CHANGES IN INTERNAL CAROTID PRESSURE DURING CAROTID AND JUGULAR OCCLUSION AND THEIR CLINICAL SIGNIFICANCE*

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The surgical treatment of intracranial aneurysms has been advanced in the past 15 years by several distinguished studies, amongst which may be mentioned (1) the description of cerebral angiography by Moniz; (2) the proof that elective ligation of the carotid artery in the neck is relatively safe in cases of aneurysm, by the collective work of British neurological surgeons21 and by the work of Dandy6 and Matas15; (3) the reporting of several series of daring intracranial operations by Dandy,7 Dott,9,10 Jefferson,13 and others.

However, there remain a number of uncertainties related to the problem, particularly with respect to the long-term prognosis, the best type of operation, and the effectiveness of proximal ligation in reducing pressure in a weakened portion of the wall of an intracranial artery.

Available data are particularly meager with respect to the long-term prognosis, especially in those cases that have been characterized by one or more episodes of bleeding before completion of recovery from a previous subarachnoid hemorrhage. A patient with no localizing symptoms or signs following the first burst of subarachnoid bleeding is not commonly considered a candidate for angiography because of the alleged good prognosis. However, if a second outflow of blood occurs in the next few days or weeks, it is generally felt that the prognosis worsens a good deal, although we lack any good statistical study of the mortality in such cases. Gamsu is now following up the cases of subarachnoid hemorrhage in a group of Boston hospitals with the intent of making such a study upon which a prognosis might be based.

Further uncertainty prevails when an angiogram has demonstrated the precise location of an aneurysm on the internal carotid or one of its branches and a decision must be made as to whether the case requires no operation, ligation of one of the carotid arteries in the neck, or an intracranial approach to the aneurysm. Dandy,7 in his last work, regarded only a direct approach to the aneurysm itself as surgical treatment, and saw fit to carry out such direct operations on 8 infraclinoid and 13 supraclinoid aneurysms of the internal carotid and on 4 aneurysms of the anterior cerebral vessels. He classified ligations of the internal carotid artery in the neck as a "preoperative procedure." Dandy’s results may be summarized by

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CHANGES IN INTERNAL CAROTID PRESSURE

stating that 7 of his 8 patients with infraclinoid aneurysms survived a direct clipping or a trapping of the aneurysm between intracranial and cervical closures of the internal carotid, whereas the results of similar treatment of supraclinoid aneurysms were less encouraging. In 3 of 12 cases of the latter type in which the operation was not an emergency, the patients died from the operation; 1 of the survivors had a permanent hemiplegia and aphasia, and in 8 the aneurysm was effectively treated without producing disabling sequelae. In 3 of the successes a massive hemorrhage occurred from the internal carotid during operation and required emergency occlusion of that vessel, which happily was well tolerated. Although these brilliant operations represent a major contribution, the difficulty of the procedure and the discouraging ratio of 4 disasters to 8 cures make advisable a consideration of the effectiveness of simpler and less dangerous measures, such as the ligation of the internal or common carotid arteries in the neck.

Accordingly, the measurements reported in this paper were designed to shed light on the uncertainties related to the effectiveness of carotid ligation in reducing the hydraulic pressure in an intracranial aneurysm. We have carried out direct measurements of intracranial arterial pressure in the internal carotid artery before and after closure of various vessels in the neck pertinent to the problem. The principal objective of these measurements was to determine whether or not a major fall in blood pressure in the distal portion of the internal carotid artery was obtained consistently by occlusion of that vessel in the neck. From the data obtained we have deduced whether or not the pressure on a weak intracranial aneurysmal wall might be significantly reduced.

Direct measurement of intracranial arterial pressure changes in the region of the circle of Willis would have been preferable, but we were unable to pass a catheter suitable for pressure measurements past the tortuositities of the intrapetrous and intracavernous portions of the internal carotid artery. Accordingly, we reluctantly contented ourselves with the methods described below.

MATERIALS AND METHODS

Measurements were taken on 13 patients ranging in age from 5 months to 65 years. The common, internal and external carotid arteries were exposed to direct vision under procaine anesthesia in the cases of the 11 adults, and under general anesthesia in the cases of the 2 children.

The pressure-recording system was connected to the lumen of the internal carotid artery by means of a #16 or #18 gauge needle whose point was inserted into the common carotid artery and then passed on up into the internal carotid artery. The hydraulic pressure was transmitted from the needle by flexible non-distensible Saran tubing filled with fluid to a glass membrane optically recording manometer equipped with a hydraulic integrator bypass system. The apparatus has been fully described by Bennett, Bassett, and Beecher. This recording system permitted one to record faithfully and continuously at will either a full pulse pressure or an integrated mean pressure, with ready and speedy conversion from either form of recording to the other. The needle was frequently irrigated with normal saline solution containing heparin in order to preclude the formation of any thrombi. Needles with stilets were used, and following the pressure studies the stilet was inserted into the needle and the patient
taken to the X-ray department for angiography. Upon return to the operating room the hole in the artery made by the needle was routinely closed with arterial silk. The effects on the intra-arterial pressure of occlusion of the common carotid artery alone, of common plus external carotid (equivalent to internal carotid) occlusion, of occlusion of internal jugular vein, and of simultaneous occlusion of both common carotid arteries were measured. Pressures just after occlusion of common and internal carotid arteries were compared with pressures obtained during 90 minutes of such continuous occlusion. The possibility that change of pressure in the region of the carotid sinus might set up reflexes from this structure was forestalled by having a pool of 1 per cent procaine in the region of the carotid bifurcation in the neck before the pressure recording was carried out.

