The System Approach to Brain Blood Flow

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The clinical approach to ischemia of the brain is burdened by practices established by custom and by conflicting terminologies and perspectives tailored to the individual needs of diverse medical specialties. There is a need for a common terminology resting on basic definitions and concepts that precisely reflect anatomical, physiological, and pathological states. The integration of multiple disciplines is necessary to understand evolving practices.

By accurately delineating the structure of the brain, the CT scan has markedly changed the practice of medicine. Pneumoencephalography has been abandoned. A minute brain infarction can be localized. Intracranial bleeding can be diagnosed without lumbar puncture. Clinical judgment yields to more precise information. Brain structure is yielding to brain blood flow because blood flow is more closely related to function (73,143,144), and because abnormalities of blood flow precede structural changes (67,88,119). The reversibility of neurologic deficit can be anticipated if brain structure is preserved (CT scan) and ischemia is identified by either noninvasive (25) or regional perfusion studies (22,60,104,127) and corrected.

The measurement of brain perfusion (44), which has recently become available to the authors, has added a new dimension to our approach to problems of brain ischemia. The angiogram is limited to its role as an anatomical study with serious limitations even in this area (94,114). The complexity of brain blood flow and the need to "rethink" basic concepts have become apparent.

BRAIN BLOOD FLOW AND PERFUSION

Our fundamental concern is blood flow to the brain, not only the blood flow through the arteries of the carotid and vertebral system, but also the blood flow through the tissues of the entire brain, the cerebral hemispheres, the deep nuclei, the brainstem, and the cerebellum. Blood flow through arteries must be carefully distinguished from tissue perfusion. Blood flow through arteries is expressed in volume flow per unit of time and is not associated with metabolism. When tissue is perfused, blood courses through capillaries where metabolic exchange takes place and perfusion is expressed as volume flow per unit of tissue per unit time. In arteriovenous malformation, the blood flow in the arteries may be high while the tissue perfusion is low.
Circulation involves, at the very least, tissue perfusion and blood flow in the arteries and veins. *Blood flow within the carotid and vertebral systems is interdependent* (11, 27, 104, 111, 135) by virtue of the circle of Willis, and it follows that *neither one hemisphere nor one system can be validly studied in isolation*. Intracranial stealing (77, 136, 155) is common with cerebral artery occlusion, arteriovenous malformation, and tumors (49). Stealing is always created by the surgical clamping of the carotid artery so that blood flow through an artery in the neck need not perfuse its customary intracranial vascular bed.

Perfusion of the brain is pathological when it compromises brain function, structure, or growth and development. Reduced brain perfusion, i.e., ischemia, as well as excessive perfusion may be pathological.

In general, the function of the brain is directly related to tissue perfusion. Progressive reduction of perfusion results in the orderly deterioration of function first, then structure. Optimal perfusion may deteriorate two-thirds before electrical dysfunction is noted. Metabolism is grossly disturbed when perfusion falls to 20% of optimal levels and cell death occurs shortly thereafter. What is significant is the wide range between optimal perfusion and cell death, and the narrow range between hypoperfusion and cell malfunction (73, 88, 143). The variables which may modify the course of events include the metabolic activity of the cell, the function considered, the duration and type of stress, and the age of the individual.

Cerebral autoregulation (96, 100, 104) is the ability of the brain to maintain its tissue perfusion at a relatively constant level irrespective of moderate changes in the circulation. Brain infarction (153), tumor, ischemia (96), diabetes (14), and aging (108, 162) may result in loss of autoregulation so that mild hypotension causes profound ischemia or hypertension results in hemorrhagic infarction (7). The loss of autoregulation is also associated with long-standing vertebral-basilar insufficiency (109), a factor that must be appreciated when contemplating vertebral artery reconstruction.

**THE SYSTEM AS A SYSTEM**

The concerns of this chapter are the factors that affect blood flow to the brain and perfusion of the brain. These factors may lie within the brain and the arteries supplying it or within the cardiovascular system. From this point of view, the cerebral vascular system consists of the brain, the arteries to the brain, the heart, and the blood (Fig. 1). The System Approach can be used to detect troubles that critically affect brain blood flow and for which a remedy may be available.

The cerebral vascular system is a part of the cardiovascular system and both share characteristics and elements in common. Characterization of the cardiovascular system as a system permits insight into patterns of disease. Often the clinical examination permits classification of the system as a system, separating the young from the old, the sick from the well, the volume-depleted from the plethoric, the aneurysmal from the occlusive, and the high-flow from the low-flow systems.

Life is dynamic with constant stress to the system. Falls in adults (32) and even in children (84) might be expected to provoke serious consequences, but the ordinary activities of eating (2, 129), standing erect (61, 92, 139), or turning the head (20, 58, 70, 74) may also cause pain.

![FIG. 1. The system approach to brain blood flow. The system consists not only of the brain and the arteries to the brain but also those factors that may critically affect regional brain perfusion, including the heart and blood. Depleted plasma volume often reduces cardiac output and brain perfusion, whereas overexpansion improves cardiac output and perfusion of the ischemic brain.](image-url)
dizziness, blurred vision, or syncope for which medical care is sought. Patients rarely can be examined under the exact circumstances which provoke their symptoms. But examination only when supine, at rest, and with the head in neutral position is not realistic.

Gravity

Man walks erect and blood flow to the brain must be maintained against gravity. The upright position can have great impact on cerebral perfusion (71,124,133). Blood flow and blood pressure in the internal carotid artery during the normal activities of walking, running, standing, bending, and lying down have been measured invasively in the wild giraffe (154,156), but never in man. Intracranial tumors (126), cerebral infarctions (100,104,120), and traumas (28) that disturb brain autoregulation increase sensitivity to the upright position and to cerebral hypotension.

In man, noninvasive hemodynamic and brain perfusion studies are, with few exceptions (27,109), performed at rest, supine, and without stress (17,81,89,144). In the upright position, cardiac output ordinarily falls (61). Arterial obstructions of the neck which are position dependent (13,89,152) may be even further aggravated when the subject is upright (99).

Symptoms of brain ischemia may not be related to brain blood flow alone but may be modified by other factors. In human subjects experiencing the graded stress of gravity in the centrifuge, dizziness does not occur prior to syncope as long as visual orientation is maintained. The brachial artery pressure may not be a reliable guide to brain perfusion pressure. In the centrifuge, cessation of blood flow in the temporal artery accurately predicts impending syncope, but does not correlate with the systemic blood flow as reflected by radial artery blood flow or the brachial blood pressure (118,121). Similar observations have been made in patients (23).

