INTRA-ARTERIAL PRESSURES IN THE NECK
AND BRAIN

LATE CHANGES AFTER CAROTID CLOSURE, ACUTE MEASUREMENTS
AFTER VERTEBRAL CLOSURE*

LOUIS BAKAY, M.D., AND WILLIAM H. SWEET, M.D.

Neurosurgical Service, Massachusetts General Hospital and Department of Surgery,
Harvard Medical School, Boston, Massachusetts

(Received for publication December 15, 1952)

In the past six years we have carried out measurements of intra-arterial
pressure in the carotid vessels in the neck and more recently in the intracranial arteries as well. Brackett and Mount2 and Woodhall, Odom, Bloor and Golden8 have reported similar studies. The intracranial studies have been facilitated greatly by the addition to our Sanborn electromanometer of a system to provide continuous minimal flushing with normal saline of the minute No. 27 gauge needle we use.† Pressure readings take place during this flushing process without introduction of inaccuracy and the problem of clotting is virtually eliminated even though we now dispense completely with anticoagulants such as heparin.

We have already reported1 on a comparison between the pressures in the cervical carotid and those in intracranial arteries down to 0.4 mm. in diameter. The pressures measured in the neck and in the main intracranial branches of the internal carotid with the flow free reveal that there is virtually no measurable pressure drop in the anterior and middle cerebral trunks. Even in the small arteries on the superolateral surface of the hemisphere the systolic pressures run from 65 to 92 per cent of those in the cervical carotid with an average of 83 per cent in the twelve vessels measured in as many patients. Upon occlusion of the common carotid the percentage drop in pressure in all portions of the internal carotid and its accessible branches is the same. These measurements include some in arteries as small as 0.4 mm. in diameter. Hence measurement of the pressure drop in the carotid in the neck proves to be a reliable indicator of the degree of fall throughout the branches of that carotid’s tree, and cervical carotid ligation has essentially the same effectiveness in the treatment of an intracranial aneurysm whether it arises from the carotid trunk or one of its smaller branches. Furthermore, since cervical carotid occlusion usually lowers the arterial pressure distal thereto by about

---

* Read at the Clinical Congress of the American College of Surgeons, September 25, 1952, New York City.
† This work has been supported by research grants from the National Institute of Mental Health of the National Institutes of Health, Public Health Service, from the American Cancer Society, and by funds from an anonymous donor, all to the Massachusetts General Hospital.

† The Sanborn electromanometer with equipment for continuous flushing is obtainable from the Sanborn Instrument Co., 39 Osborne St., Cambridge, Mass.
50 per cent, this tactic appears to represent the most rapidly controllable means of achieving hypotension in the distribution of the internal carotid artery and may prove to be the most satisfactory means of producing hypotension to facilitate surgical removal of haemorrhagic lesions in this part of the brain.

We wish to present now: (1) serial measurements of cervical carotid pressure in the neck from the time of occlusion up to several months thereafter and (2) findings on the distal pressures in the vertebral artery and its branches before and after occlusion of this main vessel.

**CAROTID PRESSURES LONG AFTER OCCLUSION**

It has long been established that collateral circulation to the brain of man develops promptly after one carotid is partially occluded. Thus Dandy found that his patients tolerated complete closure of the carotid in the neck within a week of a first operation at which such closure evoked neurologic signs of cerebral ischemia. Braden has recently reported 3 patients in whom intracarotid pressures rostral to closure of the common carotid had returned to the original level when measured 2 years, 48 hours and 24 hours respectively after the closure. In each patient there had been a substantial drop acutely immediately after occlusion.

It would appear valuable in ascertaining the efficacy of carotid ligation to know how long the falls in pressure may be expected to persist. We have data bearing on this point in 9 patients with intracranial aneurysms.* In 8 of them we have a set of pressure measurements at the time of the original exposure of the vessels in the neck, and another set 6 days to 24 weeks later when the wound was reopened for additional occlusive measures. In a ninth patient we have only the late reading nearly 2 years after total occlusion.

