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# Bilateral Internal Carotid Occlusion:

## A Clinical and Radiological Study

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SEVENTEEN cases of bilateral internal carotid occlusion have been encountered since 1959 in our investigation of cerebrovascular disease. The purpose of this paper is to ascertain whether the natural history of these cases provides information of use in the management of cases of unilateral stenotic or occlusive disease and also how closely the clinical course can be correlated with the radiological findings.

The treatment of atherosclerotic vascular disease has changed, and the surgeon now copes successfully with cases hitherto considered inoperable. Statistics are essential in appreciating the magnitude of the problem. In Canada and the United States the incidence of death due to a stroke is of the order of 80 per 100,000 population per year, which means that some 16,000 people per year in Canada die from this cause.[22] From cerebral thrombosis the initial mortality is 21%, and 50% of the remainder die within 4.1 years, compared to an 18% mortality in the general population of comparable age.[23] It is also recognized that 50% of those with cerebrovascular insufficiency have extracranial occlusive vascular disease[3] and that in up to 74% of these patients there are multiple lesions, with the commonest sites of stenotic lesions at the origins of the vertebral and internal carotid arteries.[1, 17] If one carotid sinus shows severe stenosis, there is only a 19% chance that the other carotid sinus will be normal.[25] Surgical repair of the stenotic extracranial lesion is now accomplished almost without incident. Restoration of flow in occlusive carotid disease is attained in 25% of cases when late operation (more than four days after the occlusion) is undertaken, with the percentage increasing to 66% when an early operation can be carried out.[13]

Bilateral internal carotid occlusion is not a common finding. Most cases do not come to angiography, so that there is a natural preselection of cases. In the investigation of 1500 patients with atherosclerotic disease, North *et al.*[20] encountered 13 cases with bilateral internal carotid occlusion, and five cases are described in the monograph by Yates and Hutchinson[31] which deals with 100 cases of cerebral infarction. The relatively high incidence in the latter series is because this was an autopsy series.

Clarke and Harrison[2] in 1956 reviewed 68 cases of bilateral internal carotid occlusion reported in the literature, dating back to the mid-sixteenth century. They added two cases of occlusive disease, but both were of unilateral carotid occlusion with a severe contralateral carotid stenosis. Fisher[6] reported 11 cases of bilateral carotid occlusion, six with bilateral neurological signs and coma. Four of these had a history of a hemiplegia years before. The other five cases all showed marked dementia. Fisher stated that he had not seen a case of asymptomatic bilateral carotid occlusion. Symptoms, however, can vary greatly in degree and severity. Groch, Hurwitz and McDowell[7] reported two patients in one of whom there was a minimal deficit whereas the other lapsed into coma after the second stroke and died 21 days later. The status of the collateral circulation was not stated. Mishkin[16] also reported two cases; both had a large patent vertebral artery providing a collateral blood supply. Whereas one had a complete stroke, the other showed only moderate weakness of the right arm and leg. Sutton and Davies[28] mention bilateral internal carotid occlusion as an unexpected finding in the investigation of cerebrovascular insufficiency. In the discussion of atherosclerotic vertebral basilar disease, Myer, Sheehan and Bauer[18] reported bilateral internal carotid occlusion, as well as tortuous compromised vertebral arteries, in a patient who was aphasic, hemiplegic, stuporous and blind. Gurdjian, Hardy and Lindner[8] reported nine cases briefly in discussing surgical considerations in carotid disease.

### CLINICAL MATERIAL

The clinical and radiological findings in this series of 17 cases with bilateral internal carotid occlusion are set forth in Table I.

The average age of the patients was 53 (42 to 71) and the sex incidence was 13 males to 4 females; this conforms to the 3:1 male pre-

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TABLE I.—CLINICAL AND RADIOLOGICAL FINDINGS IN 17 CASES

| *Patient No.* | *Sex and age* | *History of admission* | *Physical findings* | *EEG* | *Radiological findings* | *Course* |
|---|---|---|---|---|---|---|
| 1. L.D. | M 55 | Mar. '66—Right leg twitching (focal seizure) and transient dragging of right foot. Three weeks later lost consciousness and awoke with right hemiparesis, cleared in four days. No mental changes, alert. | 140/65 left arm. 80/60 right arm. L. carotid bruit. No neurological signs | Normal | Innominate and L. internal carotid occlusion. Stenosis L. external carotid, normal L. vertebral. R. subclavian steal. R. external carotid steal. Collateral to internal carotids via posterior communicatings from large L. vertebral artery. | L. carotid explored with no retrograde flow. Employed as butcher, five-day week. |
| 2. M.R. | F 55 | June '63—Right arm and leg weakness for two days. May '64—Marked aphasia for 24 hours, improving over two months. Mentally unimpaired. | 160/90. L. carotid bruit. No pulses right leg. Slight dysarthria. Minimal right facial weakness | Left side irregularity | R. and L. common carotid artery occlusion. Stenosis both vertebral artery origins. R. external carotid steal. Collaterals to internal carotids via posterior communicating arteries | No operation. At three years doing well and no new symptoms. |
| 3. W.H. | M 62 | Sept. '66—Sudden confusion, blurred vision, speech slurred and unable to write. Gradual improvement. Moderate mental impairment with short attention span, poor insight, flat emotionally. | 120/80 right arm. 160/85 left arm. L. carotid bruit. R. carotid not palpable. Mild right hyperreflexia and weakness. Right homonymous hemianopsia | Mild diffuse arrhythmia | Innominate and L. internal carotid occlusion. Stenosis R. vertebral and L. external carotid origins. R. subclavian steal. R. internal carotid fills via L. posterior communicating, L. internal carotid via L. ophthalmic. Rete mirabile between occipital of external carotid and L. leptomeningeal vessels and to L. internal carotid | Both arteries explored with retrograde flow. Worse postop. and then improved. Better intellectually than before operation. Not working |
| 4. G.D. | M 59 | May '65—Gradual personality change and poor memory. July '65—sudden left-sided weakness and confusion. Disorientated, unable to calculate, mental impairment marked. | 200/100. Normal pulses. No bruit. Slight left arm spasticity and weakness. Weakness of left leg | Right dysrhythmia | Bilateral internal carotid occlusion. Stenosis R. vertebral origin. Collateral to L. internal carotid via L. ophthalmic; to R. internal carotid via R. posterior communicating. Collateral between R. pial posterior cerebral and pial middle and anterior cerebrals | Bilateral exploration with no effective removal of clot. In nursing home owing to mental impairment, hemiparetic |
| 5. J.M. | M 47 | First symptom, sudden loss of consciousness. Died in 24 hours | 140/90. Spastic quadriplegia, comatose | — | No angiography done | Postmortem examination also disclosed coronary occlusion |
| 6. R.B. | M 48 | 1960—Numbness of right leg, recovered in one hour. One month later period of confusion and admitted to psychiatric hospital for one week. On admission, alert and co-operative. Normal intelligence | 170/90. Normal carotid pulses. No neurological signs | Arrhythmia in temporal area | Bilateral internal carotid occlusion. Stenosis in L. vertebral origin. Collaterals to internal carotids via posterior communicatings bilaterally | Bilateral exploration, no retrograde flow. Mild intellectual impairment. Working as carpenter |
| 7. F.J. | M 42 | Intermittent R. frontal headache for a few days. Slurred speech followed two hours later by left-sided weakness. Dysphagia. One-half hour loss of consciousness at onset. Confused but orientated | 150/90. Left paresis with cortical sensory loss. Left hemianopsia | — | Bilateral internal carotid occlusion. Collaterals to internal carotids via posterior communicatings bilaterally | No operation. Personality change and nursing care problem. Belligerent, coarse and boisterous |
| 8. L.T. | M 44 | Headache one year. Poor memory for two months. Two weeks with weak right foot and paresthesia of left hand. Orientated but mentally impaired | 140/90. No bruits. Pulses normal. Paralysis right foot | Asymmetry with slower activity from right hemisphere | Bilateral internal carotid occlusion. L. middle cerebral occlusion. Rete mirabile in L. frontal area. Collaterals to internal carotids via ophthalmics bilaterally | No operation. Marked right hemiplegia, coma and death |
| 9. H.P. | M 48 | June '63—Visual disturbance. Difficulty reading. Then left Jacksonian seizures. Emotionally unpredictable. Aug. '64—Sudden loss of consciousness and left hemiparesis | 170/120. Bilateral carotid bruit. Pulses equal. No pulses below femorals. Left hemiparesis. Left lower quadrant-anopsia | Disorganized rhythm, worse in temporal area | Bilateral internal carotid occlusion. Occlusion R. vertebral origin, stenosis of L. vertebral and external carotid origins. Collaterals to internal carotids via ophthalmics bilaterally | Operation unsuccessful with drop in B.P., coma and death |
| 10. W.S. | M 48 | Mar. '63—Numbness of left shoulder and arm, impaired position sense in left hand. Apr. '63—Increased left hand weakness. Mar. '63—Confusion, left hemiparesis. Grand mal seizure after admission. Disorientated to time and place, restless, confused, mentally impaired | 130/80. Occipital and orbital bruits. Spastic left arm. Brisk reflexes. Left facial weakness | Diffuse disorganization with no lateralization | Bilateral internal carotid occlusion. Stenosis R. external carotid origin. Collaterals to internal carotids via ophthalmics bilaterally | No operation. Demented and admitted to mental hopsital |
| 11. A.B. | F 61 | Aug. '65—Left stroke, sudden onset. Jan. '66—Sudden right-sided weakness with numbness. Incontinent. Mild dysphasia. Repetitive. Forgetful. Slow | 105/90. No pulse below femorals. R. carotid bruit. Normal carotid pulses. Mask-like facies. Brisk reflexes, especially left side | Normal | Bilateral internal carotid occlusion. Stenosis of external carotid origins bilaterally. Stenosis of R. subclavian and R. common carotid origins. Collaterals to internal carotids via ophthalmics bilaterally | Bilateral exploration with no retrograde flow. Doing own housework |

