical efforts. However, in one patient (Case XI), aged 55 but with only slightly elevated blood pressure, the right vertebral artery was ligated between the atlas and axis. It was hoped that the lessened blood flow through the basilar artery might lessen the strain on the aneurysm and prolong life. The patient remains well two years later. With more advanced age, I have been fearful that, with the great degree of arteriosclerosis and consequent reduction in size of the lumen, ligation of one vertebral artery might too greatly reduce the blood supply to the brain stem. As there is no known test by which the patency of these vessels can be determined beforehand, one might well ligate the only patent vertebral artery. One patient (Case XIII, Fig. 26), had symptoms of brain-stem involvement, principally tinnitus and dizziness, so distressing that we ligated the left vertebral artery and a few weeks later compressed the exposed right vertebral artery with the forceps (certainly not longer than two seconds). She died instantly without another heart beat or respiration; there could be no more rapid death or one so silent. However, it is known from Alexander's ligations of the vertebral artery (1882) that both vertebral arteries can be ligated—even at the same operation—with impunity. Doubtless the excessive arteriosclerosis in our case was a determining factor.

VI

OPERATIVE RESULTS

Of the 108 cases of aneurysm in this series 64, or about 60 per cent, were found at operation and the remainder (44) were disclosed at necropsy. The necropsy reports of the hospital cover a period of 50 years, the operative results 20 years.

Our first cured aneurysm was 6¼ years ago, and this was the first time that an operation had been performed with the diagnosis of an aneurysm beforehand. Thirty-six aneurysms have since been disclosed at operation. Before that date the aneurysms found at operation were findings made accidentally in the course of search for tumors. With three exceptions all since that time have been diagnosed beforehand. It is therefore evident that the diagnosis of aneurysms can be made with a high degree of accuracy, and the results of this initial effort are much better than the character of the lesion would have indicated.

The results of the series in the past 6¼ years are as follows:

Type of Aneurysm	*Number of Cases*	*Cured*	*Probably Cured*	*Not Treated*	*Dead*
Carotid Canal	10	9	0	0	1
Internal Carotid (intracranial)	16	9	1	1	5
Anterior Cerebral	4	2	0	1	1
Middle Cerebral	1	0	0	0	1
Posterior Communicating	1	0	0	0	1
Posterior Cerebral	0	0	0	0	0
Basilar and Vertebral	4	0	0	4	0
Total	36	20	1	6	9

The mortality rate therefore is 25 per cent; cures from the total number of operations, 55.5 per cent, plus one additional probable cure; in 16.8 per cent there was no attempted treatment (except that in one the vertebral artery was ligated). If the number of aneurysms in which operative treatment was attempted (30 cases) is used for this statistical basis, the cures are 70 per cent. It is believed that, with due appreciation of the errors of the past, the subsequent results should be improved.

Literature on Surgical Treatment

Ligation of the carotid in the neck had long been occasionally employed when aneurysms were suspected. Holmes (1881) reported a case of aneurysm (Operator Coe) in the carotid canal as cured by carotid ligation, but the aneurysm was clearly arteriovenous, for which this treatment had been practiced much earlier and with a fair percentage of cures. Holmes said at the time: "We know nothing at present of the diagnosis of intracranial aneurysms so that no treatment can as yet be directed especially to it. And looking at the four large trunks that nourish the brain it seems unlikely that surgical measures directed to any of these would produce the consolidation of an aneurysm situated on any one of the main branches."

Dott (1933) reported two cases as well after carotid ligations, but the lapse of time since the operations was short in each instance. Walsh and Love (1937) reported that a case—probably of the carotid in the carotid canal but not verified—had been treated in this way and that shortly thereafter the extraocular palsies and trigeminal pain had disappeared and the patient was apparently cured. Vincent *et al.* (1937) added two cases in which improvement followed ligation for aneurysms of the carotid, demonstrated by angiography and by operation, but no claim was made that the patients were cured. Jefferson (1938) ligated the carotid in three cases, with subsequent improvement in two; paresis of the arm and face followed the ligation in one case. Improvement, or even disappearance of signs and symptoms, is not uncommon in the natural history of intracranial aneurysms. The ligation of the carotid therefore may or may not have had even a beneficial influence.

Application of muscle to an aneurysm was used by Dott (1933). He said that it was the only alternative to ligation of the carotid. Tönnis (1936) split the corpus callosum to expose a small cherry-sized aneurysm of the anterior cerebral artery. The aneurysm was covered with muscle. One of the most impressive results with muscle was by McConnell (1937). He opened the aneurysm and packed the sac with muscle, which was held in place by the finger until the bleeding stopped. The symptoms improved rapidly after operation and eleven months later the patient was reported to be in excellent health. This may well be a cure.

The introduction of the muscle into the aneurysmal cavity, prob-

ably inducing a filling thrombus, is a far more rational treatment than the mere application of muscle to its exterior. At the present time coagulation of the muscle within the sac, by the electrocautery, makes the cessation of bleeding much more secure and at the same time doubtless greatly favors the complete filling of the cavity by a thrombus (as in Fig. 54).

Fincher (1939) cured an aneurysm of the carotid by trapping it between an intracranial clip and a ligature in the neck. This is the only other report I have found in which this method of attack has been used.

Russell (1939) reported the actual removal of an aneurysm on the anterior cerebral artery (operation by Dr. William V. Cone of Montreal) with an excellent result and, of course, a permanent cure. This is the only extirpation of an aneurysm in the literature and antedated our case.

German (1938) notes without comment that he excised an aneurysm of the posterior cerebral artery—with recovery.

My former associate, Dr. Barnes Woodhall of Duke University, has just presented a paper, not yet in print, in which he reports the cure (15 months) of an intracranial aneurysm of the carotid by "trapping" between two intracranial clips. Another patient has been well for 15 months after clipping the carotid only on the proximal, i.e., the cardiac side of the aneurysm. A third case (not included in his report) is well two years after exposing the aneurysm and extirpating blood clot and the sac except for the part attached to the vessel; he encountered no bleeding.

Summary on Treatment of Intracranial Aneurysms

An absolute prerequisite to any form of treatment of an intracranial aneurysm involving the internal carotid or its branches is the knowledge of potential collateral circulation through the anterior cerebral and posterior communicating arteries. This is determined by the Matas test, i.e., obliteration compression of the internal carotid in the neck by the finger. If this temporary occlusion cannot be tolerated for 10 minutes, an attack upon any type of aneurysm would be hazardous and probably fatal, or at least the patient would be left permanently crippled, for in the vast majority of

aneurysms treated surgically the internal carotid must be occluded intracranially. On the other hand, an adequate collateral can be established by partially occluding the internal carotid with a band of fascia lata, reducing the lumen about one-half. A week or ten days later total occlusion can then be done without risk.

Aneurysms may be cured by one of the six following methods:

(1) Clipping the neck of the aneurysmal sac.

(2) Trapping the aneurysm between an intracranial clip and a ligature in the neck.

(3) Trapping the aneurysm between two intracranial clips.

(4) Excision of the aneurysm and closure of the entering vessel.

(5) Opening the aneurysm and quickly inserting a piece of muscle large enough to fill the sac; the muscle is then thoroughly coagulated with the electrocautery.

(6) Turning back the aneurysm and coagulating the neck of the sac and the aneurysm itself.

One of the greatest risks attending any treatment of intracranial carotid aneurysms is injury to the posterior communicating artery. If both the internal carotid and the posterior communicating arteries should be sacrificed, the entire circulation of the brain must be carried from the opposite side through the anterior communicating and anterior cerebral arteries, and this link may or may not be adequate. It has been adequate in two cases and inadequate in four.

Simple ligations of the cervical internal carotid may occasionally affect an aneurysm, but it is a shot in the dark and is usually ineffective. Without actual exposure of an aneurysm, the diagnosis cannot be established; the carotid ligation may therefore be based on a misconception.

Pieces of muscle placed upon or around an aneurysm can scarcely be effective in stopping hemorrhage from a leaking aneurysm, and cannot play any role in curing one. If more direct attack is not possible, the electrocautery is a far better means of closing the point of rupture and offers a chance of cure.

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NOTICE

The following charts are an important part of this book. They have been prepared carefully and folded exactly so as to be of the greatest possible service. Equally careful refolding after use will insure continued serviceability of the charts.

