CHAPTER 10

Head Injury and Cerebral Circulatory Arrest

David W. Newell, Rolf W. Seiler, and Rune Aaslid

The evaluation of cerebral blood flow (CBF) following head injury is of great interest to clinicians and investigators. Various abnormalities in CBF have been studied in human head injury and in experimental animal models. Some of the abnormalities noted include impaired autoregulation, altered CO_2 reactivity, increased CBF (hyperemia), decreased CBF (oligemia), and vasospasm (1–4). It is believed that certain CBF abnormalities can lead to secondary damage to the brain in head-injured patients (5,6).

Various methods to evaluate CBF following head injury available for clinical use include 133-xenon clearance using portable detectors, single-photon emission computed tomography (SPECT), and thermal dilution probes (7–10). Transcranial Doppler (TCD) can be used to monitor relative changes in CBF as well as to detect vasospasm following head injury (4,11). This emerging technology promises to be very valuable in the acute care setting.

This chapter discusses some background information on physiological and hemodynamic changes that occur following head injury and how this information can be applied to clinical management. It also discusses the potential role of TCD in the evaluation and assessment of these hemodynamic changes.

BLOOD FLOW ABNORMALITIES FOLLOWING HEAD INJURY

Most of the studies performed to evaluate CBF following human head injury have been performed using

D. W. Newell: Department of Neurological Surgery, University of Washington School of Medicine, Harborview Medical Center, Seattle, Washington 98104.

R. W. Seiler: Department of Neurosurgery, University of Bern, Inselspital, CH-3010 Bern, Switzerland.

R. Aaslid: Department of Neurosurgery, University of Bern, Iselspital, CH-3010; Department of Neurological Surgery, University of Washington School of Medicine, Seattle, Washington 98195.

external detectors to detect the washout of injected 133-xenon. Obrist et al. (9), using this method, have shown that CBF can be normal, increased, or decreased following severe head injury and may or may not be as closely linked to cerebral metabolism as it is under normal circumstances. When CBF is markedly elevated beyond the metabolic demand, the term "hyperemia" is applied. Hyperemia has been found to be associated with high intracranial pressure (ICP) and may lead to secondary hemorrhages (9,12). CBF can be abnormally low following head injury due to low cerebral metabolism or, in some instances, to high ICP.

Autoregulation has been defined as the ability of the brain to maintain a constant blood flow in the face of changes in blood pressure. This response was initially observed by Lassen (13), who measured CBF at different blood pressures using 133-xenon blood flow methods. Impairment in cerebral autoregulation has been demonstrated in head-injured patients (2). The result of this impairment is that CBF can change passively with alterations in blood pressure instead of being tightly regulated as under normal circumstances. In patients with impaired autoregulation, decreased cerebral perfusion pressure can lead to secondary ischemia, or unwanted increases in perfusion pressure may lead to vasogenic edema (12).

Another frequently observed abnormality is impairment in the CBF response to changing P_aCO_2 (1,2), which is seen especially when the brain tissue is severely damaged. This may have clinical implications in guiding the use of hyperventilation in the intensive care setting and may also have value in predicting prognosis for recovery from brain injury.

Clinical Aspects of Cerebral Blood Flow Evaluation

Langfitt and Obrist (5) have stated four clinical applications of 133-xenon CBF measurements following

head injury (5). These are: (a) insuring adequacy of CBF, (b) use in outcome prediction, (c) assessing cerebrovascular responses such as autoregulation and CO_2 reactivity, and (d) assessing the effects of various treatments on CBF. TCD sonography does not yield absolute CBF values at present and therefore cannot be used to try to quantitate the adequacy of CBF following head injury. However, by observing relative changes in flow using TCD, autoregulation and CO_2 reactivity can be assessed (14–16), and TCD monitoring can potentially be used to assess the effects of various treatments on CBF.

MANAGEMENT OF HEAD INJURY

The strategy for the management of closed head injury is to provide the brain with the optimal conditions required for the recovery of neurological damage and to prevent secondary injury from occurring. Recovery of neurological function following severe head injury can occur to a remarkable degree if secondary insults do not compound the initial damage (6,17). Secondary injury can occur as a result of ischemia from hypotension or increased ICP during the postinjury period. Ischemic brain damage has been found in a large number of patients dying from head injury (18). Neurosurgical intervention is often needed to remove mass lesions such as epidural, subdural, and intracerebral hemorrhages and contusions that threaten to cause additional neural damage by compression, distortion, and ischemia of brain tissue. ICP monitoring is commonly used in the management of head injury to detect expansion of mass lesions and to guide therapy directed at providing optimal conditions for cerebral perfusion. An essential consideration in management of head injury, whether in the emergency room or the intensive care unit, is to maintain adequate cerebral perfusion and oxygenation. Aggressive measures to treat secondary injury and insure adequate cerebral perfusion during the resuscitation and intensive care phases of head injury treatment can be beneficial (19). It is therefore desirable to be able to detect changes in the cerebral circulation that can lead to secondary damage, and TCD may prove to be useful for this purpose.

TCD MONITORING IN HEAD INJURY

Theoretical Considerations

Monitoring of the blood flow velocity of the middle cerebral artery (MCA), both intraoperatively and in the intensive care unit, has been reported (11,16). In order to record continuously, the Doppler velocity signal can be taken from the outline of the velocity spectrum, which is normally calculated by the TCD instrument.

This tracing corresponds to the blood flow velocity at the center of the MCA. During laminar flow, which will occur in the basal arteries in the absence of any significant turbulence, the flow velocity at the center of the MCA will be directly proportional to the cross-sectional velocity. Changes in velocity will directly reflect changes in blood flow through this conducting artery, provided its diameter does not change significantly, since flow is a product of the velocity multiplied by the cross-sectional area of the vessel. There is a linear correlation between the outline of the velocity spectrum of the MCA and volume flow in the internal carotid artery (ICA), measured by an electromagnetic flowmeter during flow variations (11). This finding indicates that the MCA diameter does not change significantly with variations of flow under these circumstances. Therefore, changes in the outline of the MCA velocity spectrum will directly reflect relative changes in flow. By using a fixed monitoring probe in the intensive care unit, information about cerebrovascular response in head injury patients can be obtained. Several recent publications report the use of TCD monitoring in the intensive care setting to evaluate cerebrovascular responses such as autoregulation and CO_2 reactivity in neurosurgical patients (15,16).

Clinical Evaluation of TCD Monitoring

We studied a group of 21 patients admitted to the intensive care unit with a diagnosis of closed head injury to evaluate the clinical utility of continuous TCD monitoring. Testing of autoregulation, CO_2 reactivity, and response to barbiturates was performed in selected patients. We have employed a computerized digital storage system to obtain continuous simultaneous recordings of arterial blood pressure, ICP, end-tidal CO_2 (ET CO_2), and mean velocity. A computerized analysis was then performed to evaluate cerebrovascular responses. Figure 1 shows simultaneous recording of blood pressure, ICP, CO_2, and MCA velocity in a patient following head injury.

