Collateral circulation between internal and external carotid arteries was sufficient to thoroughly opacify the middle cerebral artery on cerebral angiography in 11 of 100 patients with internal carotid occlusion. In four of the 11, the branches of the middle cerebral were intact. In two of the four, the carotid occlusion was an incidental finding; one patient presented with transient ischemic attacks and one had a stroke of the border zone variety. The remaining seven patients all had clinical strokes and showed occlusion of one or more branches of the middle cerebral artery. The stroke appeared to be the result of the intracranial occlusion rather than that of the internal carotid.

ADDITIONAL KEY WORDS collateral circulation asymptomatic middle cerebral artery branches ophthalmic artery stroke

The reason for this report is to emphasize a combination of extracranial and intracranial occlusions which appear to correlate with the clinical phenomena produced and can be recognized on cerebral angiography. This particular combination is occlusion of the small intracranial branches of the middle cerebral artery after occlusion of the internal carotid artery on the same side and is recognizable when the intracranial branches are opacified by collateral circulation. There is no reason to suppose that such occlusions are limited to the carotid-middle cerebral circulation, but the pathways of collateral circulation and anatomical factors allow easier recognition in this area. Since the type of collateral circulation in these particular cases may screen the brain from systemic emboli, thrombosis possibly arising on areas of preexisting atherosclerosis may be the cause.

Intracranial embolic occlusions are also found in conjunction with carotid occlusion, but these arise from large mural thrombi in the carotid which progress to occlusion after the embolus has been shed. The embolic fragments are usually large, causing proximal intracranial occlusions and angiographically can seldom be differentiated from carotid thrombosis alone unless studies are sufficient to opacify all components of the circle of Willis. These have been described as "local embolism," "forward embolism," and "concealed emboli," and are an entity quite distinct from that here presented.

Very briefly, the mechanisms of collateral circulation may be divided into three main pathways: (1) the circle of Willis, (2) pial anastomoses between the various intracranial arteries, and (3) anastomoses between musculocutaneous or dural and intracranial arteries, frequently (but not limited to) the branches of the external carotid and the ophthalmic branch of the internal carotid. This study is confined to...
cases in which the intracranial arteries are well opacified, but the degree of intracranial opacification does not necessarily reflect the effectiveness of the collateral. It is well established that the combination of these collateral pathways may be remarkably effective and that occlusions of one or more extracranial cerebral arteries may be asymptomatic.

When the common carotid artery is opacified at angiography, the branches of the external carotid artery are filled. In the presence of internal carotid occlusion the intracranial arteries are opacified to some degree depending on the predominant route of the collateral, if the collateral has developed between the extracranial and intracranial circulations. When the major flow is from extracranial to intracranial circuits through the ophthalmic artery, the intracranial portion of the internal carotid becomes densely opacified. The anterior and posterior cerebral arteries on the affected side are ordinarily supplied through the circle of Willis from the arteries nearer their origin, so that the contrast is usually confined to the area of the middle cerebral artery. Frequently, the main trunk of this artery becomes densely opacified and, since the artery at this point has no direct connection with the circle of Willis to produce dilution of contrast, there is, of necessity, opacification of all the branches of this vessel. It is true that the total area irrigated by the middle cerebral may be reduced by the pial collateral coming down from the midline arteries and up from the posterior cerebral, but the consistent localization of border zone infarcts and the angiographical findings in cases of chronic stenosis of the middle cerebral artery indicate that this reduction is in the form of a symmetrical shrinkage about the periphery rather than involving any one branch in its entirety. Consequently, nonfilling of a distal artery must be considered due to an occlusion.

A theoretical objection to this statement might be that of a preexisting gradual stenosis of an intracranial branch, with the development of retrograde pial collateral to any one artery so that its nonfilling in an antegrade manner would not be associated with ischemia. This cannot be denied, but such asymptomatic occlusions of intracranial arteries are rare, since progressive reduction in the lumen of an artery 1 mm or less in diameter is likely to lead to thrombotic occlusion before adequate retrograde collateral has had time to develop.

The problem of recognizing the intracranial occlusions can be solved by the same means as are employed in recognizing arterial occlusion anywhere else, namely, by seeing the point of occlusion in the artery or absence of the blood supply to any particular area. From previous anatomical studies a system of identifying areas of the brain that have a constant blood supply has been devised, and by concentrating on the area rather than trying to identify the branches proximally, absence of an arterial branch becomes obvious. The use of a template to outline the areas is not essential to the diagnosis, but a knowledge of anatomy is, and the template is an aid, not a substitute, for the study of anatomy. Secondary signs of an occluded branch, such as retrograde filling, cannot be expected to be as helpful as it might be when the intracranial arteries are filled directly through a patent carotid.

