Effects of carotid ligation on the size of internal carotid aneurysms

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Aneurysms of the intrathecal portion of the internal carotid artery are commonly treated by ligation of one of the carotid arteries in the neck. After ligation of the internal carotid artery, subsequent angiographic demonstration of the site of such an aneurysm may prove difficult or impossible; but if ligation has been confined to the common carotid the internal carotid artery usually remains patent, and its injection either by percutaneous puncture or after its exposure then gives good filling of the aneurysm site.

This study has been undertaken to examine 25 such cases in detail, to discover what happens to these aneurysms following carotid ligation. With the exception of cases 6, 21, and 24 they were extracted from a consecutive series of carotid aneurysms reported elsewhere (Gibbs, 1962).

In the illustrations, traced in the first instance on polythene film, the posterior communicating and ophthalmic arteries have been reproduced whenever filled on angiography. In each case the anterior cerebral artery is identified by dots: two dots if it is continued on to fill the pericallosal artery and one if it fades away at that level. Clinical features have been summarized in the table.

FIG. 1. Case 1. Left carotid angiogram, (a) before and (b) three months after common carotid ligation. In (b) the anterior cerebral artery fades away early at the dot, but evidently reaches the midline before doing so.

FIG. 2. Case 2. Left carotid angiogram. (a) before, and (b) nine months after common carotid ligation.

FIG. 3. Case 3. Right carotid angiogram. (a) before, and (b) three months after common carotid ligation.

FIG. 4. Case 4. Left carotid angiogram. (a) before, and (b) four months after common carotid ligation.

TABLE I

CLINICAL SUMMARIES OF SERIES

Case No.	Sex	Age	Blood Pressure	Date of Subarachnoid Haemorrhage	Clinical State	Interval Subarachnoid Haemorrhage to Common Carotid Ligation	Secondary Ligation	Follow-up (yr.)	Clinical State at Last Follow-up	Remarks
1	F	26	120 70	Jan. 1954	Third nerve palsy four days after subarachnoid haemorrhage	3 weeks	No	6	Well, occasional diplopia	
2	M	51	130 80	Sept. 1953	Third nerve palsy four days before subarachnoid haemorrhage	12 days	No	10	Well, partial third nerve palsy	
3	F	65	160 90	July 1956	Third nerve palsy seven days after subarachnoid haemorrhage	6 weeks	No	7	Well, diplopia upwards	
4	F	29	120 80	May 1956	Third nerve palsy six days before subarachnoid haemorrhage, 38 weeks pregnant	9 days	No	8	Well, or; third nerve palsy had recovered completely three mth. after subarachnoid haemorrhage	Caesarian section five days after onset of third nerve palsy. Placenta praevia with haemorrhage, subarachnoid haemorrhage 28 hours later when blood pressure was 100/70
5	F	64	180 100	July 1958	Initial coma	4 weeks	No	6	Well	
6	F	59	200 90	Aug. 1959	Well, mild third nerve palsy at onset	6 mth.	No	5	Well	Two weeks after subarachnoid haemorrhage had intermittent common carotid occlusion for 10 days
7	M	48	180 100	Nov. 1957	Confusion, third nerve palsy at second subarachnoid haemorrhage 11 days later	10 days after second subarachnoid haemorrhage	No	7	Partial third nerve palsy	Attempted check internal carotid angiography unsuccessful—vessel thrombosed
8	F	54	160 80	None; Third nerve palsy Mar. 1953	Third nerve palsy seven days before confusion and dysphasia, supraorbital pain	3 weeks after onset	Internal carotid	12	Very mild left third nerve palsy. In Mar. 1965 developed complete right third nerve palsy due to similar fresh aneurysm on other side	Readmitted 13 mth. after left common carotid ligation because of recurrence for two mth. of supraorbital pain; relieved by internal carotid ligation. Later opposite aneurysm presented with supraorbital pain
9	M	60	120 80	Aug. 1957	Well	10 days	Internal carotid	5	Well	Intracranial trapping after vertebral angiography successful. Died 1963 bronchial carcinoma
10	M	34	130 80	Aug. 1958	Stupor after first subarachnoid haemorrhage. Third nerve palsy hemiparesis and dysphasia at second subarachnoid haemorrhage	4 days after second subarachnoid haemorrhage	No	5	No hemiparesis; speech falters when excited; mild third nerve palsy	Craniotomy five days after first subarachnoid haemorrhage unsuccessful—brain too tense to retract. Second haemorrhage 10 days later. Epilepsy
11	F	55	160 90	Dec. 1954	Right supraorbital headache for one month, then mild ptosis. Subarachnoid haemorrhage six weeks later with coma and completion of third nerve palsy	6 mth.	No	9	Still general debility, partial recovery of third nerve palsy	Judged too ill for treatment for 6 mth., check angiogram then showed no change