TABLE A.—Aneurysms of the Internal Carotid in the Cavernous Sinus

Number, Name and Date	Age	Sex	Location of Aneurysm	First Symptoms	Duration of Symptoms	Headache	Convulsions	Convulsions Focal or General?	Extra-ocular Palsies	Cerebral Hemorrhage	Other Outstanding Symptoms	Outstanding Signs	How Diagnosed	Course of Aneurysm	Result	Was Operation Performed?	Remarks
I Schooley 3/3/13 Figs. 1, 3	26	M	Bilateral in cavernous sinus and protruding far upward into brain, left 10 times as large as right	Loss of vision in left eye; 3 years later vision in right eye began to fade	4½ years (probably much longer, as noticed accidentally)	Severe frontal 3 mos. after visual loss first noticed; in bed 6 mos. on account of headache and vomiting; lost 65 lbs.; then blind in left eye. Only occasional headaches since	No		Left III, IV, and VI	No	Forgetfulness and loss of memory	Marked exophthalmos left; retinal veins markedly dilated left and right; optic atrophy left; papilloedemas right (4 D.); some mental changes; right hemianopsia temporal for colors, acuity 20/40. Long linear shadows in x-ray at base of brain; sella destroyed	Necropsy	Congenital		Decompression 14 months before death	Enormous left and smaller right calcified aneurysm in carotid canals. Mass filled base of brain and left middle fossa; all beneath the dura. Patient lived 14 mos. after first visit to hospital and worked steadily as a telegraph operator, but he says he had made more mistakes in past year than in all remaining years of work—absent-minded and casual and forgetful. Died 3 hours after attack of terrific headache and vomiting; then coma. There had never been pulsation of eyeball or any thrill or murmur
II Brunner 9/26/36 Fig. 39	28	F	Cavernous sinus and intracranial extension, left	Following mild headache for 3 mos., sudden severe one of 24 hours left side, blurring vision. After 3 more with semicoma had ptosis and diplopia	3 mos. and 5 weeks	Left frontal and parietal	No		Third nerve total	Probably	Recurring attacks of pain and ptosis		Operation	Congenital	Well 6¼ yrs.	Cured by clipping carotid intracranially and later in neck	When aneurysm gently separated from carotid with forceps, sharp bleeding; stopped when sac fell back on carotid. Repeated three times. Patient is perfectly well except for slight residual third-nerve palsy and blindness left eye. This was due to plug of muscle inserted in carotid (Brock's method). It is the only visual loss in the series. This is the first cured aneurysm; operation 9/26/36
III Ryan 4/11/38	37	F	Cavernous sinus and intracranial extension, right	Pain right eye and facial neuralgia with severe headache, nausea and vomiting, drooping right lid, diplopia	4 months	Terrific frontal	No		Third nerve total	No	Three attacks like that at onset in period of 3 weeks, noise in ear, enlarged right pupil	Complete ptosis and third nerve paralysis (rt.), faint systolic bruit over right orbit	Clinically and by operation	Congenital	Well 4½ yrs.	Cured by clipping carotid intracranially and nine days later internal carotid ligated in neck	Aneurysm protruded through into cranial chamber. Functions of third nerve markedly improved but not complete return
IV Smith 4/20/38	37	M	Cavernous sinus and intracranial extension, left	Left frontal headache, tingling left nostril, closure left eyelid	8 months	Frontal and pain in left eye recurring	No		Third nerve	No	Recurring attacks of headache and lid closure; left eye waters and left nostril runs clear fluid	Blood pressure 120/70. Diplopia with headaches, partial left third, exophthalmos 2 mm.	Clinically and by operation	Congenital	Well 5 yrs.	Cured by clipping carotid intracranially and five days later internal carotid ligated in neck	Patient has been perfectly well to date—5½ years. All signs and symptoms have disappeared
V Futch 4/17/39 Figs. 40, 41	24	M	In left cavernous sinus, calcified	Coma, after which left lid drooped	22 years	Left frontal pains and behind left eye	+ Five years	General, probably not related to aneurysm	III, IV complete, and VI partial	At age of 2 (history)	Lid has remained drooped since onset but has improved from time to time. Convulsions (5 years). B.P. 120/70. Pains left side head	Vision 10/400 left eye, 20/15 right eye. Exophthalmos 4 mm. left; calcified shadow left cavernous sinus; left pupil dilated and does not react to light; left visual fields somewhat contracted. Complete III and IV paralysis and partial VI palsy	Operation	Congenital	Well 3½ yrs.	Ligation left internal carotid intracranially and in neck (same day). Calcified mass exposed, blood aspirated	Patient was seen 14 months later. Lid could be opened partially, but other paralyses of extraocular muscles not changed. Vision 20/400 left eye (was 10/400 before operation)
VI Davis 1/23/39 Fig. 36	20	M	Right internal carotid probably bulging from carotid canal	Sudden severe pain right frontal while lifting heavy load. Ptosis (2 mos.)	3 months	Severe right frontal	+ Three seizures at time of onset	General	Complete III, IV, and VI	Probably	Thumping, synchronous with heart beat; stiff neck 2–3 days after onset	Spinal fluid bloody at onset of pain. Blood pressure 100/70	Operation	Congenital	Well 4 yrs.	Ligation internal carotid intracranially and in neck (both same day)	Hazel-nut-sized mass on inner side of internal carotid, probably from carotid in canal. Ptosis has cleared and marked improvement in extraocular movements. Patient is well
VII Hitchens 4/17/38 Figs. 6, 7	53	M (C)	Left internal carotid in cavernous sinus; ruptured causing arteriovenous aneurysm	Those of arteriovenous aneurysm, i.e., exophthalmos, murmur, and huge dilated pulsating veins over forehead and orbit	16 years	Severe frontal	No		Complete III, IV, and VI		Blind left eye	Those of bilateral arteriovenous aneurysm	Necropsy	Congenital	Dead		Patient had a carotid cavernous arteriovenous aneurysm and was operated for this. The carotid artery ruptured when applying a clip. The arterial aneurysm of the carotid within the cavernous sinus was found at necropsy
VIII Bugbey 11/20/41 Fig. 5	52	F	Huge aneurysm from carotid canal bulging into and filling right middle fossa	Double vision (4 years) disappeared and recurred 3½ years later. Pain right side head; pain right cheek bone 4½ mos.; nausea and vomiting; ptosis and third-nerve paralysis. Exophthalmos	4 years 4½ months	Right face and headache	No		Complete III, IV, VI, and partial V	No	B.P. 192/120 (3–4 years). Numbness right face. Wassermann negative	Spinal fluid at onset negative. Vision 20/50 right eye, 20/20 left. Central scotoma right. Sella destroyed. Exophthalmos (42 mm.)	Clinically and by operation	Congenital	Well 13 mos.	(1) Aneurysm exposed. (2) 8 threads left dangling in aneurysm. (3) Partial closure internal carotid. (4) Later carotid ligated intracranially and in neck	Since carotid compression was not tolerated, wound closed (after inserting 8 dangling silk threads 2 cm. long); next day internal carotid partially occluded with fascial band, and one week later ligated in neck and intracranially
IX Prokop 9/20/42 Fig. 38, B	32	M	Huge aneurysm from carotid canal bulging into and filling left middle fossa	Double vision; ptosis 4 years ago lasted 1 week and disappeared; returned again 1 year; tingling forehead 2 mos.	4 years	None	No		Complete VI, partial III; dilated pupil	No	B.P. 120/90	Exophthalmos 3 mm., vision normal, hyperesthesia forehead (N.V.); erosion floor middle fossa (x-ray)	Operation	Congenital	Well 4 mos.	Aneurysm exposed. Internal carotid clipped intracranially and ligated in neck (one operation)	Enormous aneurysm (same size as case above) filling middle fossa with thin layer of temporal lobe over it. Aneurysm bulged over carotid and optic nerves intracranially; retracted to expose and clip carotid. Blood aspirated from pulsating mass
X Shanahan 10/6/42 Fig. 38, A	21	F	Small protrusion (caruncle) projecting along anterior border of N III, from cavernous sinus left	Double vision and pain left eye in recurring spells. Pain in face 2 mos.	2 years	Left eye paroxysmal	No		Slight III and VI	No	Pain in left ear; pain in face	Hypoesthesia 1st and 2d branches of left trigeminus	Operation	Congenital	Well 3 mos.	Small red caruncle protruding from cavernous sinus into cranial chamber above 3d nerve	One month after operation the extraocular palsies had cleared and she was perfectly normal in every way
XI Murphy 11/7/42 Fig. 38, C	39	F	Hazelnut dome-like aneurysm above sella in midline and attached to left internal carotid artery	Frontal headaches 3½ mos., sudden loss of vision both eyes, more left, 2½ mos.; aching pain in eyes	3½ months	Frontal and in both eyes	No	No	No	No	Some polyuria and polydipsia. B.P. 114/72. Wassermann negative	Vision left eye 20/200. Color lost except small segment upper nasal field. Temporal hemianopsia right eye; acuity 20/50; x-ray negative	Operation	Congenital	Well 1 mo.	Exploration for tumor, aneurysm disclosed, blood aspirated, partial closure carotid in neck; one week later totally occluded in neck and clipped intracranially	

