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INTRAOPERATIVE USE OF TRANSCRANIAL DOPPLER ULTRASONOGRAPHY

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Transcranial Doppler (TCD) ultrasonography is a diagnostic tool that is well established in the fields of neurology and neurosurgery. Its potential application as a monitor of intracerebral hemodynamics during anesthesia and surgery has only just begun. Although theoretical as well as practical limitations exist, TCD ultrasonography represents the first step toward achieving the goal of noninvasive continuous cerebral blood flow (CBF) monitoring during anesthesia. Both the flow velocity (or change in flow velocity) and the characteristic profile of the waveform (pulsatility) can provide valuable information to the anesthesiologist and the surgeon. There are many potential applications of TCD ultrasonographic monitoring in the perioperative care of the patient at risk of cerebral ischemia, the most important of which are detailed in this article. The use of TCD ultrasonography in patients undergoing cardiopulmonary bypass, although extensively investigated, is beyond the scope of this article and will not be discussed.

PRINCIPLES OF TCD ULTRASONOGRAPHY

TCD Ultrasonography as an Intraoperative Monitor

TCD ultrasonography makes it possible to measure CBF velocity in the middle cerebral artery (Vmca) in a noninvasive and continuous manner. The Vmca is proportional to CBF only when the diameter of the vessel insonated remains constant.[1, 3, 9, 56] Moreover, because the angle of insonation influences the accuracy of the flow velocity estimation, and as the exact diameter of the basal arteries is unknown, TCD ultrasonographic measurements can only give relative indices of CBF. This is reflected in the normal Vmca which ranges from 35 to 90 cm/s in the awake resting state.[3, 5] This large range is a consequence of the variation in diameter of the basal cerebral arteries, the inherent variation in CBF, and the varying angle of insonation. Although correlation between absolute flow velocity and CBF in any given population is poor, good correlation between relative changes in flow velocity and CBF has been demonstrated.[5, 9]

Carbon Dioxide and Blood Pressure Do Not Significantly Affect the Diameter of the Insonated Vessel

The basal cerebral arteries are conductance vessels and, as such, do not dilate or constrict as the vascular resistance changes under most circumstances. It has been shown angiographically that change in carbon dioxide tension, one of the most important determinants of cerebrovascular resistance, has no significant effect on the diameter of the basal arteries.[31]

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Moreover, studies on cerebrovascular carbon dioxide reactivity using TCD ultrasonography have yielded similar values to those obtained with conventional CBF measurements.[17, 49]

Similarly, blood pressure appears to have negligible influence on the diameter of the basal arteries, at least when the proximal segments are insonated. Intraoperative measurements using magnified video imaging demonstrated that vasoactive agents such as sodium nitroprusside and phenylephrine do not change the diameter of the proximal segments by more than 4%.[22] On the other hand, Dahl et al[10] have demonstrated significant vasodilation when nitroglycerin is administered to healthy volunteers.

Commonly Used Anesthetics Have Little Effect on the Diameter of the Vessel Insonated

There have been few studies on the correlation between Vmca and CBF during changes imposed by anesthetic agents. Nevertheless, available evidence indicates that, at least qualitatively, the correlation is good.[43] Intravenous anesthetic agents do not vasodilate or vasoconstrict the basal cerebral arteries.[69] Commonly used inhaled anesthetics do not seem to dilate the MCA appreciably,[45] although this remains controversial.[70] Certainly, during steady-state anesthetic conditions, changes in Vmca can be interpreted to mean corresponding changes in cortical CBF.[6, 46, 79]

Spectral Outline Instead of Weighted Mean Velocity As the Flow Velocity

Although the most accurate physiologic correlate with actual CBF is theoretically the weighted mean velocity (Vmean) which takes into consideration the different velocities that the formed elements in the blood vessel are traveling, the maximal flow velocity (Vmax; as depicted by the spectral outline) is generally used because of the higher signal-to-noise ratio. As the flow is usually laminar in nature in the basal cerebral arteries, there is also good correlation between the Vmax and Vmean. The Vmax is generally displayed in most commercially available instruments. Thus time-mean velocity usually refers to the mean velocity of Vmax and can be determined using the area under the curve method or approximated by the equation:

$$\text{mean Vmca} = [(\text{Vsys} - \text{Vdias})/3] + [\text{Vdias}, \quad (1)]$$

where Vsys = systolic flow velocity and Vdias = diastolic flow velocity.

Pulsatility of Flow Velocity Can Estimate Cerebrovascular Resistance

In the absence of stenosis or vasospasm, the pulsatility of the flow velocity profile reflects the distal cerebrovascular resistance. The pulsatility can also be affected by cardiac factors, however. Two derived indices have been used to quantify the resistance: the pulsatility index (PI or Gosling index) which equals (Vsys − Vdias)/mean Vmca[23] and the resistance index (RI or Pourcelot index) which equals (Vsys − Vdias)/Vsys.[62] Although the Vmca is used in the equations, they can be applied to any of the basal arteries. In general, PI and RI correspond to each other during changes in resistance. Neither index provides meaningful information regarding the cause of the change, however; for example, an increase in PI can be due to cerebrovasoconstriction (intrinsic, as in hyperventilation) or to high intracranial pressure (ICP) (extrinsic, from obstruction).

