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Syndrome of Carotid Artery Insufficiency
Early Clinical Recognition and Therapy

By Lamar E. Crevasse, M.D., R. Bruce Logue, M.D., and J. Willis Hurst, M.D.

DR. LOGUE: Carotid artery insufficiency is a relatively common but frequently unrecognized syndrome. Fisher in 432 consecutive autopsies in which the brain and carotid arteries were studied found 28 cases of complete occlusion of one or both carotid arteries. Thirteen cases showed very severe narrowing of the arterial lumina, making a total of 9.5 per cent with advanced carotid disease. The classic concept of carotid artery thrombosis, namely monocular blindness and contralateral hemiplegia, is the exception rather than the rule, since visual disturbances occur in only 15 per cent. Furthermore, about 15 per cent of patients with carotid artery thrombosis may have no symptoms. Like atherosclerotic occlusions in other vessels, occlusion of the internal carotid artery is more common in the male over 40, although the age group varies from 13 to 85 years. Occlusions in the younger age group are not infrequent. Any disease process producing occlusive vascular disease may be implicated. By far the most frequent offender is atherosclerosis with a normal blood pressure. Hypertensive persons for some unknown reason are less frequently affected by this syndrome. Angiitis of any cause, trauma, embolus, compression of the internal carotid by the lateral process of the atlas, and rarely syphilis have been implicated.

The arterial blood supply to the head is illustrated in figure 1. The internal carotid artery bifurcates from the common carotid artery distal to the carotid bulb and courses through the retropharyngeal space into the cranium, giving off as its first branch the ophthalmic artery. The central retinal artery branches from the ophthalmic coursing through the optic nerve furnishing the blood supply to the retina. It is an end artery with no collateral circulation. Other branches of the ophthalmic, however, have rich collateral circulation, with both the ipsilateral external carotid and contralateral internal and external carotids. The internal carotid artery subsequently branches into the posterior communicating, anterior choroidal, anterior and middle cerebral, thus providing the blood supply for the homolateral retina, frontal, temporal, parietal lobes and posterior limb of the internal capsule. The clinical pattern of the syndrome and specific symptoms are determined by the adequacy of the collateral circulation. Approximately 15 per cent of the patients with complete carotid occlusion may have no neurologic symptoms because of adequate collateral circulation.

In the past, the finding of thrombosis of the internal carotid artery has commonly been attributed to retrograde extension from the middle cerebral artery, rather than originating from the cervical portion of the carotid artery. Routine autopsy examinations of the cervical portion of the internal carotid is seldom done. It is well to note that in Fish-
er's analysis, thrombotic occlusion of major vessels adjacent to the circle of Willis was seen only 12 times in a total of 218 cases with vascular lesions. In striking contrast, carotid occlusion or severe stenosis was present in 41 cases, approximately the same frequency as cerebral hemorrhage and hypertensive atherosclerotic encephalomalacia. Necropsy examination of patients with "cerebral thrombosis" in the past has frequently shown confusing pathologic findings. Areas of extensive softening or infarction have been noted without anatomic obstruction of the cerebral vessels. It is becoming quite clear that with complete examination of the brain and carotids that carotid artery thrombosis and stenosis is a commonly overlooked etiologic factor.

It is apparent that the problem of carotid disease, its incidence, and the spectrum of clinical findings associated with carotid artery disease is today poorly formulated. The following cases are representative of this broad spectrum.

Case 1

M.P., a 55-year-old white woman, experienced 4 transient episodes of monocular blindness 15 months prior to admission. Three involved the right eye and 1 the left eye, and the attacks were always associated with the upright position. The episodes consisted of a sensation of a window shade descending gradually over the eye until total monocular blindness occurred. After approximately 1 minute the vision would return rather jerkily by levels until there was complete restoration of vision. There were no associated motor or sensory disturbances. The patient remained symptom free until 3½ months prior to admission, when she noted progressive difficulty in the use of the right hand. Two months prior to admission there was a transient episode of left-sided weakness. There was no history of diplopia, vertigo,
or dysphasia. Her father died of a cerebral vascular accident at age 73 and a sister died of coronary thrombosis at age 43. The blood pressure was 140/90 mm. Hg in the left arm and 110/90 mm. Hg in the right. The right radial pulse was diminished. The carotid vessels pulsed vigorously to the angle of the jaw. Auscultation over the right carotid revealed a harsh grade III systolic murmur. There was a palpable thrill over the left carotid bulb, and a grade III continuous murmur was present. Changes in position, jugular compression, and the Valsalva maneuver produced no appreciable changes in the murmurs. There was no evidence of venous collaterals. Carotid compression just proximal to the bulb obliterated the murmur on the ipsilateral side and accentuated the contralateral murmur. On compression of the left carotid artery the previously noted systolic murmur over the right carotid bulb became continuous. There was bilateral diminution of the pharyngeal carotid pulsations and this was more evident on the left.

