INTRACRANIAL PRESSURE AND CEREBRAL BLOOD FLOW

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INTRODUCTION

The physiological changes that maintain cerebral blood flow (CBF) and accommodate alterations in brain volume are relatively simple to understand. Following trauma or in the presence of major intracranial disease additional changes occur. Major advances in the care of patients with major neurosurgical problems have been developed over the last 10 years. These advances have evolved from a sound understanding of basic physiological rules and the pathological process of different disease situations as well as the pharmacology of anaesthetic drugs. Successful management of these patients relies on a clear understanding of these physiological mechanisms and of the added effect of anaesthesia and the manipulation of arterial pressure, CO_2 and O_2 tensions. Poor anaesthetic technique which allows coughing, straining, hypotension, exaggerated hypertension, hypoxia and hypercarbia will seriously damage the brain. Better results can be obtained by careful monitoring of the patient and attention to simple details than by complex pharmacological interventions. It is the purpose of this article to explain these factors and how an understanding of them can be applied to patients following head trauma or intracranial disease.

The brain is only able to withstand very short periods of ischaemia, unlike the kidney, liver or muscle. Thus cerebral blood flow must be maintained to ensure a constant delivery of oxygen and glucose as well as the removal of "waste" products. Maintenance of cerebral blood flow depends on a balance between the pressure within the skull, intracranial pressure (ICP) and the arterial pressure of the blood, mean arterial pressure (MAP). It is important to maintain a constant blood flow. Thus when blood pressure falls, physiological mechanisms attempt to maintain flow to prevent ischaemia. This process is autoregulation and is explained in detail later. Similarly, when blood pressure rises, the same mechanism stops the blood flow from increasing to excessive levels. If this did occur, cerebral oedema could develop and the brain would enlarge because of the increase in cerebral arterial blood volume.

A number of terms will be used in this article and are defined:-

ICP intracranial pressure is the pressure within the rigid skull.

CBF cerebral blood flow is the flow of blood through the brain, important for delivery of oxygen and removal of "waste" products

CPP cerebral perfusion pressure is the effective pressure driving blood through the brain. It is discussed in detail later

INTRACRANIAL PRESSURE

> *Teaching point*
> High intracranial pressure (ICP) will cause internal or external herniation of the brain, distortion and pressure on cranial nerves and vital neurological centres. Cerebral perfusion will be impeded and operating conditions difficult or impossible. Loss of CSF and reduction of venous blood volume act to compensate for increases in brain volume. Once these mechanisms are exhausted, any further increase, however small, will cause a large increase ICP.

The principle constituents within the skull are brain (80%), blood (12%) and CSF (8%). The total volume is 1600ml. The skull is thus a rigid fluid filled box. If the volume of the contents of a rigid fluid-filled container increase, the pressure inside will rise considerably unless some fluid is able to escape. So it is with the skull and brain within it. If the brain enlarges, some blood or CSF must escape to avoid a rise in pressure. If this should fail, or be unable to occur there will be a rapid increase in ICP from the normal range (5-13 mmHg). If there is an increase in the volume of either the brain or blood the normal initial response is a reduction in CSF volume within the skull. CSF is forced out into the spinal sac. Thus the pressure within the skull, ICP, is initially maintained. If the pathological process progresses with further increase in volume, venous blood and more CSF is forced out of the skull. Ultimately this process becomes exhausted, when the venous sinuses are flattened and there is little or no CSF remaining in the head. Any further increase in brain volume then causes a rapid increase in ICP. This chain of events is represented by the sequence in

Fig 1a and 1b.