TABLE B.—Aneurysms of the Internal Carotid Artery

Number, Name and Date	Age	Sex	Location of Aneurysm	First Symptoms	Duration of Symptoms	Headache	Convulsions	Convulsions Focal or General?	Extraocular Palsies	Cerebral Hemorrhage	Other Outstanding Symptoms	Outstanding Signs	How Diagnosed	Cause of Aneurysm	Result	Was Operation Performed?	Remarks
I Kinyon 10/26/27 Fig. 8	60	M	Fusiform dilatation right internal carotid as it enters cranial chamber	Delirium, blindness, and high fever for 10 days 4 years ago	4 years	No	No		None	No	Visual	B.P. 110/70 Blind right eye, temporal hemianopsia left 4 year duration Sella destroyed	Operation	Congenital? Arteriosclerosis?	Dead	Aneurysm exposed; nothing done	Patient blind after sudden attack 4 years ago. Recovery of nasal field left eye; remained stationary. Aneurysm exposed at operation; nothing done. Died four days later of extradural hemorrhage
II Green 12/28/37	47	F	Right internal carotid	Migrainous headaches, spells of mental confusion	5 years	+ Migrainous		No	None	Few hours before death; ruptured into frontal lobe	Only migrainous headaches (right side) until coma		Necropsy	Congenital	Dead		Patient died at home; brought into hospital for necropsy. Carotid might have been ligated above the diffuse enlargement
III de Oliveira 10/20/40	67	M	Bilateral fusiform dilatation of both internal carotids (entire length) and extreme thickening	Loss of vision	3 months	No	No			No	Only loss of vision; right eye almost blind (acuity 1/200); vision in left reduced to tiny field with suggestion of temporal hemianopsia (acuity 20/70)	Small area of calcification above right anterior clinoid and calcification in right carotid in carotid canal	Operation	Arteriosclerosis	Dead	Right optic nerve divided to free optic tracts	Both carotid arteries like rigid pipestems. Right adherent to optic nerve and elevated optic chiasm. Three days later developed hemiplegia (left) and died two days later
IV Silverstein 6/1/39	58	F	Fusiform dilatation of left internal carotid as it enters cranial chamber	Blindness, left eye; slight pain in left eye	5 months	No	No		None	No	Almost blind left eye; small central area remaining with 20/100 vision; right normal	B.P. 110/70	Operation	Arteriosclerosis	Living 3½ yrs.	Aneurysm exposed, but nothing done	Fusiform aneurysm of entire intracranial carotid to first branch—about twice the size of normal artery and firmly adherent to optic nerve which was almost functionless. On account of age and thick walls of aneurysm it was thought inadvisable to close artery in neck or intracranially. Complete hemiplegia followed operation, also aphasia—cause? Recovery complete in 4 weeks (beginning return in 6 days), but has since had convulsions
V Crouch 4/17/32 Fig. 9	64	F	Bilateral diffuse aneurysms of carotid; also basilar aneurysm	Loss of vision in left eye; almost gone	8 months	None from aneurysm	None from aneurysm		None	No	Loss of vision	B.P. 200/120	Extensive calcification of both carotids	Arteriosclerosis	Dead	Aneurysm exposed but patient had a large dural tumor that was missed	Patient had diffuse symptoms referable to both sides of the cerebral hemisphere and brain stem. Large dural tumor was found at necropsy, also large left basilar aneurysm (S-shaped)
VI Smith 7/8/25	52	M	Bilateral dilatation of both internal carotids	None						No	Patient had huge hypophyseal tumor and homonymous hemianopsia from the tu-	B.P. 120/80. Sella destroyed by tumor	Operation and necropsy	Arteriosclerosis	Dead	For hypophyseal tumor	Aneurysms were found at operation for huge hypophyseal tumor and confirmed at necropsy. Although greatly dilated carotids, they probably did not affect vision

											hemianopsia from the tumor							
VII Healey 3/22/37 Fig. 45	43	M	Sacculated aneurysm of right internal carotid trunk below posterior communicating	Right frontal pain and in eye. Ptosis next day	6 days	Right frontal	No		Right III	No	Diplopia and ptosis	Nystagmus; dizziness	Clinically and at operation	Congenital	Cured. Lived 2½ yrs.	Neck of aneurysm was clipped with silver clip	Ptosis and 3d-nerve palsy completely cleared in 6 weeks. No symptoms for 2½ years, when he died of pneumonia
VIII Russell 5/30/40 Fig. 47	60	M	Sacculated aneurysm left internal carotid below branches	Sharp pains and throbbing headaches left temporal; diplopia 2 weeks. Ptosis 24 hours later and pain left eye	5 weeks	Left temporal and behind eye	No		Left III complete	No		B.P. 180/120. Vision 20/40 left; 20/20 right	Operation and clinically	Congenital	Well 2½ yrs.	Aneurysm clipped 26 days after partially occluding band of fascia to internal carotid in neck	Matas test caused faintness after 5 minutes. Partial occlusion left internal carotid in neck by fascial band May 31, 1940. Clipped neck of aneurysm June 25, 1940
IX Fitch 2/10/41 Fig. 48	29	F	Left internal carotid trunk	Severe sudden pain in head, not localized. Ptosis next day. One month later coma and right hemiplegia and aphasia. Cleared in one month	5 months		No		Partial III	One 4 months ago with hemiplegia and aphasia		Blurring vision left eye; vision 20/20 left; right 20/15; B.P. 115/70. Theotrast carotid injection (Dr. Francis Grant) shows aneurysm back of left internal carotid intracranially	Operation	Congenital	Cured. Died 11 mos. later of anemia	Matas test negative. Neck of aneurysm clipped and cut through. Terrific hemorrhage resulting. Internal carotid thrombosed on each side of aneurysm. Internal carotid tied in neck	Pea-sized aneurysm with well constricted neck. Anterior part of temporal lobe was brown stained and necrotic from old hemorrhages. This extended along Sylvian fissure to outer surface of brain. Descending horn of ventricle open in track of necrotic area. Adhesions around aneurysm
X Clemence 12/19/40 Fig. 49	34	M	Small sacculated aneurysm arising from posterior wall of right internal carotid about ½ cm. intracranially	Headache (3 weeks), diplopia (2 weeks), ptosis right eyelid (1 week)	3 weeks	Right frontal; later occipital	No		Right III complete	Yes	Several chills. Headache developed 2 weeks after onset of chest cold (coughing); and when it had about run its course	B.P. 132/84; daily rise of temperature to 100; white blood count 16,400. Large hemorrhage covering right disc and two others in retina (came on overnight 24 hours ago). Also small hemorrhage left fundus	Operation	Congenital	Dead one mo. after return home	Aneurysm and carotid artery (intracranial) coagulated obliterating both	Blood 2 cm. deep on frontal lobe at time of operation. Small pea-sized aneurysm, bulging backward from internal carotid (posterior wall), reaching third nerve which was not actually seen. When forceps gently passed along neck of aneurysm, it ruptured on mesial surface. Carotid then thrombosed with cautery—also aneurysm. Uneventful immediate recovery without motor loss. Twenty-four hours later facial weakness appeared, and 59 hours after operation left hemiplegia developed suddenly. No loss of consciousness immediately or subsequently. Five days after onset of hemiplegia, function began to return and improved rapidly. One month later he was reported to be walking without assistance. Forty days after operation, while at home, he died a few hours after a cerebral hemorrhage due to rupture of the internal carotid that had been coagulated at operation
XI Strahlin 1/29/41 Fig. 47	47	M	Right internal carotid trunk	Occipital headaches in attacks; in one 4 mos. ago, especially severe, became comatose several hours, then irrational for 1 mo. Third-nerve palsy 2 mos. later	4 years; 4 months	Severe occipital in attacks—more right, about two per week	No		Complete III	One 4 months ago	Diplopia	Bloody spinal taps in period of coma 4 months ago. Vision right eye 20/40	Operation	Congenital	Living 22 mos.	Band fascia on internal carotid; aneurysm trapped between clips and clip on neck of sac, 2/19/41. Internal carotid closed in neck	
XII Valan 7/3/41	27	F	Left internal carotid	Sudden severe right frontal headache. Third nerve palsy. Delirious and drowsy	5 months 3 months	Severe right frontal	No		Complete III right	?			Operation	Congenital	Well 18 mos.	Hazel-nut-sized round aneurysm filling region of 3d nerve. Clips applied above and below neck of aneurysm. Aneurysm shrunk with cautery applied to surface	
XIII Douglas 9/9/41 Fig. 50	31	F (C)	Right internal carotid at entrance to cranial chamber	No history						Cause of death			Necropsy	Congenital	Dead		Died on way into hospital
XIV Lewis 8/20/41	47	F	Left internal carotid at entrance to cranial chamber	Blurring vision left eye, then blindness. Pains left eye and temple	1 year	Not severe left eye and temple	No		Third-nerve palsy	No	Visual loss and headaches only	B.P. 140/80. Haemoglobin 52%. Temporal defect for color right eye. Total blindness left. Visual acuity 20/20 right	Operation	Congenital	Living but hemiplegic and aphasic	Carotid clipped intracranially and tied in neck one hour later	Matas' test; very dizzy after 5 minutes. At operation the internal carotid was clipped. Hemiplegia and aphasia resulted immediately; recovery with permanent loss of these functions
XV Myshrall 6/25/42	42	F	Internal carotid (left) just after entering cranial chamber	Sudden left ptosis, hemiplegia and aphasia 6 years ago; again 7 mos. ago; coma 4 hrs., gradual improvement since, except ptosis which is complete	6 years 7 months (2d)	Severe left frontal and occipital	No	No	Left III and IV	+ 5 days after onset 7 months ago. Another 6 years ago with bloody spinal fluid	B.P. 130/80. Partial motor aphasia hypoesthesia left side of body (functional?)	Complete III and IV; reflexes normal	Operation	Congenital	Well	Neck of aneurysm clipped; aneurysm coagulated. Internal carotid partially occluded before operation	Hazel-nut-sized aneurysm at entrance to cranial chamber. Tightly fused to dura at third nerve. No evidence of earlier hemorrhage. Cause of hemiplegia and aphasia and hypoaesthesia not ascertainable at operation. Patient perfectly recovered after first stroke six years ago
XVI Weitman 12/23/42	39	F	Left internal carotid at posterior communicating	Pains supraorbital region left side; nausea, vomiting, ptosis in attack	2 months	Left frontal and general	No	No	Left III complete	Bloody spinal fluid		Vision 20/10 left. No color vision. Fields far from normal B.P. 124/95	Clinically; angiography; operation	Congenital	Well 2 wks.	Carotid ruptured when clipped, tied in neck and coagulated intracranially. Aneurysm turned back to its neck which was coagulated with cautery	Angiogram by Dr. Irwin Levy of St. Louis: beautiful demonstration of aneurysm running posteriorly on intracranial portion. Posterior communicating artery probably thrombosed with neck of aneurysm
XVII Gardner 5/9/39 Fig. 52	41	F	Right internal carotid at posterior communicating	Mild pain right orbit. Ptosis	2½ months 3 weeks	Right frontal	No		Partial III	Cause of death	Pain gradually more severe. Vision 20/40 right eye	Diminished vision to 20/40	Clinically and at operation	Congenital?	Dead	Aneurysm and carotid artery were thrombosed after it ruptured during dissection. Posterior communicating artery arose below aneurysm	Patient had hemiplegia following operation; died 2 days later. Posterior communicating artery was included in thrombosed aneurysm. This branch arose from carotid nearer base of skull than normally
XVIII Cogwell 5/22/42	32	M (C)	Left internal carotid at posterior communicating	Left occipital headache; closure left eye; coma (20 minutes after coitus)	2 years 3 months	Left occipital and later frontal, worse past 3 mos.	No	No	Left III	+ Bloody spinal fluid	Vision in left eye 20/200 (congenital)	Complete III developed in 4-5 days after period of coma	Operation and Necropsy	Congenital	Dead	Aneurysm exposed; carotid clipped above and below neck of aneurysm; posterior communicating included between clips	Immediate postoperative hemiplegia; death in 2 days. Anterior communicating artery tiny cord, no lumen. Posterior communicating artery entered carotid alongside aneurysm