Clinically, patients who are vulnerable to the upright position can be detected simply by use of the tilt table.

BLOOD-THE COMMON DENOMINATOR

Blood is a tissue with complex metabolic and physical roles in changing vascular compartments. Our concerns are with plasma volume, red blood cell mass, viscosity, and micro-emboli.

Plasma Volume

Blood volume depletion has long been recognized as a contributing factor to hypotension. In recent years, diuretics that dehydrate by depleting plasma volume have been used to control hypertension. When plasma volume depletion is excessive, cerebral perfusion may be severely compromised by the reduction of cardiac output (61) and by the increase in blood viscosity.

Red Blood Cell Mass and Increased Viscosity

Elevation of the hematocrit reading and increased blood viscosity have been associated with an increased incidence of thrombotic strokes (147,148,151). Chronic obstructive pulmonary disease and elevated carboxyhemoglobin levels (137) due to smoking are common problems. Neurological symptoms may be relieved by the reduction of red blood cell (RBC) mass and blood viscosity. This may be achieved by whole blood donations, isovolemic whole blood removal, or RBC removal with a centrifugal cell separator (80) (Haemonetics Corp., Natick, Mass. 01760).

CASE E. A.
Isovolemic hemodilution and surgery within 48 hr-1,000 cc whole blood removal

A 49-year-old white man was admitted for carotid and vertebral artery surgery. Past history: right thoracotomy and decortication for postpneumonitic empyema. Cigarettes: 2 packs per day. Hemoglobin 17.3 and 16.5 g/100 ml. Carboxyhemoglobin (COHb) was 14% of all hemoglobin (normal: non-smoker 0.5 to 1.5%; smoker 4 to 5%). One unit of whole blood was removed while volume was re-
placed with 750 cc of albumin and electrolyte solution. The hemoglobin at the time was 14.8 g/100 ml. After the second unit of whole blood was removed and the volume replaced, the repeat hemoglobin was 13.1 g/100 ml. The following day, the hemoglobin was 12.5 g/100 ml. The patient underwent uneventful surgery 48 hr after removal of 1,000 cc of whole blood with plasma volume replacement.

Reduction of Normal Viscosity—Cerebral Vasospasm

Brain ischemia due to profound cerebral vasospasm (J64) has been associated with subarachnoid hemorrhage and intracranial aneurysm surgery (J34). An aggressive approach to increase blood flow through the high-resistance arteries has been based on the reduction of normal blood viscosity by plasma expanders (J34) and the increase of cardiac output by volume loading (J13). Both principles have been applied to vascular procedures as well (J21, J91, J132). Symon (J43) has advocated monitoring the central venous pressure as a guide to maintaining blood volume for 5 to 14 days following surgery for cerebral aneurysms.

The place of plasma volume expansion in the management of acute neurological episodes or in the preoperative preparation of the precarious patient undergoing neurovascular surgery remains to be defined. Although plasma volume expansion may be an alternative to the use of intraoperative shunts during carotid surgery, it has, in our experience, been critical to the safe conduct of vertebral artery reconstructions where shunting is not possible.

Microembolism

Although the diagnosis of embolism to the retinal artery can be verified by fundoscopy, the diagnosis of microembolism to the brain is less certain. Control of symptoms presumes that the pathological process is controlled (Fig. 2).

Recognition of the significance of microembolism was crucial to the development of carotid artery surgery (J47, J51), which has estab-
lished its effectiveness in the control of such embolism, a reputation acknowledged even by ardent advocates of other forms of therapy (10). But embolism within the vertebral-basilar system is another matter, for the diagnosis has not been as simple and the alternative of surgery has not been tested.

Embolism within the vertebral artery selectively seeks out the posterior cerebral artery because of its terminal origin from the basilar artery (140). The acute onset of visual field defects and focal lesions of the occipital lobe on CT scan (Fig. 3) are consistent with microembolism. Although the origin of the emboli may be ulcerations, more often it is the intima at the site of acute kinks where intimal damage occurs due to shear stress as suggested by Blaumanis et al. (16).

CASE A. E.
Acute hemianopia-Occipital lobe infarction
Embolism from vertebral artery at C-2
Vertebral artery surgery

A 68-year-old white man presented with acute right homonymous hemianopia. For the 3 days prior to admission, the patient had painted the basement ceiling with his head hyperextended. On the morning of the fourth day, he noted loss of the right visual field. Angiogram revealed acute constriction of the vertebral artery at C-2. At surgery, a platelet aggregate thrombus was found at this site of acute angulation. Segmental resection and end-to-end anastomosis were performed. Twenty-two months later, the reconstruction was satisfactory and no embolism had recurred. The hemianopia was unchanged.

Although the defects of the vertebral-basilar system (33,55,63,69,167) and surgical corrections (15,26,29,36,63,159) have been described, the development of the bypass to the distal vertebral artery, posterior inferior cerebellar artery (PICA), and anterior inferior cerebellar artery (AICA) has provided added incentive to pursue the precise diagnosis. The potential application is only now becoming appreciated (28). Impetus in this direction has been added by the development of dynamic angiography and brain perfusion techniques; but more important, the better understanding of vertebral symptoms (66) and brain hemodynamics and the utilization of neurophysiologic diagnostic techniques have stimulated interest.

THE HEART IS A PUMP

William Harvey (65) approached the heart as a pump. Primarily the mechanical effectiveness of the heart, rather than its electrical activity, is relevant to cerebral perfusion. The ECG cannot assess left ventricular function or cardiac hemodynamics (Clauss, Chapter 16).

Syncope and dizziness can be quantitatively related. Cardiac dysrhythmia can give rise to either symptom, depending on the severity of the dysrhythmia, the degree of associated obstruction of the arteries to the brain, and the pumping capacity of the heart.

The vertebral artery is especially sensitive to reductions of cardiac output. Vertebral-basilar artery symptoms may be the first to herald the failing
heart. On the other hand, both vertebral artery reconstruction and cardiac management often are required for the relief of vertebral-basilar symptoms.

CASE C. W.
Stokes-Adams Syndrome due to bradyeardia and vertebral artery occlusion
Treatment: Right ventricular pacing, vertebral artery bypass, and atrial ventricular sequential pacing

A 65-year-old physician presented with syncope associated with bradyeardia. A right ventricular pacemaker relieved the bradyeardia but precipitated intractable vertigo which he had never before experienced and which occurred only when he was upright and when the pacemaker fired. The amplitude of the ocular pulse of the paced beat was 60% of that of the sinus beat. Subclavian proximal vertebral artery bypass provided relief of vertigo. Three years later, petit mal-like episodes were associated with RV pacing. Atrial pacing with an atrial ventricular sequential pacemaker restored the amplitude of the paced beat to that of sinus rhythm with relief of symptoms (27) (Fig. 4).