Autoregulation

TCD can be used to record changes in flow velocity in the MCA, which reflect relative changes in CBF. The response of the CBF to blood pressure fluctuations can be used to evaluate the autoregulation response. Correlations can be observed between the velocity change and spontaneous changes in blood pressure, or, alternatively, changes in blood pressure can be induced while observing the velocity response. The effects of many antihypertensive medications on the MCA diameter has not been adequately studied at this time and therefore it may not be appropriate to use
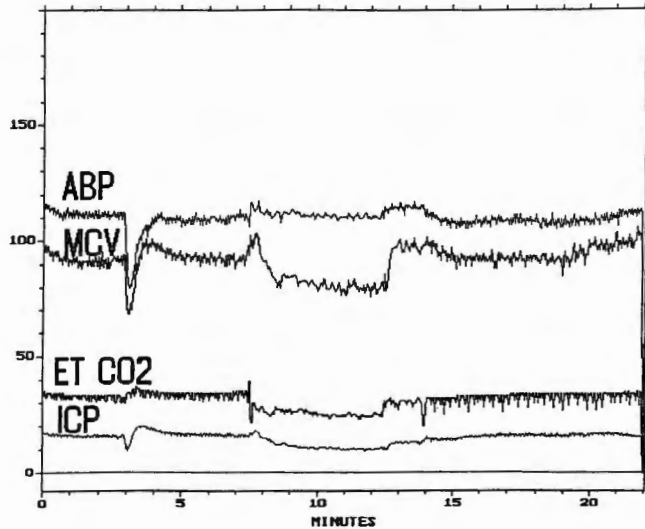
FIG. 1. Continuous recording of arterial blood pressure (ABP) in mm Hg, middle cerebral artery velocity (MCV) in centimeters per second, intracranial pressure (ICP) in mm Hg, and end-tidal CO_2 (ET CO_2) concentration in mm Hg in a patient after closed head injury.

these drugs to induce blood pressure changes for this purpose.

We have used the method of Aaslid et al. (14) to study the autoregulatory response by inducing rapid blood pressure changes. Recent experimental evidence supports the validity of this method (20). The rapid blood pressure changes are produced using large blood pressure cuffs placed around the thighs. These are then inflated for a period of time and quickly released, inducing a rapid blood pressure drop. The velocity response is measured and then a calculation is made of the change in cerebrovascular resistance (CVR) in response to this blood pressure drop to evaluate the effectiveness of the autoregulation. The CVR is calculated by dividing the mean velocity (used as an index of relative CBF) by the arterial blood pressure (ABP) for each time point. The CVR changes in a linear fashion over the period of time between 1 and 5 sec following the blood pressure drop. A regression line is then fitted to the slope of this change, which defines the rate at which the CVR changes. The CBF would be regulated back to the initial value if the change in resistance fully compensated for the drop in blood pressure. An index of the rate of regulation (RoR) was therefore defined as the change in CVR divided by the change in time over the change in ABP. The final index (RoR) gives the percentage of the adjustment required to correct the CBF back to its baseline value, which occurs per second. In a group of normal volunteers at normocapnia, Aaslid et al. (14) found the normal value for RoR to be $20.0 \pm 3.0\%$.

Using this method we have evaluated the autoregulatory response in 20 head injury patients in the in-

tensive care unit. The RoR value was analyzed separately for the individual hemispheres. The values ranged from 0 to 29.4 in the 118 studies performed. The range of values indicated that in head injury the autoregulatory response could be completely absent, completely intact, or impaired to various degrees. In the patients with RoR values of 15 or above, spontaneous blood pressure changes of at least 20 sec duration produced minimal changes in velocity. In patients with RoR values of 5 or below, similar spontaneous blood pressure changes produced passive changes in MCA velocity, indicating that the CBF was pressure passive. Figure 2 illustrates examples of intact and absent autoregulation, assessed by this method, in patients following head injury.

CO_2 Reactivity

Reactivity to CO_2 was tested using continuous recording methods by observing the velocity change in the MCA following hyperventilation, which reflects blood flow changes caused by changes in the regulatory arteries. Experimental evidence strongly supports the rationale for this method. Measurements of the cerebral arteries in humans during angiography indicate that the diameter does not change significantly in the large basal arteries in response to changes in arterial CO_2 concentration (21). In contrast, the more distal arteries undergo marked changes. The results of CO_2 reactivity in normal volunteers, as well as in patients with pathological conditions, yield results similar to those obtained using other methods (22–24).

The response was measured in 143 tests performed in the same group of 21 patients with closed head injury. Patients were temporarily disconnected from ventilators and hyperventilated using Ambu bags for a period of at least 2 min. The percentage of change in velocity was used as an index of the relative change in CBF. This value was then divided by the change in ET CO_2 in mm Hg to yield a % change/mm Hg. Hypoventilation was also performed in selected patients but often caused significant increases in ICP. Figure 3 illustrates examples of intact and absent CO_2 reactivity in head injury patients.

The MCA response to hyperventilation varied between 0% and 4.63% change/mm Hg in this group of patients. Absent CO_2 reactivity was generally found in patients with very severe brain injury or in MCA distributions with extensive focal lesions. Knowledge of the integrity of the CO_2 response was helpful in determining the potential effectiveness of hyperventilation for ICP control in individual patients.