ABLE B (*continued*).—Aneurysms of the Internal Carotid Artery

TABLE B

Number, Name and Date	*Age*	*Sex*	*Location of Aneurysm*	*First Symptoms*	*Duration of Symptoms*	*Headache*	*Convulsions*	*Convulsions Focal or General?*	*Extra-ocular Palsies*	*Cerebral Hemorrhage*	*Other Outstanding Symptoms*	*Outstanding Signs*	*How Diagnosed*	*Cause of Aneurysm*	*Result*	*Was Operation Performed?*	*Remarks*
XIX McGill 3/13/39 Fig. 15	50	M (C)	Right internal carotid at junction of posterior cerebral arising in position of posterior communicating. Another tiny one in same location on other side	Intermittent right frontal headaches over 6-month period. Ptosis and diplopia after second headache	4 years	± Periodic right frontal, finally bifrontal from arteriovenous aneurysm	No		Partial right III	Cerebral hemorrhage from arteriovenous aneurysm left occipital lobe—not from arterial aneurysm	Stiff neck after each headache, blurred vision right eye. Ptosis has remained since onset, also extraocular palsies. Severe bifrontal headaches past 2 weeks. Period of coma	Partial right homonymous hemianopsia for color only. Vision 10/200 right eye. Bloody spinal fluid	Operation	Congenital	Dead	Aneurysm exposed	Patient died of rupture of a left occipital arteriovenous aneurysm into cerebral substance and ventricle. Arterial aneurysm 12 mm. long and neck 8 mm. wide was filled with thrombus and apparently cured. Small unruptured aneurysm left internal carotid
XX Kraussaul 6/13/40	58	M	Right internal carotid at posterior communicating artery. Large mass back of carotid	Sharp pain back of neck, felt weak and ill, then chilly, then coma	2 days	Sharp in neck then frontal	No		Complete right III 24 hours after onset	± Bloody fluid. Recovery	Irrational. Paralysis left side	Rigid neck; B.P. 120/120. Wassermann negative. Babinski and ankle clonus on left	Clinically and at operation	Congenital? Arteriosclerosis?	Living 2½ yrs. later; condition unchanged	Aneurysm disclosed back of internal carotid filling the region. Carotid clipped intracranially	Patient would not tolerate total occlusion of internal carotid (Matas' test). Partial ligation by fascial band May 16, 1940. Ligation (clip) internal carotid intracranially June 13, 1940. No after effects
XXI Dreschler 4/7/38	34	F	Right internal carotid at junction of posterior cerebral which is in position of posterior communicating artery	Severe occipital headache. Convulsion (1) preceding	5 weeks	Occipital	Three	General opisthotonos preceding convulsion	None	Died of hemorrhage and had had others	Occipital headache and two convulsions. Nausea and vomiting. Died when being prepared for a lumbar puncture which was not done	Many hemorrhages in both eyegrounds but no papilloedema. Bilateral Kernig and Babinski on left. Positive 4+ (blood) Wassermann	Necropsy	Congenital	Dead	No	Patient had syphilis, acute sores dating back few weeks and still present. Aneurysm, however, was congenital—3 mm. in diameter
XXII Parham 7/24/40 Fig. 11 (a)	35	F (C)	Bilateral: right internal carotid at posterior communicating; left internal carotid at anterior choroidal	No history. Died when being brought into hospital						Yes		B.P. 230/150	Necropsy	Congenital	Dead	No	Patient died before reaching hospital—sudden onset of coma. No further history. Two aneurysms, one on each internal carotid: (1) right at posterior communicating ruptured; (2) left at anterior choroidal branch; both pea-sized