The visual fields were normal. The fundi revealed grade II changes. The heart was not enlarged and there was a grade I to II aortic systolic murmur. The neurologic examination was negative except for motor weakness of the right arm. The cranial nerves were intact, save for slight flattening of the left nasolabial fold. The remainder of the neurologic examination was normal. The electrocardiogram was normal. X-rays of the skull and neck revealed calcium plaques in the carotid arteries. The serum cholesterol was 299 mg. per cent. The electroencephalogram was normal. Following compression of alternate carotids for 10 seconds frontal dysrhythmic changes occurred on the side of carotid compression.

The patient experienced one episode of left-sided weakness concomitant with a drop in blood pressure from 140/90 to 120/80, probably induced by oversedation. Function rapidly returned with restoration of blood pressure to the previous levels. The patient's condition has remained stable on short-term anticoagulant therapy.
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Dr. Crevasse: The history and physical findings are quite typical of bilateral carotid artery insufficiency. Premonitory signs and symptoms such as transient, localized motor and sensory disturbances, hemiparesis, monoparesis, and visual difficulties may be observed from days to years before a permanent vascular accident occurs in the majority of patients. This patient illustrates the long duration of premonitory symptoms evincing in a progressive neurologic picture simulating brain tumor. This pattern occurs in 15 to 25 per cent of reported patients. Early recognition of this group with premonitory symptoms is paramount in regard to therapy.

A Physician: How can this be differentiated from the aortic arch syndrome or "reversed coarctation"?

Dr. Crevasse: The clinical features and symptoms of these 2 syndromes may closely overlap. Briefly the aortic arch syndrome occurs in the younger age group, is usually associated with muscle group ischemia and atrophy of the muscles supplied by the great vessels arising from the arch.

The asymmetry or absence of blood pressure in the upper extremities is well known, but blood pressure variation or absence of a radial pulse not infrequently occurs simultaneously with carotid artery thrombosis alone and represents segmental obstructions. In the aortic arch syndrome the carotid pulsations are usually diminished at the base of the neck and all the reported bruits have been maximal at this site (fig. 2). By contrast in carotid artery insufficiency, an occlusion may be detected high in the neck or the pulsations may be vigorous to the angle of the jaw and diminished or absent in the pharynx. The bruits are maximal over the carotid bulb (fig. 3).

Case 2

J.P., a 75-year-old white man, suddenly developed paralysis of the right arm and leg and inability to speak. The blood pressure was 130/80 bilaterally. There were aphasia and right hemiplegia. The left carotid pulsations terminated abruptly at the carotid bulb. The right carotid pulsated vigorously to the angle of the jaw. Palpation of the pharyngeal segments of the internal carotid revealed good pulsations on the right and none on the left. Auscultation of the right carotid bulb revealed a faint systolic bruit. The heart was normal.

Dr. Crevasse: About one half of the cases of carotid artery thrombosis simulate the
routine stroke. Aphasia is present in approximately 50 per cent of cases and may be quite prominent, even though the carotid thrombosis may be on the side opposite the dominant hemisphere.

A Physician: How can carotid occlusion be differentiated from the routine middle cerebral artery thrombosis?

Dr. Crevasse: Preceding episodes of transient motor weakness or sensory symptoms occurring primarily in the upright position or after maneuvers that provoke hypotension, postural or otherwise, are quite typical of impending carotid thrombosis.