Limitations and Uses of TCD Ultrasonography

The use of TCD ultrasonography as an intraoperative monitor has only just begun, so the indications remain to be defined. There are currently three major factors that limit its use as a routine monitor in patients considered to be at risk of cerebral ischemia or embolism. These include equipment, operator, and patient factors.

A number of commercial vendors of reliable TCD ultrasonographic equipment now exist. In the past, most instruments were designed for diagnostic, not monitoring, purposes. Recently, the equipment has been adapted for monitoring purposes. There is a need for a fixation device that would allow continuous, reliable recording that does not interfere with the surgical field. Devices such as the headband or head strap are difficult to use during neurosurgical procedures. Giller[21] used a transducer mounted on a flexible retractor attached to a head clamp with considerable success, but he found frequent intraoperative adjustment necessary with such a device. On the other hand, we have found a specially designed frame attached to

the ear canals and the bridge of the nose (developed in conjunction with DWL Electronische, Sipplingen, Germany) more reliable, and it has provided us with usable recordings in virtually all neurosurgical procedures, with the exception of those involving subtemporal incision which precludes the use of the transducer probe (Fig. 1*A* and 1*B*).

The successful transmission of ultrasound through the skull is dependent on the thickness of the skull, and the temporal bone thickness (for MCA insonation) varies with gender, race, and age.[15] In elderly patients, the failure rate can be as high as 20% to 30%. Increasing the energy output of the TCD ultrasonography may increase the success rate in these difficult patients, but the current output is limited to 100 mW/cm^2, and the safety of exceeding this limit has not been adequately addressed.[24] Intravenous contrast agents are now available which can increase the rate of successful recording for short durations in difficult patients.

CAROTID ENDARTERECTOMY

The ideal monitoring technique for carotid endarterectomy should be able to detect events associated with all of the potential complications during and following surgery. Previous monitoring strategies have mainly focused on monitoring for hypoperfusion and possible ischemia during cross-clamping. Other complications may be as or more important than cross-clamp ischemia in producing neurologic deficits following endarterectomy. Riles et al[65] recently analyzed the complications leading to neurologic deficits in a large number of patients undergoing carotid endarterectomy. The most common complications were postoperative thrombosis, intraoperative and postoperative embolism, intraoperative ischemia, and postoperative hyperperfusion (Table 1). Monitoring the MCA using TCD ultrasonography has the potential to provide sufficient warning to prevent all of these recognized complications associated with carotid endarterectomy.

