Cerebral Blood Flow in Patients with Intracranial Pressure Elevation due to Traumatic Brain Edema

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SUMMARY: The object of this study was to determine if traumatic brain edema (BE) and increased intracranial pressure (ICP) reduce cerebral blood flow (CBF). Two groups of patients were studied, one with slight BE and ICP less than 20 mm Hg, the other with pronounced BE and ICP over 20 mm Hg. Although ICP was higher and cerebral perfusion pressure lower in pronounced edema there was only a small and non-significant reduction in CBF and no difference in cerebro-vascular resistance. Since traumatic BE does not increase resistance to blood flow through the brain, cerebral perfusion can be maintained if an adequate perfusion pressure is established. This in turn, demands the monitoring and control of ICP.

RESUME: Cette etude avait pour but de determiner si l'oedeme cerebral post-traumatique et l'elevation de la pression intra-cranienne reduisent le debit sanguin cerebral. Pour ce faire, nous avons etudie 2 groupes de sujets, l'un oedeme cerebral modere et une pression intra-cranienne inferieure a 20 mm Hg; et l'autre, avec un oedeme cerebral important et une pression intra-cranienne superieure a 20 mm Hg. Quoique la pression intra-cranienne etait plus eleve et la pression de perfusion diminuee dans le groupe avec oedeme cérébral prononcé, nous n'avons note qu'une diminution minime et non significative du débit sanguin cérébral. De plus, il n'y avait aucune difference au niveau des resistances cérébro-vasculaires. Puisque l'oedème cerebral post-traumatique n'augmentent pas les resistances vasculaires cérébrales une pression de perfusion adequate assurerait une perfusion cérébrale. Mais ceci impliqueun monitoring et un controle de la pression intra-cranienne.

Although traumatic brain edema (BE) and increased intra-cranial pressure (ICP) are common complications of head injury, there are few studies of cerebral hemodynamics in patients with these complications. This is a report of clinical investigations in patients with ICP elevation and BE following severe closed head injuries. Twenty-six patients were studied within 14 days after injury, with measurements of cerebral blood flow (CBF), mean arterial blood pressure (MABP), mean intraventricular pressure (MIVP), and arterial blood gases.

METHODS

CBF was measured by the intracarotid xenon injection method, recording the washout of xenon from the brain with 35 externally located scintillation detectors (Meditronic Cerebrograph), arranged in an array over the ipsilateral cranium. The average hemispheric CBF was calculated by the initial slope method (Olesen et al., 1971) from the single washout curve that was obtained by continuously recording the average counting rate of the 35 separate channels. CBF derived by the initial slope method is designated CBF init. to distinguish it from other values for CBF which can be obtained by applying different analyses to the same washout curve. CBF init. is higher than the mean CBF calculated by the height over area method, and is closer to, but not identical with, grey matter flow in a normal brain. The advantage of using CBF init. in clinical studies is that it requires only two minutes of steady state recording while other methods require 10 minutes or more of recording time.

During each CBF measurement MABP was continuously recorded.
from the carotid artery catheter, and
MIVP from a catheter inserted
through a burrhole into a lateral
cerebral ventricle. Cerebral perfu­
sion pressure (CPP) was calculated
as the difference between MABP
and MIVP and cerebrovascular re­
sistance (CVR) across the brain as
CPP/CBF init.

An anaerobic sample of arterial
blood was taken after each xenon in­
jection and analyzed in a radiometer
gas analyzer for blood gases and
acid-base.

The difficulty of estimating BE
clinically is well known. We asses­
sed BE at each CBF examination by
the following criteria:
1. angiographic and ventriculog­
ographic displacements not due to
hematomas.
2. direct observation of brain swel­
ling at operation when possible.
3. persistent elevation of MIVP over
20 mm Hg. during normocapnia,
and after hematomas had been
evacuated.

Although the first two criteria may
give a qualitative estimate of BE, it
was only possible to rank edema
quantitatively by its effects on
MIVP. Since all hematomas were
evacuated before MIVP monitoring
began, persistent elevation of MIVP
in the acute phase after injury was
considered indicative of BE. (Later
elevation of MIVP may be due to
post-traumatic hydrocephalus).
Since we could not be certain that
any patient had no BE we have clas­
sified them as slight edema (MIVP
less than 20 mm. Hg. with the first
and second criteria negative), or
pronounced edema (MIVP over 20
mm. Hg. plus criteria 1 or 2). CBF
was always measured in the hemis­
phere with the most evident edema.