By angiographic studies about 75 per cent of the complete or partial occlusions are just above the bifurcation of the common carotid and 15 per cent at the siphon, with the remaining 10 per cent in varied locations (fig. 1). This produces several interesting physical findings that are most useful in the clinical diagnosis. It is well to remember, however, that physical findings including neurologic deficits may be absent, particularly in the premonitory phases.

Palpation of the neck at the angle of the jaw may reveal a diminished or absent pulsation. Occasionally a thrill is felt over the partially occluded vessel. More important, however, is the gentle palpation of the lateral portion of the pharynx using a wet glove. Absence or diminution of pulsation of the pharyngeal segment of the internal carotid is a consistent and reliable physical finding. Thus, when an occlusion is suspected and the carotid pulse is absent in the neck or on pharyngeal palpation, the diagnosis would appear unequivocal. Since approximately 15 per cent of people with occlusion of the carotid artery will have no neurologic symptoms, cases of expanding intracranial lesions may coexist with asymptomatic carotid artery occlusion.

The finding of a pulse in clinically suspected cases should not deter one from the diagnosis but indicates the need for further study by laboratory methods. Bruits over the ecarotid and carotid pulse are quite helpful in that they are unlikely to occur with small-vessel occlusions at the circle of Willis.

A Physician: How do you explain the presence of good pulsations in the neck and pharynx in some cases of carotid thrombosis?

Dr. Crevasse: The explanation for a palpable pharyngeal pulse in proved cases of occlusion are threefold. Since the occlusion is at the carotid siphon in approximately 15 per cent of cases (fig. 1), pulsations in the neck and pharynx, of course, will be normal. Secondly, a palpable pharyngeal pulse may represent transmitted pulsations from the more laterally positioned external carotid artery. Lastly, spasmatic contractions from the stimulated pharyngeal musculature may simulate arterial pulsations. For these reasons, the finding of a normal carotid pulse in the neck or pharynx only makes the diagnosis more elusive, but certainly not untenable.

Case 3

J.S., a 59-year-old white diabetic man, developed episodes of numbness and weakness in the right arm and leg associated with a stumbling gait 4 months prior to admission. On one occasion there was transient aphasia. These episodes occurred primarily in the upright position while
working, lasted 4 to 5 minutes, and promptly cleared with rest. After being given a Rauwolfia preparation because of mild hypertension, the patient noted a marked increase in number of these episodes. He discontinued his medication and there was an abrupt decline from 10 to 15 to approximately 1 such episode each day. The blood pressure was 190/90. The fundi showed grade II changes. The right posterior tibial pulse was absent. The left carotid pulsations were absent below the angle of the jaw. The right carotid pulsed vigorously. The pharyngeal segments were not palpable. There were no bruits over the head or carotids. The neurologic examination was normal. The electrocardiogram revealed left ventricular hypertrophy. The serum cholesterol was 360 mg. per cent. Anticoagulant therapy was instituted and there was no recurrence of symptoms during a short follow-up period.

Dr. Crevasse: This case demonstrates a phenomenon that may occur in patients with cerebral vascular disease treated with hypotensive agents. The pathophysiology of these symptoms now seems clearer. In general, 55 to 60 mm. Hg is the critical systolic blood pressure to maintain adequate cerebral circulation, but with a compromised cerebral vascular circulation the critical blood pressure is considerably higher, and seemingly insignificant falls in blood pressure may cause significant symptoms. Shanbrom and Levy7 observed the appearance of hemiplegia in a case of carotid thrombosis when the blood pressure fell below 160 mm. Hg following carotid arteriography. When the blood pressure was maintained above this level by vasopressor agents, the hemiplegia would resolve, only to reappear when the pressure fell again below this level. We have recently observed a patient with basilar artery insufficiency with tetraplegia precipitated by a fall in blood pressure from 200/110 to 130/110 incident to myocardial infarction. Restoration of function on 1 side with clearing of the sensorium rapidly occurred when the blood pressure was again maintained with vasopressor agents at 200 mm. systolic.