Comment: The electrocardiogram provides limited insight into the heart as a pump. Syncope associated with bradyeardia may not be solely of cardiac origin. Vertebral artery obstruction increases the sensitivity to reduced cardiac output. Vertebral revascularization may reduce this sensitivity but does not alter the cardiac status.

Right ventricular pacing increases the heart rate but may significantly decrease the stroke output (31). In most it is tolerated; in a few it is catastrophic (62). In some the dramatic symptom of syncope may be replaced with dizziness and ataxia. To what extent the increased venous pressure raises intracranial pressure and resistance to arterial inflow is uncertain (4, 6, 163). Atrial pacing increases heart rate and maintains the stroke output (131). The sicker the heart, the more the atrial contribution is needed.

ARTERIES TO THE BRAIN

Pathology

In man, the primary pathology of the artery arises from the media or the intima. Intimal

FIG. 4. Worsening of stroke output by right ventricular pacing. In this case, C.W., atrial pacing relieved pacemaker symptoms. Volume-calibrated ocular pulse recordings with the ECG and common carotid pulse tracing taken in a continuous strip. A: Normal sinus rhythm-normal carotid pulse contour. B: Atrial pacing-physiologic QRS complex with ocular and carotid pulses comparable to those of normal sinus rhythm. C: Right ventricular pacing-grossly altered QRS complex, significantly reduced ocular pulse amplitude.
diseases include atherosclerosis, hyperplasia, ulceration, and shear injury. *Media degeneration* is associated with loss of elasticity, dilatation, elongation, and the formation of kinks and aneurysms. Obstruction may also be produced by *extrinsic* constriction and compression. Arterial *trauma* includes blunt and penetrating injury, traction, intracranial aneurysm surgery, and subarachnoid bleeding. Catheterization or contrast irritation may result in vasospasm, intimal tears, dissection, aneurysm formation, thrombosis, and embolism.

The *intimal thickening* of atherosclerosis tends to develop at sites (42,88,166) of turbulent flow, where the direction of flow is altered and kinetic energy is converted into thermal energy. The atherosclerotic process involves a long segment, is static or slowly progressive, and gradually occludes the vessel, which increases tolerance to the obstruction. A sustained pressure gradient fosters the development of a collateral system. When the collateral system is not adequate, the pressure gradient persists unchanged. The existence of this constant pressure gradient makes microvascular surgery possible. Yasargil feels that the high gradient fosters flow so the grafts do not become occluded early and permits the bypass to dilate and mature in size (103).

*Degeneration of the media* results in the loss of elasticity and dilatation and elongation of the arterial system. The arteries of the young are thick walled with excellent elasticity. The most extreme of head and neck positions are accommodated. But as the body ages, the elasticity is lost, the arterial tree increases in size, and the hydraulics of blood flow are markedly altered (48). The points where nerves encircle or are attached to the artery are points of fixation for pivoting, rotation, kinking, and angulation. Dilatation of an artery beneath a nonyielding nerve produces constriction. Elongation of an artery between two points of fixation produces a kink in the midportion (39,157). Head motion can produce not only intermittent obstruction but also wear and tear at the site of maximum motion.

The *dynamic* obstruction differs from the static obstruction in many respects: the dynamic obstruction is of short length, its severity is not constant, and pressure gradients are fleeting. Altered head position can produce acute catastrophic symptoms, and these symptoms can be completely relieved with correction of the head position. Because of the obstruction's inconstant nature, *collaterals do not develop* and reconstruction must be with a full-capacity flow system because the pressure gradient is intermittent and a low-flow microvascular reconstruction will fail. Malfunction usually precedes catastrophe, so that acute aggravation is usually superimposed on some degree of existing obstruction, which may proceed to occlusion.

Although *extrinsic constriction and compression* are well accepted as causes of neuropathy, arterial compression and entrapment by bone, tendon, nerve, or inflammatory disease are poorly appreciated. The hypoglossal carotid entrapment syndrome (Carney and Anderson, *this volume*) and the pathology of the vertebral artery require further elaboration.

Sites of predilection for trauma to the carotid artery are at the base of the skull and the atlas (12,18,37,141). Sites of entrapment include peritonsillar inflammatory scar and lymph nodes (158), anomalous muscle (101), and the hypoglossal nerve (102,130; Carney and Anderson, Chapter 18). Compression of branches of the vertebral artery may occur intracranially (93), but more often the vertebral artery at the level of C-2 has been widely implicated (3,32,57,87,107,140). Compression by osteophytes within the cervical spine (140) and at the attachment of sympathetics at C-6 (29) occurs, but shearing of the segmental branches during delivery also appears to be common (167).

The dissecting aneurysms of the arteries in the neck (35,52) have not been subjected to thorough analysis for mechanical and hydraulic stresses as the neural structures (20) have been analyzed for mechanical stress.

**Significant Arterial Obstruction-Criteria**

The existence of a pressure gradient, i.e., the difference in pressure between a point proximal
to a stenosis and a point immediately distal to it, is usually considered evidence of significant arterial obstruction (149,160). From this observation has evolved the theory of critical arterial stenosis. The underlying reasoning for its use appears to be as follows: Significant obstructions produce a pressure gradient. Occlusive disease of an artery produces a pressure gradient when angiographic obstruction is equal to or greater than 65 to 70%. Therefore, the angiographic quantitation of the degree of occlusion can identify significant arterial obstruction. The concept is simple and practical but inadequate.

Studies often cited to support this theory were performed with ligation of the common carotid artery for intracranial aneurysm (4,74) in the absence of occlusive disease. Correlation is less impressive when applied to carotid stenosis alone (41). But when the flow of both internal carotid arteries is studied simultaneously during the graded closure of one artery, the importance of the reciprocal increase of flow in the contralateral internal carotid artery and vertebral system becomes more apparent (111).

Cardiac output, blood viscosity, peripheral resistance, collateral capacity, extent of vascular bed (75), and volume of blood flow may markedly modify the pressure gradient (116, 146). The pressure gradient is directly related to flow. For the same obstruction, low flow may produce no pressure gradient and high flow a marked gradient (168).

When a totally occluded artery has well-developed collateral circulation, there may be no pressure gradient and no disability. Clinically, cardiac and vascular stress studies utilize high blood flow states to determine significance of arterial obstruction. The hemodynamic consequences of obstruction, the functional disability, and the threat to life and to tissue are more significant and more accurate than judgments based on the angiogram.