Barbiturate Reactivity

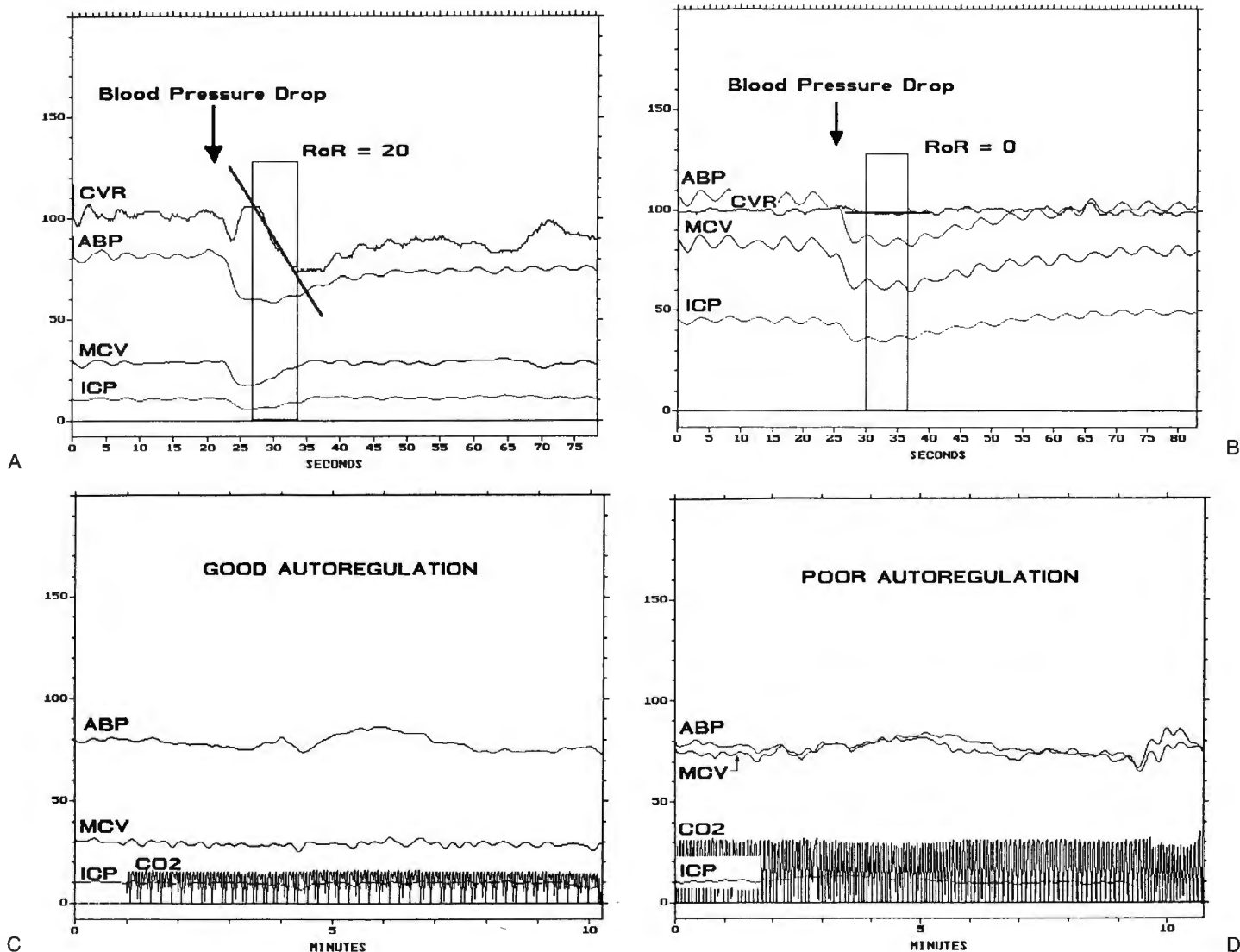Barbiturates have been used to lower elevated ICP in patients with head injuries. The mechanism of action

FIG. 2. A: Illustration of the MCA velocity response to an induced blood pressure drop, using leg cuffs, in a patient with intact cerebral autoregulation (rate of regulation = 20). **B:** Response in a patient with absent cerebral autoregulation (rate of regulation = 0). Middle cerebral artery velocity response to spontaneous blood pressure in patients with good **(C)** and poor **(D)** cerebral autoregulation. APB, arterial blood pressure in mm Hg; MCV, middle cerebral artery velocity in centimeters per second; ICP, intracranial pressure in mm Hg; ET CO_2, end-tidal CO_2 concentration in mm Hg; RoR, rate of regulation; CVR, cerebrovascular resistance.

is believed to be secondary to barbiturate-induced reduction of cerebral metabolism. This in turn reduces CBF and thus cerebral blood volume, which results in a reduction of ICP. The change in CBF in response to barbiturate infusion has been found to correlate with the potential effectiveness of barbiturates to lower ICP in individual patients (25). In head injury patients, barbiturates are often ineffective in lowering ICP. This can be either because of already severely reduced cerebral metabolism or failure of CBF to respond to a reduction in metabolism (1). Although changes in metabolism cannot be assessed using TCD monitoring, this technique can be used to evaluate relative blood

flow changes and also to assess blood pressure and ICP, before and after the administration of a test dose of barbiturates. The available evidence indicates MCA velocity closely reflects CBF changes following barbiturate infusion (11), but further studies are needed to confirm these results.