Meyer et al.8 studied a group of 36 patients with recurrent signs and symptoms of major arterial insufficiency to the head in the light of the electroencephalogram, electrocardiogram, and clinical manifestations as the blood pressure was lowered by repeated postural tilting on the tilt table and by carotid artery compression. When hypotension was produced on the tilt table or more commonly following carotid compression, neurologic symptoms could often be reproduced. Striking abnormalities of the electroencephalogram occurred during hypotension or carotid compression, whereas changes were not reproducible in normal subjects, patients with surgical ligation of the carotid artery, or elderly subjects with normal cerebral vascular function. Patients with carotid or basilar obstruction developed cerebral dysrhythmias over the regions supplied by the respective vessels when seemingly insignificant falls in blood pressure were produced on the tilt table (figs. 4 and 5).

Carotid artery insufficiency or thrombosis is usually present when transient or persistent hemiplegia occurs in relation to shock. Total or partial blockage of 1 carotid can exist without symptoms and be unmasked during a period of abnormal lowering of the blood pressure. Patients with a combination of cerebral vascular and cardiac disease are prone to episodes of postural ischemia to the brain, since postural fall in blood pressure is much greater in these patients.8

Postural hypotensive maneuvers and agents such as Rauwolfia, chlorpromazine, and ganglionic-blocking agents are closely analo-

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*Fig. 5. Electroencephalogram of a 78-year-old man with basilar artery insufficiency before and after postural tilt. (Reproduced from Neurology 6:464, 1956, by permission of the authors and the publishers.)*
gous to the tilt-table observations and should be avoided in all patients with cerebral vascular insufficiency. Furthermore, one should correct promptly in these patients hypotension incident to cardiac arrhythmia, myocardial infarction, blood loss, acute left ventricular failure, anesthesia, or other less common etiologies.

A Physician: What is the role of cerebral angiospasm in the production of symptoms?

Dr. Crevasse: From clinical observations in man and laboratory evidence in animals where the cerebral vessels can be viewed through a skull window, alterations in blood flow related to changes in systemic arterial pressure rather than cerebral angiospasm seem to be the major factor. It is difficult, however, to explain the transient neurologic deficits accompanying migraine headaches and, of course, arteriospasm has been observed by neurosurgeons operating on the circle of Willis.

A Physician: Can carotid artery thrombosis be established without resorting to angiography?

Dr. Crevasse: Carotid artery insufficiency and thrombosis is rapidly becoming a clinical diagnosis. Ophthalmodynamometry is a valuable adjunct in the diagnosis and consists of insertion of a gage with a spring-loaded plunger between the lid and sclera for the measurement of intra-ocular pressure. The retinal arteries are observed with the ophthalmoscope as pressure is gently increased. When the retinal arteries begin to pulsate this represents the diastolic pressure and can be interpreted from the gage. The intra-ocular pressure is further increased until pulsations cease, and this reflects systolic pressure. This is repeated in the opposite eye. There is a striking pressure differential between the involved and the opposite eye, a reflection of carotid arterial obstruction. The average retinal artery pressure has been found to be 30 to 45 mm. Hg diastolic and 65 to 75 mm. systolic, that is, approximately half the systemic blood pressure. It seems that a reduction of 25 to 30 per cent in both systolic and diastolic pressures is diagnostic of impaired carotid circulation.

Case 4

J.A., a 47-year-old white man, developed episodes of sudden blindness in the right eye, characterized by a fog rolling in from the temporal side until vision was completely obscured. The blindness lasted 4 to 5 minutes and vision returned gradually from the nasal to the temporal side. During the past year he has had repeated episodes of weakness and numbness of the left arm and leg, not accompanied by visual symptoms. There have been persistent and severe frontal headaches for the past 8 months. Mental acuity has progressively diminished.

The blood pressure was 160/90. There was slight aphasia with moderate weakness of the left arm and leg. There was a systolic bruit over the left eye ball. The fundi were normal. The right pharyngeal pulse was absent. The reflexes were hyperactive on the left. The right carotid pulsation ended abruptly at the bulb, and the left pulsedated vigorously. A grade II systolic murmur was present over the left carotid bulb.

A right carotid arteriogram demonstrated occlusion below the right carotid bulb. A thrombendarterectomy was performed by Dr. Garland Perdue but despite removal of the thrombus and passage of a catheter into the carotid siphon no back flow from the distal carotid occurred. The immediate postoperative course was characterized by recurrent episodes of monocular blindness at which time his blood pressure was 10 to 20 mm. Hg lower than when he was asymptomatic.