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TABLE I.—CLINICAL AND RADIOLOGICAL FINDINGS IN 17 CASES—*Continued*

| *Patient No.* | *Sex and age* | *History of admission* | *Physical findings* | *EEG* | *Radiological findings* | *Course* |
|---|---|---|---|---|---|---|
| 12. L.H. | F 57 | 1960—Intermittent weakness and clumsiness right arm and leg. 1961—Some recovery but memory defect with three episodes of loss of consciousness. Intellectual decline | 200/100. No bruits. Carotid pulses faint. Right homonymous hemianopsia. Left spastic hemiplegia | Silent area in left frontal and temporal areas | Bilateral internal carotid occlusion. Stenosis L. external carotid origin. Collaterals to internal carotids via ophthalmics bilaterally | No operation. Demented, hemiplegic |
| 13. G.L. | M 42 | Transient right arm weakness. Two weeks later, right hemiplegia with aphasia. Drowsy and confused | 130/80. Normal pulses. No bruits | — | Bilateral internal carotid occlusion. Bilateral external carotid origin stenosis. Stenosis of R. vertebral origin. Aberrant R. subclavian, collaterals to internal carotids via ophthalmics bilaterally | No operation. Ten days after admission, into coma with quadriplegia and died |
| 14. A.W. | M 64 | Slurred speech. Left-sided weakness with numbness and tingling left hand and face. Alert and co-operative | 140/80. Normal pulses. R. carotid and bilateral femoral bruits. No pulses below femorals. Left spastic hemiparesis | — | Bilateral internal carotid occlusion. L. external carotid steal. Collaterals to L. internal carotid via L. ophthalmic, to R. internal carotid via posterior communicating | Operation—no retrograde flow. Mild disability but not working |
| 15. T.M. | F 71 | Left arm weak—three years previously. Left-sided stroke two weeks earlier with confusion and dementia. Sudden loss of consciousness 12 hours before admission | 160/100. Normal pulses. No bruits. Right spastic hemiplegia. Comatose | Silent on left side with slow activity on right side | Bilateral internal carotid occlusion. Collaterals to internal carotids bilaterally via ophthalmics | No operation. Died 14 days after admission. |
| 16. M.L. | M 57 | 1959—Sudden loss of consciousness, incontinent | 120/80. Bruits over both carotids | Normal | Bilateral internal carotid occlusion. Collaterals to L. carotid via ophthalmic | No retrograde flow established. To mental hospital where he died five years later after leg amputation |
| 17. A.P. | M 50 | Intermittent claudication two years. Increasing weakness left arm and leg for eight weeks. Slow improvement then change in mental state with peculiar behaviour, difficulty in use of common objects. Withdrawn, confused, restless | 160/85. Weak right arm and leg. Right homonymous hemianopsia. Right sensory inattention | Mild diffuse irregularity, worse on left side | Bilateral internal carotid occlusion. Collaterals to internal carotids via ophthalmics bilaterally | No retrograde flow established at operation |

ponderance in cerebrovascular disease.[28] The earlier age of onset in the male patient is also seen in our group, with the average male age 47 and the average female age 61 at the time of diagnosis. Only six were hypertensive.

In two patients a one-sided internal carotid occlusion was associated with an innominate artery obstruction on the opposite side; in one there was a bilateral common carotid obstruction. The other 14 cases were all of bilateral internal carotid occlusion occurring at the origin of the vessel. Nine of the 17 never did have a history or physical signs to indicate bilateral disease. Of the other eight, two had a history of a stroke on the opposite side previous to their present episode; the other six had recognizable evidence of bilateral disease, and even in these the clinical evidence was predominantly unilateral in three.

The two cases with a clear-cut history of a previous stroke on the opposite side are of interest, for they allow dating of each separate clot and may give guidance in prognosis. Case 15 might be used as an argument in favour of the vigorous pursuit of initial symptoms. This woman was 71 years of age at her death; in 1961 she had a transient left hemiparesis, from which she recovered in a few days; in 1964 she presented with a sudden right hemiplegia, became confused and stuporous, developed bulbar signs and a spastic quadriplegia, and died within two weeks.