TABLE I—continued

CLINICAL SUMMARIES OF SERIES

Case No.	Sex	Age	Blood Pressure	Date of Subarachnoid Haemorrhage	Clinical State	Interval Subarachnoid Haemorrhage to Common Carotid Ligation	Secondary Ligation	Follow-up (yr.)	Clinical State at Last Follow-up	Remarks
12	F	25	110/84	Mar. 1955	Retroorbital pain for one week before subarachnoid haemorrhage; coma, aphasia, third nerve palsy, hemiparesis	18 days	Internal carotid	9	Speech and limbs normal, partial third nerve palsy	
13	F	44	140/90	Aug. 1955	Second subarachnoid haemorrhage two weeks after first; confusion	6 days after second haemorrhage	External carotid	8	Well	
14	M	56	130/80	Aug. 1956	Well	5 weeks				Readmitted two years later for persistent headache; clipping of intracranial carotid artery to trap aneurysm led to its rupture and death
15	F	52	210/120	(1) Feb. and (2) June 1957	Well	6 weeks	Internal carotid	7	Well, blood pressure 180/100	
16	M	29	130/70	Dec. 1957	Initial coma	12 days, partial closure only	Internal carotid	7	Well	Originally test closure of internal carotid caused syncope
17	F	55	170/100	(1) 1951 (2) April 1955	Well	7 weeks	External carotid	9	Well	Also had posterior communicating aneurysm on the other side—not treated
18	F	36	120/70	(1) April 1954 (2) 17 days later	Coma, temporary aphasia and hemiplegia, seven mth. pregnant	8 days	External carotid	4	Well—then lost to follow-up	Caesarian section one month after ligation
19	M	6	100/70	(1) June 1955 (2) 5 weeks later (3) 4 days later	Coma, hemiplegia, and aphasia. Left eye almost blind, right hemianopia	3½ weeks after third subarachnoid haemorrhage	External carotid	9	Left optic atrophy and right hemianopia; no hemiparesis or dysphasia	Left handed. Common carotid ligation in presence of extreme carotid spasm made dysphasia no worse
20	M	30	120/65	(1) 1952 (2) May 1955	Well	4 weeks after second subarachnoid haemorrhage	Internal carotid	9	Well	
21	F	61	240/140 to 170/100	None Radiograph May 1959	Well	—	—	5	Well	Chance discovery of calcification on radiograph led to treatment
22	M	37	150/100	May 1957	Temporary left hemiparesis	4 weeks	—	6	Well	
23	M	54	120/70	(1) 1936 (2) 1953 (3) 1954	Stupor, hemiplegia, and aphasia after third subarachnoid haemorrhage	10 weeks	—	10	Well	Craniotomy after contralateral check angiogram. Found inoperable
24	F	32	100/80	Feb. 1959	Decerebrate coma	17 days	—	5	Feeble, but no neurological signs	Refrigeration, tracheostomy
25	M	20	160/90	August 1958	Well	4 days (partial internal carotid closure)	Internal carotid	6	Impaired writing and fine movements of right hand; drags foot when tired	Eight hours after internal carotid ligation, aphasia and hemiplegia

FIG. 5. Case 5. (a) Right carotid angiogram. Five months after common carotid ligation vertebral angiography (b) fills the carotid syphon through channels other than the posterior communicating. (The vertebral artery filled strongly as far as the axis, where it faded away and carotid filling replaced it; c.f. Fig. 12.)

FIG. 6. Case 6. (a) Left carotid angiogram unchanged six months after intermittent carotid occlusion for 10 days. (b) Vertebral angiography four months after common carotid ligation.

FIG. 7. Case 7. (a) Left carotid angiogram and (b) right carotid angiogram six months after left common carotid ligation. The head had been tilted to throw the right carotid syphon above that of the left, which is now patent only above the neck of the aneurysm, marked by an arrow.