In all of the 13 cases studied a neurological examination was carried out after 30 minutes of occlusion of the external and common carotid arteries. In 10 of the cases, this included a check on the patient's mental alertness, strength, coordination, proprioceptive sensation, and graphesthesia in all 4 limbs and a note of the plantar responses. In all 10 cases, the findings remained normal. In the other 3 cases, a man who had been stuporous for 2 months and 2 small children, less complete examinations showed no change from the status prior to the occlusion. Hence, in each of these 13 cases therapeutic occlusion of the internal carotid might have been carried out if indicated; and the pressure changes are representative of those that may be expected immediately after such occlusion. It will be noted that the maximum systolic pressures we recorded with our special equipment were above 150 mm. of mercury in 8 of the cases and over 200 mm. in 2 of these 8 cases. However, the pressure in these cases as read at the same time by auscultation in the usual manner below a cuff around the arm was under 150 in 6 of the 8 cases, and between 150 and 200 by the usual clinical method only in the 2 cases in which we recorded pressures over 200 mm. of mercury. So our material was not unduly weighted with cases of hypertension.

CRITICISM OF METHOD

Since the needle point lay in the cervical portion of the internal carotid artery, the systolic and diastolic pressures recorded are those in the artery at that point and these pressures in the intracranial internal carotid would be slightly different due to the frictional pressure drop and to the energy consumed in expanding the intervening arterial wall with each pulse beat. Inasmuch as there is no significant branch of the internal carotid until the ophthalmic branch is given off, the pressures recorded after occlusion are essentially those at that intracranial point in the vessel, the remainder of the internal carotid from there proximally to the needle point simply forming part of the tubing to the manometer. Hence with the manometer damped and with common and external carotid arteries occluded on the side of the recording, the pressure measured is that of the internal carotid artery at the point of origin from it of the ophthalmic artery. But the undamped pressure cannot be taken as a faithful representation of the pulse pressure at that point, because of the additional damping effect of the elastic walls of the
internal carotid artery from its ophthalmic branch to the point of the needle. Likewise, the undamped pressures recorded with the flow free would probably be somewhat less in the supraclinoid portion of the internal carotid than at the needle point. For these reasons we regard our quantitative data as more reliable when integrated mean pressures were recorded.

RESULTS

For each case several hundred determinations of blood pressure were measured off from the continuous tracings taken during operation. The main results are summarized in Tables 1 and 2.

Note. In Figs. 1–4: Top row of dots—time in 5 sec. intervals. Top black line—a signal marker. 3rd line—tracing of the intra-arterial pressure. 4th line—for reference. Numbers at left refer to pressures in mm. of mercury.

![Graph](image-url)

Fig. 1. Case C. J. Recording starts with full pressure in internal carotid (external carotid occluded) At 1st signal the common carotid occluded also, producing marked fall in pressure. Ten seconds before 2nd signal the recording changed to integrated mean pressure. At 2nd signal occlusion of common carotid released, giving a reading of integrated mean pressure with flow free.

The figures indicate the type of tracing obtained, and are selected to show typical pressure responses to each of the main procedures carried out. Fig. 1 shows the type of pressure fall obtained when the common and external carotid arteries are occluded. Fig. 2 demonstrates the absence of significant change produced by opening or closure of the external carotid artery during occlusion of the common carotid. Fig. 3 illustrates the absence of change in pressure in the internal carotid observed whenever the ipsilateral internal jugular vein was closed for 2 or 3 minutes while the common and external carotid arteries were occluded. A result of this type was obtained in every case in which such determination was made. Fig. 4 represents one type of result obtained when the carotid on the second side was compressed percutaneously, the common and external carotids on the side of the open wound being already occluded. This represents the maximal fall
obtained. In another group there was no noticeable fall from this procedure, and in other cases a fall of intermediate degree ensued.

FIG. 2. Case I. C. Recording starts with common carotid occluded, external carotid open. At 1st signal external carotid also occluded revealing no measurable change in undamped pressure. At 2nd signal the external carotid was released showing a slight rise in pressure.

DISCUSSION
EFFECT OF OCCLUSION OF INTERNAL CAROTID ARTERY

From Table 1 it is evident that occlusion of common and external carotid arteries produced a reduction in both maximum and minimum systolic pressures to 50 per cent of the original values prior to occlusion, and corresponding decreases of maximum and minimum pulse pressures to 25 per cent and of integrated mean pressures to 58 per cent of the original values. We had not anticipated results of this nature, as in previous animal experiments\(^3\) we found the pressures in the arch of the aorta to be virtually identical with those in the superior and inferior mesenteric arteries. Hence we expected to find but little pressure fall in the internal carotid arteries of patients who had no neurological sequelae when the internal carotid was occluded. The surprising magnitude of the fall actually measured is interpreted by us as evidence that occlusion of that vessel may indeed be effective therapeutically in reducing the hydrostatic pressure and pulse pressure in the intracranial arteries in question.

