Relation of cerebral blood flow to neurological status and outcome in head-injured patients

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Noninvasive studies of regional cerebral blood flow (CBF) were performed on 36 head-injured patients in varying degrees of coma, using the intravenous xenon-133 method. Serial examinations, averaging four per patient, were begun during the acute phase of illness and continued until death or recovery of normal consciousness. Comparison of the initial and final studies revealed that CBF declined to very low levels in all nine patients who died, and remained subnormal in a patient with persistent vegetative state. In contrast, 25 of 26 patients who recovered consciousness showed increases in blood flow.

Because of the presence of both reduced and elevated blood flows on the initial study, CBF was not predictive of outcome. Absolute or relative hyperemia, observed in nine acute cases, was associated with either diffuse cerebral swelling (observed on computerized tomography) or recovery from systemic shock. Cerebral metabolic studies in hyperemic patients yielded a very low oxygen uptake and arteriovenous oxygen difference, indicating that the high blood flow was a true "luxury perfusion." When instances of presumed luxury perfusion were excluded, CBF was positively correlated with level of consciousness, assessed on a four-point coma scale.

KEY WORDS - acute head injury • coma • cerebral blood flow • computerized tomography • cerebral metabolism • hyperemia • outcome of head injury

REGIONAL cerebral blood flow (CBF) studies have been carried out in acute head injury by a number of investigators, using the xenon-133 ($^{133}$Xe) intracarotid injection technique. These studies have contributed to a better understanding of brain trauma by providing insight into its hemodynamic consequences. Most importantly, they have shown that posttraumatic coma is associated with marked variations in CBF, ranging from abnormally low values to pronounced hyperperfusion, and that the homeostatic regulation of blood flow is frequently impaired following injury. Continued investigation of these parameters, together with other physiological variables, is clearly indicated.

The invasiveness of the intracarotid technique imposes a limitation on the amount of information obtained. Both the number and timing of blood flow studies must be restricted, and with few exceptions, confined to a single hemisphere during carotid angiography. In contrast, the recent development of a noninvasive method of CBF measurement offers the possibility of making serial, bilateral observations with minimal patient risk. Not only are homologous regions of the two hemispheres readily compared, but repeated determinations can be made during the course of illness. The present paper describes our preliminary experience with the noninvasive $^{133}$Xe intravenous technique, based on 153 examinations in 36 patients with head injury. Emphasis will be given to the relationship between CBF, level of consciousness, and clinical outcome. Correlations with computerized tomography (CT) have been reported elsewhere.

Clinical Material and Methods

The sample consists of 36 patients with acute head injury (27 males and nine females) admitted to the Hospital of the University of Pennsylvania with varying degrees of coma and neurological deficit. Their ages ranged from 14 to 76 years (mean, 31 ± 15 years). All patients were examined by CT scan at the time of admission, and all had at least one follow-up examination. Sixteen patients underwent emergency surgery, primarily for evacuation of hematomas.

Following the CT scan or surgery, the patient was transferred to the Neuro-Intensive Care Unit where
appropriate therapy was instituted. Intracranial pressure (ICP) was monitored by a subarachnoid bolt in 22 patients, half of whom had elevated pressures while acutely ill. In 17 patients, the clinical condition required endotracheal intubation and controlled ventilation.

All CBF studies were carried out in the Neuro-Intensive Care Unit. More than four tests per patient were performed on the average. Each patient was followed until just before death (nine cases), or until recovery of normal consciousness (27 cases). In 15 patients the initial CBF study took place on the day of injury, while in 18 it occurred between the second and seventh days. Three patients with chronic hematomas were initially examined between 2 and 5 weeks after trauma. A final CBF study was obtained in the survivors at the time of hospital discharge or during a subsequent outpatient visit (median = 61 days after injury).

Measurements of Cerebral Blood Flow

Regional CBF measurements were obtained noninvasively by a modification of the technique of Obrist and coworkers. A 20- to 30-mCi bolus of $^{133}$Xe was injected intravenously, following which gamma radiation was monitored for 15 minutes by means of 16 extracranial detectors, eight over each hemisphere. The clearance curves were subjected to a two-compartment computer analysis that employed a correction for recirculation, based on isotope concentration in the expired air. This yielded an estimate of blood flow for the faster clearing compartment, $F_1$, which in normal subjects corresponds to cerebral gray matter.