CONCLUSIONS

Of 24 aneurysms on the intrathecal carotid artery in this series, at the check angiograms after carotid ligation four appeared completely thrombosed, three were much smaller, 13 smaller, and four unchanged. The aneurysm of case 25 was unchanged after partial internal carotid occlusion. In 13 of the 16 aneurysms showing reduction in size, the reduction took the form of a more or less concentric shrinkage of the cavity, rather than as of clot build-

ing up at the site of the original puncture (or calcification) as happened in cases 8, 16, and 21.

The designation 'posterior communicating aneurysm' includes by common consent aneurysms at the usual site of origin of the posterior communicating artery, whether in fact such an artery is present or not.

Harris and Udvarhelyi (1957) reported a study of 66 posterior communicating aneurysms treated by common carotid ligation; check angiography at an interval of three months showed the aneurysm 'to

FIG. 8. Case 8. (a₁) Left carotid angiogram and (a₂) film taken two seconds later showing spasm with no aneurysmal filling except for a stagnant pool at the fundus; (b) internal carotid angiogram 13 months after common carotid ligation: the only part of the sac thrombosed is that formerly stagnant. The anterior cerebral artery is not shown in (a) because it filled in only one of four relevant films; it did not fill at all in (b).

FIG. 9. Case 9. (a) Left carotid angiogram. The small forward-going vessel is not the ophthalmic artery. (b) internal carotid angiogram four months after common carotid ligation. (c) vertebral angiography five months after internal carotid ligation.

FIG. 10. Case 10. (a) Left carotid angiogram; (b) vertebral angiogram one month after common carotid ligation. A small posterior communicating artery fills for the first time. Repeat check vertebral angiogram, (c), five months later, shows the posterior communicating artery to have enlarged, and though the internal carotid artery is considerably smaller, only one locus of the aneurysm appears to have shrunk. The anterior cerebral artery fills distally, beyond that shown, in one out of four films in (b), and in none out of three in (c).

FIG. 11. Case 11. (a) Right carotid angiogram, unchanged over six months with no treatment. (b) Internal carotid angiogram one year after common carotid ligation. The large posterior communicating artery now fails to fill, being presumably strongly forward-flowing. The anterior cerebral artery continues to fill particularly well in its whole course. The internal carotid artery is much reduced in size.

FIG. 12. Case 12. (a) Left carotid angiogram. (b) Internal carotid angiogram four months after common carotid ligation. No change except reversal of anterior cerebral and posterior communicating arteries. (c) Vertebral angiogram one year after internal carotid ligation; the vertebral artery fades away in all three lateral films and fills the affected carotid tree only, by a route unknown.

FIG. 13. Case 13. (a) Right carotid angiogram. (b) internal carotid angiogram four months after common carotid ligation. As in case 12, reversal of flow in anterior cerebral and posterior communicating arteries has occurred, but only the 'daughter' loculi appear clotted.

FIG. 14. Case 14. (a) Left carotid angiogram. (b) vertebral angiogram two years after common carotid ligation. Clotting appears to have occurred in the fundus of the sac, which has enlarged in an upward direction.

FIG. 15. Case 15. (a) Left carotid angiogram. (b) internal carotid angiography five months after common carotid ligation; the posterior communicating artery does not fill (though it had done so at vertebral angiography the previous day). (c) Vertebral angiogram five months after internal carotid ligation done by injecting the patent stump of the left common carotid. The anterior cerebral artery now does not fill, but the ophthalmic artery is seen throughout the series.

FIG. 16. Case 16. (a) Left carotid angiogram and (b) repeat left carotid angiogram eight months after subtotal closure of common carotid. In (b) the anterior cerebral artery fills as far as the dot in one film, not at all in two.

FIG. 17. Case 17. (a), above, left carotid angiogram; (b) below, internal carotid angiogram eight months after common carotid ligation. The anterior cerebral artery flow is unstable, filling beyond the dot in one film and in two not reaching the midline, which in Towne's views is dotted.

FIG. 18. Case 18. (a) Left carotid angiogram showing spasm beyond the (probably double) sac. (b) Internal carotid angiogram five months after common carotid ligation, and (c) a later film of the same series.

FIG. 19. Case 19. (a) Left carotid angiogram showing marked arterial spasm four weeks after subarachnoid haemorrhage. (b) Internal carotid angiogram four months after common carotid ligation. Obliquity of the view has thrown the middle cerebral branches upward.

FIG. 20. Case 20. (a) Left carotid angiogram. The opposite anterior cerebral artery is already dominant. (b) Internal carotid angiogram six months after common carotid ligation.