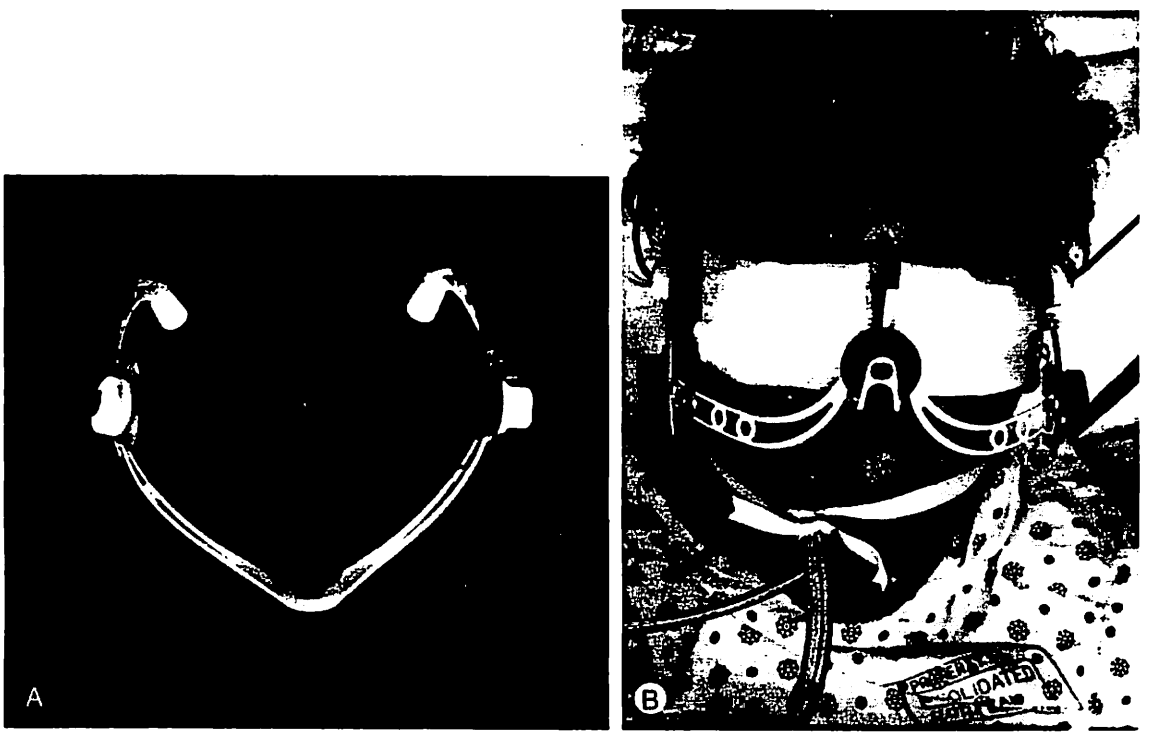

Figure 1. Method of probe fixation using TCD for intraoperative use during neurosurgical procedures. *A.* Isolated frame showing the ear-inserts for anchorage and the bilateral probes (patent pending). *B.* Placement of the frame on a patient for intraoperative monitoring. This probe fixation device does not interfere with most cranial incisions for exposure of the supratentorial region.

Table 1. CAUSE OF STROKE AFTER CAROTID ENDARTERECTOMY

Cause of Stroke After Carotid Endarterectomy (2.2% total of 3062 operations)	Number of Patients (% of all strokes)
Postoperative thrombosis	15 (23%)
Intracranial bleeding	12 (18%)
Postoperative embolus	10 (15%)
Cerebral ischemia	10 (15%)
Other surgery related	8 (12%)
Unrelated to operated artery	8 (12%)
Unknown	3 (5%)

Data from Riles TS, Imparato AM, Jacobowitz GR, et al: The cause of perioperative stroke after carotid endarterectomy. J Vasc Surg 19:206–214, 1994.

Detection of Cerebral Ischemia During Cross-Clamping of the Carotid Artery

This is one indication where the use of TCD ultrasonography perhaps has come close to being an established and important tool.[25, 26, 35, 40, 64, 71, 72, 74] It fulfills most of the requirements of an ideal monitor because it permits continuous, noninvasive monitoring of the changes in cerebral hemodynamics that take place during surgical occlusion of the carotid artery. Moreover, various fixation devices to anchor the transducer probes can be used successfully without infringing on the surgical field. The method for calculating changes in flow velocity with cross-clamping of the carotid artery is shown in Figure 2.

In a multicenter study involving 1495 patients undergoing carotid endarterectomy, Halsey[25] has attempted to define the role of TCD ultrasonography as an intraoperative monitor for cross-clamp ischemia. In this study, ischemia developing during cross-clamping of the carotid artery is considered to be severe if mean flow velocity (Vmca) after cross-clamping is 0% to 15%, mild if velocity is 16% to 40%, and absent if velocity is greater than 40%. All values are relative to the flow velocity recorded immediately before cross-clamping. Although it was not a randomized, controlled trial, and the anesthetic technique and surgical protocol were not standardized, this report nevertheless demonstrates that insertion of a shunt is associated with a significant risk of stroke (presumably embolic in origin) and that the best results are obtained using selective shunting in patients with severe ischemia based on TCD ultrasonographic criteria (for example, decrease in flow >60% with cross-clamping (Table 2). Unnecessary shunting is thus avoided. The correlation between flow velocity changes and electroencephalographic changes appears to be excellent in other studies.[35, 74] In contrast, the correlation with stump pressure is less consistent, but low stump pressures are generally associated with low flow velocity during cross-clamping.[42, 54, 72] We used TCD ultrasonography at our institution and found excellent correlation between the two (unpublished data). Moreover, TCD ultrasonography can instantly detect malfunctioning shunts due to kinking or thrombosis.[54]

Detection of Microemboli

Microemboli occur frequently during carotid endarterectomy, and both air and particulate matter can be detected with TCD ultrasonography. Recent advances in embolic

1) Record pre-clamp value
2) Allow 15-20 seconds for autoregulatory response
3) Record post-clamp value

$$\frac{\text{Post-clamp time averaged mean velocity}}{\text{Pre-clamp time averaged mean velocity}} \times 100 = \%\ \text{Baseline value}$$

e.g. Pre-clamp 40 cm/sec; Post-clamp 18 cm/sec = 45% residual velocity

Figure 2. Method for calculating the degree of velocity reduction in the MCA following cross-clamping for carotid endarterectomy.

Table 2. CORRELATION BETWEEN ISCHEMIA AND INCIDENCE OF STROKE

Degree of Ischemia (Percentage of residual MV after cross-clamping)	Incidence of Stroke	Probability (P)
Severe (MV = 0%–15%)		
Shunt	0/74 (0%)	< 0.0001
No shunt	6/13 (46%)	
Mild (MV = 16%–40%)		
Shunt	3/77 (3.9%)	0.1 > P > 0.05
No shunt	1/159 (0.6%)	
None (MV > 40%)		
Shunt	6/136 (4.4%)	< 0.001
No shunt	7/1016 (0.7%)	

MV = Mean velocity.
Data from Halsey JH Jr: Risks and benefits of shunting in carotid endarterectomy. The international transcranial Doppler collaborators. Stroke 23:1583–1587. 1992.

