SPECIAL ARTICLE

Interarterial Shunts in the Cerebral Circulation

By JAMES F. TOOLE, M.D.

GEORGE ELGIE BROWN of the Mayo Clinic, one of the founders of the Council on Circulation of the American Heart Association, was a physician whose particular interest was vascular diseases, primarily those of the lower extremities. This lectureship was endowed to honor his memory by helping to keep alive the widespread interest which he kindled in the peripheral circulation. My presentation is the twenty-sixth in this series but the first relating to the cerebral circulation, the most complicated vascular bed in the body.

The situation in which we find ourselves today concerning the cerebral circulation is so reminiscent of that described for the peripheral circulation by Dr. Isaac Starr in his introduction to the thirteenth Brown Memorial Lecture delivered in 1952 that I would like to introduce my subject by quoting it to you:

Let us recall the background under which Brown was working in the years preceding 1928. At the time he and I were in medical school, clinical medicine in the United States was dominated by the great pathologic school of thought whose primary interest was in the lesions found at necropsy. It was under the aegis of this school that the obstructive lesions of peripheral vascular disease had been discovered and described. As a result of such knowledge when a clinician detected diminution or lack of pulsation in a peripheral vessel, he could confidently predict that at necropsy the vessel would be found to be obstructed by something solid and tangible, by a clot perhaps, by a sclerotic lesion of the vessel's walls, either alone or accompanied by a clot, or possibly by pressure from without. This concentration of thought on the demonstrated arterial obstruction had led to a completely pessimistic therapeutic outlook. The vessel was obstructed and nothing could be done about it; the disease ran its malignant course, more vessels would become involved, the part supplied would suffer increasingly from lack of blood until eventually gangrene was inevitable.

Since 1928, cardiology has been transformed from an art in which the stethoscope was the only tool and digitalis the major therapy into a dynamic specialty commanding the full resources of electronics and physics, of biochemistry, physiology, radiology, and surgery. The tremendous advances that brought about this transformation have yet to be applied, on a large scale, to the cerebral circulation and its diseases. Too many present-day physicians have a 1928 attitude toward circulatory diseases of the brain, and the field continues to be dominated to a certain extent by pathology, just as was general medicine in the 1920's. We neurologists are just now learning to use the stethoscope, and the hemodynamics of the arteries in the thorax, neck, and head are still a mystery to us.

On the other hand, many internists have an oversimplified concept of the aortocranial circulation, considering it to be analogous to the coronary and peripheral vascular beds. Phrases like "cerebral angina" and "intermittent claudication of the brain" have crept into their vocabularies. Tempting as it is, this holistic concept is dangerous, because the cerebral circulation is unique in many ways. One of its distinctive features—interarterial shunts—is the subject of this paper.

Adapted from the Twenty-sixth George E. Brown Memorial Lecture delivered at the meeting of the American Heart Association, Miami, Florida, October 15 to 17, 1965.
SHUNTS IN CEREBRAL CIRCULATION

For convenience I have divided these arterial anastomoses into intracranial and extracranial varieties, but the two do not act separately in the body and one compensates for deficiencies in the other. Together they constitute the only means for transferring large volumes of blood from one hemisphere or lobe of the cerebrum to another.

Intracranial Shunts

Normal shunts take place through leptomeningeal (surface conducting) arteries such as the anterior, middle, and posterior cerebral arteries and their pial branches or through the circle of Willis. In rare cases persistent embryonic anastomoses between the carotid and vertebral basilar systems form still another interarterial shunt. All of these arteries and their anastomoses lie on the surface of the brain where they form an interconnecting network containing a pool of blood which flows into the brain parenchyma according to its needs.

Systemic blood pressure is transmitted into these arteries, and pressure differences among branches of the surface network equilibrate with one another. Even though mean arterial blood pressure may fluctuate widely, so long as the changes are not too abrupt total flow through this system remains remarkably constant at about 55 ml/100 g of tissue per minute when mean arterial pressure ranges between 70 and 200 mm Hg. Therefore, blood from this pool arrives in the capillary network at constant flow and perfusion pressure.