Having covered the preliminaries, the material is as follows: In 100 patients with occlusion of the internal carotid artery, diagnosed by direct carotid angiography, 11 cases demonstrated sufficient collateral circulation, primarily through the ophthalmic artery, to allow identification of the branches of the middle cerebral artery. All patients were Caucasian. In two, the carotid occlusion appeared to be an incidental finding unrelated to the patients' immediate problem. One patient had stroke, with apparently normal intracranial branches, although clinically the residual neurological changes suggested that this was of the "border zone" variety. Another patient with intact intracranial branches had symptoms suggesting transient ischemic episodes rather than infarction. The other seven all showed varying degrees of arterial occlusions and had appropriate symptoms. Three of these patients died. The nomenclature used in describing the arterial branches is given under the diagram of the first illustration. The artery (or arteries) in the central sulcus are those supplying the motor and sensory strips.

Only one example of "normal" middle cerebral artery filling is shown in the illustrations, and the radiographs of two of the abnormal (cases 9 and 11) have been previously published.
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**Case 1**
**INCIDENTAL FINDING**
(M.C.) Male, 44, hypertensive and diabetic, admitted for evaluation of seizures with olfactory hallucination. EEG suggested a left temporal lobe focus. Angiogram: Left carotid study normal. Right carotid: Occlusion of the internal carotid, complete opacification of normal middle cerebral artery branches by collateral circulation. Brain scan and pneumoencephalogram were normal; there was no evidence of atrophy.

**Case 2**
**INCIDENTAL FINDING ON THE SIDE OPPOSITE A LETHAL EMBOLUS**

**Case 3**
"NORMAL" INTRACRANIAL FILLING WITH A CLINICAL STROKE (FIG. 1)
(M.O.) A 75-year-old hypertensive female. Noted "weakness" in left arm, acute stroke with left hemiplegia four days later. Angiogram: The angiogram was performed within four hours after the onset of hemiplegia. Occlusion of internal carotid, complete opacification of branches of middle cerebral artery and no evidence of occlusion. Repeat angiography was not performed. Course: Some function returned within 24 hours. After physical therapy there was good return of function with residual weakness limited to the left shoulder and left leg. Patient is ambulatory with a cane and short leg brace.

**Case 4**
"NORMAL" INTRACRANIAL FILLING WITH EPISODES SUGGESTING TRANSIENT ISCHEMIA
(O.M.) An 80-year-old male with recurrent brief episodes of right-sided weakness, and blurred vision for four weeks. Angiogram: Left carotid: Occlusion of the internal carotid, complete opacification of branches of the middle cerebral artery with moderate arteriosclerotic changes but no evidence of occlusion. Right carotid angiography was not performed. The patient was discharged without specific therapy and no follow-up information was obtainable.

**Case 5**
INTRACRANIAL OCCLUSION WITH GOOD RECOVERY (FIG. 2)
(H.C.) A 63-year-old normotensive, nondiabetic male. Acute stroke with left hemiplegia and...
Left. (Case 5) The angiogram shows collateral filling of only the anterior and posterior components of the middle cerebral artery. Right. From the drawing, the absence of arteries to the central sulcus, posterior parietal and angular areas is obvious.

mild mental confusion. Angiogram: Right carotid: Occlusion internal carotid, good intracranial collateral, with a large avascular area, due to absence of arteries to central sulcus, posterior parietal and angular areas. Left carotid angiography was not performed. The brain scan was positive in the frontoparietal area. Improvement was pronounced. Six weeks later only a mild spastic hemiparesis persisted, and the patient felt able to return to work.

Case 6
INTRACRANIAL OCCLUSION WITH GOOD RECOVERY (FIG. 3)
(F.W.) A 75-year-old normotensive nondiabetic male with episodes of left-sided weakness associated with difficulty in thinking or expressing himself for two months. Minimal left-sided weakness found on examination. Angiogram: Right carotid: Showed occlusion of the internal carotid with good collateral circulation and absence of angular

Left. (Case 6) The angiogram shows collateral filling of anterior as well as middle cerebral artery, the more posterior components of the latter being absent. Right. On the drawing of the middle cerebral artery, the occlusion of the angular and posterior temporal branches is more obvious. The point of occlusion of the posterior temporal is well seen.
and posterior temporal arteries. Left carotid angiogram was not performed. Course: The patient was treated with anticoagulants and did well with no further episodes. The weakness of the left extremities disappeared.

**Case 7**

**INTRACRANIAL OCCLUSION WITH SEVERE NEUROLOGICAL DEFICIT (FIG. 4)**

(E.L.) A 47-year-old obese, severely hypertensive diabetic female who suffered an acute stroke with left hemiplegia. Angiogram: Right carotid angiogram performed within eight hours after the onset of the left hemiplegia. It showed occlusion of the internal carotid with intracranial collateral, filling the proximal middle cerebral, the proximal portion of the anterior cerebral and a large frontopolar branch of the anterior cerebral. The middle cerebral was occluded just after the origin of the orbitofrontal and operculofrontal branches. Left carotid angiography was not performed. Course: The patient improved slightly but had persistent left hemiparesis, was mentally obtunded and required nursing home care.