Case 11 illustrates how difficult it may be in an individual case to predict the outcome of a second carotid obstruction, even when other vessels are diseased. This 62-year-old woman had a left hemiplegia in 1965 and in a few weeks had made a good recovery. In 1966 she presented with sudden right-sided weakness, numbness and dysphasia, and was forgetful and incontinent. She had a shuffling gait and fixation of facies. Bilateral carotid exploration was undertaken without restoration of flow. She recovered, however, and one year later had resumed her normal domestic duties. Her angiograms reveal a right subclavian stenosis on the right retrograde brachial injection (Fig. 1). Bilateral carotid injections demonstrate stenotic lesions at the origin of both external carotids—vessels which are the prime source of collaterals to her intracranial vessels via the ophthalmic artery bilaterally (Figs. 2a, 2b, 3a and 3b). Her collaterals cannot be described as abundant, but in spite of this, and the bilateral carotid occlusion and the multiple stenotic lesions, she is able to carry out her normal domestic activities. Her reserve of circulation potential must be extremely low, and any operative procedure that could have opened up a carotid would have been advantageous.

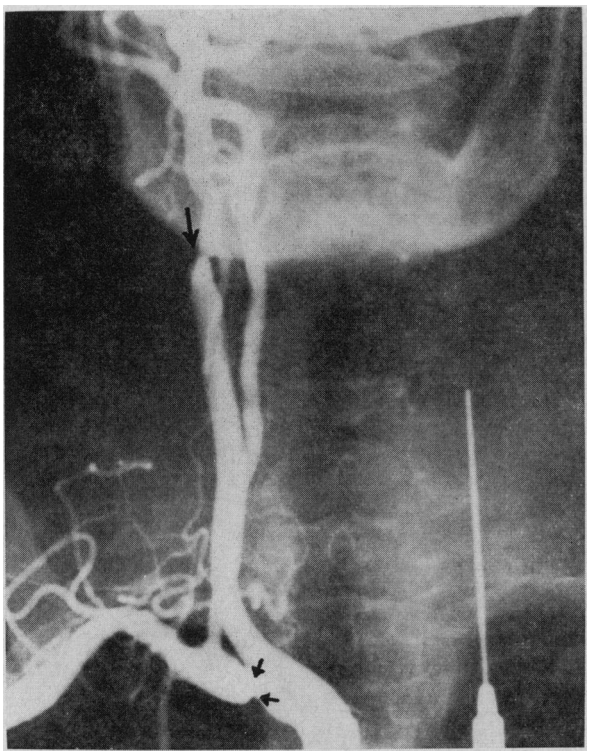

Fig. 1.—Case 11. Right retrograde brachial injection showing a stenosis at the origin of the right subclavian artery (arrows) and occlusion of right internal carotid at its origin (upper arrow).

The mode of onset was not generally distinguishable from that of unilateral occlusion. Episodic progression was the most common mode of presentation and occurred in nine cases. Slow insidious progression occurred in two. An abrupt onset was seen six times, four times with a calamitous result and twice with development of a less complete deficit.

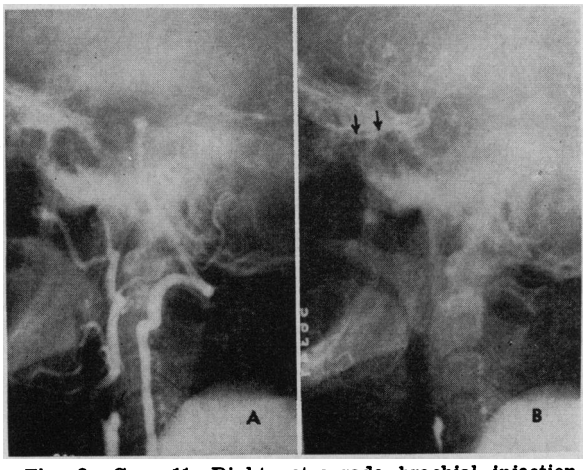


Fig. 2.—Case 11. Right retrograde brachial injection, lateral views. (a) Right internal carotid occlusion (arrow) with severe right external carotid stenosis. Large right vertebral artery not supplying collaterals via posterior communicating. (b) Later film shows collaterals from internal maxillary branch of external carotid to ophthalmic branch of internal carotid (arrows) with subsequent filling of distal right internal carotid and its anterior and middle branches.

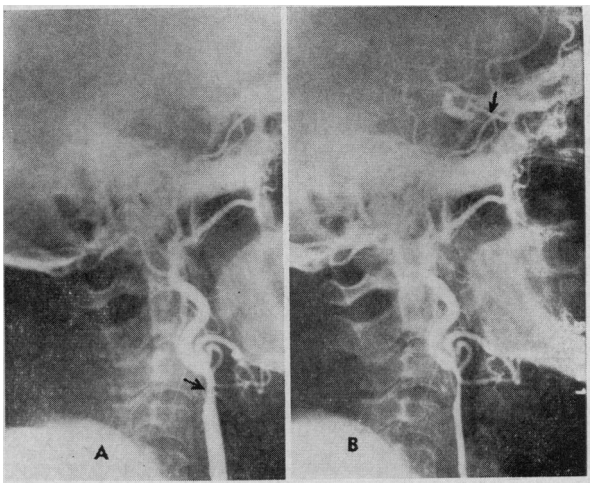


Fig. 3.—Case 11. Percutaneous left carotid injection: (a) Left internal carotid occlusion with stenosis of left external carotid origin (arrow). (b) Collaterals to left internal carotid via its ophthalmic branch (arrow) from internal maxillary of left external carotid.

The duration of the warning symptoms varied, as it does in unilateral cases, from days to weeks or months. However, one can see from reviewing Table I that eight of these patients had months of warning and that tragedy might have been averted had they been brought earlier to neurological attention. Their course was punctuated by the usual wide variety of initial symptoms, including headache, localized transient weakness and numbness, but also included two unusual symptoms—focal seizures and intellectual decline.

As a matter of interest, the incidence of seizures was higher in this small series than is generally seen in unilateral carotid obstruction. Three patients (Cases 1, 9 and 10) had focal spreading seizures, one had a major seizure before the development of the major neurological disability and one as a sequel to it. Perhaps the relatively hypoxic state of these brains resulted in this higher seizure incidence.

Impairment of intellectual function of sufficient severity to justify the term dementia occurred in 10 of these cases, while two others had definite intellectual decline of lesser degree. In four this was the presenting symptom and only twice did it improve significantly.

The outcome for the patients in the series was poor. Five patients died and another died five years later in a mental hospital; six had major residual disability; only four were able to return to useful employment, and in these return of function was almost entirely spontaneous; one had minor disability but is not working. Effective operative treatment, with restoration of flow, was accomplished only once in the nine patients who came to operation. These results are poor and considerably worse than the overall

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results from surgery of unilateral occlusion already reported from this centre,[13] where, if the operation was undertaken within three days, restoration of flow was possible in 66% of cases. One may be misled here because of the smallness of the series, or it may be that the occlusions were of such long standing that restorative surgery had a relatively small chance of success. In one instance the operation probably contributed to the patient's death (Case 9) and twice the preoperative angiography added materially to the neurological deficit. These two latter cases occurred early in the series, and more sophisticated angiographic methods might well have obviated these complications.[13]

In contrast, in a group of patients with unilateral occlusion together with a severe stenotic lesion of the opposite carotid, the outcome and clinical course were relatively good and only two of 10 such patients showed intellectual impairment. In six out of eight patients surgical correction of the stenotic lesion was carried out successfully.[12]

Electroencephalography was performed in 13 of the 17 patients; the tracings were considered normal in three and showed varying degrees of abnormality, such as silent areas of diffuse disorganization, in the other 10.