Most authorities attribute this remarkable autoregulation of the cerebral circulation to diffuse change in the caliber of arterioles interconnecting the surface arteries and the capillary bed. A few contend that it is effected by sphincters located at the origin of small arteries and large arterioles. Whatever its mechanism, there is ample evidence that this autoregulatory capacity normally responds both to systemic stresses and to regional stimuli.

It is suspected that smaller branches of the surface arteries change their caliber in response to neuronal activity, but there is little to suggest that shunting of blood from one to another branch of the same artery occurs under normal conditions. However, this possibility has not been completely excluded, because precise methods for detecting regional flow in man have not yet been devised.

Angiograms performed on the immobilized patient with face front show that the two carotid arteries and the vertebral-basilar system function independently despite the rich interconnections described above. Yet change in pressure relations between the two carotid arteries, or between the vertebral-basilar and the carotid system, causes blood to shift immediately into the system with the lower pressure. Because head rotation alters flow through the vertebral and probably the carotid arteries, it may reasonably be suspected that the areas of intracranial distribution of these arteries vary when the head is turned. Once more, however, there have been no investigations of this possibility.

When one or both carotids are occluded by disease, remarkable intracranial shunts can develop to protect the brain. One example is illustrated by the following case.

A 49-year-old man (Hospital of the University of Pennsylvania, case no. 20 17 81) was evaluated for recurrent episodes of transitory neurological deficit which had begun in 1951 when he was 39 years old. The first manifestation was right facial paralysis, and after a customary neurological evaluation he was diagnosed as having multiple sclerosis. Subsequent to this he experienced episodic right hemiparesis, dysphasia, and dyslexia, and in 1960 episodes of left hemiparesis at which time his wife began to notice intellectual deterioration.

Over the years he had been examined by several physicians all of whom concurred in the diagnosis of multiple sclerosis.

On examination in the hospital he had bilateral spastic hemiparesis and hesitant speech but he was lucid and oriented. His recall of the recent and remote past was adequate to testing even though his wife averred that his memory had deteriorated. Pulsation was stronger in his left superficial temporal artery than in the right. Pulsations in his carotid arteries in the neck, the subclavians, and the radial arteries seemed to be intact. He had a grade III holosystolic murmur.
over the region of the origin of the left vertebral artery from the subclavian. This murmur was transmitted up the left vertebral artery.

Blood pressure in the right arm was 160/100 mm Hg while that in the left was 120/90. Ophthalmic artery pressures were 60/10 g in the right eye and 20/0–10 in the left. The internal carotid artery in the pharynx could not be palpated.

Because of the low ophthalmic artery pressures, a tentative diagnosis of bilateral carotid artery disease was made, and arteriography of the aortic arch and great vessels was carried out through a retrograde femoral catheter. This demonstrated occlusion of both internal carotid arteries and very large vertebral arteries which appeared to be free of atherosclerosis. The origins of the innominate, left common carotid, and left subclavian arteries appeared to be normal. There was no evidence of a subclavian steal.

To delineate the intracranial ramifications of the aortocranial arteries, percutaneous puncture of his common carotid arteries (figs. 1 and 2), and the left vertebral artery were carried out (figs. 3 to 5).

Drs. Christian Lambertson and Harry Wolman performed studies of the cerebral blood flow and cerebral oxygen consumption. The results are tabulated in table 1. Strangely enough, cerebral vascular resistance was low despite the bilateral carotid occlusions and cerebral blood flow increased remarkably in response to the inhalation of air containing 5% carbon dioxide possibly due to increase in systemic blood pressure and cerebral vasodilatation.

In addition to proving that occlusion of extracranial portions of both carotid arteries need not affect the capacity for normal cerebral blood flow, this case demonstrates

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**Figure 2**

Left internal carotid artery is occluded just distal to its origin.

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**Figure 1**

Arteriogram of right common and external carotid arteries. Right internal carotid is occluded just distal to its origin.

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**Figure 3**

Right vertebral artery opacified. Early phase demonstrating dilated vertebral and basilar arteries. Note the large posterior communicating arteries.
the phenomenal extent to which intracranial shunts can compensate for extracranial occlusive disease.