**FIGURE 4**

*Left* (Case 7) The angiogram shows collateral filling of only the most anterior portion of the middle cerebral. One large branch of the anterior cerebral is also seen. *Right* From the drawing, in which the branch of the anterior cerebral is omitted, the middle cerebral is seen to be absent distal to the origin of the orbitofrontal and operculofrontal branches.

**FIGURE 5**

*Left* (Case 8) The angiogram shows good collateral opacification of both anterior and posterior components of the middle cerebral artery with an avascular area between. There is faint opacification of the pericallosal artery as well. *Right* From the drawing, the avascularity is seen due to absence of central sulcus and posterior parietal arteries.
Case 8
INTRACRANIAL OCCLUSION WITH SEVERE NEUROLOGICAL DEFICIT (FIG. 5)
(M.C.) A 52-year-old hypertensive diabetic female, with episodes of intermittent weakness of the right leg for one week and then a sudden onset of aphasia and complete right hemiplegia. Angiogram: Left carotid: Occlusion of internal carotid, good intracranial collateral with occlusion of central sulcus, and posterior parietal branches. There was also transient filling of a portion of the pericallosal artery on later films. Course: The patient had no return of function after two months, and requires nursing home care.

Case 9
INTRACRANIAL OCCLUSION WITH SEVERE NEUROLOGICAL DEFICIT
(R.O.) A 43-year-old obese, hypertensive diabetic male. The patient had transient left hemiparesis three years previously and transient aphasia one week prior to admission. He developed sudden right arm weakness with progressive hemiparesis and aphasia the morning of admission. Aortic Arch Study: Occlusion of both internal carotids; right vertebral was small and vestigial, and the left vertebral was large and appeared normal. Angiogram: Right carotid: Occlusion of internal carotid, no intracranial filling. Left carotid: Occlusion of internal carotid. Good intracranial collateral, with occlusion of the central sulcus, posterior parietal, and posterior temporal arteries. Course: The patient was anticoagulated but did very poorly, being totally hemiplegic on the right, and severely obtunded. He was discharged to a nursing home.

Case 10
INTRACRANIAL OCCLUSION WITH FATAL TERMINATION (FIG. 6)
(S.K.) A 70-year-old normotensive nondiabetic male who had a "small stroke" with speech difficulty and right-sided weakness one week prior to admission. The weakness improved spontaneously but the speech difficulty persisted. Aortic Arch Study: Occlusion of the left internal carotid, other vessels essentially normal. Angiogram: Left carotid: Occlusion of the internal carotid with good intracranial collateral and absence of the operculofrontal branches. Course: Surgery was performed with anastomosis of the superficial temporal artery to cortical arteries. The postoperative course was uneventful for two days; then systemic emboli developed, and subsequently embolic occlusions to the right cerebral hemisphere, documented by right carotid angiography. Patient expired two weeks after the original procedure.

Case 11
INTRACRANIAL OCCLUSION WITH FATAL TERMINATION
(J.L.) A 70-year-old normotensive, nondiabetic but obese male had vague headaches for one week prior to acute onset of weakness in the right arm on the evening of admission. Angiogram: Left carotid: Occlusion of the internal carotid with good collateral filling of the middle cerebral artery and its branches. There was absence of the artery in the central sulcus. Course: The patient was treated vigorously with a thrombolytic agent, but signs progressed and he was anticoagulated. Progressive deterioration continued, and the patient expired four days after
admission. Extensive encephalomalacia in the left temporal and occipital lobes was found at postmortem.

The distribution of these occlusions is similar to that seen in occlusive disease with a patent carotid, and the wide variation in location and extent of the intracranial occlusions suggests that the nonfilling of the various branches cannot be attributed to anatomical or technical factors. The clinical picture suggests that there is some correlation with the site and extent of the occlusions although, since collateral circulation is in itself a modification of the normal, it is reasonable to presume that the effect produced by the occlusions may also be modified. Despite the reservations, the reasonable conclusion is that the stroke is the direct result of the intracranial process and the extracranial carotid occlusion is only indirectly responsible.

A second inference is that this is probably common as a mechanism of stroke. The process is seldom documented because: (1) only a minority of patients with occlusion of the internal carotid opacify the intracranial arteries sufficiently to allow a diagnosis, and (2) some 20% to 25% of occlusions of the supratentorial arteries involve the anterior or posterior cerebral vessels and occlusions in these locations can rarely be recognized even though the middle cerebral artery branches are well seen. Finally, it seems likely that the finding of internal carotid artery occlusion is usually accepted as a suitable diagnosis in itself and the possibility of recognizing additional occlusions when the intracranial arteries are opacified is seldom considered.

Although the recognition of the intracranial components of the process may do little to modify existing therapy, this process does appear to be a basic and not uncommon factor in stroke production and as such is worthy of further interest and investigation.

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