												not done						
XXII Parham 7/24/40 Fig. 11 (a)	35	F (C)	Bilateral: right internal carotid at posterior communicating; left internal carotid at anterior choroidal	No history. Died when being brought into hospital							Yes		B.P. 230/150	Necropsy	Congenital	Dead	No	Patient died before reaching hospital—sudden onset of coma. No further history. Two aneurysms, one on each internal carotid: (1) right at posterior communicating ruptured; (2) left at anterior choroidal branch; both pea-sized.
XXIII Height 4/4/34	32	M (C)	Right internal carotid at junction with posterior communicating. Another on anterior cerebral	Convulsions. Pains in head (terrific). Coma	24 hours	Terrific	Yes	General	None	Cause of death	No cervical rigidity. Deep coma. Wassermann negative	One round hemorrhage right disc. B.P. 104/70; bloody spinal fluid	Necropsy	Congenital	Dead	No	Side of localization was subhyaloid hemorrhage in disc
XXIV Kratz 6/11/38	68	F	(1) Small pouch on internal carotid below branches. (2) Middle cerebral. (3) Junction basilar and posterior communicating artery	None							No						No	Three small unruptured aneurysms discovered at necropsy. No symptoms from any
XXV Barker 10/5/37 Fig. 12	46	M	Left internal carotid at junction of posterior cerebral arising in position of posterior communicating	Severe pain in occiput for 5 days, then recurred a week later	20 days	Severe in attacks	One	General	None	Cause of death	Comatose 7 days after severe occipital pain (second time), then worked 3 days; then general convulsions and death in 6 hours. Said to have had hypertension	Large round hemorrhages in left eye and papilloedema *left*	Necropsy	Congenital	Dead	No	Saccular aneurysm 14 mm. long, ruptured into temporal lobe and into descending horn of lateral ventricle. Ventricles filled with blood, also subarachnoid space. Hemorrhage into sheath of left optic nerve.
XXVI Jones 1/31/40 Fig. 14	37	M (C)	Fusiform dilatation of anomalous vessel in position of anterior choroidal beyond junction with internal carotid right	Blindness right eye. Pain right eye and head. Coma	3 months 2 months 8 days	Severe right frontal	No		Partial right III	Cause of death			Necropsy	Congenital	Dead	No	Ruptured fusiform aneurysm with thick wall filled with thrombus. Another aneurysm on right anterior cerebral artery. His symptoms of three months ago due to retinal thrombosis, and not associated with rupture of aneurysms (8 days ago)
XXVII Bender 6/5/39 Fig. 11 (b)	51	F	Right internal carotid at posterior communicating	Pains in and watering of right eye. Drooping eye (2 days)	2 weeks	+ Severe, and nausea	No		Right III	Cause of death	Slightly dizzy blurring vision	Ptosis right, and limitation of N III two days. Wassermann negative. Diplopia	Clinically and necropsy	Congenital	Dead		After partial ligation of internal carotid with fascial band. Ruptured aneurysm with intraventricular and subarachnoid hemorrhage
XXVIII Ingram 6/24/37 Fig. 11 (c)	44	F	Right internal carotid at posterior communicating	Pain right side of head, nausea, and vomiting	6 days	Right sided severe	No		None	Cause of death	Photophobia, stiffness of neck, projectile vomiting	Hypertension 212/112 for at least 4 years. Bilateral papilloedema with hemorrhages	Necropsy	Congenital	Dead	Decompression because of papilloedema	6-mm. aneurysm from trunk of internal carotid before it branches; ruptured. At operation brain was very tight. No hemorrhage seen at operation
XXIX Blackwell 3/5/41	36	F (C)	(1) Anterior choroidal artery in right lateral ventricle near foramen of Monro (ruptured). (2) Junction of right internal carotid and posterior communicating (small nubbin, intact). (3) Anterior communicating	Headaches, dizziness, blurred vision (had hypertension 260)	10 days (previous hemorrhage from anterior communicating 5 months ago)	Severe	No		None	Cause of death (2nd hemorrhage)	Bloody spinal fluid on previous admission 5 months ago	B.P. 260/140 (6 years at least). Bilateral Kernig, bilateral papilloedema, and hemorrhage about both discs	Necropsy	Congenital	Dead	No	Earlier hemorrhages (5 months ago) from anterior communicating aneurysm. Ventricles now filled with blood. Aneurysm on choroid plexus near foramen of Monro
XXX Small 3/25/41	62	F	(1) Left internal carotid from posterior communicating and middle cerebral, 2.5 cm. (2) Anterior communicating, 4 mm. Neither ruptured	Died of carcinoma of gall bladder. None referable to aneurysm							No	No symptoms. Necropsy specimen. Died of carcinoma of gall bladder	B.P. 170/100. Two small hemorrhages in left eyeground	Necropsy	Congenital	Dead	No	2.5-cm. aneurysm on internal carotid extending from terior communicating to middle cerebral artery; a aneurysm on anterior communicating; neither ane ruptured
XXXI Lancaster 6/12/28	33	M	Left internal carotid near first branch; another smaller on other side in same position	Pain over left eye; closure left lid	6 weeks 3 weeks	Left eye and forehead	No		Complete III	Cause of death		B.P. 118/80. Eyegrounds negative; 10 days later rigidity of neck and Kernig	Necropsy	Congenital	Dead	No	Bilateral symmetrical aneurysms of internal carotid; b ruptured. There was a neck to the sac
XXXII Christian 7/29/38	44	M (C)	Small aneurysm at junction with posterior communicating artery (right), but on carotid	None		No	No		None	No	None	None	Necropsy	Congenital	Dead of another cause	No	Aneurysm found at necropsy; no symptoms; cardi death; hypertension
XXXIII Paudera 9/2/39 Fig. 13	38	M	Right internal carotid at junction of anterior communicating	Intermittent pain right temple	1 months	Severe four weeks. Right side throbbing, worse at night	Yes	General	Right III	Cause of death	Blurring vision (right), diplopia, ptosis right	Ptosis and Right III complete, 4 days	Clinically and by necropsy	Congenital	Dead	No	Patient died soon after entering hospital
XXXIV Johnson 2/20/40 Fig. 11 (d)	34	F (C)	Right internal carotid at junction of anterior choroidal	Headache	6 days	Severe 4 days	No		None	Cause of death		Bilateral Babinski, arms rigid, opisthotonus, coma, absence of knee-kick	Clinically and by necropsy	Congenital	Dead	No	Patient entered hospital in coma; died next day. Patie had pulmonary tuberculosis
XXXV Penick 7/3/35	43	F (C)	Right internal carotid 3 mm. above posterior communicating	Sudden coma	Day of admission	None before	Numerous when in coma	General	Right III	Cause of death		Arms spastic and flexed. Bloody spinal fluid. Pressure 245	Clinically and by necropsy	Congenital	Dead		There were several small unruptured aneurysms alo circle of Willis. Patient entered hospital in coma and di next day
XXXVI McLean 11/1/41	52	M (C)	On *both* carotids at posterior communicating; another anterior communicating	None		No	No	No	No	No	Blood pressure over 200 for past 7 yrs.		Necropsy	Congenital	Dead	No	Two small berry aneurysms, one on each carotid at po terior communicating, causing no symptoms. Another anterior communicating ruptured into frontal lobe a subarachnoid space and caused death
XXXVII Chase 6/9/42	42	M (C)	Internal carotid (left) between posterior communicating and middle cerebral	Coma	6 hours		No	No	No	Cause of death	Hemiplegia total (right). B.P. 250/120	Bloody spinal fluid	Necropsy	Congenital	Dead	None	Aneurysm had relatively long neck that could have be clipped. Aneurysm ruptured at fundus
XXXVIII Brown 10/20/37	19	F (C)	Left internal carotid at origin of middle cerebral	Hemiplegia Coma	1 month 2 days	No	No		None	Cause of death	Said to have had a transient right hemiplegia without aphasia one month ago	Positive Wassermann, endocarditis, rheumatism	Clinically and by necropsy	Congenital	Dead	No	Aneurysm 5 mm. in diameter; ruptured. Middle cerebr artery thrombosed
XXXIX Fisher 11/23/42 Fig. 51	51	F	Left internal carotid at posterior communicating	Loss of consciousness lasting 2 weeks. Two additional spells since each lasting a week. Headaches and vomiting	2½ months	Generalized frontal and occipital, not unilateral	One spell of contracture of both arms	No	None	3 attacks	Has had spells of severe headaches for 10 years. Dizziness on bending. Loss of memory and mental confusion for one month	Right homonymous upper quadrantal defect in visual fields. Visual acuity normal. B.P. 134/76. Bloody spinal fluid	Clinically, angiography, operation	Congenital	Well 6 wks.	Aneurysm against left optic nerve and chiasm. Carotid tore when clip applied. Coagulated. Aneurysm turned back and neck coagulated with posterior communicating artery. Carotid tied in neck	Angiogram by Dr. W. Gayle Crutchfield of University Virginia showed aneurysm on carotid and well below a terior cerebral artery. After thrombosis of the posteri communicating artery and the carotid, the anterior cer bral artery carries the entire blood supply to the side cerebral

TABLE C.—Aneurysms of the Anterior Cerebral and Anterior Communicating Arteries

Number, Name and Date	*Age*	*Sex*	*Location of Aneurysm*	*First Symptoms*	*Duration of Symptoms*	*Headache*	*Convulsions*	*Convulsions Focal or General?*	*Extra-ocular Palsies*	*Cerebral Hemorrhage*	*Other Outstanding Symptoms*	*Outstanding Signs*	*How Diagnosed*	*Cause of Aneurysm*	*Result*	*Was Operation Performed?*	*Remarks*
I Crisp 7/2/03	81	M	Left anterior cerebral	Sudden coma	1 day	Frontal	No	None	No	Cause of death	Cardiovascular		Necropsy	Congenital	Dead	No	Patient was under treatment for cardiovascular disturbances when hemorrhage occurred
II Thomas 7/19/27	28	M (C)	Right anterior cerebral just beyond internal carotid (ruptured). Three aneurysms left middle cerebral (intact)	Headaches (found in coma)	Several days	General	No		No	Cause of death	Found in coma. Well the night before. Lived only few hours	B.P. 220/140. Bilateral Babinski, bloody spinal fluid, rigid tortuous peripheral arteries. Eyegrounds negative	Necropsy	Congenital	Dead	No	Ruptured aneurysm, 1 cm. in diameter, on right anterior cerebral—broad base on vessel; given off just beyond carotid. Three other aneurysms on left middle cerebral, one at origin, other two lying side by side in Sylvian fissure. Ruptured in brain and ventricle
III Baker 4/22/30 Fig. 19	52	M	Anterior communicating	Fainting spell, headache and vomiting one week later, and then confusion since, and drowsiness	2 months	Severe, generalized		Left-sided convulsion one week before death	Right 3d nerve, 11 days before death	Cause of death	B.P. 150/90; has been 180 past week	Bloody spinal fluid	Necropsy	Congenital	Dead	Operation for suspected tumor. Aneurysm not found	Aneurysm, 3.5 x 0.5 cm., between two anterior cerebral arteries where anterior communicating artery should be; ruptured posteriorly
IV Butler 3/14/31	25	M	Right anterior cerebral	Severe headache and stiff neck	2 weeks	Severe, right temple	Not known			Probably one a week before fatal one	Heavy lifting at time of onset. Patient had 2d hemorrhage in hospital	Left Babinski +, then right +, Kernig + + bloody spinal fluid	Necropsy	Congenital	Dead	No	Aneurysm, 4 mm., ruptured into ventricle (anterior horn)
V Davis 2/2/32	45	F	Left anterior cerebral	severe pain in head and lumbar region, blurring vision right eye, irrational	8 days	+ Severe, occipital, and backache	No	None	None to account for double vision	Yes	Diplopia, vomiting	Stiffness neck and spine, papilloedema bilateral, bloody spinal fluid, B.P. 150/80 to 180/100; Wassermann +	Necropsy	Congenital (possibly syphilitic)	Dead	No	Aneurysm on left—visual disturbance on right. Hemorrhage burst through brain into left anterior horn. Aneurysmal sac 3-4 mm.
VI Foster 8/7/32 Fig. 16 (a)	33	F	Anterior communicating	Sudden cry for water, rigidity and coma for 20 minutes. Vomiting, dull and apathetic for several days. Headaches	68 days	+ Severe, *base of brain*	No, but rigidity		No	Yes	Blurring vision; 5 weeks after first attack had another like first. Loss of power in right arm and leg and slowness of speech. Arm improved but not leg. Bedfast	Bilateral papilloedema. Weakness right leg and arm. Babinski on left, not right; absent left knee-kicks, rigidity of neck	Necropsy. Lesion localized by ventriculography	Congenital	Dead	Pineal approach for suspected tumor in third ventricle. Haematoma evacuated	Supposed tumor located by ventriculography in 3d ventricle. Pineal approach; bulging mass protruding into third ventricle; opened and haematoma as large as walnut evacuated. No tumor or aneurysm found. Died next day. Necropsy showed aneurysm, 1 cm., of anterior communicating artery, and involving both anterior cerebral arteries; thinned wall at point of rupture
VII Rawlings 11/22/31	39	F	Anterior communicating	Headaches (1 year); something terrible happened; excited, drowsy; urinary retention; bradycardia (3 days, sudden)	3 days 1 year	; General one year, more at night	No		No	Thirteen days later died in 3 hours	B.P. 136/80. Comatose on admission, regained consciousness and bladder control. Died 3 hours after sudden coma 10 days later	Pulse 40–50	Necropsy	Congenital	Dead	Cerebellar exploration for suspected tumor	Patient had cerebellar exploration (negative). Later (3 days) air injection (negative). Ten days later hemorrhage and death (3 hours). Aneurysm anterior communicating artery 1 x 0.4 cm., ruptured. Second, unruptured aneurysm anterior tip of basilar (3 mm.), patent and not thrombosed