We call attention particularly to the marked drop in recorded pulse pressure. In spite of the recognized uncertainties inherent in our recordings of undamped pressure, we regard our data as reflecting a corresponding
significant though less marked fall of pulse pressure in the circle of Willis. Such a fall would not only relieve the aneurysmal wall of rhythmic expansile forces it is not stressed to bear, but may also be a major factor in permitting a healing clot to develop in the aneurysm.

One may inquire how long-lasting are the pressure falls produced by internal carotid occlusion. We have direct evidence bearing on this question only in relation to the 30-minute period during which the vessel was occluded at operation, and during which records of pressure were taken. We found no difference in the internal carotid artery at the beginning and again at the end of the half-hour period of occlusion, indicating that the extensive pressure drop is maintained during at least that brief time. We have only indirect evidence bearing on the long-term effectiveness of internal carotid closure. In this connection we present the following case report:

In J.M., a 36-year-old male, a saccular aneurysm was demonstrated by arteriogram on the intracranial portion of the left internal carotid artery on Dec. 18, 1945. A 30-minute period of direct occlusion of this vessel in the wound in the neck produced no abnormal neurological signs and a tantalum clip, 8 mm. wide, was then used to occlude the vessel. No additional neurological signs ensued, but the paralysis of the left oculomotor nerve present pre-operatively persisted with but slight improvement. The left internal carotid artery was re-exposed in the neck on Mar. 20, 1946, with the intent of measuring the intra-arterial pressure in the vessel distal to the point of occlusion. To our surprise, the vessel rostral to the tantalum clip was contracted down to an avascular tiny cord about 2 mm. in diameter. This was followed up toward the base of the skull and remained bloodless and of the same caliber. It was transected but no bleeding occurred and a hair-sized lumen without clot was seen. There could scarcely have been an error in identification because the large tantalum clip, and the powerful pulsation of the internal carotid artery proximal to it, were unmistakable. This patient has since shown slow steady improvement in the function of the left 3rd nerve. His only complaint is diplopia on upward and downward gaze.

Apparently the application of the metal clamp to the vessel was followed by a contraction distal thereto sustained for over 3 months. If this contraction persists and extends up to the level of the aneurysm, he has of course been cured. Such a sustained contraction extending up to its first major branch, the ophthalmic artery, would probably make superfluous the application of a clip to the internal carotid at the anterior clinoid process.

The question of the gradual development of anastomotic circulation increasing over weeks and months must be considered. In such a case the intracarotid pressure on the side of the ligation might gradually rise. We have no measurements bearing on this question, but plan to take some if a suitable case should appear. Indirect evidence is presented in Dandy’s work. He found that when he made only a partial closure of the internal carotid artery in the neck because a complete closure provoked neurological signs, a subsequent complete closure could be carried out in 1 to 6 weeks without neurological sequelae. This may indicate that collateral circulation to the brain can build up rapidly in man. Development of such collateral circulation may in part vitiate the helpful effect of the initial fall in pressure occasioned by carotid occlusion.
TABLE 1

**Summary of results**

<table>
<thead>
<tr>
<th></th>
<th>Systolic Pressure</th>
<th>Diastolic Pressure</th>
<th>Pulse Pressure</th>
<th>Integrated Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum</td>
<td>Minimum</td>
<td>Maximum</td>
<td>Minimum</td>
</tr>
<tr>
<td>G. R.  ♂</td>
<td>184</td>
<td>147</td>
<td>105</td>
<td>98</td>
</tr>
<tr>
<td>Age: 53 yrs. (1)</td>
<td>72</td>
<td>67</td>
<td>48</td>
<td>44</td>
</tr>
<tr>
<td></td>
<td>45%</td>
<td>46%</td>
<td>50%</td>
<td>60%</td>
</tr>
<tr>
<td>B. S.  ♂</td>
<td>195</td>
<td>105</td>
<td>108</td>
<td>98</td>
</tr>
<tr>
<td>Age: 54 yrs. (2)</td>
<td>85</td>
<td>74</td>
<td>64</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>39%</td>
<td>37%</td>
<td>61%</td>
<td>55%</td>
</tr>
<tr>
<td>I. C.  ♂</td>
<td>195</td>
<td>171</td>
<td>87</td>
<td>78</td>
</tr>
<tr>
<td>Age: 64 yrs. (3)</td>
<td>75</td>
<td>64</td>
<td>54</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>38%</td>
<td>37%</td>
<td>62%</td>
<td>68%</td>
</tr>
<tr>
<td>D. H.  ♂</td>
<td>137</td>
<td>125</td>
<td>88</td>
<td>82</td>
</tr>
<tr>
<td>Age: 4 yrs. (4)</td>
<td>64</td>
<td>60</td>
<td>58</td>
<td>56</td>
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<tr>
<td></td>
<td>47%</td>
<td>48%</td>
<td>66%</td>
<td>68%</td>
</tr>
<tr>
<td>R. S.  ♂</td>
<td>113</td>
<td>95</td>
<td>67</td>
<td>57</td>
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<td>Age: 19 yrs. (5)</td>
<td>48</td>
<td>48</td>
<td>40</td>
<td>30</td>
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<tr>
<td></td>
<td>42%</td>
<td>42%</td>
<td>60%</td>
<td>53%</td>
</tr>
<tr>
<td>H. M.  ♂</td>
<td>123</td>
<td>118</td>
<td>79</td>
<td>74</td>
</tr>
<tr>
<td>Age: 41 yrs. (6)</td>
<td>68</td>
<td>58</td>
<td>44</td>
<td>41</td>
</tr>
<tr>
<td></td>
<td>51%</td>
<td>52%</td>
<td>66%</td>
<td>55%</td>
</tr>
<tr>
<td>A. B.  ♂</td>
<td>174</td>
<td>162</td>
<td>71</td>
<td>68</td>
</tr>
<tr>
<td>Age: 65 yrs. (7)</td>
<td>81</td>
<td>79</td>
<td>58</td>
<td>54</td>
</tr>
<tr>
<td></td>
<td>47%</td>
<td>49%</td>
<td>82%</td>
<td>79%</td>
</tr>
</tbody>
</table>