signature analysis have made it possible to differentiate air and particulate emboli. Embolic causes probably outweigh hemodynamic causes as an etiologic factor for perioperative strokes during carotid endarterectomy.[44, 65] Embolization can occur during carotid dissection, but more commonly, it occurs upon release of common carotid artery cross-clamping.[36, 71] In one study, the number of embolic signals during carotid artery dissection was positively correlated with the occurrence of intraoperative infarcts.[36] Although at the present time it is not possible to quantify the emboli, the use of TCD ultrasonographic monitoring frequently allows the surgeon to improve his technique to decrease the incidence of embolic episodes.[54]

Diagnosis and Treatment of Postoperative Hyperperfusion Syndrome

Approximately 1% of patients may develop hyperperfusion syndrome following carotid endarterectomy, resulting in cerebral hemorrhage.[61] Studies with TCD ultrasonography have identified the fact that hyperemia tends to occur in patients with high-grade stenosis and is characterized by sustained elevated flow velocity following release of the carotid occlusion. Other factors predictive of the hyperemia include low stump pressure and low flow velocity during cross-clamping.[37, 39, 53, 63, 68] Whereas in most patients the release of occlusion leads only to a transient hyperemia and the flow velocity quickly returns to the baseline value, in these patients, the flow velocity remains elevated. The period of elevation can range from 1.5 hours to as long as 14 days.[39] The incidence of hyperemia as identified by TCD ultrasonography ranges from 10% to 20%, and the increase in flow velocity can range from 30% to 230%.[39] Not all patients with evidence of hyperemia develop cerebral hemorrhage. Some patients remain asymptomatic, some develop severe headaches, and in the most severe cases, they may develop hemorrhage or seizures. These findings suggest that there is defective autoregulation in the ipsilateral hemisphere after carotid endarterectomy. This hypothesis is supported by the fact that reduction of blood pressure is effective in normalization of the ipsilateral flow velocity as well as alleviation of the symptoms. Thus, the use of TCD ultrasonography allows the identification of patients at risk and immediate institution of therapy with tight blood pressure control.

Diagnosis and Treatment of Postoperative Intimal Flap or Thrombosis

Postoperative occlusion of the ipsilateral carotid artery due to clot formation or the presence of an intimal flap are disastrous but potentially avoidable complications of carotid endarterectomy. TCD ultrasonography can detect the formation of thrombus or a nidus of emboli at the end of the operation or in the recovery room, thus allowing early reexploration to prevent an impending stroke.[20, 66] Clinically, sudden development of symptoms in the recovery room should prompt an immediate TCD ultrasonographic examination. This avoids an invasive angiography procedure

and allows early re-exploration. Some surgeons advocate continuous monitoring in the recovery room for a period of 2 hours. A recent study revealed that embolic signals in the MCA are common following carotid enarterectomy. Large numbers of embolic signals in the immediate postoperative period are correlated with the development of postoperative neurologic deficits.[47]

CEREBRAL ANEURYSM SURGERY

Deliberate Hypotension

Although the use of induced hypotension during surgical clipping of cerebral aneurysms is in decline, in a recent survey of 45 North American neurosurgical centers, 48% of the centers surveyed continue to use it in some patients.[8] Systemic hypotension decreases the transmural pressure across the aneurysmal sac and thus decreases the risk of rupture during surgical dissection and clipping of the aneurysm. Hypotension also decreases bleeding from surrounding small vessels which allows better visualization of the anatomy of the aneurysm and the perforating vessels. The reasons for this decline in the use of induced hypotension during clipping of cerebral aneurysms include the increasing use of temporary occlusion and the unpredictable cerebrovascular response to induced hypotension in patients with subarachnoid hemorrhage. Defective autoregulation may be present following subarachnoid hemorrhage and systemic hypotension would potentially increase the risk of ischemic complications. Moreover, carbon dioxide reactivity may be agent dependent when hypotension is employed, rendering the change in CBF during systemic hypotension and hyperventilation unpredictable.[51] Continuous monitoring with TCD ultrasonography allows the anesthesiologist to determine the patient's ability to tolerate induced hypotension as well as the effect of hyperventilation on CBF during this vulnerable period.

Detection of Perioperative Rupture of a Cerebral Aneurysm

Although the incidence of aneurysmal rupture during induction of anesthesia with modern techniques is low (< 1%), the diagnosis may not be readily apparent until the time of dural incision. Unrecognized preoperative rupture of a cerebral aneurysm often results in difficult operating conditions with a swollen congested brain. The authors have used TCD ultrasonography intraoperatively to confirm the diagnosis in a patient in whom rupture was suspected shortly after induction of anesthesia, and the monitoring facilitated the perioperative management of this patient.[16]

Assessment of the Adequacy of Collateral Flow During Temporary Occlusion of Major Vessels

Temporary occlusion of major feeding vessels is being increasingly used instead of induced hypotension during clipping of cerebral aneurysms. One of the main concerns regarding temporary occlusion is the adequacy of distal flow which is dependent on collateral circulation. Monitoring of a major vessel distal to the temporary occlusion with TCD ultrasonography will theoretically allow assessment of the adequacy of this collateral flow. In practice, however, this is seldom possible due to the inaccessibility of the distal site for monitoring (encroachment in the surgical field) as well as the presence of intracranial air insulating the ultrasound beam. The gas-sterilizable microvascular Doppler, however, is particularly suited for this purpose.