In the following 2 cases complete closure of a carotid artery was not carried out at the first operation.

**Case 1.** In Donalda B., aet. 40 years, the interval between measurements was 6 days. At the first session complete common carotid occlusion had reduced the pressure from 130/95 to 30/28 and additional occlusion of external carotid had no further effect. A residual systolic pressure of only 23 per cent of that with the flow free may be followed by a late thrombosis spreading from the aneurysm into the main arterial branches (see our case G.D.M. in Sweet, Sarnoff and Bakay). Hence we should not have employed complete occlusion here even if the patient had tolerated it for 30 minutes. However, in this patient, we had to open the occluding tantalum band almost completely in order to avoid the appearance of neurological signs. The pressure distal to it at that time was the same as with the flow free. Six days later the intracarotid pressure was about the same, 132/110, but now common carotid occlusion caused less reduction, to 63/58, or a remaining systolic level 48 per cent of the original. The addition of external carotid occlusion produced no further change and the patient remains well after permanent closure of the common carotid.

---

* For the opportunity of obtaining some of these readings, we are greatly indebted to Dr. John Drew, Chief of the Neurosurgical Service, Veterans Administration Hospital, Boston, Mass. and to Dr. Hannibal Hamlin, Clinical Associate in Surgery, Massachusetts General Hospital, Boston, Mass.
INTRA-ARTERIAL PRESSURES IN THE NECK AND BRAIN

Case 2. In Ann B., aet. 54 years, the initial readings were: with flow free 142/85, with common carotid closed 59/38, and with common plus external closed 59/38. Although the residual systolic pressure was 42 per cent of the original, signs of cerebral ischemia were apparent within 10 minutes of clamping the common carotid. This patient was left with the artery about half closed by the tantalum band, a degree of closure that had no effect on the systolic pressure. At the reopening of the neck, 11 days later, there was extreme fluctuation in the blood pressure distal to total occlusion of the common carotid, which we shall describe below. Complete occlusion of both common and external carotids has been well tolerated.

In the next 7 patients the common carotid artery was completely occluded at the first procedure.

![Fig. 1](image)

**Fig. 1. Case 6.** Pressure recording in right common carotid artery. (A) 1, 5 = free flow, 2, 4 = proximal occlusion of common carotid artery, 3 = additional occlusion of external carotid. (B) Recording in distal part of the same artery 3½ months after proximal occlusion. 6, 8 = no significant change in pressure drop as compared with 2, 3, 4, 7 = effect of manual compression of contralateral carotid artery.

Case 3. In Margaret S., aet. 40 years, the period intervening between the two operations was 12 days. The percentage drop distal to occlusion remained the same although the systemic systolic pressure was 175 mm. Hg the first time and only 140 at the next session.

Case 4. In Floyd G., a 28-year-old man, whose two measurements were made 5 weeks apart, essentially the same maintenance of percentage drop ensued.

Case 5. In Samuel C., a 32-year-old male, a major fall to 33 per cent of the initial level followed common carotid occlusion acutely. Six weeks later this level had risen somewhat, but only ½ the way to the general systemic arterial pressure. The actual figures were a fall from 118 to 39 mm. Hg acutely with a recovery to 62 mm. Hg in 6 weeks, so that a substantial reduction was still present. In this patient the rise in pressure was the result of the development of intracranial collateral channels because the level remained at 62 mm. Hg. when the external carotid was closed.