When the criterion of critical arterial stenosis is applied to the highly collateralized cerebral vascular system, it is grossly inadequate because it cannot define the hemodynamic con-sequences to the brain of total occlusions (25,27,14) or of kinks (114; Carney and Anderson, Chapter 25); it cannot be applied to the obstruction of short length (94), to the dynamic obstruction, to the vertebral artery, or to the vulnerable vascular beds. The X-ray appearance of the carotid artery in the neck cannot predict the brain perfusion in the head, irrespective of the obstructing mechanism. The measure of the significance of carotid or vertebral artery obstruction is the impact on arterial flow, brain perfusion, and brain function.

The most common failure is in the application of the "critical arterial stenosis" criterion to the kink and to the vertebral artery. Numerous studies (40,112,114,115,157) have documented the clinical relevance of the carotid kink to cerebral vascular events, but no angiographic criteria for the selection of the hemodynamically significant kink are available because the obstruction of short length often cannot be seen. If the criterion of angiographic critical arterial stenosis is valid, then it should be uniformly applicable to all arteries, but it is not. Stenosis of the vertebral artery is not judged by the same standards as is stenosis of the carotid artery (117).

Reduced regional brain perfusion, i.e., ischemia, is pathological when it compromises function or structure, irrespective of the etiology, the artery supplying the region, or the mechanisms involved. This criterion, the significant reduction of brain perfusion, can be applied to all arteries to the brain and to any mechanism of obstruction without exception. The correlation of noninvasive hemodynamic evaluation with brain function and perfusion studies is described elsewhere (45; Carney and Anderson, Chapter 25; Fuster et al., Chapter 27). Impaired brain function and preserved brain substance with regional ischemia due to arterial stenosis may benefit from corrective surgery. Impaired brain function with global ischemia is due to a system problem and may not benefit from reconstructive surgery irrespective of the degree of arterial stenosis present.
Hemodynamic Significance of the Neck

How does the neck work? The head is mounted on a universal joint, the occipital-atlanto-axial articulation, with a flexible extension from the body, the neck. There exist a wide range of motion and a variable terrain across the head and neck through which four high-pressure arterial lines that are fixed in position by investing fascia, nerves, dura, and bone pass into the rigid skull. These arteries are stretched over bone (18,32,87), pinched by nerves (Carney and Anderson, Chapter 25), and crushed into tight coils with head motion, with hardly an indication to the individual of these dynamic processes.

Is head and neck motion really a problem? It definitely is, but only the field of neurotology directly addresses the impact of head position on the function of the brainstem (9,52,64). Hemodynamic studies have been specifically addressed to symptoms produced by head position (89; Fuster et al., Chapter 27). Although neurological symptoms and pain aggravated by head position are commonplace (9,18,20,58,64,70) and traumatic thrombosis of the cervical arteries is well recognized, there is great reluctance to regard head position as a cause of brain ischemia. Commonly encountered stresses to the neck include head rotation (3), chiropractic manipulation (87), hyperextension of the neck (107), such as in football injuries (128) and intubation during general anesthesia (57), whiplash (64), head position during surgery (34; Fuster et al., Chapter 27), and clamping of the carotid artery. Normally, any extreme Lead position obstructs one or two arteries depending on the direction of the motion by the process of kinking, compression, or stretching. When this occurs, the remaining vessels in-flow to the circle of Willis to maintain blood perfusion. In effect, stealing by one part of the brain from the blood supply of another via the circle of Willis seems to occur a thousand times a day. If this dynamic concept is valid, then peak flow of a carotid or vertebral artery (27,145) is more important than resting flows in determining the significance of arterial obstruction, and the reserve capacity of the cerebral vascular system (105) is a concept to ponder.

The Neck Is Never Autopsied

When a patient dies with a stroke, the heart and the brain are carefully examined, but the neck is never autopsied. Detailed examination of the neck vessels is rare (32,33,69,167). Corrosion specimens are never used to detail the internal structure of arteries of the neck, although they are commonly used elsewhere. The surgeon is the only physician who routinely examines the pathological anatomy of the arteries of the neck of a symptomatic patient in vivo, and his colleagues in medicine and radiology should defer to his anatomical and clinical observations (29,30,112,115). More important, clinical practice is strongly influenced by the conclusions of large review series which are based on the angiogram rather than on actual pathology (50,157). For this reason, the validity of the angiogram must be critically scrutinized.

Validity: Angiogram Versus Pathological Anatomy

CASE A. J.

"Normal" carotid angiogram, cerebral infarction and death

A 67-year-old white man presented a transient right hemiparesis involving the left cerebral hemisphere. Noninvasive evaluation demonstrated obstruction of the left carotid artery. The angiogram (Fig. 5) revealed a kink, a dynamic obstruction, of the left internal carotid artery which was interpreted as "normal" because no discrete obstruction could be seen. The noninvasive test was considered a "false positive." Fifty-nine days following the transient ischemic attack (TIA), the patient developed an infarct in his left cerebral hemisphere. Twelve days later, he was dead.

False Negative Angiogram

The angiographer has no criteria for assigning clinical or hemodynamic significance to "kinks" or dynamic obstructions of the carotid or vertebral artery. Often his judgments and in-
FIG. 5. "Normal" carotid arteriogram-The fatal kink. Carotid bifurcation. Left: Lateral projection. Right: AP projection. In the case of A. J., not correcting a carotid kink was fatal. The obstruction of short length, or of a single plane, may be missed if the projection axis is not perpendicular to the defect or if the exposure time is prolonged. Motion of the arterial wall will blur an area of obstruction. Close up, magnification views and rapid exposure times are of value. The impact of the obstruction on brain perfusion determines the significance of the obstruction.

Interpretations intimidate the clinician. Repeatedly, false positives in noninvasive testing may prove to be obstructions of short length for which the angiographic imaging has not been adequate (94).

**Limitations of Angiography - Current Systems**

Current X-ray imaging systems were designed to meet the technical requirements of static intracranial lesions (45,86,123) because the technology was not sufficiently advanced in 1944 to permit dynamic imaging (68). The long atherosclerotic plaque lent itself to static imaging, but the obstruction of short length (94), the diaphragm, the constriction, or the dynamic kink did not, for motion readily blurs and obscures detail (83,138).