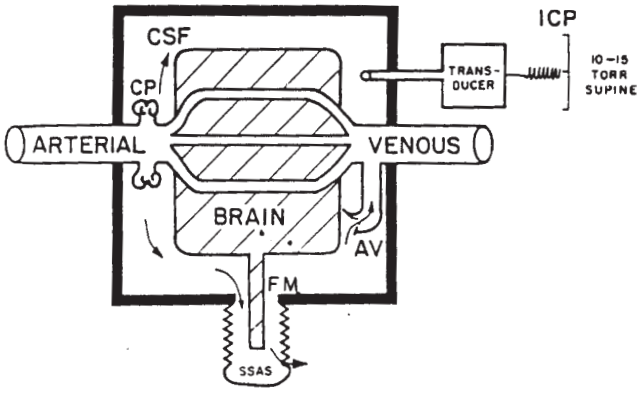

Fig. 1a. Schematic representation of normal intracranial contents SSAS= spinal subarachnoid space, FM = foramen magnum, AV = arachnoid villi, CP = choroid plexus. Arrows indicate direction of CSF flow, heavy lines the skull

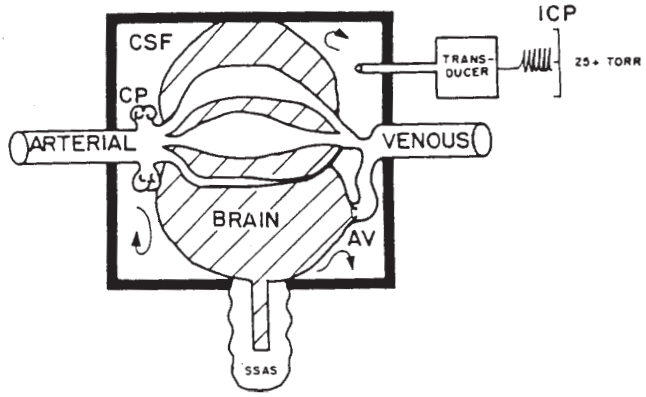

Fig. 1b. Schematic representation of contents of skull during raised intracranial pressure

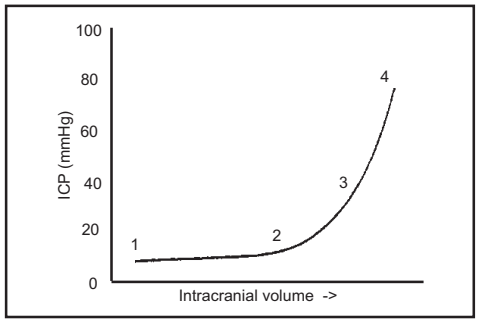

Fig. 2. Brain volume-intracranial pressure relationships: 1-2 compensation phase; 3-4 decompensation phase

The pressure changes within the skull are drawn in the classical curve Fig. 2 which indicates an increase in volume with little change in pressure until a certain point is reached when a further small change in volume results in a large increase in pressure: 1-2 compensation phase; 3-4 decompensation phase.

It is interesting to note that this classic curve represents the alterations in pressure when the volume of a single compartment within the skull, in this case CSF, changes. Therefore it is a CSF-pressure volume curve. In practice when the enlargement of the brain is due to a tumour or haematoma the curve is less steep. Pressure gradients develop within the brain substance and the compliance or "squashiness" of the tumour is different from that of brain leading to this altered curve.

Cerebral swelling leads to herniation of the brain either internally, when the temporal lobe is pushed down onto the mid-brain through the tentorium incisura or externally, with the cerebellar peduncles being forced down through the foramen magnum. This causes torsion of the brain stem and a reduction of local cerebral blood flow as the unrelenting rise in ICP opposes arterial pressure. Ultimately cerebral perfusion pressure falls to a point when there is no cerebral blood flow, no cerebral perfusion and death. The rise in ICP may be accelerated because of acute hydrocephalus. This is caused by brain-stem torsion leading to sudden obstruction of CSF flow.

The volume of blood contained within the venous sinuses is reduced to a minimum as part of the compensatory process. However, should free flow of venous blood be impeded by a number of simple causes (Table 1) then this increase in volume of the venous system in a critically swollen brain will lead to a rapid rise in ICP. In practice, it is imperative to ensure that when the patient is in the supine or lateral position that a head up tilt to a maximum of 30^{o} is obtained. This improves venous drainage with minimal effect on arterial pressure [1].