RESECTION OF ARTERIOVENOUS MALFORMATIONS

Arteries leading to an arteriovenous malformation (AVM), or feeding vessels, convey blood through the shunt and into the venous side. The size and flow rates of these arteries are far out of proportion to the low metabolism within the AVM. These vessels are termed "feeders" to distinguish them from normal vessels conveying purely nutrient flow to the neural tissues, and they are characterized by high flow velocity, low pulsatility, and low perfusion pressure.[27-29] Cerebrovascular reactivity to carbon dioxide is also decreased in the feeding vessels.[11, 13, 50] In principle, as the resistance to flow in an AVM is low, it is possible to diagnose AVMs and to locate individual AVM feeder arteries from findings of flow velocity, waveform pulsatility, and carbon diox-

ide reactivity outside the ranges for expected normal variation. Lindegaard et al[48] have shown that there is normally less than 10% difference in MCA flow velocities on opposite sides in the same individual[48]; based on this, they were able to diagnose AVMs in 26 of the 28 patients examined using TCD ultrasonography.

The changes in flow velocity following embolization and resection of AVMs have been well described, with normalization of flow velocity, pulsatility, and carbon dioxide reactivity.[7, 41, 60] The potential use of intraoperative TCD ultrasonography with AVM resection lies with detection of residual AVM and diagnosis and treatment of the hyperfusion syndrome.

Detection of Residual AVM

If the feeding vessel can be monitored (this criterion primarily restricts the application to AVMs supplied by anterior cerebral artery, MCA, and posterior cerebral artery because it is currently not possible to continuously monitor the vertebrobasilar system), important information can be obtained. By simultaneously monitoring the vessels bilaterally, theoretically, one can observe the preoperative difference in velocity, as well as in pulsatility, between the two sides progressively disappearing as resection is completed. In our series of eight patients, we have identified in one patient the presence of residual AVM based on TCD ultrasonographic observations, which was confirmed by postoperative angiography. Nevertheless, TCD ultrasonography cannot replace angiography for this purpose, because a 10% side-to-side difference is considered to be within normal limits. Persistent large differences at the end of resection, however, may alert the surgeon to the need for intraoperative angiography. This information may be less useful in institutions where intraoperative angiography is used routinely.

Diagnosis and Treatment of the Hyperperfusion Syndrome

It is well recognized that some patients are at risk of brain swelling and hemorrhage after AVM resection. Risk factors include the volume of the AVM (> 20 cm^3), the presence of deep feeders, the location of the AVM (rolandic, inferior limbic, and insular regions), and the pre-excision mean feeder flow velocity (> 120 cm/s).[59] According to the normal perfusion pressure breakthrough theory initially proposed by Spetzler et al,[73] the hyperemia occurs as a result of the loss of autoregulatory capacity in normal brain tissue adjacent to the AVM. Young et al[81, 82] were not able to demonstrate this loss of autoregulation with xenon washout studies, however. Occlusive hyperemia from venous obstruction has also been proposed as the cause of this hyperperfusion.[4] Regardless of the underlying mechanism, there is little doubt that brain swelling/hemorrhage occurring after AVM resection is related to an increase in hemispheric perfusion,[82] and the only effective treatment is adequate control of blood pressure and optimal cerebral vasoconstriction facilitated with hyperventilation I anesthetic agents. Perioperative TCD ultrasonographic monitoring during AVM resection would allow early diagnosis of the development of hyperemia and prompt institution of treatment.

We have performed continuous bilateral intraoperative TCD ultrasonographic monitoring in a series of eight patients with supratentorial AVMs. With the exception of one patient in whom we monitored the posterior cerebral arteries, the MCAs were monitored. In all patients but one (residual AVM as described above), we observed equalization of flow and pulsatility at the end of resection. In one patient with a parieto-occipital AVM, we observed an immediate increase in flow velocity in the ipsilateral MCA with a spontaneous increase in blood pressure, and this was associated with sudden brain swelling not responsive to hyperventilation. High-dose propofol infusion (300 μg/kg) was initiated to control the blood pressure and to effect maximal cerebral vasoconstriction. This brought the flow velocities down with equalization bilaterally, and the brain swelling subsided. The patient was maintained on propofol infusion for another 24 hours and subsequently made an uneventful recovery. Monitoring flow velocities in this patient had allowed us to treat this potentially fatal complication promptly; thus, the advantages of intraoperative TCD ultrasonographic monitoring during AVM resection are amply illustrated. We have since developed a protocol to routinely test autoregulatory capacity and the risk of hyperfusion at the end of resection by raising the systemic blood pressure by 20 mm Hg using a phenyl-

ephrine infusion. The majority of patients studied had intact autoregulation (unpublished data). Because of the inability to predict with certainty which patient is at high risk of developing hyperfusion, we believe TCD ultrasonography as a noninvasive monitor is a promising tool.