Case 6. In Agnes S., a 31-year old woman, the identical degree of pressure drop was maintained for the 3½ months that elapsed between the two studies (Fig. 1).
Case 7. In Eleanor W. (Fig. 2), a 65-year-old hypertensive arteriosclerotic female, signs of cerebral ischemia developed when her common carotid artery was occluded completely, as one might expect from a pressure drop of 216 to 54 mm. External carotid occlusion caused no further change. The common carotid was then partially closed by a tantalum band, but in the succeeding 9 weeks scar tissue completed the closure of this artery. Measurement then showed that the maximal systolic pressure distally had risen to 77 mm. This was shown to be caused by collateral back flow from the external carotid artery since the pressure fell almost exactly to 54 again when the latter vessel was occluded.

![Pressure recording in right common carotid artery. (A) 1, 5 = free flow; 2, 4 = proximal occlusion of the artery, 3 = additional occlusion of external carotid artery. (B) Pressure distal to occlusion 10 weeks later shows some increase (6, 8) since previous recording; occlusion of external carotid artery reduces pressure to original level (7).](image)

In contradistinction to the maintenance of a marked reduction in pressure in the previous 5 cases, is the behavior of our next patient.

Case 8. Robert K., aet. 35 years, showed pressure of 144/89 with the flow free, 69/58 with the common carotid occluded and 63/54 with common plus external occlusion. Five and a half months later his systemic arterial pressure was essentially the same but the pressure above complete occlusion of the internal carotid had now risen strikingly to 127/95. The residual pressure had come up from 44 per cent to 84 per cent of the systemic arterial level.

Case 9. In Elvira P., another 60-year-old hypertensive woman, the initial occlusion of the common carotid artery was carried out elsewhere without pressure measurements. Twenty-two months later, following a second subarachnoid haemorrhage, the carotid in the neck was exposed again. The systolic pressure distal to the ligation was 71 per cent of the systemic pressure—125 and 175 mm. Hg respectively.

Comment. In 10 of the first 38 patients in whom we made such pressure measurements acutely we found the levels distal to the occlusion of the internal carotid to be greater than in 60 per cent of those with the flow free. Consequently there is about a 25 per cent chance that the figure of 71 per cent in our Case 9 is nearly what it was immediately after the first operation.
In summary one notes that while the majority of this small series showed only slight to moderate late rises in the carotid pressure distal to full occlusion, the patients in our Cases 2 and 8 had a pronounced recovery of their pressures amazingly close to the previous level. The first two patients (Cases 1 and 2) who did not tolerate complete occlusion initially, but who did so 6–11 days later when their pressures distal to occlusion were higher, demonstrate another point, namely that the prompt development of an improved collateral circulation does not require a high degree of occlusion of a trunk artery.

SERIAL MEASUREMENTS DURING OPERATION

In view of the fluctuations seen in our late measurements we have made many more records during the initial 30–60 minute period of trial occlusion. In our first paper on this subject (Sweet and Bennett), we found in a few instances that the pressure drops on occlusion were maintained at the same level 30 minutes later. The cumbersome apparatus we used at first made serial measurement difficult, but with the electromanometer we have made such studies, recently, in 8 patients. In 4 of these the pressures were almost exactly the same at the end as at the beginning of the period of common or common plus external carotid occlusion. In our Case 1, we studied the effect of bilateral cervical sympathetic block with xylocaine, since occlusion of common carotid alone or common plus external had produced such a marked drop in pressure to 30/28. The effective block appeared to improve slightly the collateral supply via the external carotid since the pressure with common alone occluded now rose to 40/35, while it remained at the original level distal to occlusion of the internal carotid. However, a conclusion that the sympathetic block improved the circulation rests on dubious grounds because there was a similar or even greater degree of spontaneous rise in 3 other patients.