Table 1. Non-Pathological causes of raised ICP

Increased Venous Volume	
	Coughing
	Obstructed airway
	Head-down position
	Obstructed neck veins
Cerebral oedema	
AVOID	Hypotonic IV solutions
	5% Dextrose, Dextrose-Saline, Hartmann's Solution
USE	0.9% Normal Saline
Increased CBF	
	Anaesthetic drugs - *see next article*

Venous drainage is passive and thus maximised by ensuring there is no pressure on, or kinking, of the neck veins. In addition the higher the head, the greater the effect of gravity on the flow of venous blood. However, as the head is raised, the gravitational effect on the arterial pressure at the brain is also increased. This is a disadvantage as it reduces the pressure of blood perfusing the brain. The best compromise is the position described above of 30°.

Teaching point
If the patient is lying in the supine position, and it is necessary to turn the head laterally, a sand bag should be placed under the shoulder to reduce the pressure of the sternomastoid on the jugular vein. When patients with severe head injuries are nursed or transported it must be with a 30° head up tilt, and the blood pressure maintained.

The extent of the change in ICP caused by an alteration in the volume of intracranial contents is determined by the compliance or "squashiness" of the brain. In other words if compliance is low, the brain is stiffer or less "squashable". Therefore, an increase in brain volume will result in a higher rise in intracranial pressure than if the compliance were high. Compliance affects the elastance or "stretchiness" of the walls of the ventricles. When the elastance is reduced the walls are stiffer. Therefore there is a greater change in pressure for a given alteration in brain volume. If a catheter is inserted into a lateral ventricle via a burr hole, this can be assessed by injecting 1ml of saline and observing the change in intracranial pressure. After the injection, if the rise in pressure is more than 5 mmHg then the patient has become has become decompensated and is at the right hand end of the pressure-volume curve (Fig 2).

CEREBRAL PERFUSION PRESSURE

Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial and intracranial pressures. Mean arterial pressure is the diastolic pressure plus one third of the pulse pressure (difference between the systolic and diastolic). MAP is thus between systolic and diastolic pressures, nearer diastolic. It is used as it is the best value to estimate the "head of pressure" perfusing in the brain:

CPP = MAP - ICP

Normal cerebral perfusion pressure is 80 mmHg, but when reduced to less than 50 mmHg there is metabolic evidence of ischaemia and reduced electrical activity. There have been a number of studies on patients with severe head injuries which have shown an increase in mortality and poor outcome when CPP falls to less than 70 mmHg for a sustained period [2,3]. Continuous monitoring of jugular venous bulb saturation is another tool used to monitor the adequacy of the cerebral circulation when it is at risk. Jugular venous bulb saturation is the oxygen saturation of venous blood in the jugular bulb which is at the base of the skull. The normal range is 65%-75%. If blood flow to the brain is reduced below a critical point there is a fall in venous saturation. As the flow of blood and delivery of oxygen is reduced, the brain, in order to maintain its oxygen supply, extracts more oxygen from the blood, leading to a fall in venous oxygen saturation.

Teaching point
Cerebral perfusion pressure (CPP) = MAP - ICP

Inadequate CPP (less than 70 mmHg) has been shown to be a major factor in the poor outcome of patients with raised ICP. Assessment of CPP is vital and possible either by measurement of both ICP and MAP (mean arterial pressure - see text) or by measuring MAP and making a reasonable estimate of ICP. During anaesthesia therefore, if ICP is raised a fall in blood pressure must be avoided or treated quickly by volume replacement or catecholamines whichever is relevant.

More specifically, when CPP is inadequate the oxygen saturation of jugular venous blood falls (normal range 65%-75%) because of increased oxygen extraction. Does the jugular venous bulb measurement give an indication of the minimum level for CPP? Chan [4] in another study of head-injured patients showed that when CPP was below 70 mmHg, there was a rapid decrease in jugular venous bulb saturation. It was concluded that when CPP was less than 70 mmHg cerebral perfusion was insufficient.

In the head injured patient, CPP should not fall below 70 mmHg.