DETERMINATION OF CEREBRAL AUTOREGULATION

Cerebral autoregulation refers to the ability of the brain to maintain constant CBF despite changes in cerebral perfusion pressure (CPP). Below a mean arterial pressure of about 50 mm Hg, CBF begins to fall with further reductions in pressure. The static, or steady-state, methodology for determining cerebral autoregulation in the past has been cumbersome and invasive, requiring radioisotopes for CBF measurement and vasoactive medication to change the blood pressure. TCD ultrasonography can now be used to noninvasively determine autoregulation in the MCA perfusion territories, using static changes or rapid dynamic changes in blood pressure as a stimulus. Aaslid et al[2] introduced a method whereby blood pressure and MCA velocity are monitored noninvasively, the relative change in flow through the MCA in response to a rapid step change in arterial blood pressure (ABP) is observed, and the dynamic autoregulatory response can be measured. Large thigh cuffs which are inflated to suprasystolic pressures are rapidly released to produce a transient drop in blood pressure. Recent comparisons between MCA velocity and internal carotid artery flow have confirmed the validity of this method.[56] We have completed an intraoperative study which indicates that the static and dynamic methods yield similar results when used to measure intact or impaired autoregulation, and either can readily be used for intraoperative testing of autoregulation (Fig. 3).

We have utilized noninvasive testing of autoregulation recently to determine whether patients with minor or moderate head injuries had normally functioning autoregulation. Thirty-one patients with minor and moderate head injuries (MMHIs), as defined by Glasgow coma scale (GCS) scores of 9 through 15 (average GCS, 14) underwent testing of dynamic cerebral autoregulation using continuous TCD ultrasonographic velocity recordings and blood pressure recordings within 48 hours of their injury. This study indicated that a significant number of patients with minor head injuries can have impaired cerebral autoregulation and may be at increased risk for secondary ischemic neuronal damage. It may therefore be advisable to assess autoregulation preoperatively or intraoperatively in patients who have had head injuries and are undergoing surgical procedures.

CARDIOPULMONARY BYPASS

This is an important application of TCD ultrasonographic monitoring for a nonneurosurgical procedure, particularly in patients with ischemic cerebrovascular disease. TCD ultrasonographic monitoring during cardiopulmonary bypass allows optimal management of perfusion pressure to maintain CBF as well as diagnosis of cerebral emboli. A detailed discussion of these issues, however, is beyond the scope of this article.

NONINVASIVE MONITORING OF INCREASED ICP AND INADEQUATE CPP

As ICP increases and CPP correspondingly decreases, the cerebral vascular resistance is elevated due to distal obstruction, resulting in a characteristic highly pulsatile flow velocity pattern. Continuing increase in ICP would initially result in a loss of diastolic flow, progressing to systolic spike and eventually to an oscillating flow pattern which signifies the onset of intracranial circulatory arrest.[30, 31, 33, 67] Thus, although not specific and not quantitative, TCD ultrasonographic monitoring allows the detection of this phenomenon of increased resistance due to inadequate cerebral perfusion and may facilitate anesthetic management of patients with increased ICP, avoiding further cerebral injury. We have documented a good correlation of pulsatility index with ICP and CPP in a series of patients undergoing general anesthesia for a variety of surgical procedures,[52] as did Hamburg et al[33] (Fig. 4). This noninvasive monitoring is particularly useful in institutions where ICP monitoring is not routinely used or in patients in whom ICP monitoring is not deemed to be indicated, and yet the patient may have decreased intracran-

ial compliance (that is, after a concussion or mild closed-head injury).

DETECTION OF INTRACRANIAL CIRCULATORY ARREST IN TRAUMA PATIENTS

Patients with multiple injuries having concomitant cerebral injury may require emergency abdominal or thoracic surgery prior to appropriate radiologic assessment of the extent of head injury. Although the mere presence of an oscillating flow pattern in the intracranial arteries as detected by TCD ultrasonography, consistent with intracranial circulatory arrest, is not a criterion of brain death, its persistence would inevitably result in death.[18,57,80] Thus, diagnosis of intracranial circulatory arrest with TCD ultrasonography in patients with head injuries as well as severe systemic injuries will aid the overall decision-making process.

MISCELLANEOUS USES

Seated or Head-up Position

In elderly patients and in those with poorly controlled systemic hypertension, the seated