In patients with intracranial pathology, estimates of $F_1$ are sometimes unstable due to variations in the relative size of the fast compartment. A second blood flow index was therefore used that is less sensitive to shifts in compartment size. This estimate, designated $\text{CBF}_{15}$, represents the mean flow of all tissue seen by a given detector, and includes a small extracerebral component in addition to cerebral gray and white matter. Although based on parameters derived from compartmental analysis, it is theoretically equivalent to the height-over-area method previously applied to intracarotid injection data. The effect of extracerebral contamination was reduced by integrating the curves to 15 minutes rather than to infinity.

In order to compare blood flow findings over time, all CBF measurements on a given patient were corrected to the median arterial $pCO_2$ obtained for that individual in the test series. Based on normal control data, correction factors of 3.0% and 2.5%/mm Hg change in $pCO_2$ were applied to $F_1$ and CBF $\text{CBF}_{15}$, respectively. Although such a correction did not equalize tests between subjects, it permitted inter-test comparisons within a subject. This procedure was preferred, rather than correcting to some arbitrary $pCO_2$ level, since it minimized adjustment of the blood flow values and reduced the effect of any error in the correction factors. Because the patients tended to hyperventilate spontaneously, correction to a normal $pCO_2$ of 40 mm Hg would have entailed adjustments of considerable magnitude. After deleting instances of induced hyperventilation, the average median $pCO_2$ across subjects was 33.3 mm Hg (SD = 3.5).

Neurological Examination

A detailed neurological examination was administered at the time of each CBF study, which included assessment of consciousness by means of the Glasgow Coma Scale. The latter was supplemented by a simple four-point grading system developed by us to classify the depth of coma. The four coma grades were assigned on the basis of responses to pain and to verbal commands, plus evidence of clouded consciousness. Definitions of these grades are as follows:

Grade 1: the patient is alert and interacts with his environment
Grade 2: the patient obeys simple verbal commands, but shows definite signs of somnolence, manifested by deficient arousal with stimulation, inappropriate lapses into sleep and slowed reaction times
Grade 3: the patient does not obey verbal commands, but gives appropriate withdrawal or localizing responses to pain
Grade 4: pain elicits reflex decorticate or decerebrate responses, or no response at all

In assigning grades, allowance was made for specific motor, sensory, and cognitive deficits that might interfere with test reactions. It should be noted that, in contrast to the Glasgow Coma Scale, Grade 4 is based on the worst rather than the best response.

Results

Neurological Findings

Table 1 classifies the patients according to coma grade at the time of their first CBF study. Whereas 27 of the patients were unresponsive to commands (15 in Grade 3, and 12 in Grade 4), nine revealed only a mild disturbance in consciousness (Grade 2). Agreement with the Glasgow Coma Scale was excellent, in that only two of the Glasgow scores (6 and 11) overlapped between grades.

As shown in Table 1, the initial coma grade also correlated well with survival and with the occurrence of intracranial hypertension. Among the 12 patients in Grade 4 (decerebrate or decorticate responses to pain), nine died and one remained in a persistent vegetative state (PVS). This contrasts with no instances of death or PVS when the initial coma grade was 2 or 3. Similarly, eight of 12 patients in Grade 4 developed an elevated ICP (above 20 mm Hg), as op-
Fig. 1. Fast compartment blood flow ($F_1$) in ml/100 gm/min on the initial and final cerebral blood flow studies of 10 patients with poor outcome. All patients were in Grade 4 coma during the initial study. **Dotted lines** indicate the normal limits of $F_1$ at the mean PaCO$_2$ of 33.3 mm Hg. DS = diffuse cerebral swelling; SH = systemic shock; PVS = persistent vegetative state; BD = brain death.

Among the 27 survivors, all but one patient (in PVS) returned to normal Grade 1 consciousness by the time of their final CBF study. Complete recovery was not achieved in all cases, however; nine patients continued to show focal neurological deficits and several had obvious cognitive impairment.

**TABLE 1**

*Description of sample of 36 patients with head injury*

<table>
<thead>
<tr>
<th>Initial Coma Level</th>
<th>Glasgow Coma Score (Range)</th>
<th>No. of Cases</th>
<th>Total</th>
<th>Elevated ICP</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 2</td>
<td>11–15</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Grade 3</td>
<td>6–11</td>
<td>15</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Grade 4</td>
<td>3–6</td>
<td>12</td>
<td>8</td>
<td>9*</td>
<td></td>
</tr>
</tbody>
</table>

*Plus one additional poor outcome in a patient in persistent vegetative state.