VIII Page 6/27/33	30	F (C)	Right anterior cerebral ruptured	[illegible] scious while ironing			known		[illegible]	hemorrhage	Brought into hospital in deep coma and died 1½ hours later	coma)					
IX Kaintuck 3/19/35	42	M (C)	Left anterior cerebral and anterior communicating arteries—small, ruptured	Severe pain right side of head and neck. Rigid neck	3 weeks and few hours	Severe right (aneurysm left)	Probably one	General?	No	Apparently one 3 weeks ago and present fatal one	Attack following coitus	Hypertension 12 years; now 225/125. Wassermann negative	Clinically and necropsy	Congenital	Dead	No	Sudden fatal hemorrhage
X Porter 9/1/38 Fig. 16 (b)	36	F (C)	Left anterior cerebral	Headaches—frontal, occipital and vertex, steadily increasing	9 days	Severe; most occipital	No, but cramps in right leg	Focal attacks (cramps)	No	Cause of death	Drowsiness, dizziness, vomiting (once) on admission, stiff neck, general cramps right leg	No hemorrhages in fundi. B.P. 185/110 (later 110/60); bloody spinal fluid; cervical rigidity (slight). Spinal pressure 330 mm.	Necropsy	Congenital	Dead	No	Three weeks after admission patient had another hemorrhage (conscious at time). B.P. rose to 280/140; sudden severe headache; spastic extremities; rapid tremor, right hand; coma. Bilateral Hoffmann and ankle clonus. Hemorrhages both fundi noted 10 minutes after previous negative examination of eyegrounds. Died 41 hours
XI Hemer 8/25/37	68	M	Junction right anterior cerebral and communicating arteries	Sudden coma	6 days	No history	No	None		Yes	Fainted week before; since then headache and abdominal discomfort, but kept working	Left hemiplegia Wassermann ±. B.P. 145/105 to 190/130. Bloody spinal fluid	Necropsy	Congenital	Dead	No	Aneurysm, 2 mm., between anterior cerebral and communicating vessels on right, ruptured
XII Grimm 1/11/39 Fig. 17	40	M	Junction left anterior cerebral and anterior communicating	Pain in eyes, Photophobia, and loss of vision; 3 mos.	5 months, but loss of vision 1 year	Frontal and in eyes	One lasting 20 minutes	General	Diplopia 5 weeks	Probably not until fatal hemorrhage	Hypertension 182/104 for 19 years, visual loss left eye more, acuity 10/200 left; 30/40 right	Bitemporal scotomata (large). Sella destroyed	Operation; aneurysm large; cauterized by passing needle into it; hoped to thrombose	Congenital	Dead	Aneurysm exposed and coagulated on exterior and with needle penetrating interior	Patient had heart trouble (?) for years. (Shortness of breath.) Coarctation of aorta diagnosed on ward. Because of this Dr. Ford made a diagnosis of intracranial aneurysm. Large mass, about 2.5 x 3 cm., filled hypophyseal region—blood aspirated. Cautery to vessel in aneurysm. Ruptured 2 months later
XIII Hutton 6/8/40	26	M	Anterior communicating left	None	None	No	± but not from aneurysm		No	No	Epilepsy from another congenital cause; x-ray shadow	B.P. 170/70	X-ray and operation	Congenital	Living	Aneurysm disclosed at operation but nothing done	Patient had epilepsy, and x-rays showed calcified shadow (small, round) just above sella and to left of midline. Diagnosed as an aneurysm but not causing his epilepsy. Hemisphere explored and aneurysm exposed at same time. A small club-like aneurysm with white tip (calcified) dangled between carotid artery and optic nerve—½ cm. long. No indication for ligating it; not disturbed
XIV Ogle 7/25/32 Fig. 18	53	M	Anterior communicating at junction with anterior cerebral	Cerebral hemorrhage with bilateral VI and left facial, followed sexual intercourse	8½ years	+ Generalized and back of neck	No	None	Bilateral VI	Cause of first symptoms; probably several since	Seven subsequent spells of headache, coma, vomiting. Stiffness both legs	B.P. 110/70-140/90; said to have been 170 before attacks; bilateral Babinski; right facial weakness; loss of memory; comatose	Operation. Ventriculography (third ventricle absent; large filling defect). Necropsy	Congenital	Dead	Aneurysm excised	Ventriculography showed large filling defect in region of third ventricle which was obliterated. Smooth walled mass, 4 x 4 cm., exposed under foramen of Monro after right frontal resection. Solid mass of fibrin sliced away, piece by piece; finally reached pulsating area which finally ruptured. Aneurysm partially excised; bleeding entirely controlled by silver clips. Died following day; patient had not responded since operation despite absence of pressure and bleeding. At necropsy both anterior cerebrals, which were included in the aneurysm, had been sacrificed
XV Stone 8/10/41	70	F	Anterior communicating at junction with left anterior cerebral	None	None	No	No	None	No	No	Accidental finding at necropsy	B.P. 170/100	Necropsy	Congenital	Dead	No	Patient had three other aneurysms, one at anterior end of basilar, and two symmetrical ones on middle cerebral arteries at first branch
XVI Horns 11/8/16	33	M	Right anterior cerebral at anterior communicating	Something popped in head; then coma one day; disoriented and irrational	2 years	General and back of neck	No	None	No	Cause of death (2 years)	Two years ago left hemiplegia; cleared in 3 months; hand weak since	B.P. 130/75; three days after onset complete left hemiplegia. Died 11 days after onset	Necropsy	Congenital	Dead	No	Hemorrhage into right Sylvian fissure and under frontal lobe. Aneurysm right anterior cerebral at anterior communicating, about 1 cm. in diameter
XVII Woods 10/30/40	46	F	Anterior communicating	Severe headache and coma		General	No	None	No	Cause of death		B.P. 158/104. Bloody spinal fluid. Stiff neck	Necropsy	Congenital	Dead	No	Pea-sized ruptured aneurysm on anterior communicating artery
XVIII Blackwell 3/5/41	36	F (C)	Anterior communicating at junction of anterior cerebral	Headache, stiff neck, dizziness, blurred vision	Not from this aneurysm (2 others)					Cause of death	Another attack 5 months earlier; bloody spinal fluid, headache from hypertension	B.P. 260/140 (6 years at least); positive Kernig. Bilateral papilloedema and hemorrhages; spinal pressure 520	Necropsy	Congenital	Dead	No	Patient died of ruptured aneurysm in choroid plexus. Had had another hemorrhage 5 months earlier, probably from this aneurysm, as region about it was covered with orange pigment. Aneurysm not filled with thrombus. Measured 8 mm. in diameter
XIX McLean 11/1/41	52	M (C)	(1) Anterior communicating at junction of anterior cerebral (ruptured). (2) Left and right internal carotid; neither ruptured		3 days	No	No	None	No	Cause of death	Hypertension of over 200 for 7 yrs.	Bloody spinal fluid pressure 480 mm.	Necropsy	Congenital	Dead	No	(1) 4-mm. aneurysm anterior communicating (ruptured). (2) 21/2-cm. aneurysm on left and right internal carotid at junction with posterior communicating arteries
XX Hyzer 8/9/42	58	F	Right anterior cerebral	Severe pain right eye and forehead	4 attacks in 12 years	At time of attack right forehead and eye	No	None		Cause of death; 3 previous hemorrhages	History of hypertension	Bloody spinal fluid on 4 occasions	Necropsy	Congenital	Dead	No	Aneurysm 4.5 x 3 x 2.5 cm. on right anterior cerebral artery, capsule calcified. Ruptured into right frontal lobe and subarachnoid space. Aneurysm almost filled with clot, only narrow space remaining; ruptured into this space. (Kindness of Dr. Wm. Vandergrift, who performed the autopsy.)
XXI Lynch 5/15/42	32	F	(1) Right anterior cerebral, unruptured. (2) Posterior inferior cerebellar, ruptured (right)	Severe headaches; coma. Earlier attacks 11 years before	11 years 10 days	Slight in past few months	No	None	No	Hemorrhage 11 years ago from one of the aneurysms	None	Bloody spinal fluid	Necropsy	Congenital	Dead	No	Patient had been perfectly well 11 years after first hemorrhage (bloody spinal fluid) after strenuous day, headache and coma. Two aneurysms; it was the basilar aneurysm that ruptured
XXII Hitchens 4/19/38 Figs. 6, 7	53	M (C)	(1) Small right anterior communicating. (2) Left internal carotid in sinus. (3) Post. cerebral left at post. communicating. Only aneurysm in carotid sinus had ruptured	None	None	None	No	None	No	No			Necropsy	Congenital	Dead	No	Aneurysm disclosed at necropsy after operation for carotid cavernous arteriovenous aneurysm (due to rupture of arterial aneurysm)
XXIII Small 3/25/41	62	F	(1) Left anterior cerebral at anterior communicating. (2) Left internal carotid. Both unruptured														
XXIV Greenberg 9/13/41 Fig. 54	49	M	Anterior communicating or at anterior cerebral; lies in and above sella	Headaches	2 years	Bifrontal, bitemporal, posterior left frontal	No, but spells of weakness of right side	Focal	No	No	Motor aphasia; memory poor	Partial hemiplegia; partial motor aphasia; right bitemporal hemianopsia for colors; quadrantal defect for form. Sella entirely destroyed. Vision 20/20; B.P. 125/80. Wassermann negative	Operation	Congenital	Living	Aneurysm exposed, opened muscle inserted, and coagulated	Huge mass in front of optic nerves, opened, blood spurted. Large piece of muscle inserted and coagulated. Partial ligation internal carotid next day. Total ligation seven days later. Condition unchanged on discharge three weeks after first operation. Midline position of aneurysm reason for assuming origin from anterior cerebrals or anterior communicating arteries
XXV Byrn 12/3/41 Fig. 53	45	F	Right anterior cerebral artery	Dimming vision	2 months	None	No	None	No	No	None	Vision 15/200 right; 70/70 left. Loss of color right eye; almost total left central scotomata; altitudinal hemianopsia right. B.P. normal	Operation	Congenital	Well	Aneurysm excised. Internal carotid artery clipped intracranially	Aneurysm shape of hour-glass, size of pigeon's egg. Largely filled with thrombus. Aneurysm ruptured during dissection; internal carotid clipped, bleeding stopped at once. It was necessary to bisect the right optic nerve in the longitudinal plane to free the mass. The vision in this eye improved in 6 months from 15/200 before operation to 20/70, and the fields for both form and color were normal except for a lower temporal quadrantal defect. The vision in the left eye was then 20/15 (20/70 before operation)