Therefore continuous consideration of changes in CPP are vital when anaesthetising patients who may have raised ICP and a fall in arterial pressure occurs as a result of anaesthetic agents or blood loss. Ideally ICP should be monitored, but often this is impossible or impractical. However a reasonable estimate can be made in head injured patients who are not sedated: (Drowsy and confused (GCS 13-15)ICP=20 mmHg, Severe brain swelling (GCS <8) ICP=30 mmHg).

Teaching point
The following example illustrates the point. A 28-year old patient who has had a recent head injury where he was unconscious briefly, requires urgent abdominal surgery. He is confused, restless and drowsy. It would be reasonable to estimate his ICP to be 20 mmHg. Following induction of anaesthesia his systolic arterial pressure (SAP) falls to 80 mmHg. In this situation MAP will have fallen to 65 mmHg and therefore CPP will have fallen to less than 45 mmHg, significantly below the critical value of 70 mmHg with a significant risk of causing cerebral ischaemia and a poor cerebral outcome.

CEREBRAL BLOOD FLOW

The normal cerebral blood flow is 45-50ml $100g^{-1}$ min^{-1}, ranging from 20ml $100g^{-1}$ min^{-1} in white matter to 70ml $100g^{-1}$ min^{-1} in grey matter. There are two essential facts to understand about cerebral blood flow. Firstly, in normal circumstances when the flow falls to less than 18-20ml $100g^{-1}$ min^{-1}, physiological electrical function of the cell begins to fail. Secondly, an increase or decrease in CBF will cause an increase or decrease in cerebral arterial blood volume because of arterial dilatation or constriction. Thus in a brain which is decompensated as a result of major intracranial pathology, increases or decreases in CBF will in turn lead to a significant rise or fall in ICP. The physiological factors which can alter CBF and hence ICP are listed in Table 2. There are also a number of drugs which can induce arterial dilatation, the most well known being high concentrations of volatile agents. These will be discussed in detail in a subsequent article.

Autoregulation
CBF is maintained at a constant level in normal brain in the face of the usual fluctuations in blood pressure by the process of autoregulation. It is a poorly understood local vascular mechanism. Normally autoregulation maintains a constant blood flow between MAP 50 mmHg and 150 mmHg. However in traumatised or ischaemic brain, or following vasodilator agents (volatile agents and sodium nitroprusside) CBF may become blood pressure dependent. Thus as arterial pressure rises so CBF will rise causing an increase in cerebral volume. Similarly as pressure falls so CBF will also fall, reducing ICP, but also inducing an uncontrolled reduction in CBF.

Teaching point
There are a number of physiological factors which affect or change cerebral blood flow (CBF). Rises in CBF due to hypoxia, hypercapnia (raised blood CO2) and high concentrations of volatile agents will cause a rise in ICP once the normal compensating mechanisms have been exhausted. Poor anaesthetic technique during which hypoxia, hypercapnia and hypotension occur will seriously damage the critically ill brain further.

Table 2. Physiological causes of raised ICP

Hypoxia
Hypercapnia
Pain
Low Cerebral Perfusion Pressure
Exaggerated Hypertension

More recent work has shown that following trauma autoregulation may still be functioning. Bouma reported that it was present in up to 69% patients with head injuries[5]. In this situation if CPP falls below the critical value of 70 mmHg, the patient will have inadequate cerebral perfusion. Autoregulation will cause cerebral vasodilatation leading to a rise in brain volume. This in turn will lead to a further rise in ICP and induce the vicious circle described by the vasodilatation cascade (fig 3a) which results in cerebral ischaemia. This process can only be broken by increasing the blood pressure to raise CPP, inducing the vasoconstriction cascade (fig 3b). This explains why the maintenance of arterial blood pressure at adequate level by careful

monitoring and rapid correction if it falls is so important.