### RADIOLOGICAL FINDINGS

Ten years ago, restriction of the use of angiography was advised by some authors in the belief that the hazards of the procedure were too great in the presence of atherosclerotic disease.[27] At present, however, it is widely agreed that with the refinements in angiographic techniques and the use of sensible precautions, there is very little or no increased risk to the atherosclerotic patient and that angiography is essential both for the diagnosis and the possible correction of the atherosclerotic lesion.

The more recent of our 17 cases were investigated by either three- or four-vessel angiography, the method being dependent on the presenting signs and symptoms, the state of peripheral vessels, the findings in the initial angiogram and even the the anomalies found; e.g. in Case 13 an aberrant right subclavian artery limited the usefulness of a retrograde brachial procedure (Figs. 4 and 5). Selective angiography from the aortic arch was rarely carried out, owing to the opinion that a catheter may dislodge emboli and that the prolongation of the procedure with use of increased amounts of contrast medium are factors in producing increased patient morbidity.[13, 16] In most cases a percutaneous right brachial puncture with a retrograde injection

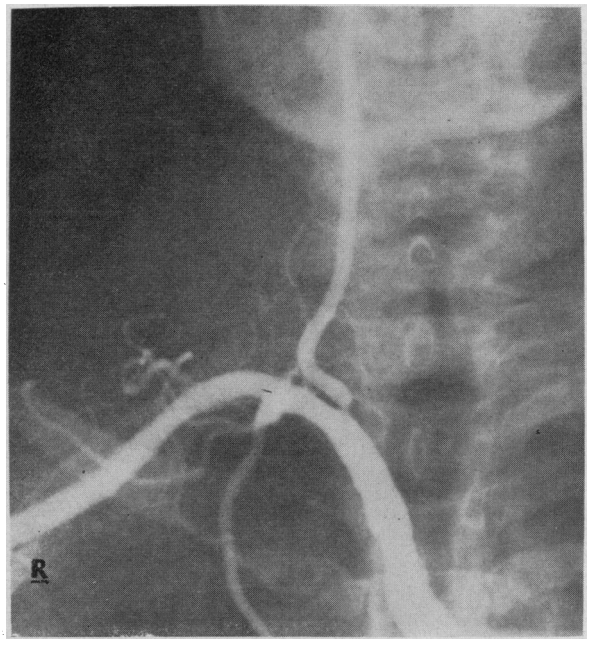


Fig. 4.—Case 13. Right retrograde brachial injection demonstrating an aberrant right subclavian artery with a stenotic lesion at the origin of the right vertebral artery.

using a pump injector allowed visualization of the right carotid-vertebral-basilar system. In addition, a percutaneous left carotid injection was done using a Sheldon cannula.[26] This is a cannula with a trocar and a 15-cm. flexible guidewire, which allows the cannula to be threaded up the vessel and markedly decreases the incidence of subintimal injections and damage. In many cases the left vertebral artery was not visualized, as operation on a stenotic vertebral lesion was not contemplated in the presence of bilateral carotid occlusion.

With this relatively complete angiography, the patterns of collateral circulation can be traced. Symptomatology is a function of the collaterals as well as of the occlusion. Pre-existing collaterals open in response to occlusion via a

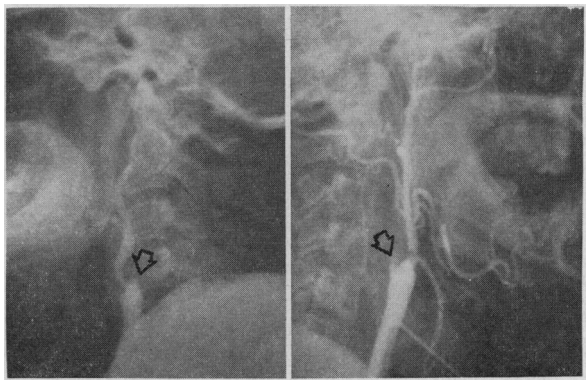

Fig. 5.—Case 13. Percutaneous bilateral carotid injections show bilateral internal carotid occlusions (arrows), as well as stenosis of both external carotid origins.

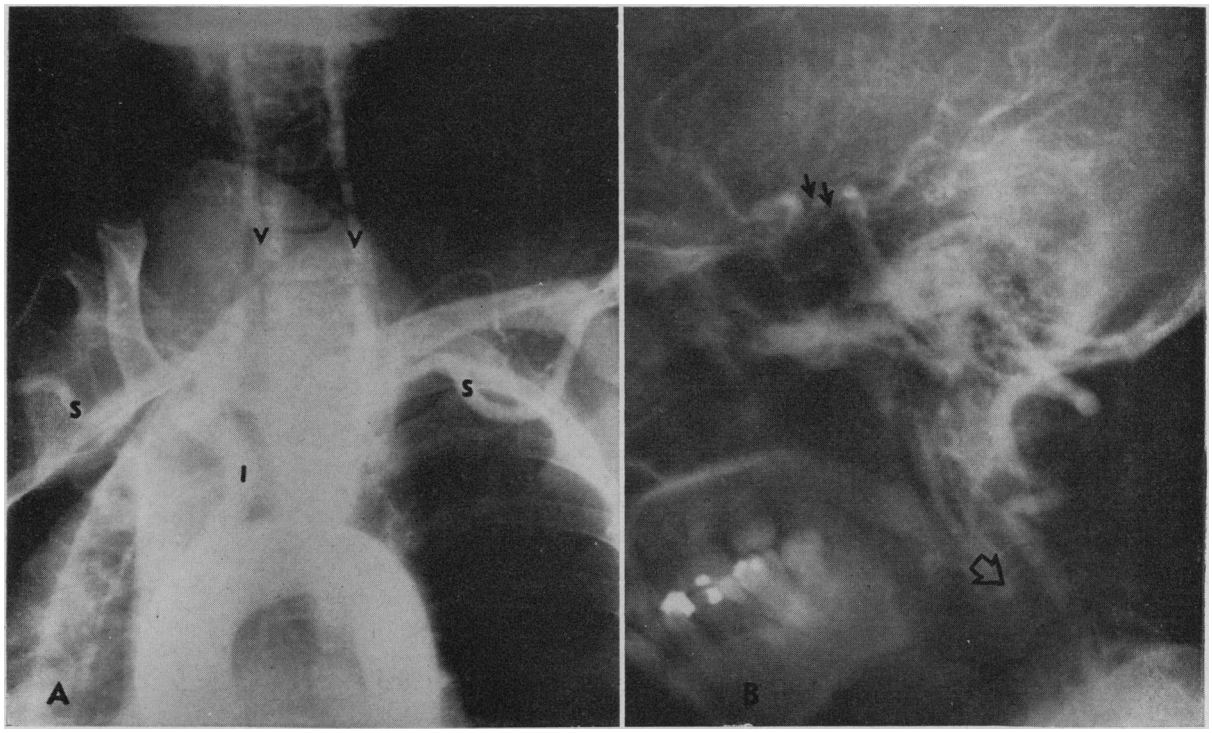


Fig. 6.—Case 2. (a) An aortic arch injection demonstrates bilateral common carotid occlusions. Vertebral arteries (V) bilaterally are large, subclavian (S) and the innominate (I) arteries are normal. (b) Catheterization of right vertebral artery shows collaterals to middle and anterior cerebral arteries via the posterior communicating arteries (closed arrows). The external carotid artery fills in a retrograde manner down to its origin (open arrow), stealing blood from branches of the right vertebral artery.