TABLE D.—Aneurysms of the Middle Cerebral Artery

TABLE D

Number, Name and Date	Age	Sex	Location of Aneurysm	First Symptoms	Duration of Symptoms	Headache	Convulsions	Convulsions Focal or General?	Extra-ocular Palsies	Cerebral Hemorrhage	Other Outstanding Symptoms	Outstanding Signs	How Diagnosed	Cause of Aneurysm	Result	Was Operation Performed?	Remarks
I Loukota 11/19/32	29	F	Right middle cerebral in brain	Hemiplegia, coma	2 days	None noted	No	No	Right VI	Cause of death	Hemiplegia (left)	Coma Hemiplegia (left)	Necropsy	Mycotic	Dead	No	Patient had (viridans) endocarditis for 7 months. Aneurysm in brain (mycotic)
II Alford 9/6/34	14	F	Right middle cerebral in brain	Headaches and coma	Few hours		No	No	No	Cause of death	Sudden coma. Patient had been treated for years for endocarditis	Signs of pressure	Necropsy	Mycotic	Dead	No	
III Potter 6/1/34	13	F	Right middle cerebral artery in Sylvian fissure and in occipital lobe (probably posterior cerebral artery)	Headaches, convulsions, opisthotonus	24 hours	+ Frontal	+ Left arm, also tonic	Left arm	No	Cause of death		Fever, bacterial endocarditis	Necropsy	Mycotic	Dead	No	Two small aneurysms were found—one Sylvian fissure, other occipital, both with necrosis brain, hemorrhage and purulent exudate about them
IV Boland 5/4/37 Fig. 20 (a)	27	M	Right middle cerebral between anterior cerebral and first branch	Headache, hemiplegia (left), convulsion, stupor	8 days 8 days 5 days	Right frontal	Yes	Left side	No	No	Stiffness of neck	B.P. 140/44; temperature 102; W.B.C. 13,300	Necropsy	Mycotic (?)	Dead	Ventriculography and exploration right hemisphere	Air injection showed filling only left lateral and third ventricle, which were pushed slightly to left. Cerebral exploration showed necrotic brain everywhere. Aneurysm with small hemorrhage at site of thrombus middle cerebral. Patient had a known endocarditis with temp. for past 8 months. Died 4 days after operation
V Herald 7/23/37	18	F	Right middle cerebral 2.5 cm. from origin	Motor aphasia cleared in 2 weeks; 2 mos. later cerebral hemorrhage with hemiplegia	2 months	Frontal several days	No	None	No	Cause of death	Endocarditis for 1 year; alpha streptococcus	Hemiplegia, coma	Necropsy	Probably mycotic	Dead	None	Patent sac 1 cm. long on middle cerebral artery 2.5 cm. from its origin. Hemorrhage into temporal lobe
VI Lewis 8/8/35	45	M (C)	Left middle cerebral artery at entrance into brain	Coma, headache	3 days	Yes	No		No	Cause of death		B.P. 200/115; bilateral papilloedema	Necropsy	Arteriosclerotis	Dead	No	Patient had arteriosclerosis and hypertension. Small bulge in arterial wall filled with thrombus. Ruptured
XXII 12/2/39	43	M	Right middle cerebral with anterior cerebral arteries	Sudden coma	Few hours		No	None	No	Cause of	Coma and	Coma and hemiplegia	Necropsy	Arterio-	Dead	No	Sudden coma in woman previously quite well. Hemorrhage of hypophysis and cortical adenomata of adrenal
XXII Bradbury 4/19/41	53	F	Right middle cerebral at first branches	Operated for carcinoma lung. Never regained consciousness	No	No	No	No	No	"Cerebral accident" 3 years ago; recovery	Transient dimness of vision	B.P. 235 for several years	Necropsy	Congenital	Dead	No	

TABLE E.—Aneurysms of the Posterior Communicating Artery. No cases in this series.]

TABLE E

TABLE F.—Aneurysms of the Posterior Cerebral Artery

TABLE F

Name and Date	Age	Sex	Location of aneurysm	First Symptoms	Duration of Symptoms	Headache	Convulsions	Convulsions Focal or General?	Extra-ocular Palsies	Cerebral Hemorrhage	Other Outstanding Symptoms	Outstanding Signs	How Diagnosed	Cause of Aneurysm	Result	Was Operation Performed?	Remarks
Potter 6/1/34	13	F	Right posterior cerebral; another on right middle cerebral	Headaches, convulsions, opisthotonus	24 hours	Frontal	Left arm, also tonic attacks	Focal	No	Cause of death		Fever, endocarditis	Necropsy	Mycotic	Dead		Necrosis in occipital lobe. Purulent hemorrhagic exudate
Hitchens 4/19/38	54	M (C)	(1) Left post. cerebral at post. communicating. (2) Left internal carotid in sinus. (3) Anterior communicating artery	None		No	No	No	No	No	None	None	Necropsy	Congenital	Dead from other cause	No	

TABLE G.—Aneurysms of the Vertebral and Basilar Arteries

TABLE G

Number, Name and Date	Age	Sex	Location of Aneurysm	First Symptoms	Duration of Symptoms	Headache	Convulsions	Convulsions Focal or General?	Extra-ocular Palsies	Cerebral Hemorrhage	Other Outstanding Symptoms	Outstanding Signs	How Diagnosed	Cause of Aneurysm	Result	Was Operation Performed?	Remarks
I Rudy 4/3/37 Fig. 25	18	F	Left vertebral artery	Left occipital pain; neck slightly stiff	8 months	Pain	No		Bilateral VI partial; slight ptosis left lid (III N)	No	Vomiting, nervousness, numbness and tingling of fingers and feet, occ-	Bilateral VI, nystagmus, Romberg to right, ptosis right; unsteady gait. No papilloedema	Ventriculography, operation, necropsy. Hydrocephalus trigeminal neuralgia	Congenital	Dead later	Aneurysm removed; thought to be a tumor at the time. Weight 26.9 gms.	Patient died soon after operation. Cerebellar pressure was so great when dura opened that there was little chance of success. Aneurysm shelled out with finger and only slight bleeding. Aneurysm arose from left vertebral artery and presented at foramen of Magendie
				mandible and teeth					plopia (2)								eration)
11/7/33 Fig. 35																	