Carbon dioxide causes cerebral vasodilation. As the arterial tension of CO_2 (fig 4) rises, CBF increases and when it is reduced vasoconstriction is induced. Thus hyperventilation can lead to a mean reduction in intracranial pressure of about 50% within 2-30 minutes [6]. When $PaCO_2$ is less than 25 mmHg (3.3kPa) there is no further reduction in CBF. Therefore there is no advantage in inducing further hypocapnia as this will only shift the oxygen dissociation curve further to the left, making oxygen less available to the tissues. Acute hypocapnic vasoconstriction will only last for a relatively short time (5 hours). While hypocapnia is maintained, there is a gradual increase in CBF towards control values leading which will lead to cerebral hyperaemia (over-perfusion) if the $PaCO_2$ is returned rapidly to normal levels [7]. When long term ventilation is required, only mild hypocapnia (34-38 mmHg: 4.5-5.1 kPa) should be induced. Worse outcome was reported in patients after head injuries at 3 and 6 months in those who had been hyperventilated to low $PaCO_2$ levels for long periods [8].

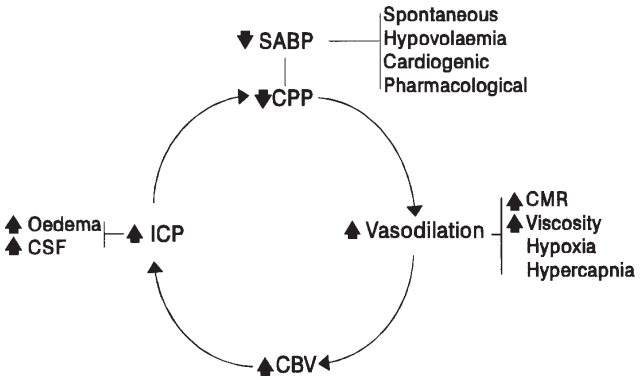

Fig 3a Vasodilatory cascade

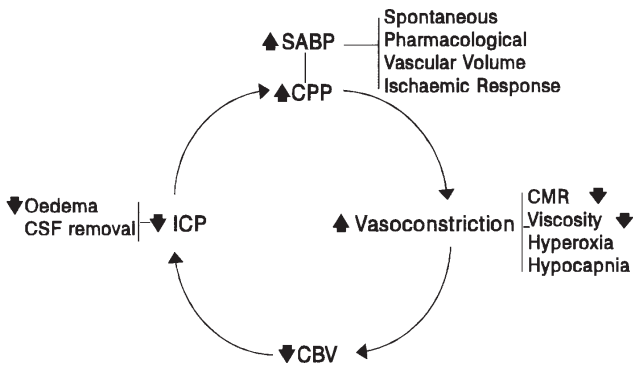

Fig 3b Vasoconstriction cascade

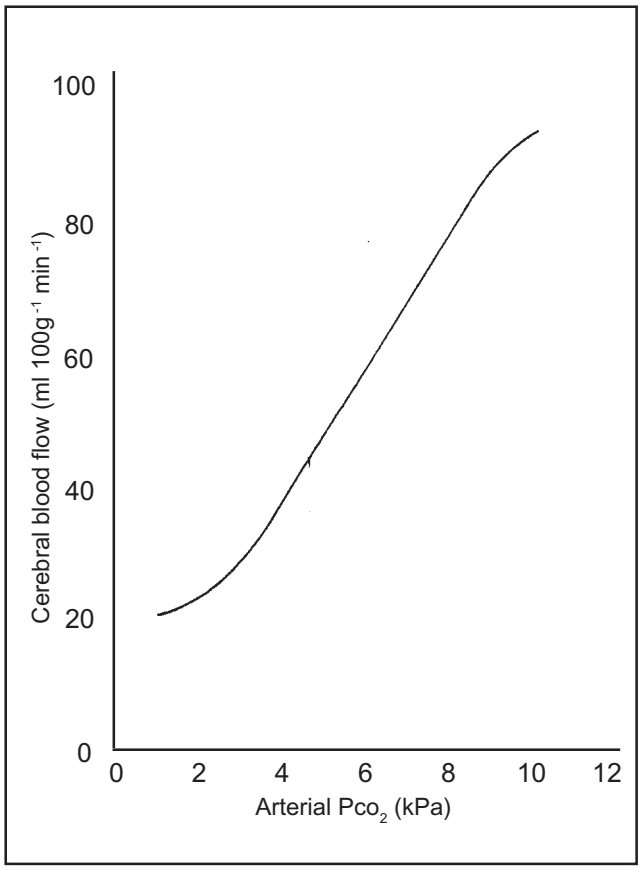

Fig 4 Schematic representation between cerebral blood flow and arterial carbon dioxide tension