vasomotor reflex. Utilizing Poiseuille's law in respect of the volumetric rate of flow of liquids, Potter[21] has shown that a relatively small dilatation of one vessel will compensate for a constriction or occlusion in another. Thus an occlusion of one carotid can occur with no symptoms if there is a healthy circulation. In other cases, obstructive lesions are found on the asymptomatic side.[28] The correlation of cerebral symptoms and the demonstrable disease is dependent on the state of all the cerebral vessels throughout their intracranial and extracranial course. Gurdjian, Hardy and Lindner[8] stated that carotid artery occlusion in the neck does not cause a stroke unless collaterals are inadequate. Fields, Edwards and Crawford[5] reviewed 16 cases of bilateral internal carotid occlusion, including the cases reported by North *et al.*[20] Their conclusions were that those with adequate collaterals had a minimal deficit, and of six patients in this category, four were able to return to work. In those with impaired collaterals, a severe deficit was found with language and motor impairment and severe neurological signs. Cronqvist,[4] however, reported a correlation between symptoms and affected neck vessels in only two-thirds of the cases of atherosclerotic disease.

Embolization may be a contributing factor in the lack of correlation between the occlusion and the degree of collateral circulation demonstrated. The emboli may be dislodged from the distal surface of an occlusion.

In our cases, good and extensive collaterals appeared to be helpful in some but ineffective in preventing a major disability in others. Collaterals represent only the vascular by-passes available and may not accurately indicate the cerebral blood flow, which may well be a factor of greater importance. Six patients had moderate or no residual disability. In all of these there was evidence of good collateral flow from the vertebral arteries and/or from the external carotid-ophthalmic-internal carotid route, or less commonly from other channels. Case 2 shows collaterals via the posterior communicating to the internal carotid in a patient who did well (Fig. 6a and b).

Case 6 shows an identical picture, with collaterals from the posterior communicating to the internal carotid arteries. This patient had minor intellectual impairment when the condition was diagnosed in 1960 at age 48; he has shown slight deterioration but was well enough to be

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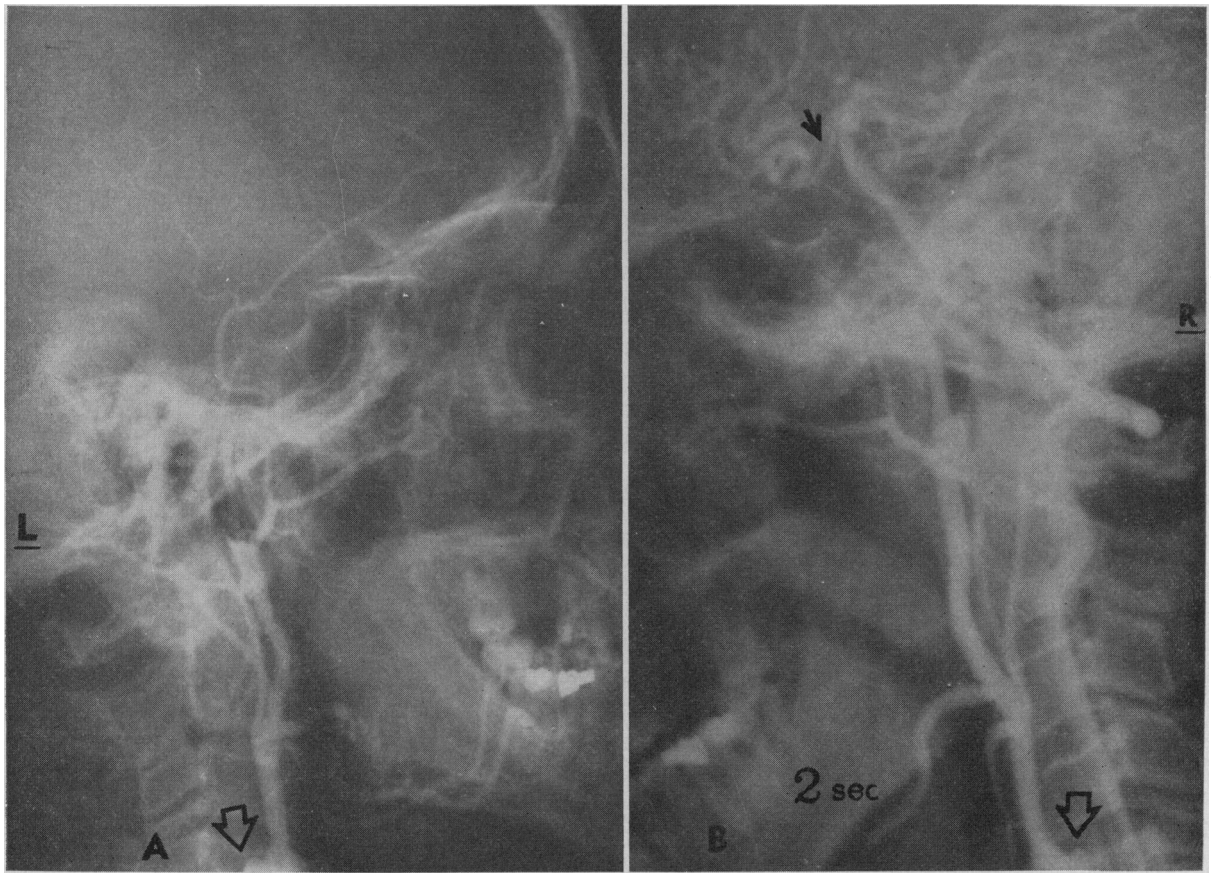


Fig. 7.—Case 6. (a) Percutaneous left carotid injection with occlusion of left internal carotid origin (arrow). (b) Percutaneous right retrograde brachial injection with occlusion of right internal carotid origin (open arrow) with collaterals from vertebral-basilar via posterior communicating (closed arrow) to anterior and middle cerebral arteries.

a candidate for the provincial legislature in 1967 (Fig. 7a and b).

In the five patients who died, the alternate circulation available was only slightly less impressive. They made use of external carotid-ophthalmic channels and some of this small group also had a good vertebral-posterior communicating-internal carotid visualization (Fig. 8). In the group of six, intermediate between those who died and those without disability at all, there were excellent vertebral collaterals in three.