We have shown that in normal men under general anesthesia each carotid artery carries about 300 ml of blood per minute and the two proximal vertebrais together carry about 100 ml per minute.\textsuperscript{15,16} When one internal carotid or vertebral artery is occluded in the neck, flow through the other one immediately increases by more than 50%—undoubtedly because of intracranial arterial shunting through the channels just described. Flow also increases somewhat in the vertebral system if one carotid is occluded and vice versa. However, this potential for shunting in response to arterial occlusion depends upon two factors: (1) the pattern of the surface network of arteries laid down within 90 days after conception, and (2) the vagaries of atherosclerosis which may destroy segments of the anastomotic system throughout the remainder of one’s life. Variations in the circle of Willis are known to all of us.\textsuperscript{17} If the posterior communicating arteries are congenitally small or if they are destroyed by disease, there can be no compensatory shunting between carotid and vertebral basilar systems, and blood must take a more circuitous and probably less satisfactory route through the leptomeningeal system.

\textbf{Extracranial-Intracranial Shunts}

When the circle of Willis is incompetent, the extracranial-intracranial channels can substitute to a certain degree.\textsuperscript{18} There are three principal anastomotic groups:

1. External to internal carotid arteries
   a. Through the orbit
   b. Through the remains of the rete mirabile which join the meningeal arteries of the external to the leptomeningeal (surface conducting)

2. External carotid to vertebral-basilar artery through the occipital branch of the external carotid

3. Subclavian-vertebral to vertebral arteries
   a. Of the same side bypassing segmental obstruction
   b. To the opposite side

This system develops in response to disease or unusual stresses. Because of a general lack of interest in these extracranial portions of
## Table 1

**Results of Studies of Cerebral Blood Flow and Cerebral Oxygen Consumption on a Patient, 49 Years of Age**

<table>
<thead>
<tr>
<th></th>
<th>Breathing air</th>
<th>Breathing 5.5% CO₂ in air</th>
<th>Expected normal values for patient's age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cerebral hemodynamics and metabolism</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebral blood flow (ml/100g/min)</td>
<td>39.8</td>
<td>58.3</td>
<td>49</td>
</tr>
<tr>
<td>Cerebral oxygen consumption (ml/100g/min)</td>
<td>2.99</td>
<td>3.00</td>
<td>3.0</td>
</tr>
<tr>
<td>Cerebral vascular resistance (mm Hg/ml/100 g/min)</td>
<td>1.85</td>
<td>1.35</td>
<td>2.2</td>
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<tr>
<td>Arterial-venous O₂ Δ (ml/100 ml)</td>
<td>7.5</td>
<td>5.2</td>
<td>6.5</td>
</tr>
<tr>
<td>Mean arterial blood pressure (mm Hg)</td>
<td>80</td>
<td>89</td>
<td></td>
</tr>
<tr>
<td>Jugular venous blood pressure (mm Hg)</td>
<td>6.5</td>
<td>10.5</td>
<td></td>
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<tr>
<td>Cerebral respiratory quotient</td>
<td>0.96</td>
<td>0.92</td>
<td></td>
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<tr>
<td><strong>Arterial blood</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O₂ content (cc/100 ml)</td>
<td>18.1</td>
<td>19.3</td>
<td></td>
</tr>
<tr>
<td>O₂ capacity (cc/100 ml)</td>
<td>19.2</td>
<td>19.2</td>
<td></td>
</tr>
<tr>
<td>% Hb saturation</td>
<td>93.2</td>
<td>99.0</td>
<td></td>
</tr>
<tr>
<td>P O₂ (mm Hg)</td>
<td>72.4</td>
<td>&gt; 140</td>
<td>90 (Breathing air)</td>
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<tr>
<td>CO₂ content (cc/100 ml)</td>
<td>45.7</td>
<td>48.8</td>
<td></td>
</tr>
<tr>
<td>P CO₂ (mm Hg)</td>
<td>40.2</td>
<td>46.1</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.366</td>
<td>7.336</td>
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<tr>
<td><strong>Internal jugular venous blood</strong></td>
<td></td>
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<tr>
<td>O₂ content (cc/100 ml)</td>
<td>10.6</td>
<td>14.1</td>
<td></td>
</tr>
<tr>
<td>O₂ capacity (cc/100 ml)</td>
<td>19.2</td>
<td>19.2</td>
<td></td>
</tr>
<tr>
<td>% Hb saturation</td>
<td>54.7</td>
<td>72.9</td>
<td></td>
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<tr>
<td>P O₂ (mm Hg)</td>
<td>31.1</td>
<td>43.2</td>
<td>40</td>
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<tr>
<td>CO₂ content (cc/100 ml)</td>
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<td>P CO₂ (mm Hg)</td>
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<tr>
<td>pH</td>
<td>7.316</td>
<td>7.296</td>
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<tr>
<td><strong>Mean brain capillary blood</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P O₂ (mm Hg)</td>
<td>45.1</td>
<td>65.8</td>
<td>60</td>
</tr>
</tbody>
</table>