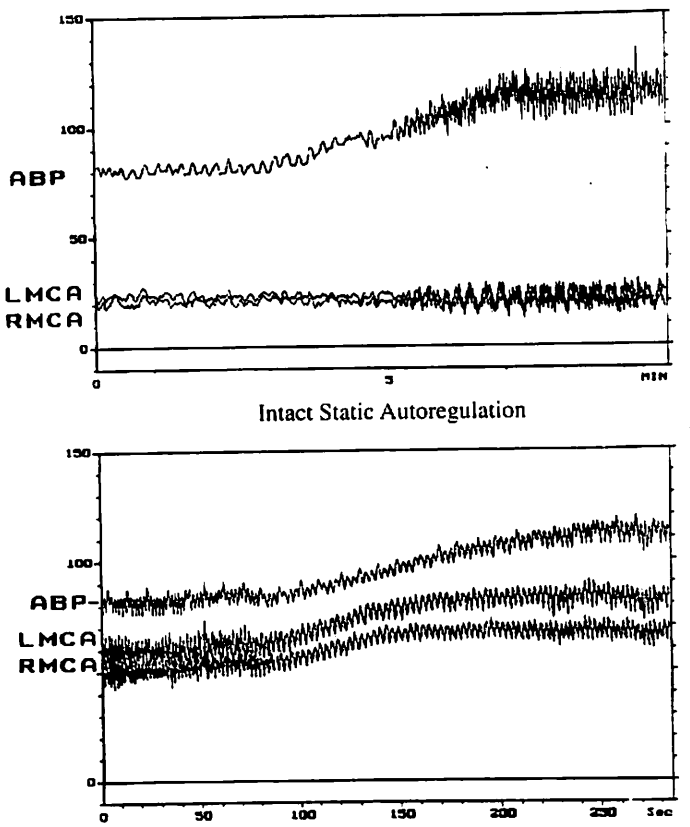

Figure 3. *A*, Measurement of autoregulation using the static method of increasing blood pressure to a new steady state using a phenylephrine infusion. The degree of MCA velocity change is illustrated with intact and impaired autoregulation using this method. *B*, Similar results are obtained using the dynamic method that uses a rapid short decrease in blood pressure induced by the release of thigh cuffs. Note the rapid return to baseline of the MCA velocities on both sides following the blood pressure drop, with intact autoregulation, and the persistent decrease with impaired autoregulation. *C*, Correlation between static and dynamic cerebral autoregulation measurements.

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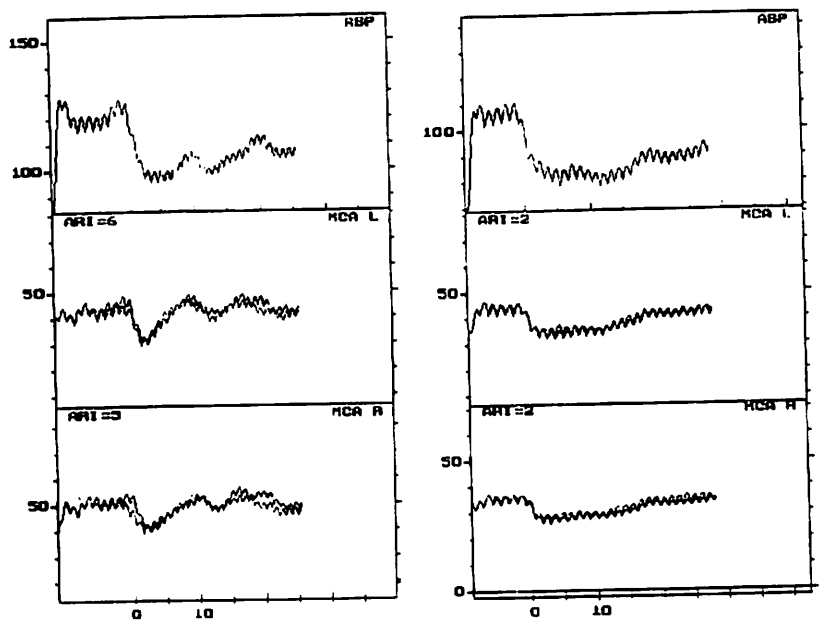
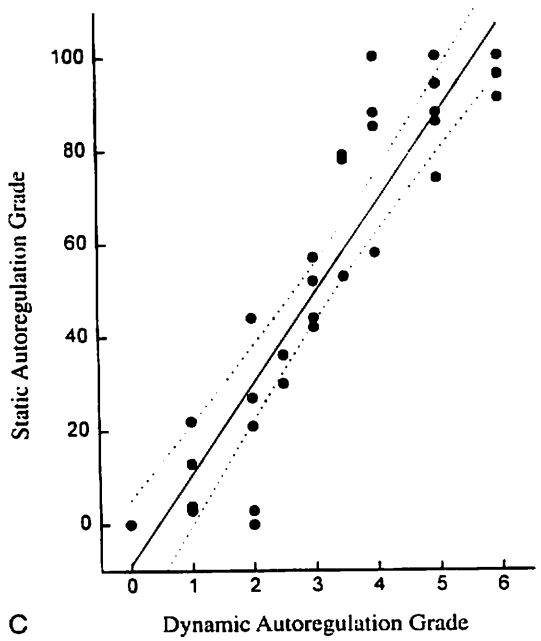


B Intact Dynamic Autoregulation Impaired Dynamic Autoregulation



C

Figure 3 (*Continued*).

position may result in an unacceptably low CBF. TCD ultrasonographic monitoring will help to determine the lower limit of autoregulation and thus the need for blood pressure support. Approximately 25% of the normal population has probe-patent foramen ovale, which places these patients at risk of paradoxical embolus should venous air embolism occur in the seated position. Recent studies have confirmed that TCD ultrasonography has similar