**Dynamic Cerebral Vascular Angiography - A Reality**

After we reviewed our unsatisfactory clinical experience and the surgical pathology, technical specifications (Table 1) were drawn up for

**TABLE 1. Requirements for head and neck angiography: static and dynamic capability**

1. Rapid exposure time, to 1 msec
2. Film: 35 mm, 100 mm, and standard cut film
3. C-Arm with compound angulation
4. Isocentric manipulation
5. Magnification
6. Conventional biplane
7. Head position manipulation
8. Cardiac and vascular hemodynamics
9. Diastolic microbolus injection, ECG triggered
10. Floating table top
11. Instant video replay
12. Patient supervision: R.N. or M.D.
imaging the cerebral vascular system. In November 1975, these specifications were presented to Siemens (A. Carney, personal communication) with surgical cine footage (29) which demonstrated that arterial pathology visible to the camera was not visible on the angigram. Subsequent presentations were made in both Sweden and Germany in 1976. In November 1978, the Angioskop system was presented to the nation, and by 1980 other manufacturers followed.

It is important to recall that the field of coronary artery surgery began with the change from static to dynamic imaging (138). Conclusions regarding coronary arteries were not possible with serial cut film angiograms. Similarly, conclusions regarding obstructions of short length cannot be adequately made with current angiographic technique (94). Angiographic technique, diagnostic criteria, and interpretations will continue to improve.

VULNERABLE VASCULAR BEDS

Intracranial vascular patterns are associated with brain dysfunction, ischemia, and infarctions in a consistent fashion. These must be considered in some detail. The development of techniques for measuring brain perfusion requires concepts that permit the integration of the vascular patterns and vessel blood flow with regional brain perfusion and function.

The Watershed

The watershed is the interdigitation of the peripheries of two adjacent vascular beds in the brain (Fig. 6). The watershed between the carotid and vertebral arteries lies within the occipital and temporal lobes (Fig. 7). EEG abnormalities in these areas are common in vertebral-basilar insufficiency. Reduction of blood flow to the watershed may be due to hypotension, hypertension, or multiple arterial stenoses. Ischemic watershed infarctions are seen with blood loss, hypotension, cardiac arrest, and hypoxic events (1,6,90,96,97). In hypertension, the small peripheral arterioles develop high resistance to flow with the identical distribution of infarction in man and the rat (54,165). The watershed type of ischemia may also occur with reduced flow due to obstruction of the carotid and vertebral arteries.

CASE W.B.

Carotid occlusion and vertebral obstruction
Parietal-occipital infarction
Carotid distal vertebral artery bypass (26)

An elderly white man presented with transient right hemiparesis, slurred speech, and pharyngeal paralysis. Angiography revealed total occlusion of the left internal carotid artery, sharp kinking of both vertebral arteries within the cervical spine, and kinking of the right internal carotid artery. Injection of the left vertebral artery preferentially filled the anterior and middle cerebral arteries but not the posterior circulation. CT scan revealed a left parietal occipital infarct. Reconstruction by carotid distal vertebral bypass was performed with marked improvement of posterior and anterior fossa perfusion (Fig. 8).

Comment: Preoperatively, the vertebral flow was diverted preferentially to the anterior and middle cerebral arteries, rather than to the posterior circulation. Infarction occurred in the parietal occipital lobe between the carotid and the...
FIG. 7. The watershed is variable. The watershed between the carotid and vertebral arteries lies within the occipital and temporal lobes. The watershed between the anterior and middle cerebral arteries, two branches from the same internal carotid artery, lies within the frontal and parietal lobes. The three-dimensional watershed need not be symmetrical and arterial anatomy determines the precise location, size, and vulnerability. Top: Right carotid-small watershed lies high in the vertex. Bottom: Left carotid-large watershed associated with small-caliber anterior cerebral artery.
vertebral vascular beds. Enhancing the vertebral artery flow capacity results in improved perfusion of both the anterior and the posterior circulations. The demand for vertebral artery flow is markedly enhanced by bilateral carotid obstruction.

The Collateral Bed

**Carotid-to-Carotid Steal**

In the brain, the collateral flow in carotid occlusion is often inadequate (17,75,98,145) to maintain total cerebral blood flow and regional perfusion within normal limits because secondary channels cannot carry the blood flow of the primary vessel. With internal carotid occlusion, the common source for collateral flow is from the contralateral carotid (75, 111), and the less common but important source is the vertebral system. Arterial stenosis at elevated flow rates has greater significance (48) than at low flows. Reduced flow in the patent carotid can result in cerebral infarction in the collateral bed on the side of the occluded carotid artery (Fig. 9A). Speed of occlusion, extent of vascular disease, autoregulation, and cardiac output are mitigating considerations.
CASE W.C.
Carotid occlusion-Transient hemiplegia and bradycardia; cerebral infarction following RV pacemaker

A 54-year-old white man presented with a transient right hemiparesis and severe bradycardia. Angiography revealed total occlusion of the left internal carotid artery with poorly developed collateral channels. A right ventricular pacemaker was implanted to "improve" cardiac output. Immediately postoperatively, the patient developed an infarct in the left frontal lobe, and bradycardia was no longer noted (Fig. 9B).

Comment: With the loss of cerebral auto-regulation, the blood flow to the left hemisphere was directly related to perfusion pressure (143). Right ventricular pacing reduced both cardiac output and perfusion pressure. If the blood flow to a collateral bed is at a critical level, mild reductions in cardiac output or perfusion pressure result in cerebral infarction (Fig. 9B).

Vertebral-to-Carotid Steal

The vertebral vascular bed is a low-volume, high-resistance bed in comparison to the carotid vascular bed (Fig. 10). In the presence of carotid stenosis or occlusion and an adequate posterior communicating artery, blood will be shunted from the vertebral bed into the carotid bed.

CASE M.P.
Carotid occlusion, cerebral infarction, hindbrain ischemia
Carotid steal from vertebral system
Relief with carotid distal vertebral bypass

In 1976, a 61-year-old white woman presented with an acute cerebral infarction of the left hemi-
sphere associated with occlusion of the left internal carotid artery and stenosis of the right internal carotid artery with right hemiplegia and aphasia. Seven weeks later, endarterectomy of the right carotid artery was accomplished uneventfully with improvement but with permanent deficit. Thirty-six months later, the patient developed agitation, ataxia, and nystagmus, and marked emotional lability. EEG revealed left temporal lobe abnormality.

Four-vessel angiography revealed extensive stealling from the posterior circulation into the carotid system through the posterior communicating artery. There was moderately severe kinking of the dominant left vertebral artery at its origin and at the level of the axis.