The aorticranial circulation, the factors influencing the volume of blood shunted through them have not been studied until very recently. Except for the carotid sinus, they have not been studied extensively by physiologists, and even at autopsy pathologists seldom examine their entire length. Clinicians, on the other hand, are as a rule even less knowledgeable because our specialty training has caused us to divide these arteries artificially into thoracic portions (cardiology and internal medicine), cervical (vascular surgery), and intracranial (neurology and neurosurgery). As a result, only a handful of investigators are conversant with the anatomy and physiology of the carotid and vertebral-basilar systems from the thorax to their intracranial capillary beds.

Until 5 years ago, it was generally thought that the cervical segments of the carotid and vertebral arteries functioned passively as conduits for blood coursing through the neck from the heart to the brain. Then Tucker and I²⁹ drawing on ideas expressed by Gegenbauer²⁰ performed studies on cadavers which suggested that the flow of blood through the vertebral and possibly through the carotid arteries can be altered by extension or rotation of the head. Our demonstration of this phenomenon in cadavers did not provide proof of this occurrence in normal man nor did our subsequent studies using flowmeters on the carotid and vertebral arteries of...
anesthetized men. Others have demonstrated this effect of head movement on blood flow by angiography, but this technique does not give a true concept of the dynamics of the change.

Within the past 6 months we have studied a patient whose left vertebral artery became obstructed whenever he turned his head to the right. The patient had platybasia, a congenital anomaly of the first cervical vertebra and the base of the skull. Whenever he turned his head to one side or looked upward, he experienced vertigo and ataxia. Cineangiography and arteriography revealed hypoplasia of his right vertebral artery, which ended in the midcervical region (fig. 6). Although his left vertebral artery was normal in caliber and was patent when his face was front or turned to his left side, it became obstructed at the level of the fourth cervical vertebra whenever he turned his head to the right. At this time the blood supply for his posterior fossa must flow from his carotid system because of his hypoplastic right vertebral artery. Customary face-front angiograms of the left vertebral artery would not have demonstrated the true cause for his symptoms but cineangiography during head turning did.

**Intracranial-Extracranial Shunts**

Up to this point we have considered shunts through the leptomeningeal arteries over the surface of the brain and through extracranial-intracranial anastomoses, all of which act to protect cerebral circulation. In addition to these local shunting mechanisms, there is a more general one which protects the brain. In his Brown Memorial Lecture in 1952, Starr described it beautifully:

> There is normally a continuous shifting of blood flow from one part of the body to another, a process which they (Dr. George Burch and his associates) dignify with the term hemo-metakinesia, and which they regard as serving the useful purpose of supplying the organs with blood according to their varying needs, with the greatest economy of effort. We still lack knowledge about shifts of blood flow between the peripheral organs in which it can be measured easily, and the abdominal organs in which, with the possible exception of the kidney, blood flow can be measured only with difficulty or not at all in man, but we have every reason to expect that an exchange of blood flow, a borrowing-lending process similar to that demonstrated in man for two parts of the periphery, will also take place between the periphery and the deep organs as many animal experiments indicate.