Fig. 2. Fast compartment blood flow ($F_1$) in ml/100 gm/min on the initial and final cerebral blood flow studies of 17 patients who recovered normal consciousness (Grade 1 on final study). Fifteen patients were in Grade 3 coma and two patients in Grade 4 coma during the initial study. **Dotted lines** indicate the normal limits of $F_1$ at the mean PaCO$_2$ of 33.3 mm Hg. DS = diffuse cerebral swelling; SH = systemic shock; HC = hydrocephalus.

**Relation of CBF to Outcome**

In order to relate blood flow changes to outcome, a comparison was made between each patient's initial and final CBF estimates, after first adjusting them for differences in PaCO$_2$ as described above. Although three-quarters of the patients showed significant hemispheral or regional differences in blood flow at some time during their illness, altered states of consciousness were invariably associated with generalized, bilateral decreases or increases in CBF. Blood flow estimates were therefore averaged across all 16 regions of both hemispheres for purposes of the present analysis.

Figures 1, 2, and 3 compare the initial and final CBF values of each patient. Since the results were essentially the same for $F_1$ and CBF$_{16}$, only the former variable is presented. Grade 4 patients who died or remained in a persistent vegetative state are plotted separately in Fig. 1. The survivors were subdivided according to coma grade at the time of their first CBF study, and are plotted in Figs. 2 and 3. In each graph...
the dotted lines represent the normal limits of F1 variation (mean ± 2 SD) for control subjects studied previously.27 These values were adjusted to the average PaCO2 (33.3 mm Hg) of the present sample, using the correction factors given above.

As shown in Fig. 1, F1 declined to very low levels in all nine patients who died, while remaining essentially unchanged in the patient with PVS. In two cases blood flows were recorded at the time of brain death (BD), manifested by a complete absence of neurological function and by electrocerebral silence. Both patients had reduced isotope uptake and prolonged monoexponential clearance, compatible with scalp circulation.

In contrast to the above findings, 25 of the 26 patients who recovered consciousness showed increases in F1 (Figs. 2 and 3). The one exception was the normalization of blood flow in a patient with very high initial values. Two patients, shown in Fig. 2 with initially depressed blood flows, failed to reach the normal range in their final study. Both revealed hydrocephalus (HC) on CT scan and showed clear-cut evidence of dementia.

Occurrence of Hyperemia During Coma

Cerebral blood flow varied considerably among individuals in their initial study. Whereas patients with mild disturbances of consciousness had slightly reduced flows distributed over a narrow range (Fig. 3), those in deeper coma presented a wide spectrum of F1 values, ranging from 20 to 100 ml/100 gm/min (Figs. 1 and 2). The latter occurred independently of clinical outcome. Of particular interest were patients who manifested either an absolute or relative "hyperemia," defined respectively as a supernormal or normal blood flow in the presence of coma. An attempt was made to characterize these patients on the basis of their clinical history and CT scans.

As indicated in Figs. 1 and 2, hyperemia occurred either in association with diffuse cerebral swelling (DS), or following recovery from profound systemic shock (SH). Independent evaluation of the CT scans revealed seven patients with diffuse cerebral swelling at the time of their first CBF study. The CT evidence for diffuse swelling consisted of an absence of CSF spaces, such that the perimesencephalic cisterns could not be visualized, and the lateral and third ventricles were either obliterated or markedly compressed. None of these patients had significant mass lesions that could account for the cisternal or ventricular compression. Shock was encountered in an additional two cases, one attributable to cardiac arrest and the other to hypovolemia following rupture of the spleen. In both patients cerebral hyperemia was detected within 24 hours of shock, after the blood pressure had returned to normal.

Taken together, diffuse swelling and shock accounted for all instances of normal or elevated blood flows in the initial study of patients with Grade 3 or 4 coma (Figs. 1 and 2). Only one deeply comatose patient with diffuse swelling had a subnormal flow, and even this was higher than found in other Grade 4 patients who did not have diffuse swelling or shock.