* 1st line: average pressures in internal carotid artery with flow free.
  2nd line: average pressures in internal carotid artery with common and external carotid arteries clamped.
  3rd line: percentage of original pressure remaining after external and common carotid arteries clamped.
  4th line: (omitted from some cases) pressures in the internal carotid artery with the common carotid clamped and the external carotid open.

Average maximum and minimum pressures refer to means of the peaks and of the troughs respectively of the cyclic blood pressure variations composing the Traube-Herring waves.
TABLE 1—(continued)

<table>
<thead>
<tr>
<th></th>
<th>Systolic Pressure</th>
<th>Diastolic Pressure</th>
<th>Pulse Pressure</th>
<th>Integrated Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum</td>
<td>Minimum</td>
<td>Maximum</td>
<td>Minimum</td>
</tr>
<tr>
<td>R. R. cΛ</td>
<td>227</td>
<td>223</td>
<td>125</td>
<td>124</td>
</tr>
<tr>
<td>Age: 42 yrs.</td>
<td></td>
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<tr>
<td>(8)</td>
<td>75%</td>
<td>70%</td>
<td>92%</td>
<td>87%</td>
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<tr>
<td></td>
<td>156</td>
<td>141</td>
<td>109</td>
<td>95</td>
</tr>
</tbody>
</table>

Common carotid clamped, external carotid open.

C. C. 2            | 113               | 95                 | 75             | 62                 | 38               | 34               | 92               | 88                |
| Age: 5 mos.       |                   |                    |                |                    | 60%              | 63%              | 73%              | 81%                |
| (9)              | 6%                | 24%                | 5%             | 23%                | 14%              | 9%               | 62               | 58                |

Common carotid clamped.

J. O'M. cλ        | 146               | 122                | 94             | 81                 | 57               | 42               | 127              | 117                |
| Age: 23 yrs.      |                   |                    |                |                    | 65               | 65               | 65               | 65                |
| (10)             | 62%               | 61%                | 82%            | 80%                | 29%              | 24%              | 29%              | 29%                |

Common carotid clamped.

D. E. cλ         | 153               | 127                | 74             | 61                 | 79               | 66               | 103              | 90                |
| Age: 32 yrs.      |                   |                    |                |                    | 74               | 65               | 65               | 65                |
| (11)            | 48%               | 51%                | 88%            | 93%                | 11%              | 11%              | 64%              | 68%                |

Common carotid clamped.

P. F. cλ         | 177               | 150                | 94             | 81                 | 80               | 68               | 129              | 113                |
| Age: 41 yrs.      |                   |                    |                |                    | 80               | 70               | 62               | 65                |
| (12)          | 55%               | 59%                | 74%            | 77%                | 41%              | 38%              | 68%              | 69%                |

Common carotid clamped.

C. J. cλ         | 175               | 136                | 102            | 91                 | 73               | 45               | 130              | 109                |
| Age: 56 yrs.      |                   |                    |                |                    | 81               | 65               | 65               | 65                |
| (13)          | 46%               | 46%                | 63%            | 66%                | 23%              | 24%              | 54%              | 55%                |

Common carotid clamped.