Testing of barbiturate response using TCD monitoring was performed on 20 occasions in a group of patients with head injury. Thiopentone, which is a commonly used barbiturate, was given intravenously at a dose of 5 mg/kg according to the method of Cold (1). The results of ABP, ICP, ET CO_2, and MCA velocity were observed for a period of at least 10 min.
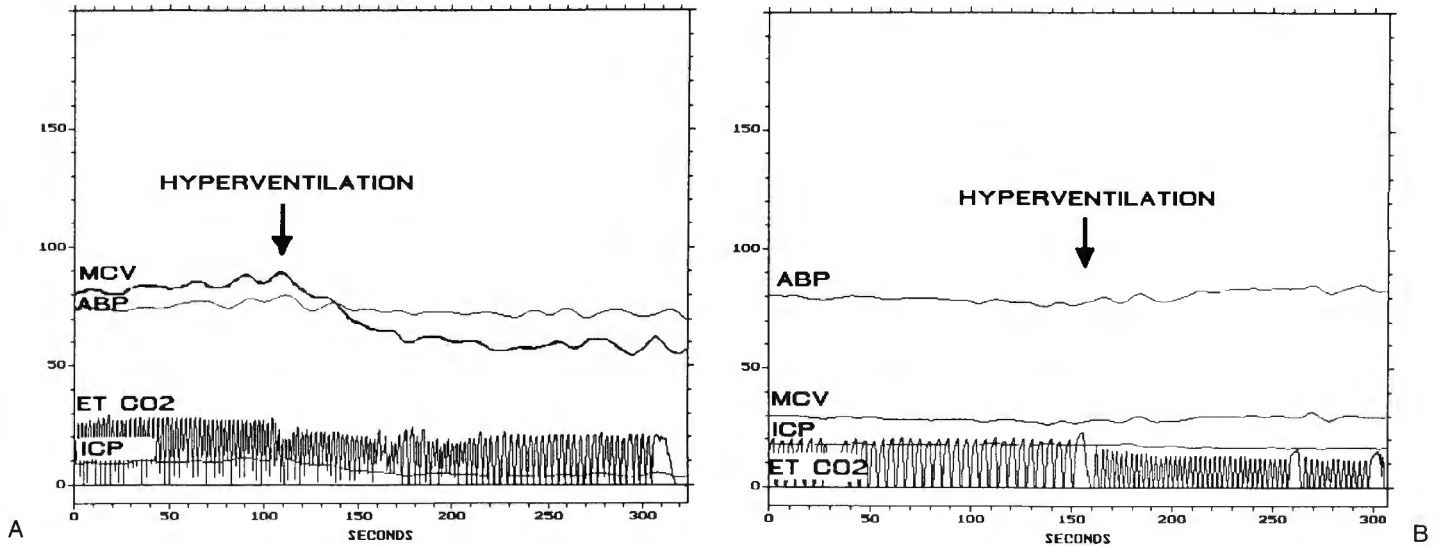
FIG. 3. Responses to hyperventilation. **A:** Good CO_2 response with marked decrease in middle cerebral artery (MCA) velocity and decreased intracranial pressure (ICP). **B:** Absent CO_2 response with no change in MCA velocity or ICP following hyperventilation. Abbreviations as in Fig. 2.
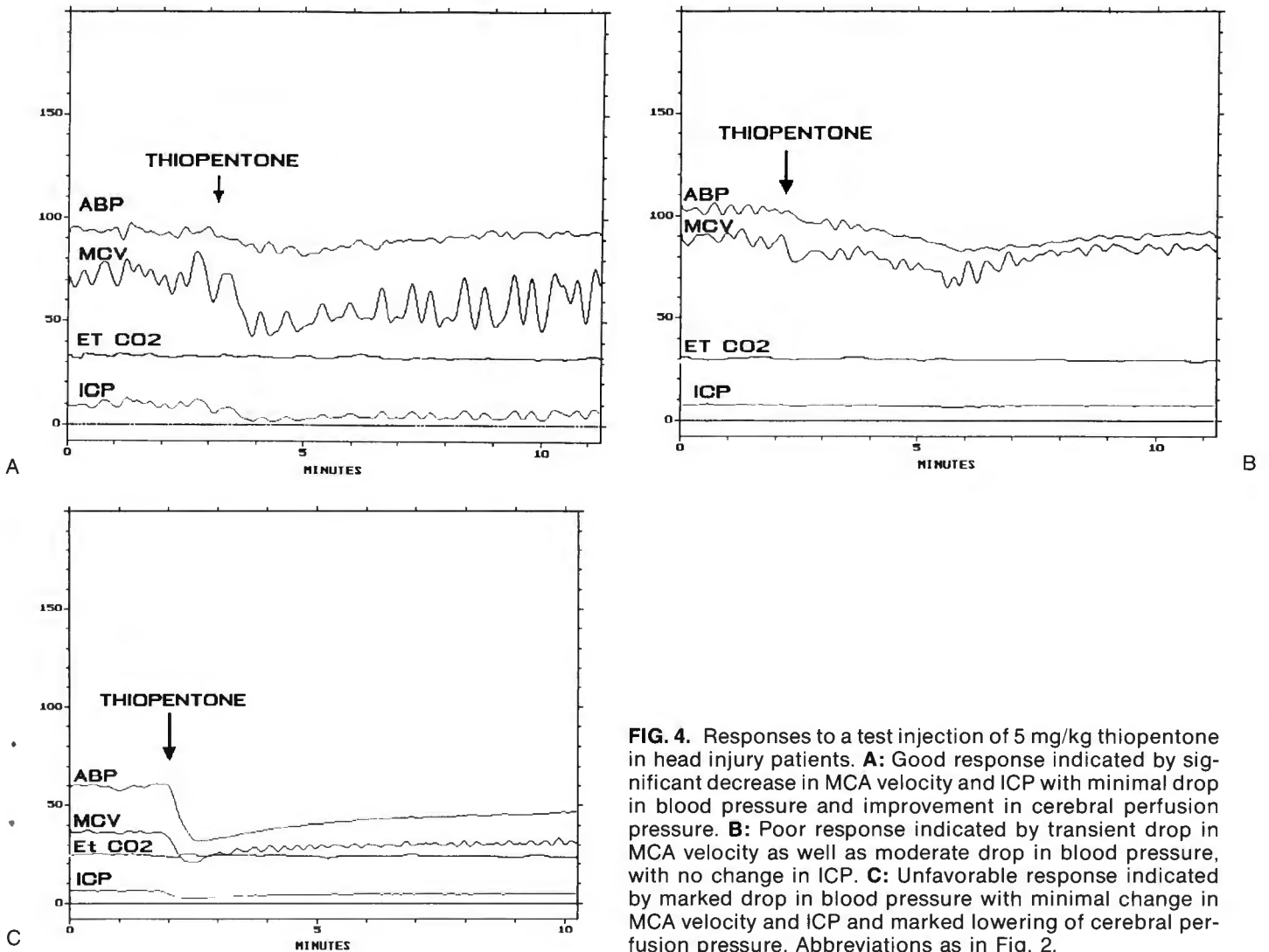

FIG. 4. Responses to a test injection of 5 mg/kg thiopentone in head injury patients. **A:** Good response indicated by significant decrease in MCA velocity and ICP with minimal drop in blood pressure and improvement in cerebral perfusion pressure. **B:** Poor response indicated by transient drop in MCA velocity as well as moderate drop in blood pressure, with no change in ICP. **C:** Unfavorable response indicated by marked drop in blood pressure with minimal change in MCA velocity and ICP and marked lowering of cerebral perfusion pressure. Abbreviations as in Fig. 2.

The percentage of reduction of MCA velocity was then calculated. Responses of ABP, cerebral perfusion pressure (ABP-ICP), and ICP were also observed to evaluate whether or not the induced changes had beneficial or detrimental effects.

All patients had a reduction in MCA velocity following barbiturate administration. The percentage of reduction varied between 3.7% and 27.8%. ICP was decreased by barbiturate infusion on 16 occasions, unchanged on three, and increased on one. The ABP decreased on all tests. The cerebral perfusion pressure was increased on three occasions and decreased on 17. It was possible, by observing individual responses, to gain insight into the potential effects of the therapy. Figure 4 shows selected responses in this group of patients.

Conclusions

These results indicate that it is feasible to use TCD monitoring to evaluate cerebrovascular responses such as autoregulation, CO_2 reactivity, and barbiturate responses in neurosurgical patients. This information may be valuable in guiding therapy in the intensive care unit.

VASOSPASM

Vasospasm is well recognized as a cause of clinical deterioration following subarachnoid hemorrhage due to aneurysm rupture and can be produced experimentally by placing blood around the cerebral arteries of experimental animals (26,27). Since subarachnoid bleeding is common in head injury, it is likely that spasm of the intracranial arteries is produced by a similar mechanism. Angiographic vasospasm has been reported in patients following closed head injury (28). Clinical deterioration following head injury due to vasospasm is not commonly reported. It is possible that the entity is not well recognized due to the underlying severe neurological dysfunction caused by head injury during the period when the vasospasm in manifest.