Until 4 years ago, physiologists and clinicians believed that this borrowing-lending process always served to protect the brain; but in 1961 Reivich and associates described a situation in which borrowing-lending worked to the detriment of the brain. It was given the colorful name “subclavian steal,” a most appropriate term when we consider that the borrowing-lending process is the normal transaction and the steal is pathophysiological or antisocial.

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*Studies performed by Drs. Richard Janeway and Henry S. Miller will be reported in detail in a separate communication.

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Subclavian steal occurs in patients whose anatomic configuration permits a reversal of flow through one vertebral artery when the proximal portion of the subclavian artery is stenosed or occluded (fig. 7). In addition to instances of such one-sided reversal, pathological siphons down both vertebral arteries and also through the right carotid artery have been documented. Of the more than 200 reported examples of this disorder, most have resulted from atherosclerosis, but some are congenital or caused by trauma or surgery.29

Once again, it is the anatomic configuration of the arteries laid down within the first 90 days after conception that determines whether a steal can develop. If the innominate artery, for example, is extremely short or nonexistent,30 so that the right subclavian and common carotid arteries arise independently from the arch, no carotidosubclavian shunt can develop. If the left vertebral artery arises from the arch rather than from the subclavian artery, as it does in approximately 1% of individuals,31 obstruction in the right subclavian or the innominate artery can cause reversal of flow, but occlusion of the left subclavian artery cannot.

When reversal of flow does develop, the vascular reserve of the brain determines whether or not it produces symptoms. If the normal intracranial shunting mechanisms previously described are adequate, a pathological siphon causes no symptoms. Obstruction located in the proximal subclavian artery is easily tolerated if the opposite vertebral artery is patent, because diversion of blood down the vertebral artery is accompanied by increased cephalic flow on the other side.

With increasing age, however, the deposition of atheromatous plaques in many locations usually results in varying degrees of reduction in cerebral vascular reserve. Let us consider the vertebral-basilar circulation as an example. In an adult, approximately 100 ml of blood per minute normally flows through the two proximal vertebral arteries to supply muscles of the neck, portions of the spinal cord, the entire brain stem, the cerebellum, and portions of the temporal lobe, as well as the entire occipital lobe, of each cerebral hemisphere. If these arteries are atherosclerotic and compressed in their foramina by osteoarthritic spurs, the blood flowing through them will be under decreased pressure and may be decreased in volume. If, in addition, the posterior communicating arteries are congenitally small, the already compromised vertebral-basilar system is isolated from the carotid circulation and will be unusually susceptible to factors (such as hypotension) which diminish cerebral blood flow. Compensatory dilatation of the carotid bed, no matter how great, cannot prevent hypovolemia and hypotension in the isolated vertebral-basilar system.

Furthermore, the vertebral-basilar circulation is in competition with the vascular bed of the upper extremities for blood pressure and flow. These two beds, one cerebral and the other musculoskeletal, are regulated independently and sometimes in reverse directions. When the muscular bed dilates in response to heat, emotional experience, and exercise of the limbs, flow to the posterior

Figure 7

Diagram of subclavian steal. Stenosis or occlusion of subclavian artery proximal to the origin of the vertebral may result in reversal of vertebral artery flow.
fossa is kept constant, under physiological conditions, by regional vasodilatation. In patients with atherosclerosis, however, this vasodilatation may not occur and factors that increase the flow of blood to the limbs may decrease that to the basilar artery and its branches, precipitating symptoms of ischemia.

Only a hint of the dynamics of this interplay has been provided by rapid serial angiography. Cineangiography, using selective catheter technique, once again provides a method for seeing the dynamic situation. The rapid transit of contrast material up one vertebral artery, down the opposite artery, and out into the arm is dramatic, but less so than situations in which proximal portions of both subclavian arteries are stenosed. In such cases, the bolus traverses the carotid circulation of the two sides, flows through the posterior communicating arteries, and passes in reverse direction down the basilar artery, down both vertebral arteries, and out into the two arms (fig. 8). The entire brain then becomes dependent upon the upper limbs for its circulation. When the arms are exercised, the volume of reversed flow increases, and if, because of disease, the cerebral arterioles cannot dilate quickly, cerebral perfusion pressure falls and the patient has symptoms caused by ischemia in the cerebrum, the brain stem, or both.