Average % of original pressure remaining after common and external carotid clamped.  
50.4% 50.8% 70.6% 70.3% 26.4% 34.3% 57.7% 58.6%

Even if the period of reduced pressure in the carotid is not of long duration, however, it may suffice to stimulate a prolonged arterial contraction or to permit a clot to fill the aneurysm and a cure to occur. Reid\textsuperscript{20} cites 4 cases (numbers 45, 47, 53, and 55) in which only proximal occlusion of common, internal or external carotid arteries for massive aneurysms in the neck was followed by conversion of the pulsating mass to a solid pulseless swelling which then gradually disappeared. This would probably be an even more likely event in the smaller intracranial berry aneurysms. Similarly, Locke,\textsuperscript{14} in a complete summary of all reported cases of carotid cavernous fistula up to 1924, found that of 34 patients in whom the internal carotid artery was ligated in the neck, 33 were cured or improved. Of course it is likely that more failures than successes remain unreported. Again, in the special case of saccular aneurysms of the intracavernous portion of the internal carotid, we know of no instance reported where occlusion of the cervical portion of the internal carotid has failed to control the advance of symptoms. However, aneurysms of this segment of the artery, protected as they are by the dural covering of the cavernous sinus, may have a materially different prognosis.
from those lying within the subarachnoid space. We cite these clinical findings to support our physiological evidence that occlusion of the internal carotid artery may well be therapeutically effective in treatment of intracranial aneurysms, but we feel nevertheless that the clinical evidence is too meager to be conclusive, and that more data are necessary before a final opinion can be expressed with confidence.

In an analysis of intracarotid pressure measurements it is also pertinent to comment on one more statement that appears in the literature. Albright (p. 2431) has remarked that, when aneurysms occur distal to the terminus of the internal carotid, it seems unlikely that much could be accomplished by ligation in the neck "because of the ready anastomoses." And Jefferson12 in a notable contribution states that one should not tie the carotid in the neck unless the aneurysm arises from it because of the possibility that the aneurysm will compress the branch from which it rises. But at least in the case of aneurysms arising from near the bifurcation (and the great majority are in this area) our measurements indicate that a major fall in systolic and pulse pressures is likely to occur upon internal carotid occlusion. The achievement of such a fall is the objective of the therapy and the basis of Albright's criticism is hence removed. Jefferson's criticism, on the other hand, is that the pressure may be reduced too much. Although we can see that an aneurysm on an artery in the depth of a sulcus is more likely to compress the vessel than one lying free in the subarachnoid space, it appears to us that this is only one of several causes of inadequate collateral circulation—to be checked by trial occlusion of the exposed carotid in the first instance and by careful postoperative observation and removal of band if late symptoms develop—just as is the case in aneurysms of the internal carotid proper. Hence, if an aneurysm shortly beyond this bifurcation is not to be treated by direct approach, we favor a trial of carotid occlusion in the neck, followed by application of a broad tantalum band if no signs appear.

**Comparison between occlusion of common and of internal carotid arteries**

It has been contended that if a common carotid artery is occluded, collateral circulation will be supplied to the brain by a flow of blood into the corresponding internal carotid from the external carotid on the same side, the latter vessel receiving its blood from anastomoses with branches of the other external carotid. Thus Dorrance8 cites 20 authors who argue that there may be significant retrograde flow from the external carotid into the internal carotid after occlusion of the common carotid. He estimated from the size of the vessels and their main branches that collateral through these channels prevented ligation of the common carotid artery from decreasing the blood flow through the internal carotid by more than about 50 per cent. Ray19 has subscribed to this point of view. Schorstein21 has said "There can be no doubt that internal carotid ligation is more dangerous (than common carotid ligation) in a comparable series of cases." Olivecrona16 stated that in supraclin-
oidal saccular aneurysms, "it is a wise policy to begin with a ligation of the common carotid." Dandy, however, has doubted that there is much of a safety factor in such a course, and in patients who had no neurological signs or symptoms after 10 minutes' compression of the internal carotid, he occluded this vessel as his initial procedure.

We do not find adequate data from these papers to support any of these statements one way or the other insofar as elective ligation for saccular aneurysms is concerned. Indeed, we regard Dorrance's measurements of the diameter of the main branches of the carotids as faultily construed; in our opinion the sum of the cross sectional areas and lengths of the small anastomotic channels between right and left side would determine the amount of collateral flow in the face of a given pressure differential, and not the size of the main branches.

Our own physiological pressure data do not support the idea of any significant collateral flow entering the internal carotid through the external carotid after occlusion of the common carotid. If there is appreciable retrograde flow of this nature, then pressure in the internal carotid with common carotid occluded should be higher with the external carotid open than with it closed. In the 8 cases in which we made such measurements (Table 2) a small rise occurred in 3; in 2 cases no pressure change was recorded; and in 3 cases a slight fall in pressure was noted. In these latter 3 cases, the data may signify that the intracranial anastomoses between the 2 internal carotid arteries were more significant than those between the 2 external carotids, and that if the external vessels were left open, the blood supply to the brain would be reduced. It is important to point out that in all of these cases a grossly deficient anastomosis at the circle of Willis had been excluded by determining that 50 minutes of internal carotid occlusion caused no change in neurological signs or symptoms. But in cases in which elective carotid occlusion is thus tolerated it would appear from these measurements that occlusion of the internal carotid is no more likely to impair immediately the

<table>
<thead>
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<th>Table 2</th>
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<tbody>
<tr>
<td>Average change in internal carotid pressure when external carotid opened during common carotid occlusion</td>
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<tr>
<td>G. R.</td>
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<tr>
<td>B. S.</td>
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<tr>
<td>I. C.</td>
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<tr>
<td>D. H.</td>
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<tr>
<td>R. S.</td>
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<tr>
<td>H. M.</td>
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<tr>
<td>A. B.</td>
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<tr>
<td>R. R.</td>
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</tbody>
</table>
cerebral circulation than is occlusion of the common carotid; hence the former vessel should be occluded in order to decrease the later development in collateral circulation.