FIG. 21. Case 21. (a) Calcification visible on radiograph of skull. (b) Left carotid angiogram. (c) Internal carotid angiogram seven months after common carotid ligation.

FIG. 22 a

b

FIG. 22. Case 22. (a) Right carotid angiogram. (b) Internal carotid angiogram four months after common carotid ligation.

FIG. 23 a

b

FIG. 23. Case 23. The calcification seen on radiograph of skull in (a) is proved on carotid angiography (b) to represent a clotted loculus only. At contralateral angiography three months after common carotid ligation the aneurysm was unchanged.

a

b

FIG. 24. Case 24. (a) above, (b) below; (a) right carotid angiogram. (b) Vertebral angiogram by the subclavian route five months after common carotid ligation. Contralateral carotid angiography had failed to fill the right carotid syphon. Dotted line represents the sagittal plane.

FIG. 25. Case 25. The ineffectiveness of partial carotid occlusion. (a) Left carotid angiogram (oblique view), and (b), above; (c) and (d) below. Five months after subtotal closure of internal carotid by tantalum clasp, check left common carotid angiography showed aneurysm quite unchanged. These films have not been reproduced except for one in the neck, (b), showing concentration of contrast as is often seen above a spontaneous internal carotid stenosis. The degree of closure may be judged from (d), an 'edge-on' view of the clasp. (c) Contralateral carotid angiogram seven months after internal carotid ligation had caused infarction.

have completely disappeared in seven, to be much smaller in eight, and only slightly smaller in the rest'. None were reported as unchanged. In this series, in two cases (cases 9 and 12) of 16 posterior communicating aneurysms the sac was unchanged after common carotid ligation and in one (case 14) it was changed in shape but no smaller.

It appears likely that the presence of a posterior communicating artery of appreciable size, *i.e.*, visible on an angiogram, may have some influence on the response of such aneurysms to carotid ligation. Thus in the six cases (cases 1, 2, 3, 4, 5, 7) in which the aneurysms, after common carotid ligation, were either very much reduced in size or appeared to have thrombosed completely, no posterior communicating artery had been demonstrated at the original angiography. In six cases in which the posterior communicating artery filled in the original angiograms, the aneurysm was reduced in size only moderately (cases 11, 13, 15) or not at all (9, 12, 14). Case 8 is an exception, and cases 10 and 11 'compromises'. Case 10 is the only example of a posterior communicating artery strongly forward-flowing, not filling on carotid angiography but filled by vertebral artery injection. It is thus uncommon for a large posterior communicating artery not to fill from the carotid, and the treatment of a posterior communicating aneurysm should not be delayed for

vertebral angiography to find it. In spite of the suggestion made above, it cannot be claimed that carotid ligation protects an aneurysm more effectively in those cases where a posterior communicating artery cannot be demonstrated by carotid injection; for haemorrhage recurred in two such cases during the period covered by this study, before check angiograms had been performed.

THE ANTERIOR VASCULAR COMMISSURE In the follow-up angiograms of cases 1, 2, 3, 5, 6, 7, 10, 12, 13, 16, 22, 24, and 25 the opposite carotid artery dominates, in varying degree, the anterior cerebral-anterior communicating complex of both sides, as might be expected; and reversal of flow in the first part of the anterior cerebral artery on the ligated side is presumed responsible in large part for supplying the middle cerebral field. Unfortunately in this series, Townes' view being considered of minor interest at the time of check angiography, proof is lacking (except in case 7) that the proximal anterior cerebral arterial flow is reversed and remains so when the steady state is reached. In eight cases (cases 4, 9, 11, 14, 15, 17, 18, 21) the ipsilateral anterior cerebral flow has either not been reversed or is reversed so feebly that the forcible injection of contrast has influenced it. In cases 11, 14, and 15 it seems that the flow has been reversed in the posterior

communicating artery rather than in the anterior.

The ophthalmic artery is well filled by contrast at the original angiography in nine cases (cases 2, 4, 8, 15, 17, 18, 19, 20, and 22), and in eight of these is as easily visible in the check angiograms, after common carotid ligation, indicating that flow in it is still in a forward direction. Only in case 22 is the flow apparently reversed after common carotid ligation, filling at the first angiography and not at the second. This suggests that the point at which the arterial blood pressure is lowest, after common carotid ligation, is on the face more commonly than within the cranium.

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