Discussion

In this survey I have attempted to develop the concept that the production of symptoms in patients with occlusive forms of cerebral vascular disease depends to a large extent upon the matrix of vessels laid down within the first 90 days of intra-uterine life and upon the atheromatous material deposited in these vessels during the next ensuing years. I have illustrated anomalies of the aortic arch and have shown what happens in the cerebral circulation of a patient with a hypoplastic right vertebral artery who obstructs his left vertebral artery by turning his head. Finally, I have mentioned the frequent variations in the circle of Willis which can prevent cross-circulation in the cranium.

Unfortunately, there have been no studies
in which anomalies of the aortic arch, the cervical segments, and the circle of Willis have been considered as a unit. Hence, I must ask you a question which I have asked myself: Are anomalies of one area frequently associated with vascular anomalies elsewhere? Since the whole circulatory system is laid down during the same period of embryogenesis, one might suspect that they would be. If they are, do any of the teratogenic agents recognized for other organ systems affect the cerebral arterial system, and do we inherit our parents’ vascular anatomy just as we inherit their facial characteristics?

In order to consider in specific terms some of the multitude of factors affecting the cerebral circulation, let us study a hypothetical case.

The patient, a vigorous 65-year-old man, has moderate atherosclerosis and diabetes. The blood pressure in his right arm is 200/100 mm Hg, while that in his left is reduced to 140/90 because of a plaque in the proximal portion of his left subclavian artery which causes reversed vertebral flow.

After a good night’s sleep in a prone position with his head turned sharply to the left (a posture which may diminish blood flow through his right vertebral and carotid arteries), our patient is awakened by his alarm clock and opens his eyes. The sudden sound and light cause an increased flow of blood to his auditory and visual pathways. With customary gusto, he arises to start the new day. Because his mild diabetes has produced neuropathy of the lower extremities, however, his vasomotor reflexes are lost, and the normal borrowing-lending process does not occur; hence, blood pressure and cerebral blood flow fall somewhat when he stands up. At the same time the blood pressure difference between the two arms is increased, so that the volume of reversed flow is augmented. The resultant reduction to cerebral blood flow, superimposed on his cerebral atherosclerosis, causes our patient to stagger and lose his vision momentarily and he decides to sit on the bed until he recovers himself.

After a few minutes’ rest has restored his sluggish circulation, the patient walks to the bathroom and, because of mild prostatism, strains somewhat to void. This Valsalva maneuver abruptly diminishes his cardiac output and perhaps precipitates momentary cerebral ischemia. Recovering from this, he begins to shave with his electric razor, massaging his carotid sinus while turning his head far to one side. The massage may initiate a cardio-inhibitory or vaso-depressor reflex, while at the same time head turning reduces flow through the vertebral and carotid arteries. I dare not allow our patient to do his usual push-ups after shaving, because I fear that the tremendous increase in reversed flow through his vertebral siphon might again produce symptoms of vertebral-basal ischemia.

Nor will I have him take a hot shower, which would cause cutaneous vasodilation and initiate hemometakinesis. These stresses occurring in the first half hour of his day are but a few of the enormous number for which our patient’s cerebral circulation must compensate minute by minute during his life.

Even the common situations described in this hypothetical case are so complex that no one can master all the variables. Like the blind men describing the elephant, each of us specialists—internist, neurologist, physiologist, surgeon—describes accurately his own segment of this magnificently complicated circulatory bed. The image that each of us has, however, is only a fragment of the whole. To map out the complete picture, we must do with the cerebral circulation what it is said that Dr. George Brown did with the peripheral:

Recognizing his limitations in certain directions, he enlisted the aid of physiologists, physicists, pathologists, surgeons and clinicians who could contribute to the process of enlightenment in the field of peripheral vascular diseases. At the same time while he was working as a student and investigator of peripheral vascular diseases, he continued to be active as a clinician in the broader field of Internal Medicine.

References
SHUNTS IN CEREBRAL CIRCULATION

Interarterial Shunts in the Cerebral Circulation

JAMES F. TOOLE

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