In order to assess the significance of these findings, the nine patients with diffuse swelling or shock were compared with all other patients in Grade 3 or 4 coma (Table 2). Although PaCO2 was comparable in the two groups, both F1 and CBF10 were almost twice as high in the patients with diffuse swelling or shock, there being only one case of overlap between the groups on each variable. Table 2 also reveals that a significantly greater proportion of patients with diffuse swelling or shock developed an elevated ICP (above 20 mm Hg) relative to the comparison group.

That age is a potentially important factor in the CBF results was suggested by the observation of a large age difference between patients with diffuse swelling and those without. Whereas six of the seven patients with diffuse swelling were 21 years or younger (mean = 19 ± 6 years), three-quarters of the remaining Grade 3 and 4 patients were older (mean = 37 ± 16 years). This difference was significant at the 0.01 level of confidence (Mann-Whitney U test).
Evidence of Luxury Perfusion

Because of earlier reports that cerebral metabolism is depressed in coma, the present finding of hyperemia in certain comatose patients raises a question concerning the relationship between CBF and metabolism in this condition. Specifically, is the elevated blood flow coupled with a high cerebral metabolic rate, or is there a dissociation between blood flow and metabolism, such that CBF is in excess of metabolic demand?

An attempt was made to answer this question by analysis of cerebral metabolic data available on a subsample of patients. Two groups of five cases each were compared: those in whom diffuse swelling or shock was present (four cases of diffuse swelling and one of shock), and those in whom it was absent. The groups were matched with respect to depth of coma, each having four patients in Grade 4 and one in Grade 3.

Arterial and jugular venous samples were drawn during the initial CBF study and analyzed for oxygen content. Cerebral metabolic rate for oxygen (CMRO₂) was estimated by multiplying the arteriovenous oxygen difference (AVDO₂) by CBF₁₅, averaged across 16 brain regions. Although it was recognized that CBF₁₅ is not strictly comparable to AVDO₂, being based on regional rather than global determinations, the mean value of CBF₁₅ was considered a reasonable approximation of global blood flow.

Table 3 compares the two groups of patients. As expected, F₁ and CBF₁₅ were significantly higher in patients with diffuse swelling or shock, there being a twofold difference and no overlap between groups. On the other hand, the two groups had essentially the same CMRO₂, which was well below the normal range of 2.5 to 4.1 ml/100 gm/min reported previously. Of particular interest was the low AVDO₂ obtained in the patients with diffuse swelling or shock. The mean value of 2.7 vol% was only half of that found in the comparison group. Since AVDO₂ represents the ratio of metabolism to blood flow, this low value indicates a high CBF relative to metabolic demand; that is, a "luxury perfusion."

Relation of CBF to Level of Consciousness

Due to the occurrence of both hyperemia and blood flow depression in comatose states, little or no relationship can be expected between CBF and level of consciousness. If, however, instances of luxury perfusion could be identified and excluded from analysis, a higher correlation might be obtained. Figure 4 presents the relationship between CBF and coma grade, after eliminating all blood flow measurements associated with either diffuse cerebral swelling or systemic shock. Nine additional instances of probable luxury perfusion were excluded; namely, transient regional hyperemia associated with seizures and/or
Cerebral blood flow in acute head injury

focal CT lesions. Also deleted were 13 measurements made during therapies that are known to affect cerebral hemodynamics, specifically those involving hyperventilation and barbiturate infusion. The data in Fig. 4 are based on the remaining 92 CBF studies in which luxury perfusion and the effects of therapy have presumably been eliminated.

Figure 4 indicates that both $F_1$ and $CBF_{15}$ increase monotonically as the level of consciousness improves. Because not all patients are represented at each coma grade, statistical evaluation was based on the direction rather than the magnitude of changes. While 25 of 27 improvements in coma grade were accompanied by increases in blood flow, only two were accompanied by decreases. Assuming that repeat CBF measurements have an equal probability of changing in either direction, this result is highly significant statistically ($p < 0.001$, binomial test).

Discussion

The use of a noninvasive method in the present study has made it possible to follow the course of CBF changes in head-injured patients from the acute phase of illness to eventual recovery or death. Unlike the intracarotid technique, intravenous injection delivers isotope to both hemispheres and to the posterior as

![Fig. 4. Cerebral blood flow (CBF) shown by $F_1$ and $CBF_{15}$ in ml/100 gm/min plotted against coma grade. Results are based on 92 CBF studies. The number of patients differs at each level of consciousness. $F_1$ and $CBF_{15}$ are defined in the text.](image)

![Fig. 5. Clearance curves recorded from the left (#13, continuous line) and right (#14, dotted line) temporal region of a patient 2 days before (upper curves) and following brain death (lower curves). Each point represents counts accumulated over a 6-second sampling interval. The same amount of isotope was injected on each occasion. Computed blood flows ($CBF_{15}$) were 36.1 and 6.1 ml/100 gm/min for the upper and lower curves, respectively. The latter is consistent with purely scalp circulation.](image)
well as anterior circulation. As reported elsewhere, this permitted detection of bilateral asymmetries in blood flow that were highly correlated with lateralized lesions on the CT scan.