There is another reason for proceeding in the first instance to closure of the internal carotid artery and that is the diminished likelihood that occlusion of this smaller artery in continuity will be followed by a fatal late erosion of the vessel just proximal to the closure. This complication has been a major bugbear in cases in which the aorta has been ligated (Bigger\textsuperscript{4}). It becomes less of a hazard the smaller the vessel occluded. There are at least 3 reported cases in which a late hemorrhage has occurred at the site of occlusion (Reid\textsuperscript{20} [Case 48], Poppen\textsuperscript{18}, Bigger\textsuperscript{4}) when the common carotid artery was closed in continuity, but we have found none from internal carotid occlusion.

Possible exceptions to this preference for internal carotid ligation might occur in cases of arteriovenous aneurysm (not saccular aneurysm). Olivecrona\textsuperscript{16} has had 2 cases of this type (Cases 2 and 6) in which ligation of the internal carotid produced severe neurological deficit requiring release of the ligature, whereas common carotid ligation in each of them was well tolerated. The rapid flow of blood out of the arterial tree into the fistulous opening makes these cases a special problem. It may be that intra-arterial pressure measurements should be carried out in the cases with fistulas to determine the relative safety of common and/or internal occlusion before either is done.

**EFFECT OF OCCLUSION OF INTERNAL JUGULAR VEIN**

Some surgeons experienced in arterial surgery contend that when a major arterial trunk to the head or limbs is ligated the accompanying veins should also be ligated in order to give the lesser amount of blood available to the part a longer time to come to equilibrium with its extracellular fluid. Thus Holman\textsuperscript{11} states, "in old adults ligation of the carotid should invariably be accompanied by ligation of the jugular vein also." If this simultaneous venous ligation does produce the desired damming back of the blood in the brain then there should be at least a slight rise in the pressure in the distal end of the occluded internal carotid. In our cases no such rise was found at any time over periods up to 3 minutes of continuous occlusion of the internal jugular vein below the entrance into it of the common facial vein (Fig. 3). This would suggest that the enormous number of veins draining the head prevents ligation of a single internal jugular vein from exerting an appreciable effect. We should point out that none of our patients had a carotid cavernous arteriovenous fistula. In this type of case, as suggested by Adson,\textsuperscript{22} ligation of the internal jugular vein might have greater likelihood of exerting some measurable delaying action on the venous return.

**PRESSURE MEASUREMENTS UPON BILATERAL CAROTID OCCLUSION AND THEIR CLINICAL SIGNIFICANCE**

In several of our cases, while we were recording internal carotid pressures with the ipsilateral common and external carotid vessels occluded in an open
wound, we then pressed in the region of the carotid on the intact skin of the other side of the neck. Our recorded pressure changes in case of contralateral carotid compression under these conditions were variable, and we believe tentatively that these variations may in part reflect anatomic variations in the region of the circle of Willis which bear on the hazards of direct operative approach to a supraclinoid aneurysm of the internal carotid. Unpredictable variations in the competence of the anterior and posterior communicating arteries constitute one major feature of the hazard, and there is at present no satisfactory method for testing the efficacy or even the presence of either of these vessels. Arteriograms demonstrate these channels only occasionally—probably because the pressure at each of their ends is usually so nearly equal.

The danger is clearly shown by the result in Dandy's cases 18 and 19, in which, when both an internal carotid and its posterior communicating artery were sacrificed intracranially, the patients were hemiplegic at the end of the operation. On the other hand, when the same procedure was carried out in his cases 9 and 39 no cerebral malfunction ensued. This indicates that the anastomoses between the anterior cerebral arteries are ample to supply the vascular beds of both internal carotid arteries in some cases but not in others.

Anatomical evidence of danger in a direct surgical interference with channels is likewise abundantly available. In a series of dissections on autopsy specimens carried out by one of us (WHS) the posterior communicating artery was found to arise from the middle third of the supraclinoid portion of the vessel in nearly all instances. Thus there is distinct hazard of loss of this source of collateral during a direct operation on an aneurysm of this part of the internal carotid. Padget, in an extensive survey of 1603 cases of his own and from the literature, found the posterior communicating artery absent on one or both sides in 6 per cent of the series, whereas in 12 per cent of the total this vessel was small—perhaps even impervious—on one or both sides.

Thus these anatomical hazards and uncertainties make desirable some objective test of the competence of the anterior communicating vessels in

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Fig. 3. Case R. S. Recording starts with common and external carotid arteries occluded. At the 1st signal internal jugular vein on the same side also occluded. At the 2nd signal internal jugular vein released. No significant change in the pressure.
cases when operative interference with the posterior communicating or the supraclinoid portion of the internal carotid is contemplated. We wish to present our few measurements as suggesting an approach to this problem. Unfortunately, we have not yet had an opportunity to study on autopsy material or under direct operative exposure the actual degree of correlation between our various types of pressure readings and the status of the vessels under consideration.