Dr. Crevasse: Monocular blindness and contralateral neurologic deficits are the hallmark of carotid artery thrombosis. As in this case, they usually do not occur simultaneously. This patient has severe headache, which is present in about half of all reported cases. The combination of headache, changes in mental acuity, neurologic deficits, and seizures frequently occurs in carotid artery thrombosis and is invariably confused with brain tumor.

The systolic bruits over the eyeball and carotid bulb opposite the occluded carotid are valuable signs in diagnosis and probably represent a compensatory increase in blood flow through the opposite partially involved carotid artery.

A Physician: What is the significance and mechanism of monocular blindness?

Dr. Crevasse: Monocular blindness is usually transient lasting from 2 to 3 minutes and is subjectively described as a fog rolling
in, a veil coming over the eye, or a curtain gradually obscuring vision. This type of blindness is typical of retinal ischemia. Since the central retinal artery is an end artery (fig. 1) with no collateral circulation, alterations in blood flow through this vessel on the side of a partially or completely occluded carotid is markedly affected by spontaneous falls in blood pressure or cardiac output. We have made observations on the preceding patient, and indeed when monocular blindness occurred his systolic blood pressure was 10 to 20 mm. Hg lower than when he was asymptomatic. With falls in blood pressure, the fundus became pale, with a decrease in caliber of the branches of the central retinal artery, and retinal ischemia and blindness followed. When the factors that have reduced blood flow were compensated or corrected, vision returned.

Paradoxically, in some cases when premonitory visual disturbances have been present and carotid thrombosis finally occurs with contralateral hemiplegia, the vision may remain unimpaired, as adequate time has elapsed for collateral circulation to maintain blood flow to the retinæ.

A Physician: What about the results of surgery and anticoagulant therapy?

Dr. Crevasse: As in this case, in general, the surgical results have been disappointing. When symptoms are present with carotid thrombosis, the collateral circulation through the basilar and opposite carotid is impaired. This concept is substantiated by the high incidence of asymptomatic carotid thrombosis and the tilt-table experiments on otherwise normal persons with carotid artery ligations without symptoms.

Rob and Wheeler have reported the largest successful series of operated cases. They have employed direct arterial surgery with and without hypothermia to restore blood flow through a partially or completely obstructed carotid in a total of 27 patients. Eleven of this series had partial occlusion with transient neurologic symptoms. They were able to re-establish good blood flow in all of these. Four patients became asymptomatic, 2 improved, and 4 showed no change. There was one death. Of the 16 patients with complete occlusion, good blood flow could be re-established in only 4 patients. After complete occlusion, as in the preceding case, blood flow could be re-established only during a short interval before the clot extended higher into the cranial cavity. In the group of complete occlusion only 1 patient became asymptomatic and the remaining 13 showed no change. There were 2 deaths. It seems that the patients most likely to benefit from surgery are those with incomplete occlusions who consult their physician because of transient symptoms of cerebrovascular insufficiency. This is the group in whom the neurologic examination will most likely be negative and the only physical finding may be a systolic or continuous murmur over the partially obstructed carotid, usually contralateral to neurologic symptoms. Restoration of flow in these patients will be rewarding not only in frequently relieving symptoms of cerebral vascular insufficiency but may in turn prevent the later development of complete thrombosis and irreversible neurologic deficits.

Though long-term follow-up and double-blind studies are not complete, promising results are seen with anticoagulants, which appear to be the medical treatment of choice at the moment. In our experience, symptoms of cerebral vascular insufficiency diminish, and the neurologic status appears to stabilize after anticoagulation.

The rational and beneficial effects are not entirely clear. Anticoagulation may further delay occlusion of a partially obstructed carotid or prevent extension of the thrombus into the cranial cavity. Autopsy material has revealed formation of thrombi in the carotid bulb and embolization of the ipsilateral cerebral hemispheres. Anticoagulation would appear more specific in this situation.

A Physician: What is the prognosis of this syndrome?