On February 6, 1980, a vein bypass to the left distal vertebral artery was accomplished. Postoperatively, brainstem and cerebellar symptoms were relieved and the patient became pleasant and cooperative. Brain perfusion studies before and after bypass were performed (45) (Fig. 11).

**Comment:** In the presence of an infarction in the territory of the middle cerebral artery, the perfusion of the peri-infarction brain may return to normal levels, but ischemia of the hindbrain may persist and be most aggravated when the patient is erect. Enhancing the flow capacity of the vertebral artery relieves hindbrain symptoms.

**Overextended Vascular Bed**

When the carotid artery must supply the carotid plus the vertebral beds, mild reductions of carotid flow result in hindbrain ischemia and vertebral symptoms (Fig. 12). Cardiac output significantly reduced by any mechanism will reduce carotid blood flow. The capacity of the carotid already supplying such a large vascular bed to serve as collateral to the opposite carotid is also reduced as evidenced by a decrease of the collateral carotid (75,145) and collateral ophthalmic artery pressure (27).

**Comment:** When the internal carotid artery is occluded, the thrombosis extends over a great distance from the common carotid in the neck to the next branch arising in the skull, the ophthalmic artery. There is little success in reconstructing this vessel when occluded. The vertebral artery has extensive segmental branches (8,167); the thyrocervical, the ascending cervical, and occipital arteries will dilate to serve as collateral channels (11). Occlusion tends to be localized and of short length so that bypass beyond the obstruction is feasible.

Brainstem ischemia associated with *basilar artery hypotension* is extremely vulnerable to reductions of carotid flow, cardiac output, carotid stenosis, or surgical occlusion. Syncope and respiratory arrest have been associated with dysrhythmia or position change (85,133). Reduction of cardiac output by right ventricular pacing in the patient described in Fig. 13 resulted in vertebral-basilar symptoms only when erect. Vertebral artery reconstruction restored primary vertebral artery flow, eliminating the overextension of the carotid vascular bed. Symptoms were relieved, though the reduction of stroke output by pacing was not altered. If the overextension is due to aplasia of the basilar artery (Fig. 14), then direct surgery of the vertebral artery is of no avail and attention should be directed to the carotid system and cardiac hemodynamics.

**Corollary**

The significance of the anatomical arterial obstruction is determined by its impact on brain perfusion and is influenced by the flow within the artery involved, the size of the vascular bed, and the cardiac output. Minimal stenosis may have great significance to a high-flow artery supplying an extensive vascular bed.

**Isolated Vascular Bed**

(Hypoplastic Vertebral System)

The hypoplastic vertebral artery which terminates in an isolated posterior inferior cerebellar artery and directly supplies the cerebellum is an end artery without collateral. These hypoplastic arteries are more sensitive to low-grade stenosis because of their high resistance. Demonstration of regional cerebellar ischemia...
FIG. 11. Carotid steal from vertebral system. Vertebral angiograms. A: Towne projection (AP). B: These angiograms show the flow into the carotid system (middle cerebral artery) from the dominant vertebral artery. An old middle cerebral infarction due to internal carotid occlusion was complicated by incapacitating vertebral-basilar symptoms. Carotid distal vertebral bypass to the distal vertebral artery at C-2 (arrow) relieved these symptoms. Case M.P. (cont.)
In 1975, before we were employing CT scans and perfusion studies, a 46-year-old white woman presented with intractable right-sided ophthalmic migraine headaches, present since 1958, which were progressive and disabling. Intravenous sedation to produce sleep was required for 5 days every month. A hypoplastic right vertebral artery which terminated in PICA and communicated with the occipital artery was obstructed in its proximal portion. Surgical decompression of the vertebral artery by dividing the constricting sympathetic fibers between the subclavian artery and the vertebral foramen has resulted in complete resolution of the headaches for the past 5 years.

**Arteries of High Resistance**

The artery of small diameter and great length is an artery of high resistance (55,83,161) (Fig. 15). The most distal and the smallest branches without significant collateralization are most vulnerable to reduced blood flow and they supply the small vascular bed. Mild reductions of flow in the mother artery may cause critical ischemia in a specific branch. Intracranially, the arteries to the brainstem, the internal auditory
artery, and atypical branches of the posterior cerebral artery may be selectively sensitive to mild reductions in flow. Extracranially, the vertebral system is of high resistance compared to the carotid, as is the hypoplastic vertebral artery which terminates in the PICA.

CASE R.T.
A typical branch of the posterior cerebral artery
Symptomatic relief with sympathetic block
Superior cervical ganglionectomy

In June 1977, a 47-year-old white woman presented with a history of right-sided migraine headaches and temporal lobe epilepsy. Marked dyslexia, difficulty with word association, and letter and digit reversal were noted which fluctuated in severity. Angiography revealed a hypoplastic right vertebral artery and a dominant left (Fig. 16). The EEG revealed focal slowing in the frontal, mid, and anterior temporal regions, maximal on the right.
The blood supply to the right occipital lobe was from two sources. The right carotid gave rise to a small-diameter branch extending from the siphon to occipital lobe where it communicated with the occipital branch of the external carotid artery. The remainder of the blood supply to the occipital lobe arose from the basilar artery.

In August 1977, sympathetic block of the superior cervical ganglion resulted in dramatic improvement of symptoms. Maintenance was achieved with tolazoline (Priscoline®) until the drug was no longer available. In December 1979, the superior cervical ganglion (46,142) was resected. Immediately postoperatively, severe vascular headache was controlled with ergotamine and caffeine (Cafergot®). Subsequently, relief of headache, digit reversal, and dyslexia was noted but vision became impaired by the dilated pupil associated with a mild Homer’s syndrome. Pre- and postoperative brain perfusion studies were performed (45) and demonstrated increased perfusion of the occipital lobe.

Comment: The artery of small diameter and great length is a vessel of high resistance to flow (46,55,83). Minor alterations of flow may produce profound ischemia in a small segment of brain without the threat of extensive brain infarction. The arteries of high resistance which appear especially sensitive supply the organs of special sense, especially the vestibular apparatus and the visual cortex. Dysfunction in these regions immediately enters our conscious life.

CASE M.D.
Acute Bell’s palsy and deafness left side
Recovery of hearing with vertebral artery surgery

On July 28, 1975, a 63-year-old white man developed total deafness of the left ear, left central facial palsy, dizziness, loss of equilibrium, and slurred speech. Dysequilibrium was extreme and the patient, with eyes open, was unable to maintain a broad-based stance with support. He failed to respond to low molecular weight dextran and heparin...
anticoagulation. Angiography revealed normal carotid arteries and two vertebral arteries; the left was the larger with a marked kink of the proximal vessel.