RESULTS
The results in patients with slight
and pronounced BE are illustrated in
figures 1/2. There was no difference
in neurological status between the
groups, and only a small differ­
ce in PCO₂, 38.9 ± 6.1 in the slight
edema group versus 36.6 ± 8.1 in the
group with pronounced edema. The
significance of the differences be­
tween the two groups was tested by
the unpaired t-test.

MIVP was significantly higher and
as a consequence CPP was lower in
the group with pronounced edema.
However, there was only a small and
non-significant reduction in CBF init.
which was due to the reduction in
perfusion pressure since calcul­
ed CVR was not significantly dif­
ferent (actually slightly less) in pro­
nounced edema. Calculated CVR
did not show any consistent rela­
tionship to the severity of edema or
the level of MIVP; that is, CVR was
not higher in those with more pro­
nounced edema.

DISCUSSION
CBF init., calculated from the ini­
tial slope of the xenon washout
curve, is a measure of perfusion in
the high flow tissues of the brain. In
the normal brain, CBF init. corre­
lates most closely with flow-grey
(Olesen et al., 1971), and we con­
sider that in clinical studies it primar­
ily indicates cortical flow. It is
linearly related to, but higher than,
the mean flow calculated by the ten
minute height over area method
(Overgaard and Tweed, 1974).
MIVP has been shown by others to
approximate very closely subarach­
noid venous pressure (Rowan et al.,
1972). Therefore CVR as we calculate it (CVR = CPP/CBF init.) represents the resistance to flow from the internal carotid artery through the cerebral cortices to the subarachnoid veins.

Studies by Bruce et al. (1973) suggest that resistance to blood flow is increased through edematous brain tissue. Our studies do not support, but do not entirely disprove, their conclusions. Although we have found no evidence of an increased resistance to flow in the presence of brain edema, it may be that we are not measuring flow through the actual edematous tissue. The initial slope method measures primarily cortical flow and edema is found primarily in white matter (Feigin and Popoff, 1962). We may therefore be measuring flow in cortical tissue that is not itself edematous but is affected by vasodilator metabolites, for example, lactic acid, diffusing from adjacent edematous white matter. We might, on the other hand, postulate that the resistance to flow at the microcirculatory level is increased in edematous brain tissue, but that this increase in resistance is more than offset by a reduction of resistance due to vasodilation in more proximal components of the vascular system, that is the resistance arteries. This would occur if the autoregulation of CBF was wholly or partially intact.

In this clinical study of BE we have not observed a significant increase in CVR across the brain with increasing severity of BE. This suggests that severe traumatic BE can be likened to an expanding intracranial mass lesion that increases the volume of the intracranial contents and elevates ICP. Since subarachnoid venous pressure closely follows ICP, resistance to flow will increase at the level of the subarachnoid veins and CPP will be reduced. Compressive or obstructive effects on the tissue microcirculation appear to be of minor importance. This discussion applies only to traumatic BE and in other cases, for example focal cortical lesions or peri-focal tumor edema, the pathophysiology may be different.

The logical conclusion from these studies is that cerebral perfusion can be maintained in the post traumatic state, even in the presence of BE, if an adequate perfusion pressure (CPP) can be established. This in turn requires continuous monitoring of ICP, and vigorous treatment of elevated ICP. On the other hand, attempts to maintain CPP by artificial elevation of systemic blood pressure are likely to prove disastrous, leading to a vicious circle of increasing edema and rising ICP. There is good experimental evidence that systemic hypertension promotes the formation and spread of BE in the injured brain (Klatzo, 1972; Marshal et al., 1969; Meiring et al., 1972). We have also shown in clinical studies that when ICP is elevated and the autoregulatory control of CBF is lost, systemic hypertension provokes an immediate further rise in ICP, due to both passive vasocongestion and increased edema formation (Tweed and Overgaard, 1975). This evidence suggests also that the episodic spontaneous hypertension, commonly observed in brain injured patients, is deleterious. In order to break this vicious cycle we propose that it is necessary not only to prevent episodic hypertension (with phenobarbital), but also to control ICP to less than 20 mm Hg. At this level passive changes of ICP with changes in blood pressure are not observed (Tweed and Overgaard, 1975), and CBF is adequate to satisfy the nutritional requirements of the brain.

REFERENCES