Although 27 of the 36 patients revealed significant hemispherical or regional differences in CBF at some time during the course of their illness, such variations were almost always superimposed on more generalized decreases or increases in blood flow. Thus, the effects of injury were global, involving both sides of the brain, even when clinical and CT evidence suggested involvement of only one hemisphere. In this respect, the findings parallel those of acute stroke, where bilateral reductions in blood flow ("diaschisis") are a common occurrence. It is just these global CBF changes that appear to be related to outcome and level of consciousness.

Without exception, CBF declined to very low levels in the nine patients who died (Fig. 1). Full recovery of consciousness, on the other hand, was accompanied by variable increases in blood flow in 25 of 26 cases (Figs. 2 and 3). The single patient who remained in a persistent vegetative state (PVS, Fig. 1) continued to show a depressed CBF 4 months after injury. His F, value of 44 ml/100 gm/min is comparable to those reported by Ingvar and Ciria in states of prolonged coma.

Two patients revealed flat electroencephalograms and clinical evidence of brain death at the time of their final CBF measurements 5 and 7 hours before cessation of cardiac function (BD, Fig. 1). In both cases isotope uptake was one-eighth normal, and blood flow was less than 10 ml/100 gm/min, compatible with purely extracranial circulation. Figure 5 presents ~3Xe clearance curves from one of these patients. Curves recorded at brain death are compared with those obtained earlier when the brain was still functioning. Of particular interest is the fact that both patients had essentially zero flow in the presence of a normal arterial pressure, normal ICP, and adequate cerebral perfusion pressure. Similar findings have been obtained by Overgaard and Tweed in two cases of "impending" brain death. We have recently observed this phenomenon in a third patient, and plan to submit a full clinicopathological report on it.

In spite of persistent neurological deficits in nine of the patients who recovered, 24 of 26 final CBF measurements were within or slightly above the normal range. The two exceptions were patients with clear-cut hydrocephalus on CT scan (HC, Fig. 2), both of whom became demented, one with a disturbance of gait. Their reduced F, values (43 and 47 ml/100 gm/min) are comparable to those reported in normal-pressure hydrocephalus.

Perhaps the most important finding is the heterogeneity of CBF on the initial examination of Grade 3 and 4 patients, such that some flows were as much as three or four times greater than others (Figs. 1 and 2). This contrasts with the relative homogeneity of values among patients with milder disturbances of consciousness (Fig. 3). The presence of very high as well as very low flows after acute head injury has been observed by a number of investigators. Unlike CMRO, which is uniformly low in comatose states and is a reasonably good predictor of outcome, these large variations in blood flow preclude its use as a prognostic indicator.

The variability of initial CBF measurements may, nevertheless, have important implications for the pathophysiology of head injury. It is a reasonable assumption that patients who present the same clinical picture but have widely divergent blood flows are somehow different with respect to the mechanisms underlying their illness. Indeed, several investigators have emphasized these differences. In particular, they have noted that many acutely ill patients pass through a phase of hyperemia which, if it persists, may lead to clinical deterioration.

In the present study, early hyperemia was found in association with two conditions: 1) diffuse cerebral swelling as revealed by CT scan, and 2) recovery from profound systemic shock. The latter observation is consistent with the experimental work of Freeman and Ingvar, who found hyperperfusion that persisted for several hours after acute reductions in blood pressure. The tendency for patients with hyperemia to develop an elevated ICP (Table 2) is also consistent with previous observations. The finding that diffuse cerebral swelling occurred primarily in younger patients agrees with the larger series of CT scans described by Zimmerman and coworkers. They observed such swelling in 45% of Grade 3 and 4 patients under the age of 18 years, which is more than three times the incidence in adults. The association of hyperemia with diffuse swelling suggests that increased cerebral blood volume may contribute to compression of the CSF spaces, a speculation supported by measurements of higher density on the CT scans.