On August 11, 1975, the origin of the left vertebral artery was reconstructed. In the recovery room, the patient noted that 85% of his hearing had returned. Subsequently, the ataxia and dizziness cleared but the facial palsy remained.

Comment: The presence of a patent right vertebral artery did not diminish the need for left vertebral artery reconstruction. Acute deafness with recovery can result only if the function of the cochlea or V11th nucleus is compromised by ischemia and not by infarction. Deafness can be a reversible ischemic neurological deficit. Acute deafness developing with head position has been noted following ear surgery. Techniques to determine the sensitivity to vertebral occlusion with head positioning on the operating table have been devised with the intent of avoiding this complication (34).

Small Vertebral System

When both posterior cerebral arteries arise from the carotid system, the vertebral system, which then supplies only the basilar and the cerebellar branches, is usually of small diameter and high resistance.

CASE F.G.
Brainstem ischemia-Small vertebral system
Right occipital PICA bypass and subsequent left carotid distal vertebral artery bypass

FIG. 17. Quadriparesis and the hypoplastic vertebral system. This hypoplastic vertebral system was associated with frightening and disabling symptoms. Occipital PICA bypass, although patent, failed to control symptoms after 2 years. External carotid distal vertebral bypass to the segment of vertebral artery seen at C-2 brought complete relief of symptoms. Case F.G.
On October 4, 1977, a 45-year-old white woman presented with transient episodes of numbness of all four extremities, near syncope, tingling of hands and face bilaterally, dizziness, and ataxia. In June 1976, she permanently lost all fine writing capability and developed slurred speech and weakness of her right eye. Head flexion produced scintillations. Standard Romberg was negative but became strongly positive with any moderate deviation of the head from neutral.

Angiography revealed a fetal type of carotid system and a small vertebral system terminating in the superior cerebellar arteries. On October 10, 1977, a right occipital-PICA bypass was performed with immediate postoperative improvement in symptoms. But in January 1978, she experienced episodes of recurrent numbness of the tips of her fingers. On October 9, 1979, she was readmitted with acute symptoms of diplopia, numbness of both hands, frequent scintillations, slurred speech, and impaired reading. Romberg was again strongly positive.

On October 17, 1979, she underwent external carotid-distal vertebral artery bypass utilizing saphenous vein to the 2.5 mm ID vertebral artery. Immediately postoperatively, there was a marked personality change with increased stability and affect, the loss of numbness and visual problems. She continued to improve over the 9 months of follow-up. Pre- and postoperative CT blood flow studies were done (45) (Fig. 17).

Comment: The small-diameter artery is more vulnerable to obstruction than to the reduction of blood flow. Although occipital PICA bypass can increase perfusion of the distal bed of the PICA, such a bypass cannot perfuse the basilar artery adequately because of the small size of the PICA in relation to the basilar artery. On the other hand, the vertebral artery at the level of the axis is usually patent and of a diameter similar to the internal carotid artery.

Arteries of Modified Resistance

Dysautoregulation

In a broad sense, autoregulation is the capability of the brain to regulate its blood supply in accordance with its needs; in the narrow sense, it is the pressure-flow relationship that permits flow to remain constant in the presence of changing blood pressure. This pressure-flow relationship is variable and sensitive to the levels of pCO₂ so that modifying the can increase or decrease regional brain perfusion. Pathologically, the brain engulfing the focus of acute infarction has disturbed autoregulation so that perfusion does not respond normally to levels; high pressure may cause hemorrhagic infarction and reduced pressure extension of the ischemic infarction. Since regional brain perfusion is interdependent, dysautoregulation may also occur at a site remote from the primary brain pathology (49,104,136).

If an ischemic infarct is represented by the center of the bull's-eye (Fig. 18), the remainder of the target represents the surrounding brain at risk due to dysautoregulation. Expanding the plasma volume may facilitate perfusion of the ischemic peri-infarct area (134); diuresis or reduction of plasma volume may result in extension of the infarction. Arterial reconstruction performed in the presence of ischemic brain and dysautoregulation may pose increased hazard (153) because of the necessary vascular clamping, though temporary, required which reduces total brain blood flow and may aggravate ischemia in the area of dysautoregulation.

FIG. 18. Dysautoregulation and the bull's-eye. If the center of the bull's-eye represents the infarcted brain, the surrounding target represents the volume of brain with disturbed autoregulation. Optimal perfusion will limit the extension of the infarction. Reduced perfusion may result in extension of the infarct to the limits of the target and the vascular bed.
CASE R.B.
Cerebral infarction and amaurosis fugax
Dysautoregulation and plasma volume

A 56-year-old white man presented with acute left-sided hemiplegia and blindness of the right eye. Recovery was complete within 12 hr of admission; he was eating well and intravenous fluids were discontinued within 48 hr. However, he was ataxic and dizzy when standing or walking even for small distances. Physical therapy failed to increase strength and stability but only aggravated his symptoms. He was discharged 9 days after admission. At home, while standing or walking, he developed dizziness, ataxia, weakness, and became ashen gray in appearance. He was readmitted to the hospital 12 days after the acute onset of symptoms. Intravenous plasma volume expansion was begun immediately despite the patient’s adequate oral intake. Within 48 hr he was no longer sensitive to the upright position. CT dynamic studies were done after 6 days of plasma volume expansion and revealed a small right parietal infarction without luxury perfusion. Carotid endarterectomy was well tolerated, and intolerance of the upright position was not encountered in his postoperative course.

PAST AND FUTURE OF BRAIN BLOOD FLOW

In 1972, EMI Ltd. announced the advent of computerized tomographic scanning (42). The impact on medicine was profound. In October 1975, the following statements appeared in a report for the National Institute of Health (106):

It is apparent that computerized tomography will revolutionize the differential diagnosis of intracranial lesions. . . . A variety of methods for measuring cerebral blood flow have been devised in the last 25 years. None of the currently used methods are accurate, reproducible and noninvasive. The methods have been used primarily in research and have provided information about cerebral vascular physiology. . . . At the present time, these methods are not significantly helpful in clinical practice.

It has become increasingly apparent that function is more closely related to blood flow than to structure (72,73,143,144), that abnormalities of brain perfusion precede structural changes (67,88), and that where brain structure and ischemia coexist, revascularization can reverse neurological deficit (22,60,119,127).

The measurement of brain blood flow had long been pursued but the clinical impact had been meagre. In 1961 Oldendorf (42), who was unhappy with the inadequacies of angiography and radiology in solving problems of the brain, advanced the concept of computerized tomography employing the radioisotope as the indicator. His concern was brain blood flow.