Several studies of serial recordings of TCD velocities following head injury have been reported, and increased velocities in the basal arteries have been recognized (4,29). Increased velocity can be due to increased CBF as well as narrowing of the intracranial arteries, and hyperemia is a common occurrence after severe head injury. Therefore, a simultaneous index of CBF is needed to differentiate the two conditions. The index of intracranial/extracranial velocity recordings may be used for this purpose. By dividing the MCA velocity by the extracranial internal carotid artery (ICA) velocity (V_{MCA}/V_{ICA}), an index can be obtained to correct for CBF changes. Lindegaard et al.
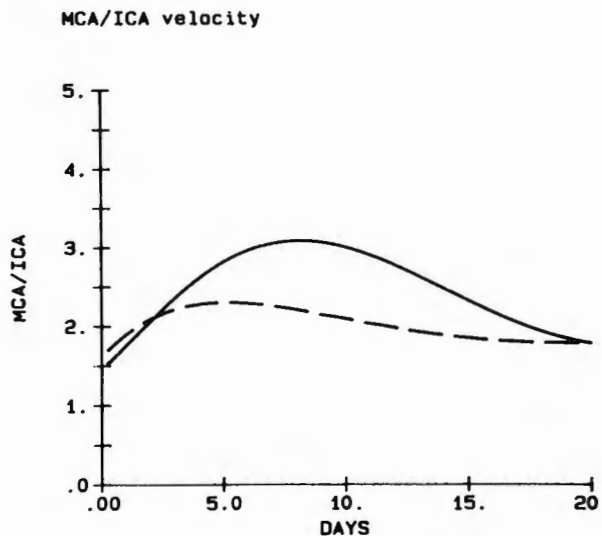
MCA/ICA velocity

FIG. 5. Mean time course of the MCA/ICA ratio of patients with CT scans predicting low risk of vasospasm based on blood distribution (*broken line*) compared to patients with CT scans predicting high risk for vasospasm (*solid line*). Abbreviations as in Fig. 2. (From ref. 4, with permission.)

(30) found the normal value for this ratio to be 1.7 ± 0.4, and a ratio greater than 3 corresponded to MCA spasm seen on angiograms. In a group of 35 patients with severe head injury examined serially with TCD, Weber et al. (4) found a 40% incidence of vasospasm during the first several weeks, indicated by a V_{MCA}/V_{ICA} ratio of greater than 3. The increase in velocity that occurred in this group of patients began as early as 48 hr and reached maximal values between 5 and 7 days after injury, which is slightly earlier than previously reported for subarachnoid hemorrhage. The occurrence of vasospasm also correlated with the amount of cisternal and intracerebral blood seen on computed tomography (CT) scan (Fig. 5). This correlation has been well established previously in subarachnoid hemorrhage (31,32).

Illustrative Case

A 43-year-old female was involved in a motor vehicle accident and was brought to the hospital in an unconscious state. Her CT scan revealed a right acute subdural hematoma, and in addition there was dense blood in the left sylvian fissure. A right craniotomy was performed to evacuate the subdural hematoma. Serial TCD examinations were performed. A postoperative scan documented removal of the subdural hematoma and showed that there were no infarctions present (Fig. 6). She improved clinically to the point where she was following commands and conversing. On the 6th postoperative day she became confused but
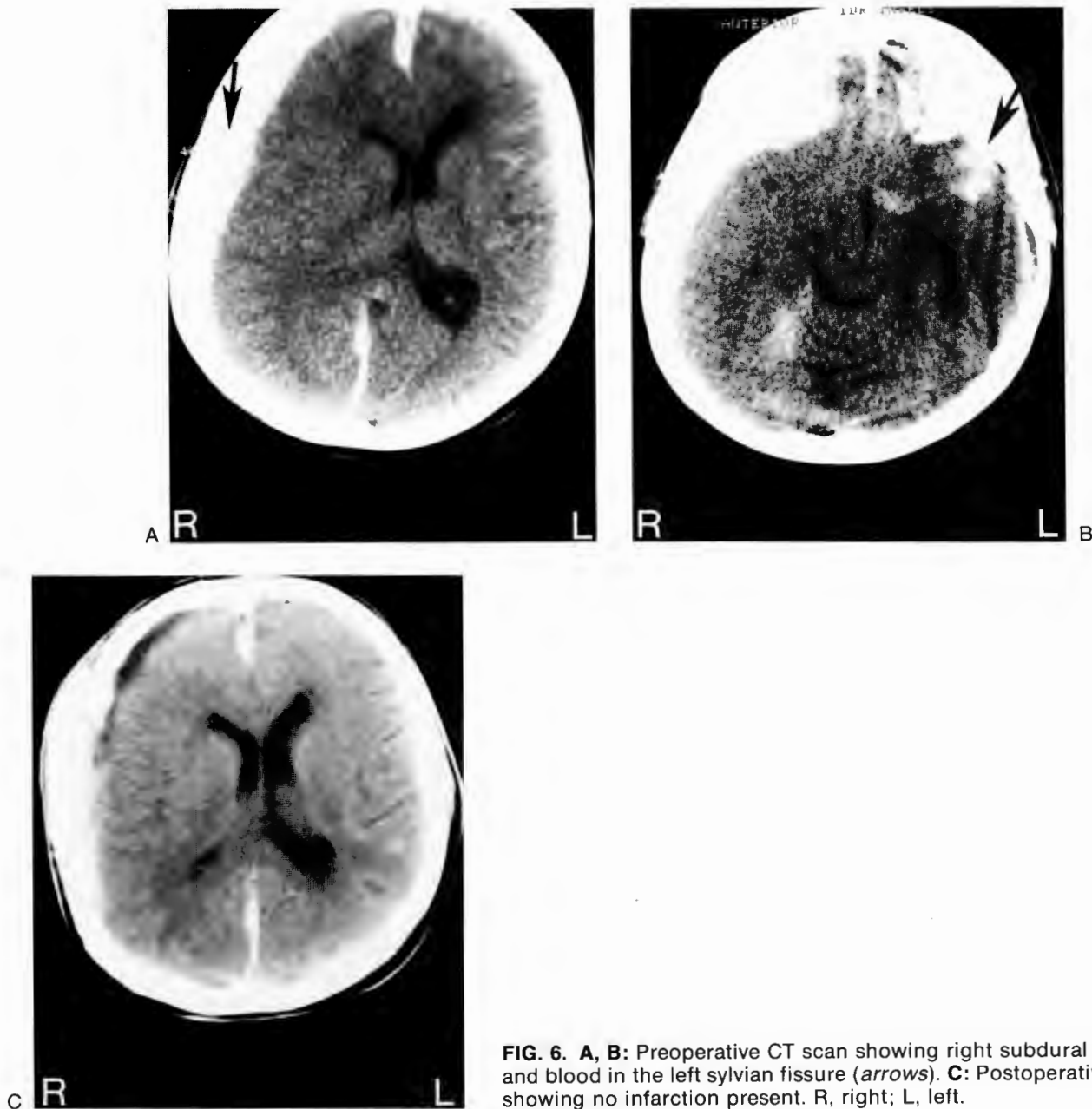
FIG. 6. A, B: Preoperative CT scan showing right subdural hematoma and blood in the left sylvian fissure (*arrows*). **C:** Postoperative CT scan showing no infarction present. R, right; L, left.

had no new focal neurological deficits. Her left MCA velocity was markedly elevated to a mean velocity of 306 cm/sec at this time, and there had been a decrease in her extracranial ICA velocity. An angiogram was performed that demonstrated severe spasm of the left MCA. A CT scan was subsequently obtained that showed two small infarctions in the left hemisphere that were not present on her postoperative CT scan, before the vasospasm occurred (Fig. 7).