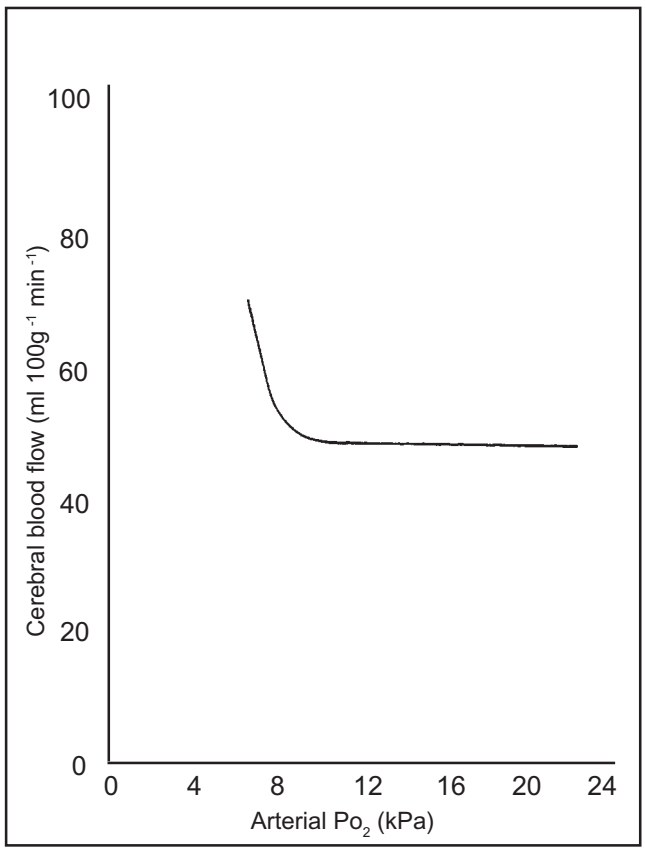

Fig 5 Schematic representation between cerebral blood flow and arterial oxygen tension

Teaching point
When there is an acute rise in ICP, for example after an acute head injury ICP can be reduced by hyperventilation to lower arterial CO_2 tension. This technique is used during neurosurgery to reduce brain size to improve access for the surgeon. In **CONTRAST** only mild hyperventilation should be used for long term ventilation of patients as described above.

Oxygen. Low arterial oxygen tension also has profound effects on cerebral blood flow (Fig 5). When it falls below 50 mmHg (6.7 kPa), there is a rapid increase in CBF and arterial blood volume.

APPLIED PHYSIOLOGY - HEAD INJURY

Following acute head injury, the term "secondary injury" has been described. The primary injury is due to the actual trauma. The secondary brain injury is caused by ischaemia due to the combination of rapid brain swelling and hypotension. Any patient who is unconscious can easily develop an obstructed airway and become hypoxic, hypercapnic, possibly hypotensive and have a rise in intrathoracic pressure. Considering the physiological changes described already, the process that results in cerebral swelling and raised ICP is clear. In addition, pain from other injuries, despite the patient being unconscious, will cause an increase in CBF as a result of both the hypertensive response and local dilatation in the relevant sensory area of the brain. Thus in the initial management of the acutely head injured patient who is unable to maintain his airway, intubation and hyperventilation should be instituted following an intravenous anaesthetic agent, and an opiate, to attenuate the response to intubation. The dose must be carefully chosen to avoid hypotension in a patient who may also be hypovolaemic.

Teaching point
In an article on the management and resuscitation of patients with serious head injuries Gentleman et al [9] noted that over an 11 year period there was significant reduction in mortality, (45% to 32%) and an increase in patients making a good recovery, (42%-58%) associated with a reduction in hypoxaemia and hypotension during treatment.

Hypotension must be treated aggressively with a rapid infusion of colloid or blood and if, necessary, intravenous (ephedrine 3-6 mg, methoxamine 1-3 mg).

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