So it comes as no surprise that computerized tomography has been extended to mapping brain blood flow, i.e., reconstructing functional, not structural, images based on the emission of radioactive particles, [emission computerized axial tomography (ECAT)], which can reflect not only brain perfusion, but also metabolic function (19,60). These ECAT scanners are extensions of the physiological approach to brain function and blood flow by those primarily oriented to the physiology of the brain.

CT dynamic brain scanning is an outgrowth of cardiac scanning. Extension of CT scanning to the heart (24,95,161) required two major modifications: rapid scanning and contrast enhancement. The brain did not possess the problems related to the motion of the heart (122), and application to the brain clinically began with the authors in August 1978. There are several distinct advantages to the use of the X-ray-based CT scanners to measure brain perfusion: (a) numerous CT scanners are already available; (b) brain structure and regional perfusion could be studied by the same instrument; (c) a variety of enhancement procedures are available; (d) a high degree of resolution can be obtained; (e) the foci can be restudied by varying the size of the pixels; and (f) the infratentorial brain circulation can be studied, a feat that remains unmatched by any other technique.

If the ability to measure brain perfusion by a variety of techniques is thus imminently available, the subject takes a practical turn. In the past, brain blood flow has been a sacred cow because the practice of medicine was largely untouched by it, but now a closer examination is in order.

All techniques of measuring blood flow require the use of indicators which are diffusable
(xenon) or nondiffusible (contrast) and which can be quantitated by some means, e.g., radioactivity (isotopes) or density (iodinated contrast media). Calculations are based on the principles of indicator dilution, the arrival time of the bolus (mean transit time), or the washout rate of the indicator, all of which may be proportional to blood flow. Indicators can be administered by artery, vein, or inhalation. The data obtained form a curve of numerical values for each focus under study. These individual curves can be displayed, numerical values can be extrapolated, and functional cross-sectional images can display relative values in gray or color scale.

The quality of the study is based on the resolution, i.e., the smallest volume of brain that can be discriminated with precision. As resolution increases, precise sampling becomes of major importance since not all brain can be studied. Multiple sections and sections of both the supra- and infratentorial brain are desirable. The most critical requirement is that the studies be practical to the clinician and meaningful for the patient.

In a substantive review of physical principles underlying the measurement of brain blood flow, Decker (38) critiques the theoretical assumptions on which the performance of the various studies is based, and the literature is pregnant with theoretical principles and technical details. But there may be a greater need to critique the clinical assumptions that have been made in the past that influence application since these have received little consideration.

In intracranial arterial disease in which the potential for major collateral flow through the circle of Willis is limited, unilateral regional perfusion studies have been reliable. For this reason, application of unilateral cerebral perfusion studies to middle cerebral artery stenosis has met with success because the technique identifies the focal ischemia and documents the success of vascular reconstruction in the area of interest where the technique can be best applied. But the unilateral technique cannot identify generalized brain ischemia, intracranial stealing involving remote areas, and hindbrain ischemia.

In extracranial arterial disease in which the circle of Willis is adequate, the potential for collateral flow is great. Regional brain perfusion in one part of the brain may markedly influence the perfusion in remote areas. Although blood in an artery tends to flow directly into its vascular bed, the circle of Willis functions as a distributor and can redistribute blood flow elsewhere, so that the carotid artery flow may perfuse the hindbrain and the vertebral artery flow perfuse the forebrain. In the past, the assumption has been made that perfusion of the total brain need not be studied, that regional perfusion in the bed of the artery of interest is adequate. Bilateral supra- and infratentorial brain blood flow studies pre- and postoperatively support the position that intracranial stealing is common and must be recognized if brain perfusion studies are to be useful in the clinical setting.

The second assumption has been that the brain can be separated from the neck. Most discussions of regional brain perfusion do not relate to the blood flow within the individual arteries of the neck. If the circle of Willis is intact, can brain perfusion stand alone without individual neck artery assessment? Is it possible to extrapolate from brain perfusion studies to lesions in the arteries of the neck without knowing the vagaries of the arterial anatomy, the resting flow, or the flow capacity (78,79) of the arterial system? It would appear that a system of evaluating flow within the arteries of the neck is desirable to complement brain perfusion studies.

Thirdly, it is assumed with few exceptions (109) that commonly experienced stresses, such as standing up, hyperextending the head, or occluding the carotid artery, need not be employed in regional perfusion studies. If a patient has profound symptoms when erect and a supine brain perfusion study is normal, we can conclude only that accurate supine studies do not reflect the state of brain perfusion when erect. Therefore, it would appear that stress evaluation should complement perfusion studies.

Finally, it might be assumed that the refinement and availability of regional perfusion tech-
nology will automatically produce patient benefit. Accurate information must be interpreted and applied. Concepts based on this information must be developed and understood. Ideally, the performance and interpretation of brain perfusion would be by someone who understands neurovascular physiology.

The measurement of brain perfusion now appears to be practical. The morphologic approach of angiography and CT scanning has failed to provide insight into problems related to brain perfusion and function. The application of microvascular (5;82,125) and vertebral artery surgery (28) to the disabled requires a precise delineation of anatomy, arterial flow, regional perfusion, and the demonstration of hemodynamic and functional improvement with treatment.

Accuracy, practical operation, and reproducibility are desirable qualities for any system of measuring brain perfusion, but it is most important that the physician performing the study understands that the phenomena being evaluated are physiological. The interpretation of CT-generated images has largely become the province of the radiologist, who is structure and image oriented. But the radiologist lacks training and discipline in the physiology and pathophysiology of brain function and blood flow.

The measurement of brain perfusion often cannot localize the artery that compromises blood flow because of extensive intracranial stealing, nor can it distinguish extensive vascular obstruction from reduced cardiac output, or identify the response of the patient to common stress. The measurement of brain perfusion is an important complement to the physiological evaluation of brain hemodynamics. Brain perfusion and equivalent functional studies such as EEG and neuropsychological testing need to be more closely related because the more common and less expensive techniques would be more satisfactory for screening and for long-term follow-up.

The most critical physician quality needed is not the capacity to analyze but the capacity to synthesize. Perfusion studies, of themselves, with wise interpretations are not capable of indicating patient management. Sophisticated analytic measurement of brain perfusion must be complemented by effective methods of treatment. Documentation of the response of brain perfusion to vascular reconstruction or medical management is necessary for the development of sound clinical judgment and of medical and surgical skills in the use of brain perfusion or equivalent functional studies.

REFERENCES