The most striking of these spontaneous changes occurred in Case 2 during the patient's second operation. At the start of this the distal pressure fell from 117/50 to 50/30 when the common carotid was occluded, a systolic drop to 43 per cent of the original. But after 30 minutes of such occlusion the distal pressure had risen to 83/50. During the next 20 minutes remarkable fluctuations occurred; at one point a level of 118/65 was reached, and at the end of this period of observation the distal pressure was 90/55. The systolic and diastolic pressures as recorded from a brachial cuff were relatively constant during this time and there was no obvious change in the patient's general status. She was fully rational, cooperative and free of pain throughout. The addition of external occlusion on several occasions did not change the immediate reading obtained. In patient Beatrice S., a pressure of 150/97 dropped to 44/42 upon occlusion either of common and external carotid or of the former vessel alone. After 30 minutes the pressure had risen to 72/58; some of this rise was caused by retrograde flow via the external carotid as shown by a fall to 63/53 when that vessel was closed. This rise was not main-
tained and at 45 minutes the pressure distal to occlusion of the common carotid only had dropped to 56/45. Blood pressures at a brachial cuff recorded nearly constant throughout this time. Patient Helen N's closure of the common carotid dropped the pressure from 116/76 to 64/52. During the next 30 minutes it fluctuated between 61/49 and 85/61. The pressures after occlusion of the external carotid as well did not change, giving evidence that no retrograde flow via this vessel was responsible for the fluctuations.

Spontaneous drops which are equally striking and of graver import than the rises can also occur. Thus in Case 1, the pressure distal to a partially closed tantalum band on the common carotid was 80/65 as compared with a level of 120/85 with the flow free. Fifteen minutes later she had become drowsier and showed contralateral weakness and numbness. The distal pressure had dropped to 38/35. We feared a thrombosis, but complete opening of the tantalum band was followed by an abrupt return to normal of the neurologic status and the distal carotid blood pressure.

It is apparent that there were moderate or marked fluctuations in carotid arterial pressure distal to occlusion within the first hour in roughly half of these patients.

PRESSURES IN THE VERTEBRAL TREE

Acting on the assumption that proximal occlusion of a vertebral artery would diminish the pressure within the corresponding posterior cerebral artery which bore an aneurysm, Falconer ligated the vertebral artery low in the neck. That this procedure will reduce at once the blood flow available to the brain stem and posterior cerebral vessels seems highly likely, and Falconer cited two instances from the literature in which ligation of one vertebral artery was followed by an operative fatality in patients who had massive space-taking lesions in the posterior fossa. It does not follow, however, that

![Fig. 3. Complete occlusion of vertebral artery at level of the atlas (1, 2) has no effect distal to the occlusion on pressure in either the vertebral or posterior inferior cerebellar artery.](image-url)
a reduction in the blood flow in this relatively tiny portion of the cardiovascular system will necessarily be accompanied by a lowering of the arterial pressure. In fact, we should expect the large basilar artery to transmit the pressures from the one open vertebral vessel without loss to both posterior cerebals and to the opposite closed vertebral artery. We have checked our reasoning during operative exposures in the posterior cranial fossa. The vertebral artery was exposed between the occiput and the atlas and the pressures were measured in both the vertebral and posterior inferior cerebellar arteries after proximal occlusion of the vertebral trunk. Fig. 3 illustrates the utter absence of any change in pressure in these vessels. One might hypothesize that a possible lessening of the blood flow in an artery decreases significantly the pressure within its aneurysm by reducing a presumptive "jet affect" at the aneurysmal opening. We know of no objective measurements to substantiate this, and submit that our findings demonstrate that no fall in pressure, clinically useful in the treatment of aneurysms of the vertebral arterial tree, is produced by occlusion of such artery, assuming that its mate on the opposite side is roughly similar in size.

SUMMARY

(1) Tentatively we find that a substantial acute drop in pressure distal to occlusion of carotid vessels in the neck is maintained for a sufficiently long period to be useful in the treatment of a majority of patients with aneurysms in that carotid system.

(2) We have found no measurable drop in pressure in the vertebral or posterior inferior cerebellar arteries distal to occlusion of one vertebral trunk at the atlas. Hence occlusion of a vertebral artery is not likely to aid in the treatment of aneurysms on vessels supplied by that artery.

REFERENCES