There is some suggestion that the occurrence of hyperemia in head injury is also a function of age. Increased blood flow was found on the initial examination of all four patients under the age of 18 years in Grade 3 or 4 coma. A predilection for younger individuals to develop hyperemia may account for the prevalence of this finding by Overgaard and Tweed, whose sample contained a large proportion of children and adolescents.

The initial appearance and time course of hyperemic responses to head injury have not been clearly delineated. Whereas Enevoldsen and coworkers stress the appearance of hyperemia within 24 hours of injury, Fieschi, et al., and Overgaard and Tweed emphasize its development over several days following an initial depression of blood flow. In the present study, a generalized elevation of CBF was found on their first examination in eight patients with diffuse swelling or shock, six within 24 hours of injury. The only other early onset was a focal hyperemia
following surgical removal of an epidural hematoma. An additional seven patients, however, developed hyperemia at later stages of their illness, five of them unilaterally. These occurred in association with persistent or evolving mass lesions on CT scan (three cases), in close temporal proximity to focal seizures (two cases), and at the time of deteriorating neurological status (two cases). A correlation between seizures and hyperperfusion has been reported previously.8

Since most hyperemias were transient, lasting 1 to 4 days, it seems probable that a number of them were missed, given the infrequent and irregular sequence of CBF measurements in the present study. A fuller understanding of the natural history of hyperemia would require early, regular, and more frequent blood flow examinations, which are now feasible with the non-invasive technique.

One limitation of the intravenous 133Xe method is its inability to visualize "tissue peaks," that is, extremely rapid components in the clearance curve that appear following an intracarotid injection. Fast "third" compartments have frequently been seen in the clearance curves of head-injured patients, particularly children.16 In the present two-compartment analysis, such fast components are usually averaged with slower clearance rates, so that their detection depends upon the presence of a sufficient volume of hyperperfused tissue. This should not present a problem, however, since clinically significant hyperemia can be expected to occupy a relatively large tissue volume.

While pointing out the sensitivity of bicompartmen
tal analysis to hyperemia, Enevoldsen and Jensen4 have stressed the difficulty of interpreting blood flow findings due to changes in compartment size. We have approached this problem by making allowance for shifts in the relative weight of the fast compartment, and by interpreting F1 values in terms of the more stable non-compartmental index, CBF.15 Although a three-compartment model may be theoretically more appropriate in some cases, the addition of two unknowns to the analysis makes it inherently less stable. This is not true in the case of very low blood flows, where a one-compartment analysis is often appropriate and easily executed.

The extremely low CMRO2 in deep coma (Table 3) was not surprising in view of similar values obtained by others11,24 in head injuries of comparable severity. The relatively high CBF and narrow AVDO2 in patients with diffuse swelling or shock confirmed the existence of luxury perfusion.18 Since an elevated blood flow is not needed to maintain metabolism in such cases, therapeutic reduction of CBF by hyperventilation becomes feasible. Indeed, several authors4,7,23 have argued that hyperventilation is beneficial in patients with hyperemia, not only because it lowers ICP, but because it may decrease capillary hydrostatic pressure, thereby impeding edema formation. Additional benefit might be derived from reduction of tissue acidosis and restoration of CBF auto-regulation.26 As reported elsewhere,9 we have studied hyperventilation in five hyperemic patients (four with diffuse swelling and one with shock) and, without exception, found it to be effective in reducing CBF. Intracranial pressure decreased significantly in three of the cases.

The correlation between CBF and coma grade (Fig. 4) depended upon excluding all instances of luxury perfusion, thus confining the relationship to blood flows that were presumably coupled with metabolic rate. Of particular interest is the finding that CBF was depressed even in mild disturbances of consciousness (Grade 2) by as much as 20%, indicating the sensitivity of blood flow to relatively minor changes in functional activity of the brain.

Although prediction of outcome by the Glasgow Coma Scale was excellent and in agreement with the findings of Teasdale and Jennett,30 the simple four-point grading system employed in the present study was equally effective (Table 1). This conforms with the experience of Overgaard, et al.,22 and Tindall and Fleischer,21 who used a similar classification based on responses to painful stimuli and verbal commands. The correlation of CBF with coma grade, particularly at higher levels of consciousness, lends further support to the validity of this grading system.

Acknowledgments

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References


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