As noted previously, in certain cases percutaneous pressure on the contralateral common carotid artery produced a marked fall in the distal segment of the ipsilateral internal carotid clamped off from its communication with common and external carotid (Fig. 4), whereas in other cases either a lesser fall, or even no significant pressure change was produced by the same manoeuvre. We would like to point out that in the first instance when a marked fall in pressure was noted, it indicated that the greater part of the collateral blood supply was derived from the contralateral carotid artery, and only a small part from the basilar posterior communicating source. This collateral supply from the contralateral carotid artery might traverse the circle of Willis either through anterior or posterior communicating channels, or both. We have already referred to the high incidence of deficiencies in the posterior communicating system. In contrast, Padgett found only 3 cases in reports of a total of 1803 cases in which the anterior communicating artery was absent. Indeed, the most common abnormality of this vessel is some form of duplication. The presence of each anterior cerebral component of the circle of Willis and its origin from the ipsilateral internal carotid can be checked by arteriography. Consequently, in cases in which a marked fall in pressure occurs on occlusion of the second carotid and in which normal origins of the anterior cerebral arteries are demonstrated by arteriography one has evidence that the flow from the second carotid could attain the opposite side by way of the anterior portion of the circle of Willis and would be so large in volume that loss of the collateral from the posterior communicating would not be attended by the disasters which befell Dandy in his Cases 18 and 19. In the other cases, where we were able to induce comparatively little or no fall of pressure by digital compression of the intact contralateral carotid, we can interpret our findings by stating that this lack of pressure change upon effective carotid compression may indicate that the basilar artery is capable of carrying an important part of the collateral supply. This group includes some cases in which direct operation on an aneurysm of the supraclinoid portion of the internal carotid carries the additional and perhaps prohibitive hazard of provoking a serious unilateral cerebral ischemia. Cases where contralateral carotid compression produced moderate falls, such as in J. O'M., where occlusion dropped the ipsilateral internal carotid pressure from 91 to 52 mm. Hg, present an intermediate situation wherein we cannot judge the competency of the collateral channels. Whether this represents a level sufficient to supply the hemisphere is of course unknown. But if in such a case of a supraclinoid aneurysm of the internal carotid we
were contemplating intracranial operation, we would begin by occlusion of that vessel in the neck and measure the pressures in the vessel distal to the occlusion some weeks later to see if there was evidence of increased collateral supply, especially by way of the anterior communicating vessels.

We suggest this possible useful application of internal carotid pressure measurements, even though its value and interpretation is as yet unproven,

in the hope that evidence for or against its utility may be assembled more speedily by its trial in a number of hands. However, we must emphasize that its successful application and interpretation depend on effective compression of the contralateral common carotid, and as will be seen in the next section, such effective percutaneous compression may be difficult to achieve and more difficult to establish. We are not certain that we attained it on every trial, and we envision the distinct possibility that ineffective compression may account for some of our cases where no pressure drop was recorded during this manoeuvre.

**EFFECTIVENESS OF PERCUTANEOUS CAROTID COMPRESSION**

An additional fact of clinical importance became apparent during the recording of pressures when carotid occlusion on the second side was attempted. This was the considerable difficulty in securing a full occlusion of
the vessel through the intact skin and hence a reliably consistent maximal pressure drop. Even though one or more doctors had made the attempt in each instance the precise area at which the compression had to be made in order to secure a maximal effect was found only after much trial and error, often with little or no pressure fall being obtained initially when the person performing the compression was "certain" he was in the right spot. Often the pressure necessary to produce a maximal fall could not be tolerated for minutes by the patient. Accordingly, we conclude that preoperative tests of the efficacy of the collateral circulation by compression of a carotid through the intact skin are unreliable, and that test should be made wherever possible by direct positive occlusion of the vessel in an open wound under local anesthesia where closure is contemplated. For the same reason, we do not bother with exercises of digital compression of the carotids several times a day in an attempt to increase collateral circulation before arteriography. Jefferson has noted in his clinical material that the test of percutaneous compression of the carotid might be negative and yet tying of the carotid produce hemiplegic signs. He suggests intracranial vascular spasm following the ligature as the cause of the discrepancy. Our experience points to difficulty in securing full compression by the percutaneous route as an important factor also.

TREATMENT OF LATE NEUROLOGICAL SEQUELAE OF CAROTID OCCLUSION

The information we have already gained makes fruitful a discussion of still another problem in connection with a complication of carotid ligation, to wit, the cases in which signs of neurological deficit appear late—over 6–8 hours—following carotid occlusion. Though our present series does not include any cases of this nature, Dandy had recently contended that a propagating thrombus or an embolus must be present to explain this complication, whereas Schorstein has presented evidence that cerebral ischemia with onset at the time of ligation, but of degree such that its cumulative effect causes clinical signs only hours or days after ligation, is the chief cause of this late neural damage. Each protagonist is able to cite postmortem material to support his contention, and the subject has more than academic interest since, if Dandy's viewpoint is correct, one is unlikely to accomplish any useful purpose by releasing a band or ligature on the carotid in the neck after the late neurological signs have supervened. In the clear-cut postmortem cases that he cites, extensive thrombi occupied some or all of the internal carotid on the side of the ligature, release of which could only have driven more blood clot into the anterior or middle cerebral arteries. This would only eliminate still more of the collateral circulation from other sources. A number of the patients with such thrombi developed their hemiplegias within a few minutes while lying quietly.