TABLE G.—Aneurysms of the Vertebral and Basilar Arteries

Number, Name and Date	Age	Sex	Location of Aneurysm	First Symptoms	Duration of Symptoms	Headache	Convulsions	Convulsions Focal or General?	Extra-ocular Palsies	Cerebral Hemorrhage	Other Outstanding Symptoms	Outstanding Signs	How Diagnosed	Cause of Aneurysm	Result	Was Operation Performed?	Remarks
I Rudy 4/3/37 Fig. 25	18	F	Left vertebral artery	Left occipital pain; neck slightly stiff	8 months	Pain	No		Bilateral VI partial; slight ptosis left lid (III N)	No	Vomiting, nervousness, numbness and tingling of fingers and feet (occasional); unsteady gait; diplopia; dizziness on change of position	Bilateral VI, nystagmus, Romberg to right, ptosis right; unsteady gait. No papilloedema	Ventriculography, operation, necropsy. Hydrocephalus 100 cc air	Congenital	Dead	Aneurysm removed; thought to be a tumor at the time. Weight 26.9 gms.	Patient died soon after operation. Cerebellar pressure was so great when dura opened that there was little chance of success. Aneurysm shelled out with finger and only slight bleeding. Aneurysm arose from left vertebral artery and presented at foramen of Magendie
II Stockdale 10/4/15	27	M	Anterior part basilar artery. Localized pouch	Headaches (1 year), dysphagia, dysarthria (4 mos.), staggering, diplopia	1 year	+ Occipital	No		No	No	Weakness both legs and rigidity	Bilateral Babinski; bilateral papilloedema	Necropsy	Congenital	Dead	Cerebellar exploration negative findings, hydrocephalus	Sharply localized pouch involving entire circumference of basilar artery at pons and midbrain; size of hickory nut, deeply imbedded in brain stem, extends to sella turcica
III Race 9/3/21	53	M	Right side of basilar artery	Headache and unconscious spell	2 weeks	Severe suboccipital	No, but unconscious and delirium following	No	No	Cause of death	B.P. 120/80	Stiff neck, coma, bradycardia. Bloody spinal fluid	Necropsy	(?)	Dead	No	Slit-like rupture of right wall of basilar artery which was not much sclerosed. A probe could be passed into a short narrow pouch (false sac)
IV Johnson 9/23/31	45	F (C)	Junction of basilar and posterior cerebral arteries. Sac 2 cm. in diameter	Sudden blindness for 10 min. (10 days); convulsion week later; coma followed	10 days	+ Severe	Yes	Not known	Left VI Left III	Cause of death	Stiffness of neck	B.P. 160/110 to 120/80. Bloody spinal fluid	Necropsy	Congenital (Wassermann negative)	Dead	No	Aneurysmal sac, 3 cm., at junction of basilar and two posterior cerebral arteries ruptured. The sac had compressed the 3d and 6th nerves
V Sutton 9/21/34	38	F (C)	Junction of basilar and posterior cerebral arteries	Headache; blind right eye (few days), convulsions and coma (1 day)	2 weeks	+ Frontal	Terminal	General (?)	No	Cause of death		Positive Babinski right. Bloody spinal fluid; 430 m.m. pressure; B.P. 226/154; stiff neck	Necropsy	Congenital (Wassermann negative)	Dead	No	3-mm. sac (ruptured) at anterior tip of basilar artery. Patient was comatose when she entered hospital; lived 12 hours.
VI Mackall 8/26/36	50	F	Sac 5 mm. on inferior medial wall right vertebral artery opposite origin of posterior inferior cerebellar artery	Sudden violent headache occipital; coma in ½ hour	4 days	Violent occipital	No	No	No	Cause of death; 2 hemorrhages (?) 3 days apart	Regained consciousness following day. Died suddenly 3 days later. Neck rigid	B.P. 110/70. Eyegrounds negative	Necropsy	Arteriosclerosis	Dead	No	Small broad-mouthed sac right vertebral artery, ruptured
VII Lynch 5/10/42	52	F	(1) Right post. inferior cerebellar (ruptured) (2) Ant. communicating at right anterior cerebral (unruptured)														
VIII Stone 2/31/41	70	F	Anterior tip of basilar	None		No	No	No	No	No	Accidental finding at necropsy	B.P. 170/100	Necropsy	Congenital	Dead	No	Patient had symmetrical (2) aneurysms on middle cerebral arteries, and a third on the anterior communicating artery in addition to one on the basilar
IX Becraft 1/19/32	67	M	Basilar artery at left 5th nerve	Trigeminal neuralgia left corner mouth and tongue	3 years	No	No	No	No	No	No cerebellar signs; no 5th nerve loss	B.P. 240/120; head enlarged. Marked loss hearing left side	Operation	Arteriosclerosis	Living	Trigeminal root sectioned	Large bulging basilar aneurysm elevating auditory and trigeminal nerves
X Hammond 6/22/36	68	F	Anterior part basilar artery, left	Pain in lower jaw, forehead, and eye	17 years	No	No		No	No	No deafness	B.P. 170/105	Operation	Arteriosclerosis	Living 4½ yrs. later	Trigeminal root sectioned	Bulging basilar artery compressing 5th nerve
XI Bedenbaugh 10/2/40	55	F	Anterior part of basilar artery, left	Trigeminal neuralgia left chin and upper jaw	11½ years	No	No		No	No		B.P. 144/88	Operation	Arteriosclerosis	Living 2 mos.	Trigeminal root sectioned	Bulge of anterior part of basilar artery at 5th nerve. was hoped that the ligation of one vertebral artery would take the strain off the aneurysm
XII Worthington 6/26/33	77	M	Basilar artery at left 5th nerve	Trigeminal neuralgia left mandible, cheek and tongue	2 months	No	No		No	No	Mild dyspnoea	B.P. 190/130; deafness both sides, more left. Cardiac enlargement	At operation for trigeminal neuralgia	Arteriosclerosis	Died 3 mos. later	Trigeminal root sectioned	The lateral bulge of the basilar artery had compressed the 5th nerve. Cause of death not known (3 months after operation)
XIII Lewellyn 11/22/27 Fig. 26	63	F	Basilar bulges to right, left vertebral artery greatly enlarged	Dizzy attacks, nausea and vomiting, bilateral tinnitus	5 weeks	No	No		No	No	Bilateral deafness severe grade, bilateral tinnitus terrific	B.P. 160/100; left corneal reflex diminished	Operation and necropsy	Arteriosclerosis	Dead	Left VIII sectioned: later ligation vertebral arteries. Huge S-shaped basilar artery at necropsy	Since tinnitus remained unbearable, and a basilar aneurysm had been found at operation, the left vertebral artery was ligated 2 weeks later. At same time the right vertebral was temporarily compressed with forceps; death followed immediately although compressed only for 2-3 seconds. Most rapid death I have ever seen
XIV Crouch 1/17/32 Fig. 9	64	F	Basilar aneurysm right, also bilateral diffuse carotid aneurysms, vertebral arteries greatly dilated both sides	Difficulty swallowing, vertigo	3 years	No	No		No	No	Dysarthria and dysphagia	B.P. 200/120	Necropsy	Arteriosclerosis	Dead	Patient had a large dural tumor, found at necropsy. The carotid aneurysms were exposed at operation; nothing done	Large dural tumor found at necropsy. Died following exploration of carotid aneurysm. Although basilar aneurysm compressed right 5th and 8th nerves there were no subjective or objective manifestations. The basilar aneurysm continued into both vertebral arteries
XV Hanson 3/18/41	72	M	Basilar artery right	Trigeminal neuralgia	1 year	No	No	No	No	No		B.P. 224/110	Operation	Arteriosclerosis	Living	Aneurysm at operation for trigeminal neuralgia	Fifth nerve entirely hidden by aneurysm. Nerve later sectioned by temporal route
XVI Kelly 2/9/42	71	F	Basilar artery right	Trigeminal neuralgia	4 years	No	No	No	No	No		B.P. 240/140	Operation	Arteriosclerosis	Living	Aneurysm at operation for trigeminal neuralgia	
XVII Webster 2/2/42	73	M	Basilar artery right	Trigeminal neuralgia	5 years	No	No	No	No	No		B.P. 150/76	Operation	Arteriosclerosis	Living	Aneurysm at operation for trigeminal neuralgia	
XVIII Smith 3/17/42	58	F	Basilar artery right	Trigeminal neuralgia	4 years	No	No	No	No	No		B.P. 180/110	Operation	Arteriosclerosis	Living	Aneurysm at operation for trigeminal neuralgia	Localized bulge of basilar artery just posterior to sensory root
XIX Kratz 6/11/38	68	F	(1) Basilar at left superior cerebellar artery. (2) Bilateral internal carotid	None													
XX Fisher 4/9/41	65	M	Right vertebral; unruptured	None	None												
XXI Walker 11/7/30 Fig. 55	66	M	Basilar artery right, 5th nerve	Trigeminal neuralgia of left mandible and teeth	5 years	No	No		No, but attacks of diplopia (2)	No	Two attacks of diplopia	B.P. 200/130 (several years)	At operation for trigeminal neuralgia	Arteriosclerosis	Died 2 yrs. later	Trigeminal root sectioned	The lateral bulge of the basilar artery had compressed the 5th nerve. Cause of death not known (2 years after operation)