Many factors are operative and of importance in the outcome of any individual case. These include the number of intracerebral and extracerebral arterial occlusions, the degree and capacity of collateral flow, the restrictions to flow via stenotic lesions affecting major alternate channels, and, finally, the steal mechanisms that may, through other collaterals, actually deprive the brain of part of its blood supply. Table II shows the incidence of associated stenotic lesions, as well as the three subclavian and two external carotid steals. Examples of these steal mechanisms are illustrated in Figs. 9a and b

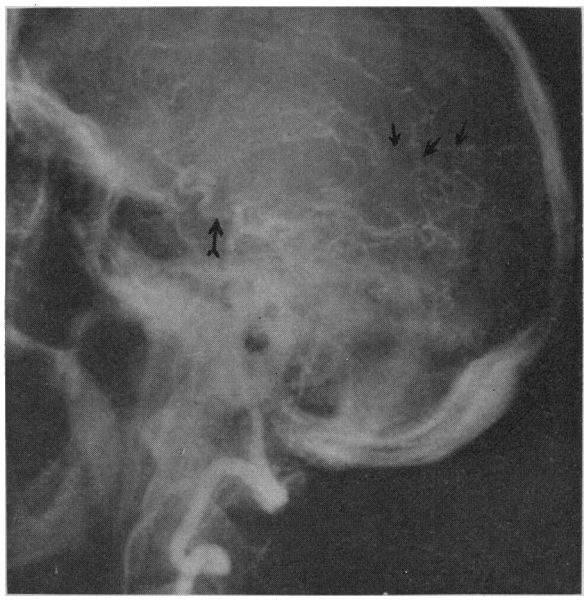

Fig. 8.—Case 4. Collaterals from vertebral-basilar system via posterior communicating (barbed arrow) to anterior and middle cerebral arteries, and also collaterals between pial branches of posterior cerebral artery and pial branches of anterior and middle cerebral arteries (arrows), This patient was hemiparetic and mentally impaired in spite of good collateral channels.

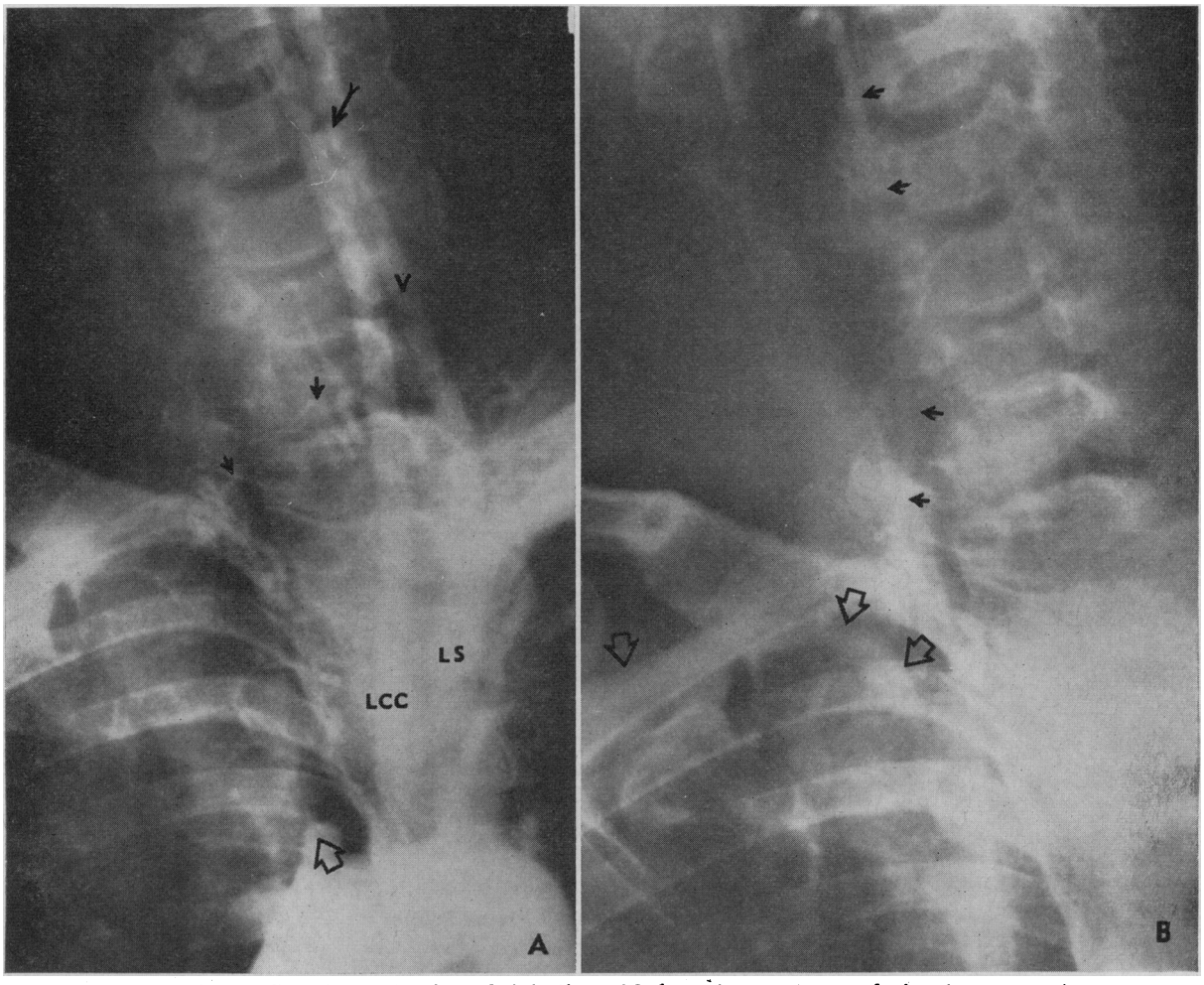


Fig. 9.—Case 1. (a) Aortic arch injection with innominate artery occlusion (open arrow), left internal carotid occlusion (barbed arrow), normal left vertebral (V), left common carotid (LCC) and left subclavian (LS) arteries. Small arrows point to thyrocervical arteries contributing to right subclavian steal. (b) Retrograde filling of right vertebral artery (small arrows) with subsequent opacification of right subclavian artery (open arrows).

and 6b (Cases 1 and 2). A detailed description of this rarely described external carotid steal will be the subject of a subsequent communication. One must consider the blood supply of the brain to be of prime importance and these steal mechanisms are therefore a misuse of collateral flow. A review of this small group, however, showed the situation to be as frequent in those who fared well as in those who fared poorly.

TABLE II.—BILATERAL INTERNAL CAROTID OCCLUSION: COMPLICATING SITUATIONS

| | |
|---|---|
| Subclavian steal | 3 |
| Subclavian stenosis without steal | 1 |
| External carotid steal | 2 |
| External carotid stenosis | 7 (2 bilateral) |
| Vertebral artery stenosis | 3 (1 bilateral) |
| Hypoplastic vertebral arteries | 2 |

This table demonstrates the multiplicity of associated lesions but indicates a low incidence of vertebral artery disease, since in the earlier cases of this series the angiography was not as complete as in later cases.