On the other hand, in the 2 cases cited by Schorstein in which late hemiplegia followed carotid ligation, the full paralysis developed only gradually
over the course of hours or days and the postmortem study in each case revealed a softening in the domain of the middle cerebral artery without thrombus or embolus. The tentative impression was that in each case the middle cerebral artery had been compressed by the aneurysm to a major degree following the carotid occlusion. A remarkable feature of one of these cases is that Dandy was the operator, and he subsequently stated that “compression (from the outside) would, of course, act precisely like an intravascular occlusion from a thrombus, and in the end the result would therefore be essentially the same.” It is clear that the result—hemiplegia or death—need not be the same if the circulation is promptly restored by the release of the occlusion of the carotid, and such cases present excellent evidence that the carotid should always be occluded in such fashion as to permit restoration of the circulation, if necessary. Moreover, the drop in pressure caused by carotid compression is so profound that we suggest that the carotid in the neck be re-explored even in those cases in which a complete hemiplegia has appeared abruptly. If a free flow of blood is obtained upon aspiration of the carotid distal to its point of occlusion then the ligature or band on it should be released. Even if small thrombi are still present in the internal carotid and are then washed on into the cerebral arteries with temporary worsening of the patient’s condition, the circulation to the hemisphere might be improved so markedly that we suggest the patient’s best chance for recovery may lie in this course of action.

**Diagnostic Applicability of the Method**

Although our method of measurement of pressures involves apparatus of sufficient complexity to preclude its routine clinical utilization, the data we have acquired appear to us to be of sufficient value to suggest frequent clinical application, and we are trying to set up a method for regular use. In addition to the applications already mentioned, there remains the possibility that, after an intracranial aneurysm has been treated by ipsilateral carotid ligation, it may prove in some types of case preferable later to ligate the contralateral carotid in the neck rather than carry out the more hazardous intracranial approach if arteriogram carried out from the second side reveals that the aneurysm still fills with contrast medium. This tactic has already been used by Ray in the treatment of inoperable arteriovenous fistulas of the cerebral hemispheres, and intra-arterial pressure measurement may prove useful in assessing its advisability in all types of aneurysms—in particular to determine whether the second common or internal carotid may be closed.

**Summary**

1. Results are given of recordings with low inertia of the intra-arterial pressure in the internal carotid artery in man before and after occlusion of various vessels.

2. In the distal portion of the internal carotid artery the systolic pressure
falls to 50 per cent, and the pulse pressure falls to 25 per cent of its original level when the vessel is occluded in the neck; hence this procedure probably has therapeutic value in the treatment of intracranial aneurysms on or near the internal carotid artery.

3. Pressures found in the internal carotid artery when the common carotid artery was occluded were compared with pressures in the same vessel when both common and external carotid vessels were occluded. The differences in pressure recorded indicated that significant retrograde flow from external to internal carotid artery usually did not occur when the common carotid artery only was occluded. In fact a flow in the reverse direction from internal to external carotid is equally likely in cases in which occlusion of the internal carotid in the neck is tolerated for 30 minutes without the appearance of neurological sequelae. Hence, in such cases, if elective ligation in the neck is decided upon, occlusion of the internal carotid rather than the common carotid is advised. Occlusion in continuity of the internal carotid has the advantage over that of the common carotid that the late fatal erosion of the vessel and rapid reestablishment of collateral circulation are less likely.

4. Occlusion of the internal jugular vein following occlusion of the internal carotid artery causes, during 30 minutes, no rise in pressure within the artery in cases without a carotid-cavernous fistula.

5. We suggest tentatively that the efficacy of the contralateral largely anterior communicating anastomotic supply as compared with the ipsilateral largely posterior communicating anastomosis may be checked by comparing the pressure there which ensues when the opposite carotid is effectively occluded in addition. If the pressure falls markedly when the carotid on the second side is occluded, this indicates the probability that the bulk of the anastomotic supply arrives by way of the anterior communicating artery. This may enable a better estimate to be made of the hazard of direct intracranial approach to an aneurysm of the supraclinoid portion of the internal carotid artery, since the posterior communicating artery may be unavoidably occluded during such an operation.

6. Difficulty was found in securing a consistent or even any fall in blood pressure distal to the point of occlusion of one internal carotid when the second side was compressed through the intact skin. The point at which application of pressure produced a maximal fall (and hence the point of occlusion of the carotid artery) was usually found only after a number of attempts. This demonstrates a gross unreliability of tests of cerebral anastomotic circulation pre-operatively and points to the need for such test by direct compression of the carotid artery in the open wound with the patient under local anesthesia.

7. In view of the marked fall in intra-arterial pressure which ensues upon internal carotid occlusion, it is advisable to close the vessel in such fashion as to permit removal of the occlusion at once if late signs of carotid ischemia develop slowly, and it is probably advisable to remove it even if such late
signs develop abruptly provided that the internal carotid at re-operation yields a free flow of blood upon aspiration distal to the occlusion.

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