Dr. Crevasse: Again, long-term follow-up studies in large series of patients are inadequate, and the relative prognosis is unknown. However, in a review of 107 cases of complete occlusion of the carotid artery, Johnson and Walker reported 15 per cent mortality with-
in several months, 15 per cent revealed no essential change, and 25 per cent showed improvement over varying periods of time. There was no follow-up study of the remaining patients. The follow-up of patients treated by anticoagulants and surgical reconstruction is again limited and inadequate. It appears, however, that their prognosis will be somewhat improved by these methods.

Case 5

E.S., a 56-year-old white woman, gave a history of benign hypertension for several years. Four weeks ago the patient developed typical angina pectoris precipitated by exertion and relieved promptly by nitroglycerin. There was no history of neurologic symptoms or visual disturbances although there had been slight impairment of memory in the past few months.

Her father died at age 45 of high blood pressure and cerebral vascular accident. Her mother died at age 59 of coronary artery disease.

The blood pressure was 160/90. The carotids pulsed vigorously to the angle of the jaw. Over the right carotid bulb a loud continuous murmur was present. This murmur was hemodynamically identical with the continuous murmur in case 1 and figure 3. There was no bruit over the head or the left carotid artery. Pharyngeal palpation of the internal carotids revealed diminished pulsations bilaterally, with no obvious asymmetry. The heart revealed a grade II aortic and mitral systolic murmur. The neurologic examination was completely negative.

Dr. Crevasse: Approximately 3 per cent of Fisher's unselected autopsy series had severe stenosis of the carotids and 11 of Rob's 27 operated cases had partial occlusion with neurologic symptoms. The pathophysiologic counterpart of these findings may be a systolic or continuous murmur over the carotid bulb, the most common site of involvement.

We have recently correlated the frequency and significance of localized carotid murmurs in 100 consecutive general hospital patients. Well localized systolic murmurs were heard over the carotid in 7 and continuous murmurs were present in 2. These murmurs occurred both in asymptomatic patients and patients with overt carotid artery insufficiency as in cases 1, 2, 4, and 5. In a 4-month period we have observed a well-localized continuous murmur over the carotid bulb in 8 patients. Five of these patients had neurologic symptoms or deficits, usually on the side opposite the carotid murmur.

The final clinical spectrum of carotid artery insufficiency and thrombosis has yet to be defined. The initial evidence of carotid obstruction may be localized murmurs over the carotid bulb and these may be noted prior to the development of symptoms. The patients may complain of "swishing sounds" or roaring in the head. When complete thrombosis ensues, the head noises and bruits cease and neurologic symptoms may appear, depending on alterations in blood flow and adequacy of collateral circulation.

A Physician: What are the mechanisms responsible for the murmurs heard in these patients?

Dr. Hurst: One can divide the bruits heard in these patients into those heard in the head and those in the neck. The examiner should establish the habit of listening over the cranium and especially over the eyeballs, in patients suspected of carotid insufficiency. It is a good plan to have the patient close his eyelids and place the bell of the stethoscope over 1 eye and have the patient "open" his eyes. This allows one to listen through a relaxed eyelid, thereby eliminating the sounds of muscle tremor.

The high-pitched bruit heard over the eyeball on the side opposite the carotid thrombosis (fig. 1) can be due to 1 of 2 mechanisms: 1. The retrobulbar arterial collateral circulation may be so abundant that a bruit is produced. 2. It is likely that a bruit produced by a partially occluded artery within the skull will be heard better over the nearest eyeball. In this case, the bony orbit acts as a megaphone. Intercranial arteriovenous fistulae and severe anemia can produce such murmurs but are not as common as vascular occlusive disease of the head and neck.

It is convenient to divide the murmurs in the neck into systolic and continuous murmurs.

Everyone is aware of the systolic arterial bruit heard in the neck vessels in patients with anemia, fever, thyrotoxicosis, and the other causes of high cardiac output. In the
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absence of these conditions a systolic bruit in the carotid artery should force one to consider the murmur of aortic stenosis, which is frequently transmitted into the neck or carotid arterial obstruction. Aortic stenosis is usually heard best maximally over the primary aortic area, may be heard at the apex, and is transmitted up both carotids, diminishing in intensity as one approaches the angle of the jaw. When atherosclerosis of the carotids occurs without significant obstruction, the systolic bruit is seldom loud and occurs with equal intensity through both carotids.