Comment

This case illustrates that vasospasm can occur following head injury and can cause secondary damage to the brain in the form of infarctions. It is probably produced by a mechanism similar to that which produces the vasospasm seen following subarachnoid hemorrhage from ruptured aneurysms, namely, a response to the presence of blood deposited around the basal arteries. It is therefore important to be aware of this potential complication, particularly in head injury patients whose CT reveals blood in the basal cisterns.

INCREASED INTRACRANIAL PRESSURE

Increased ICP is common following severe head injury, and ICP monitoring is now widely used. Sus-
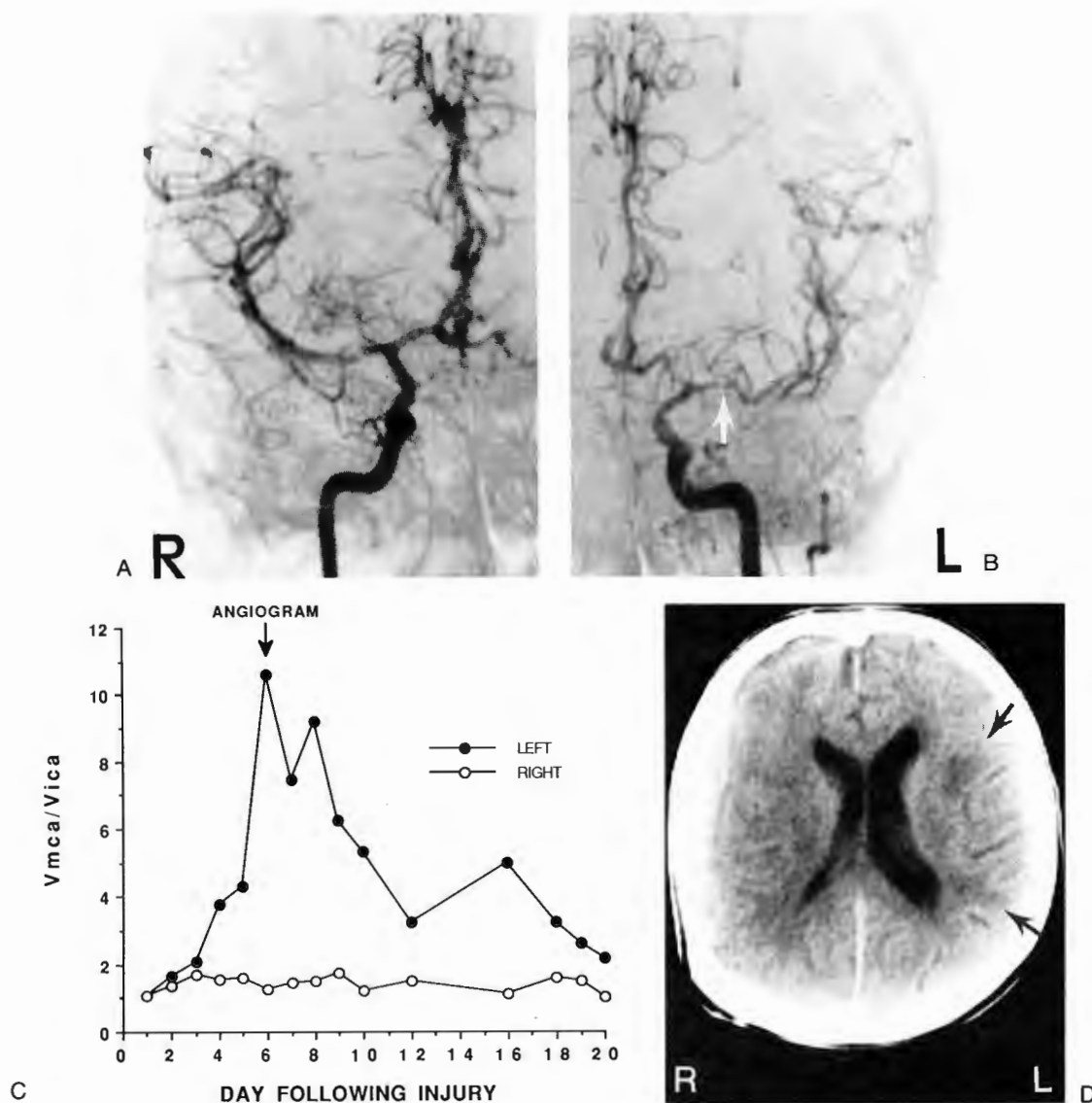
FIG. 7. Angiogram illustrating no vasospasm on the right (R) side **(A)** and severe vasospasm on the left (L) side **(B)** (*arrow*). **C:** Serial recordings of middle cerebral artery internal carotid artery velocity (Vmca/Vica) after head injury over time revealing high velocities on the left side. **D:** CT scan after vasospasm revealing infarctions in the middle cerebral artery distribution (*arrows*). R, right; L, left.

tained and severe increases in ICP following head injury indicate a poor prognosis and can cause secondary damage due to brain distortion and herniation (33,34). Elevated ICP can also cause ischemia by causing decreases in CBF. Normally, during moderate increases in ICP, compensatory mechanisms such as cerebral autoregulation and increases in blood pressure can maintain CBF at adequate levels (35). If these mechanisms are overcome by high ICP, CBF can become dangerously reduced. TCD has the potential to be used to detect reductions in CBF under certain conditions.

With increased ICP, characteristic changes can occur in the TCD waveform due to decreased per-fusion pressure (36–38). The first change is a reduction in diastolic velocity and an increase in systolic velocity, which cause an increase in pulsatility without significant changes in the mean velocity. As the ICP approaches the diastolic pressure at the level of the microcirculation, the velocity then begins to approach zero in end-diastole. Further increases cause a reversal of flow in diastole as the ICP increases above the blood pressure at the microcirculation in diastole (Fig. 8). Recognition of these characteristic TCD waveforms by experienced observers can provide valuable information and warn of compromise of the cerebral circulation. Moderate increases in pulsatility alone, however,
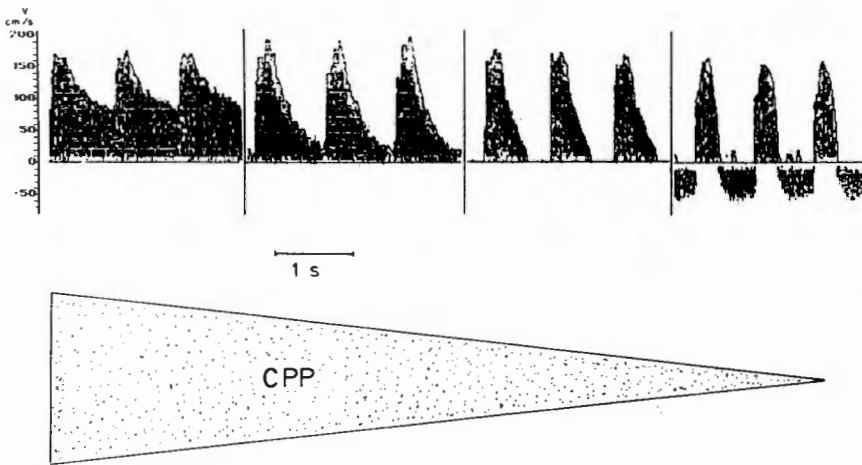
1 s

CPP

FIG. 8. Progression of the TCD waveform seen with progressive decrease in the cerebral perfusion pressure (CPP). (From ref. 37, with permission.)

are not specific to increased ICP and can be seen with vasoconstriction due to low metabolic rate or decreased CO_2 concentration.