Two cases showed collateral flow through a rete mirabile, i.e., communications between perforating or dural meningeal branches of the external carotid and the internal carotid vessels, either in the cavernous sinus area or at leptomeningeal branches. In one (Case 8) this was seen to occur between frontal meningeal and leptomeningeal arteries (Fig. 10), in the other (Case 3) at two sites—the first, between perforating occipital branches of the external carotid and leptomeningeal branches of the middle cerebral artery, and the second, between the internal carotid and the intracavernous portion of the internal carotid. This latter patient, a general practitioner, had a moderate degree of intellectual impairment. The findings in his case were an innominate and a left internal carotid occlusion, a right subclavian and a right external carotid steal with collaterals present from the left vertebral to the right internal carotid via the posterior communicating, and from the left

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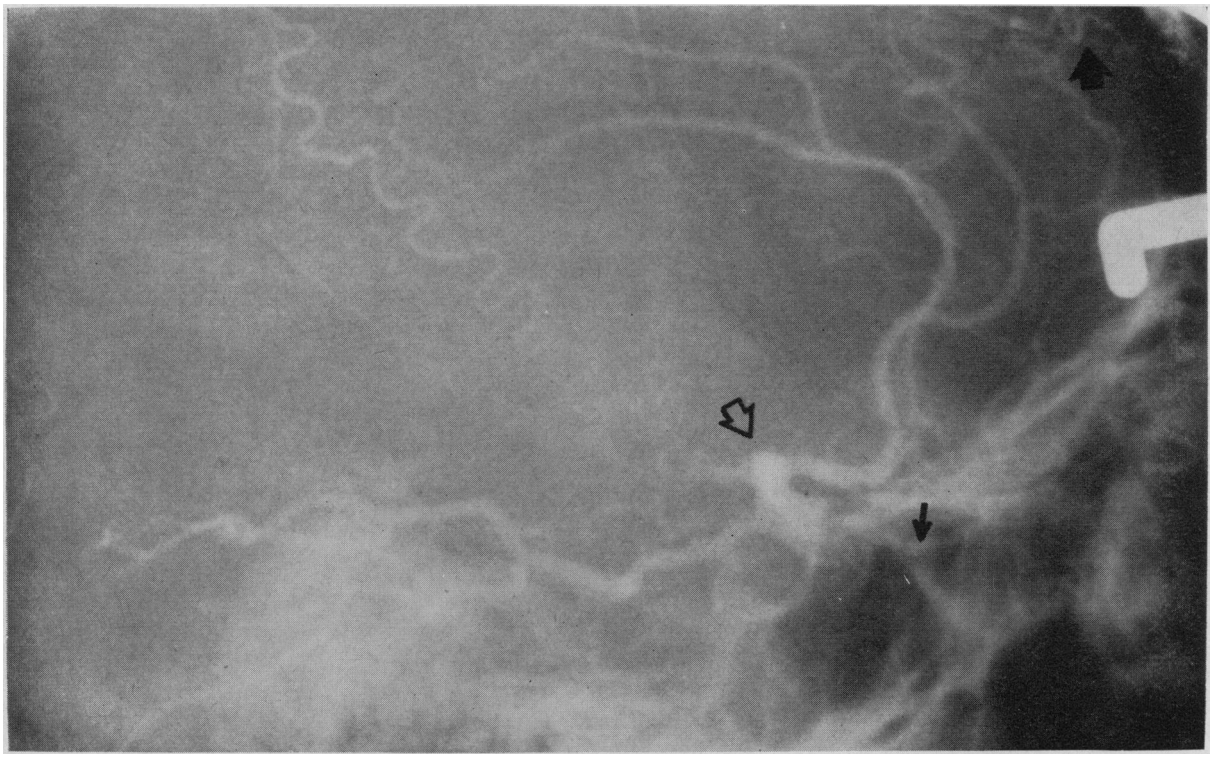

Fig. 10.—Case 8. The left internal carotid and its anterior and posterior branches are supplied by collaterals from its ophthalmic branch (small closed arrow) from external carotid branches. There is also collateral circulation via a rete mirabile between anterior cerebral leptomeningeal branches and perforating external carotid vessels (large closed arrow). An occlusion of the left middle cerebral trunk is also demonstrated (open arrow).

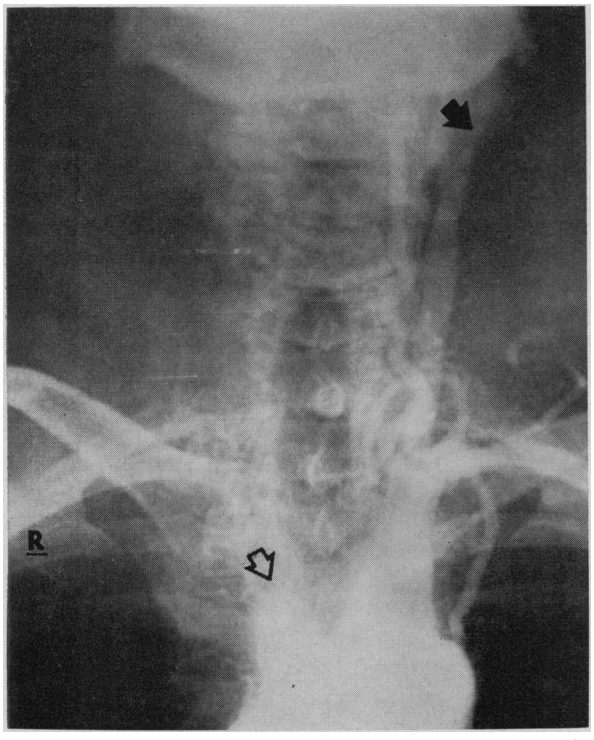


Fig. 11.—Case 3. Aortic arch injection showing innominate artery occlusion (open arrow) and left internal carotid occlusion (closed arrow). The very large left vertebral artery present is the only channel of blood supply to the brain. Enlarged thyrocervical vessels present aid in supplying the right subclavian artery, which also derives some of its blood supply from the right vertebral artery.

external carotid to the internal via the ophthalmic vessels, as well as the rete mirabile described above. The advantages of his extensive collateral circulation are being offset to some degree by the steal mechanisms which supply blood to his right arm and face (Figs. 11, 12a and 12b).

The rete mirabile has been described with occlusive disease involving the internal carotid artery;[9-11, 24, 29, 30] rarely it may be present as a congenital lesion.[15] The rete mirabile is seen in lower mammals such as the cat, sheep, goat and pig in which there is no patent internal carotid at the base of the brain but rather a primitive network of vessels supplying the circle of Willis from the branches of the external carotid. Recently, what appears to be a congenital rete mirabile has been reported in Japanese infants and adolescents, with one series reviewing 117 cases.[14, 19] The findings are of bilateral internal carotid occlusion at the carotid siphon with hemangiomatous vascular networks at the base of the brain which are bilateral and much more extensive than those seen in cases of occlusive origin. It is of interest that the two unilateral cases reported by Leeds and Abbott[11] were both in infants of Japanese descent.