When one encounters a loud and localized systolic murmur in a carotid artery, this should be considered as an important diagnostic clue of partial occlusion of this vessel. The systolic bruit is heard over the site of the arterial narrowing and can, therefore, disappear if the occlusion becomes complete. Occasionally, in complete occlusion of one carotid artery, collateral blood flow is increased through the opposite carotid and if it is partially occluded, a systolic or continuous murmur may be detected over the opposite carotid. Occasionally a faint systolic murmur is present over the site of carotid occlusion. It can be obliterated by manual compression of the branches of the external carotid and is related to an increase in collateral flow through this vessel (fig. 1).

There are several causes of a continuous murmur in the neck. The most common cause is the normal venous hum that is usually heard in children and rarely heard in adults. This type of murmur can be eliminated or altered significantly by light pressure on the jugular veins and is seldom a diagnostic problem.

Graves’ disease or an arteriovenous fistula between the carotid artery and jugular vein may cause a continuous murmur. One is usually able to identify systolic pulsations of the neighboring vein, and an increase in skin temperature and venous return in the latter.

A continuous murmur may be heard over an area of partial carotid occlusion when the collateral vessels are inadequate. Edholm et al. have shown that localized or segmental obstruction in arteries has no effect on diastolic pressure distal to an obstruction producing only a systolic pressure gradient. Flow to the segment distal to the obstruction is provided by adequate collateral circulation and equal diastolic pressures are maintained both proximal and distal to the obstruction. The turbulence of flow in systole produced by the narrowed carotid and systolic pressure gradient accounts for the systolic bruit heard. Myers and co-workers have made observations on continuous murmurs over partially occluded vessels, with particular reference to the aortic arch syndrome. The production of a similar hemodynamic situation in animals clearly substantiated their hypothesis that the continuous murmur was due simply to partial occlusion of a major artery under circumstances where collateral circulation was simultaneously diseased or inadequate to maintain diastolic pressure distal to the involved area. In this situation, a considerable pressure gradient exists both in systole and diastole, producing a continuous murmur over a partially occluded vessel. In carotid artery insufficiency when collateral circulation through the opposite carotid and the basilar system is inadequate, a continuous murmur is produced usually over the carotid bulb, the most common site of involvement (fig. 3). This is often confused with an arteriovenous fistula in the neck.

In summary, there are several points worth remembering. 1. Carotid artery thrombosis and stenosis is a common disease occurring in 9.5 per cent of one unselected autopsy series but is infrequently recognized clinically. 2. Physicians interested in cardiovascular disease are being confronted with this syndrome because of its relationship to changes in blood pressure and cardiac output. 3. The carotid arteries are neglected by the pathologist and are a no man’s land between the internist and neurologist. Careful examination and auscultation of the head and neck are rewarding in “routine strokes” and may clarify bizarre neurologic symptoms in patients with cardiovascular disease. 4. A continuous machinery murmur may be present over a partially occluded carotid and is a valuable adjunct in the early recognition of
carotid artery insufficiency. 5. Carotid artery insufficiency is a treatable disease. Its early recognition and appropriate therapy before complete thrombosis occurs will directly influence the therapeutic results.

REFERENCES


In 27 patients with symptomatic occlusions of the internal carotid artery direct arterial surgery was performed in an attempt to restore adequate blood flow. A good blood flow was obtained in all 11 patients with partial occlusion, and in only 4 of the 16 with complete occlusions. In complete occlusions blood flow could only be reestablished during the short period before the clot extended into the cranial cavity, but even then irreversible cortical damage might have occurred. The risk of surgery is not too great in these patients since only 2 of the 27 patients so treated had any postoperative exacerbation of their neurologic symptoms. The authors believe that the patients most likely to benefit from such surgery are those with incomplete occlusions who have symptoms of cerebrovascular insufficiency. In these patients restoration of adequate blood flow not only frequently relieves the symptoms but also may prevent the later development of complete thrombosis and irreversible brain damage.
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