Illustrative Case

An 8-year-old male was admitted following a bicycle accident in an unconscious state and was found to have a right subdural hematoma. This was removed by a craniotomy with subsequent improvement in clinical condition. The following day he deteriorated neurologically and a repeat CT scan showed swelling of the right hemisphere. TCD examination of the MCAs showed a pattern consistent with a high resistance to flow on the left side and a pattern of impending cerebral circulatory arrest on the right side. A right-sided decompressive craniectomy was performed, and postoperative TCD examination showed that both waveforms had returned to a less resistive pattern (Fig. 9). Despite improvement in the blood flow velocity on both sides, he developed extensive infarctions throughout the right hemisphere.

Comment

This case demonstrated impairment of the cerebral circulation due to brain swelling and increased ICP. It is of particular interest that the findings were more pronounced on one side than the other and that the patient subsequently developed infarctions on the more severely affected side. The TCD was helpful in defining the pathophysiology and may prove useful in the future to monitor changes similar to those seen in this case.

ARREST OF THE CEREBRAL CIRCULATION AND BRAIN DEATH

CBF measurements have demonstrated that at very high ICP, CBF can decrease (39). The reduction of CBF at elevated ICP will depend on each individual's blood pressure response as well as the state of autoregulation. Miller et al. (35) have demonstrated experimentally that increased ICP reduces CBF much earlier when autoregulation is impaired.

When one examines the Doppler signals from the basal cerebral arteries during increasing ICP to flow leading to cerebral circulatory arrest, a characteristic progressive change in the waveform is seen. The first change seen is progressive decrease in diastolic velocity. Subsequently diastolic velocity becomes zero in end-diastole, indicating that all of the forward flow is taking place in systole. With further increases in ICP, there is flow reversal during diastole demonstrated by negative velocity readings during the diastolic phase. The next stage seen reveals the presence of small systolic peaks in early systole, indicating a brief forward thrust of blood during expansion of the artery. In the final stage, no signals can be obtained from the basal arteries. By detecting and recognizing this characteristic progression of changes, one can use TCD to identify patients with arrest of the cerebral circulation.

Experimental studies indicate that as the distal cerebral vessels become smaller, the intravascular pressure drops (40). When the ICP reaches the level at which it obstructs the circulation, the functional obstruction probably takes place at the microcirculation. Arteriographic studies in humans indicate that during the initial stages of intracranial circulatory arrest, the larger conducting arteries remain patent despite no net forward flow (41). The presence of patent conducting arteries distal to the TCD recording site allows to and fro blood movement to occur during the cardiac cycle. Forward flow, which occurs in systole, expands the arterial tree, but due to the very high distal resistance, little or no flow occurs through the microcirculation. During diastole, the flow is forced in the reverse direction by the contractive forces of the arteries.
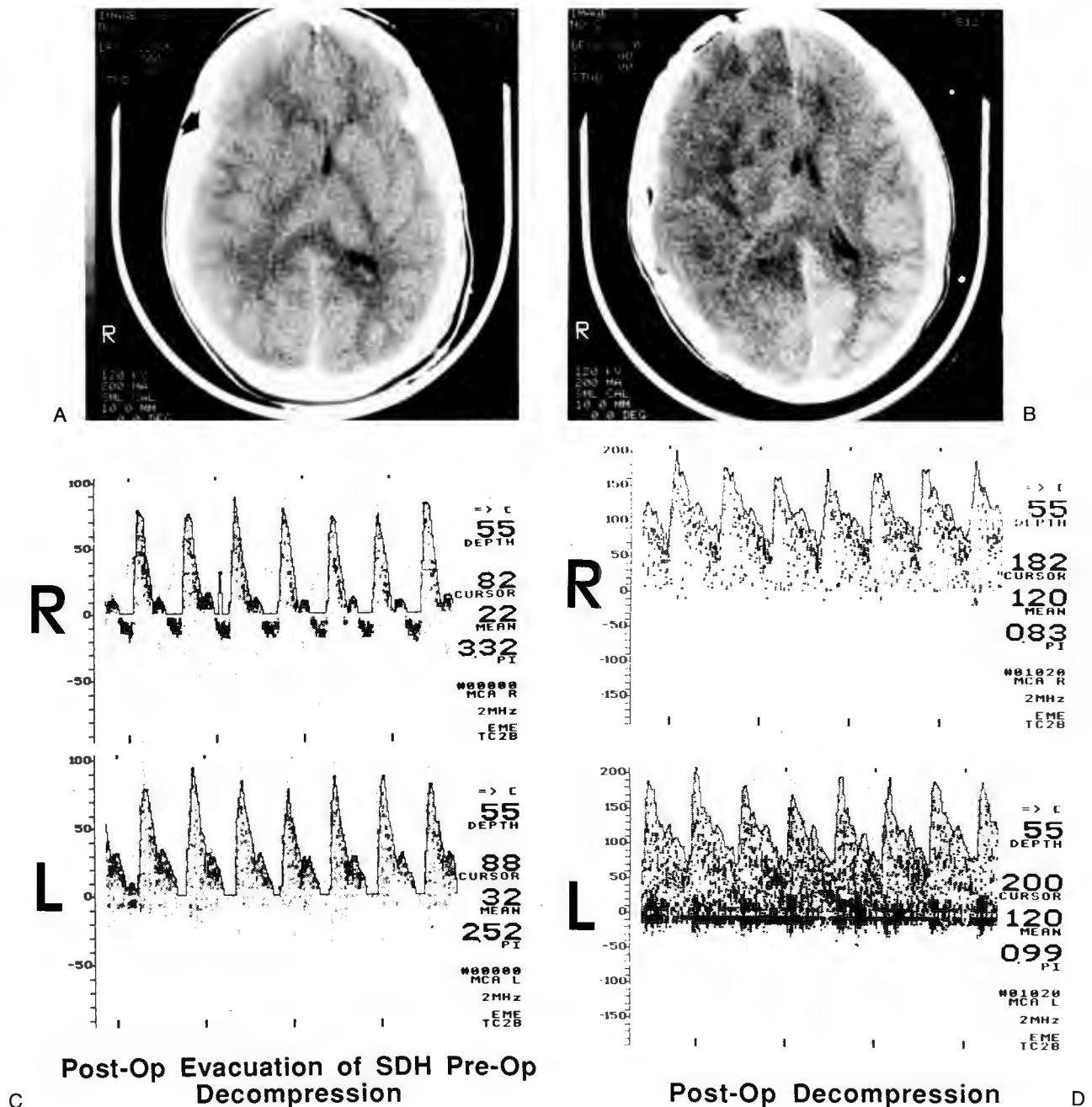
FIG. 9. A: Initial CT scan on a patient with a small right-sided subdural hematoma (*arrow*). **B:** Repeat CT scan 48 hr later showing swelling of the right hemisphere. **C:** Middle cerebral artery (MCA) velocity signals from both sides illustrating high resistance to flow on the left and impending cerebral circulatory arrest on the right. **D:** Repeat recordings after decompressive craniectomy was performed showing improved flow velocity on both sides. R, right; L, left; SDH, subdural hematoma.