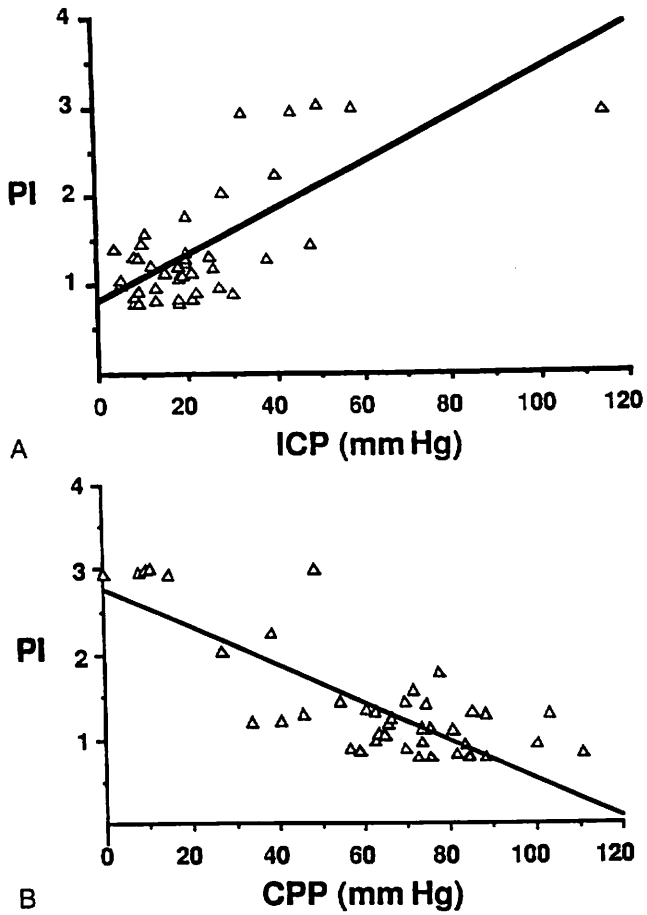


Figure 4. *A*, Correlation between perioperative measurements of pulsatility index (*PI*) and intracranial (*ICP*) in a series of 25 patients with cerebral injury undergoing surgical procedures. There is a linear correlation between PI and ICP with an r value of 0.74. *B*, Correlation between pulsatility index (*PI*) and cerebral perfusion pressure (*CPP*) in the same patients depicted in Figure *4*. There is a linear inverse relationship (r = −0.79) and the strength of this relationship is slightly better than that between PI and ICP. PI is probably more reflective of the net driving pressure (CPP) than the distal resistance or obstruction (ICP or venous pressure).

sensitivity to transesophageal echocardiography in the diagnosis of a patent foramen ovale.[12, 38, 55, 76] As TCD ultrasonography is a less invasive monitor, it would be a reasonable screening tool prior to placing a patient in the seated position.

Efficacy of Hyperventilation in Reducing CBF

Hyperventilation is commonly employed during anesthesia for neurosurgical procedures. Hyperventilation reduces CBF and cerebral blood volume and improves the operating conditions. In patients with pre-existing increased ICP, hyperventilation reduces ICP and improves CPP. On the other hand, excessive hyperventilation may lead to cerebral ischemia. In head-injured patients, carbon dioxide reactivity may be reduced, making hyperventilation a less effective means of controlling cerebral blood volume. TCD ultrasonographic monitoring thus may be useful in assessing the cerebrovascular response to hyperventilation, both in delineating the magnitude of the re-

sponse and in determining the plateau of the carbon dioxide response.

Other Uses

Intraoperative TCD ultrasonographic monitoring has been used to document increase in CBF with release of limb tourniquet.[32] A tourniquet is frequently used during lower extremity surgery to reduce blood loss. Its release and subsequent reperfusion after a period of occlusion lead to an increase in arterial carbon dioxide tension which leads to an increase in CBF. Thus sudden increase in flow, otherwise well tolerated in normal individuals, may cause an increase in ICP in patients with compromised intracranial compliance. Intraoperative TCD ultrasonographic monitoring may allow fine-tuning of ventilation to prevent or ameliorate this complication. Similarly, some patients undergoing orthotopic liver transplantation develop hyperemia following reperfusion and may suffer from intracranial hypertension. This phenomenon has been documented with TCD ultrasonography, but whether TCD ultrasonographic monitoring will aid in the clinical management of these patients remains to be seen.

CONCLUSIONS

As an investigative tool, TCD ultrasonography has been used in a variety of studies to examine the influence of anesthetic agents and techniques on CBF and cerebrovascular reactivity to carbon dioxide.[75,78] Although no direct benefits can be accrued to the patients studied, the information yielded may ultimately improve patient safety.

The introduction of TCD ultrasonography has made it possible to monitor intraoperative CBF changes in a continuous, noninvasive manner. Further refinement in the technology will delineate its appropriate use as an intraoperative monitor.

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