From the review of our 17 cases, only two firm conclusions can be drawn: (1) If a patient

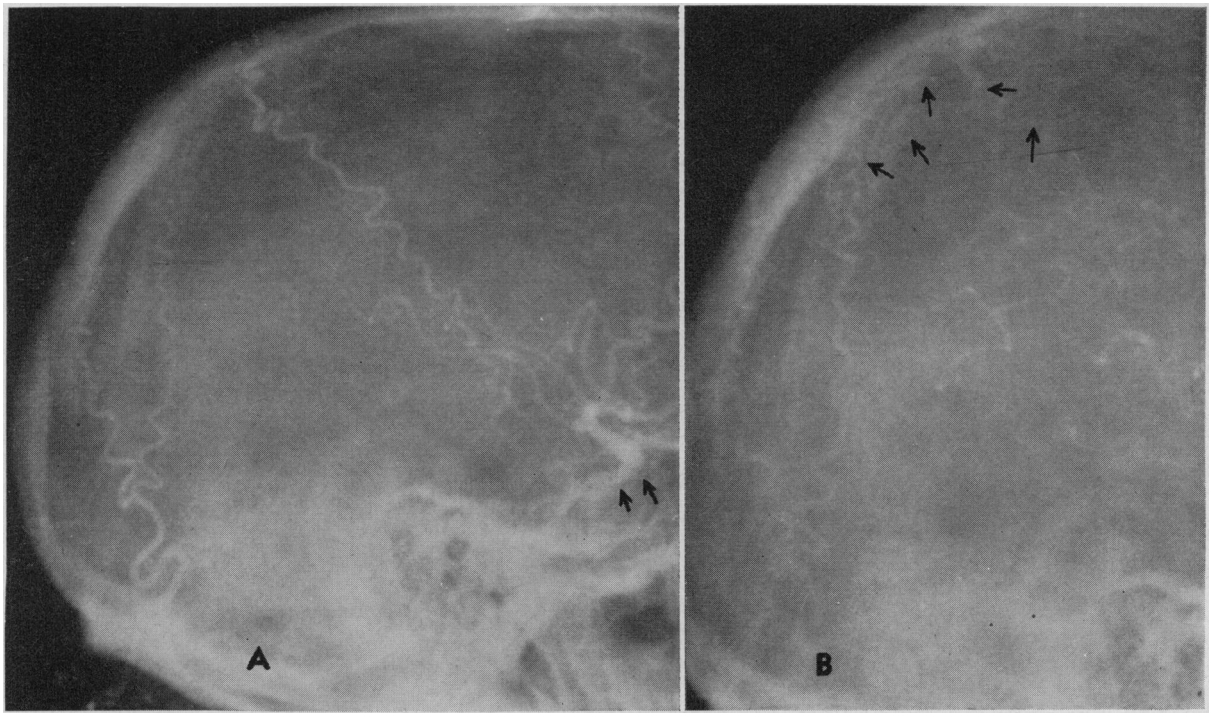


Fig. 12.—Case 3. (a) The left internal carotid artery obtains collaterals from multiple external carotid branches (arrows) in its intracavernous portion—the rete mirabile—as well as through the ophthalmic artery from left external carotid branches. (b) Perforating external carotid branches of the occipital artery are seen to form collaterals with leptomeningeal branches in the posterior parietal area (arrows). The anteroposterior view (not shown) shows that this occurs in the right parietal area, as well as in the left.

has no disability or only a slight one after losing normal circulation through both major carotid channels, it is probable that good alternate channels can be angiographically visualized. (2) Despite excellent collateral channels angiographically visualized, a major clinical disability may yet very well be apparent. This latter conclusion would reflect limitations in the significance of angiographic visualization of intracerebral vascular disease affecting smaller vessels, which undoubtedly is an important factor. It also may be that permanent brain damage may occur before the collaterals open and are effectively used.

***Summary*** A review of 17 patients with bilateral internal carotid obstruction is presented. A significant number had no history or findings to indicate bilateral disease. Most of the patients had lesions in other intracranial vessels, and steal mechanisms were operative in four. Collateral circulation of varying pattern and degree was shown in all patients but there was no exact correlation between the degree of disability and the radiological findings. Prognosis was poor and only four were able to return to useful employment.

In view of the poor prognosis for the patient with bilateral occlusive lesions, early diagnosis and surgical correction of unilateral lesions are indicated.

***Résumé*** Les auteurs présentent une revue de 17 malades souffrant d'occlusion bilatérale de la carotide. Chez un nombre assez important de ces malades, il n'y avait ni antecedent ni signe actuel indiquant une atteinte bilatérale. La majorité de ces malades présentaient des lésions dans d'autres vaisseaux intracrâniens et chez quatre d'entre eux des mécanismes "de suppléance" étaient fonctionnels. Chez tous les malades, on notait une circulation collatérale dont la forme et le degré étaient variables, mais il n'y avait pas de corrélation précise entre le degré d'invalidité et les constatations radiologiques. Le pronostic fut sombre et quatre malades seulement ont pu reprendre un travail utile.

Etant donné le mauvais pronostic chez les malades ayant des lésions bilatérales, il est indiqué de poser un diagnostic précoce et d'opérer les lésions unilatérales.

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# Influence of Modern Radiological Techniques on Clinical Staging of Malignant Lymphomas

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RADIOGRAPHIC procedures now form part of the routine pre-management investigation of patients suffering from histologically proved malignant lymphoma. The importance of accurate pre-treatment staging according to the detected extent of disease, with respect to planning of optimal therapy and accurate prognosis, is generally accepted.[14, 15] Current figures for long-term survival suggest that failures of treatment in previous years may have been due to the presence and progression of occult disease which can now be revealed by modern investigative methods.[16] This communication is an account of our experience at The Ontario Cancer Institute in the radiographic investigation of 392 patients suffering from malignant lymphomas. The findings are interpreted in the light of more recent pathological concepts.[12] Their influence with respect to clinical staging and the natural history of malignant lymphomas is discussed.

In the widely adopted system of pre-management assessment, recommended by an international committee on the staging of Hodgkin's disease,[18] the diaphragm is an important anatomical line of demarcation for lymphomatous infiltration involving lymphatic structures. Patients in clinical Stages I and II are those in whom the disease process involves lymph nodes on only one side of the diaphragm, and should disease be detected on the other side as well, they are advanced to Stage III. If structures outwith the lympho-reticular system are affected, the patient is placed in Stage IV. Similar concepts are applied to appraisal of lymphosarcomas and reticulum cell sarcomas. The relevant radiographic procedures are illustrated in Fig. 1. Lymphography has proved the most valuable investigation for detecting occult disease in pelvic and retroperitoneal lymph nodes. By outlining soft tissue abnormalities above the para-aortic nodes but still below the diaphragm, inferior vena cavography has proved a useful

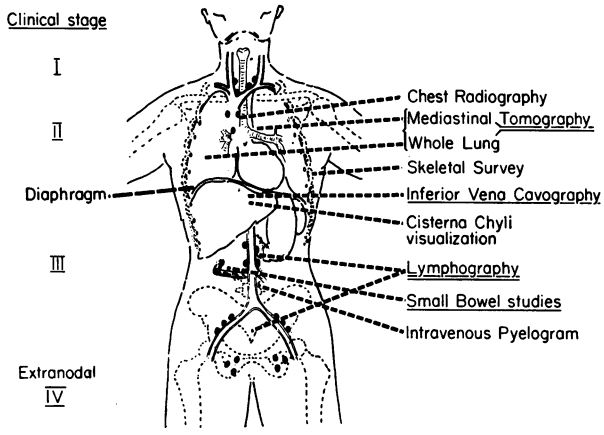


Fig. 1.—Radiographic investigations in lymphoma.

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