By comparing the TCD waveforms during the various stages of cerebral circulatory arrest with arteriographic findings, Hassler et al. (41) identified the TCD patterns that correspond to arrest of the circulation by angiographic criteria (Fig. 10). The presence of to and fro TCD signals in the MCAs indicating net zero flow indicates supratentorial circulatory arrest. This has now been confirmed by angiography as well as by technetium scanning (41–43). It has also been noted, however, that arrest of the cerebral circulation can be a reversible phenomenon if it occurs for short periods of time. Grote and Hassler (44) recorded TCD signals during rebleeding episodes in patients with cerebral aneurysms. One patient maintained a to and fro flow
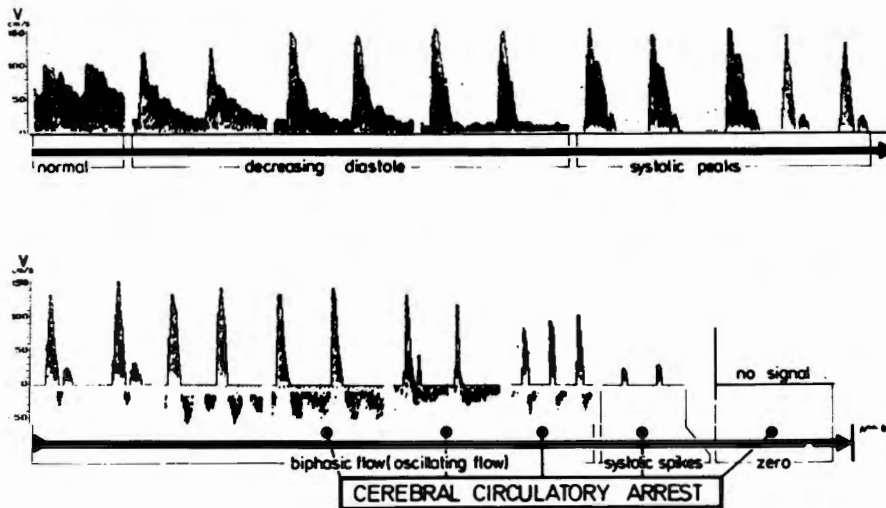
FIG. 10. The progressive changes in the TCD waveform seen with conditions leading to cerebral circulatory arrest compared with angiographic findings. (From ref. 41, with permission.)

pattern for 2 min, and then the pattern gradually returned to normal. This patient went on to recover.

The ability to detect the arrest of the cerebral circulation is useful for clinical purposes, one of which is the confirmation of cerebral circulatory arrest in cases of brain death. The guidelines set forth for the determination of brain death in the United States by the President's Commission state that the diagnosis of brain death must be made by a qualified physician on the basis of clinical findings and that CBF studies can only be used as confirmatory tests (45). Therefore, arrest of the cerebral circulation is not synonymous with brain death in all cases according to these guidelines, unless cessation of all brain function for at least 6 hr is observed. Arrest of the cerebral circulation may occur briefly due to short episodes of increased ICP and be associated with clinical recovery. Supratentorial circulatory arrest has also been noted to occur in certain cases progressing to brain death before all the brainstem reflexes have disappeared (37,46,47). Conversely brain death has been diagnosed clinically on the basis of absent brainstem function, in cases in which the supratentorial circulation is preserved for a period of time (47,48). This may occur in cases of massive destructive brainstem hemorrhage or posterior fossa lesions that compress the brainstem.

TCD as well as other types of blood flow studies can nonetheless be helpful to evaluate the intracranial circulation and confirm intracranial circulatory arrest in cases of suspected brain death. This is especially useful in cases in which clinical evaluation is made difficult due to drugs, cranial nerve damage, or other factors. It must be emphasized, however, that even though patients with intracranial circulatory arrest will all progress to brain death if the time period is sufficient, under some circumstances, the two conditions are not synonymous.

A number of publications have appeared that have addressed the usefulness of TCD in confirming the diagnosis of brain death (42,43,47–51). Kirkham et al. (51) described MCA signals obtained in 23 children with a poor outcome due to intracranial lesions. No patients survived who were observed to have an MCA mean velocity less than 10 cm/sec for longer than 30 min. Ropper et al. (48) reported TCD findings in a group of 24 adult brain dead patients. To and fro patterns or variants of them were found in 19 patients. In three patients with absent brainstem reflexes but persistent electroencephalographic activity, indicating cortical activity, TCD patterns were normal. Powers et al. (43) reported TCD findings in a group of 24 patients with cranial injuries, of which 23 progressed to brain death. No survivors were found if they had a mean forward flow velocity less than 10 cm/sec in the MCAs. Petty et al. (47) obtained results from 49 comatose patients, 23 of whom progressed to brain death. When TCD signals were found that demonstrated absent or reversed diastolic flow or small early systolic spikes in at least two intracranial arteries, these findings had a 91.3% sensitivity and a 100% specificity for brain death.

SUMMARY

Abnormalities of CBF are known to occur following human head injury, and information about these abnormalities can help in the neurosurgical management. Currently TCD cannot quantitatively measure volume flow through the intracranial arteries, but it can detect relative changes in CBF during changes in blood pressure and ICP and during changes in CO_2 concentration. This permits the assessment of physiological responses such as autoregulation and CO_2 reactivity, which can

be impaired in head injury. TCD also offers the ability to noninvasively detect vasospasm, which can be a cause of secondary deterioration in head injury patients. Decreased CBF and the arrest of the intracranial circulation from increased ICP can be evaluated using this technique, making it a valuable ancillary test in cases of suspected brain death.

ACKNOWLEDGMENTS

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