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PHYSIOLOGICAL NOTE

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# Intracranial pressure

## Part one: Historical overview and basic concepts

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## Introduction

One hundred and seventy years ago, Magendie (1783–1855) discovered a small foramen in the floor of the fourth ventricle, now bearing his name, and pointed out the connection between the cerebrospinal fluid (CSF) in the ventricular system and in the subarachnoid spaces of the brain and cord. By this momentous discovery, he led the way to understanding the circulation of CSF and to problems associated with increased CSF pressure.

## Lumbar cerebral spinal fluid pressure measurement

Physiological exploration of human CSF started in the late 18$^{th}$ century. In 1891, Quinke published his studies on the diagnostic and therapeutic applications of lumbar puncture. He standardised the technique and made it a rule always to measure the pressure of the CSF by connecting the lumbar puncture needle with a fine glass pipette in which the fluid was allowed to rise.

Subsequently, repeated measurement of lumbar cerebral spinal fluid pressure, as an assessment of intracranial pressure (ICP), was widely used (Ayer 1929, Merrit and Fremont-Smith 1937, Browder and Meyer 1938, Cairns 1939, Landon 1917, Sharpe 1920 and Jackson 1922) and this was the earliest clinical method of ICP measurement.

Jackson pointed out the neglect by surgeons of the field of acute traumatic brain injuries. He demonstrated that the pulse, respiration and blood pressure are affected only once the medulla is compressed and stated that to wait for these changes as an indication for operation on the cerebrum in acute cerebral injury is to court disaster.

Furthermore, reports emerged that some patients, even if showing clinical signs of brain compression, had normal lumbar CSF pressures or died after the procedure. Lumbar puncture fell into disuse for the diagnosis of intracranial hypertension due to the possibility of inducing brain-stem compression through tentorial or tonsillar herniation, and because, if the system does not communicate, the spinal fluid pressure is not an accurate reflection of ICP as demonstrated by Langfitt's work [1].

## Lundberg: a clinical pioneer

Researchers moved from the lumbar approach to direct cannulation of the ventricular system. Early clinical research in this field was reported by Nils Lundberg and involved conscious volunteers with a multiplicity of intracranial pathologies [2]. They were monitored by a fluid-filled transducer system attached to a ventricular catheter placed in the lateral ventricle. Recordings lasted, in some cases, several hours or days. Lundberg, enlighten by his clinical talent, reported a number of phenomena that are relevant today. However, a recording system connected to an analogue output from the ICP transducer is required for detection and this is frequently overlooked in modern ICU monitoring systems. A digital trend does not usually have sufficient resolution to detect ICP waves with a frequency of less than 2/min. The clinical importance of ventricular fluid pressure (VFP) waveform was

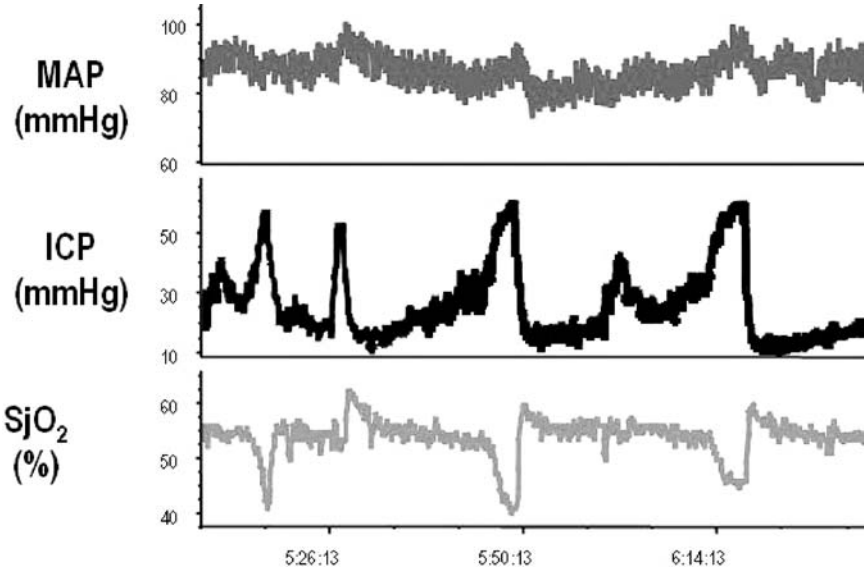


**Fig. 1** Example of plateau waves recorded at bedside. The plateau waves are a haemodynamic phenomenon associated with cerebrovascular vasodilation. They are observed in patients with preserved cerebral autoregulation but reduced pressure-volume compensatory reserve. As documented by the tracing, during plateau waves, cerebral perfusion pressure falls below the ischaemic threshold, shown by jugular saturation oximetry. *MAP* mean arterial pressure, *ICP* intracranial pressure, $SjO_2$ continuous jugular saturation

elucidated in 48 patients and it was concluded that the spontaneous changes in VFP curve were of two main types, plateau waves and rhythmic oscillations[3]. Lundberg stated that the former could cause both transient and persistent damage to the brain and therefore diagnosis, utilising a ventricular catheter, and prevention of such pressure variations were of clinical importance. The rhythmic fluctuations in VFP at the frequency of 1/min can be normal but their incidence increases with pathology and then may represent cerebral dysfunction. This may also be true for the rhythmic waves with a frequency of 6/min. The waves described by Lundberg were:

- *A waves* or *"Plateau waves"* have amplitudes of 50–100 mmHg, lasting 5–20 min. These waves are always associated with intracranial pathology (Fig. 1). During such waves, it is common to observe evidence of early herniation, including bradycardia and hypertension. The aetiology is uncertain, but it is postulated that as cerebral perfusion pressure (i.e. the difference between mean arterial pressure and intracranial pressure, CPP) becomes inadequate to meet metabolic demand, cerebral vasodilatation ensues and cerebral blood volume increases. This leads to a vicious circle, with further CPP decrease, predisposing the patient to other plateau waves and, if low CPP is not corrected, to ruinous effects.
- *B waves* are oscillating and up to 50 mmHg in amplitude with a frequency 0.5–2/min and are thought to be due to vasomotor centre instability when CPP is unstable or at the lower limits of pressure autoregulation.
- *C waves* are oscillating and up to 20 mmHg in amplitude and have a frequency of 4–8/min. These waves have been documented in healthy individuals and are thought to occur because of interaction between cardiac and respiratory cycles.

Both A and B waves require intervention to reduce ICP and preserve CPP. Without the continuous recording of ICP, judgement of correct timing and evaluation of the efficacy of the therapy will be difficult.

## Monro and Kellie doctrine

The fundamental principles of raised intracranial pressure were developed in Scotland and are condensed in the doctrine credited to Professors Monro (1783) [4] and Kellie (1824) [5], which states, once the fontanelles and sutures are closed, that:

- The brain is enclosed in a non-expandable case of bone;
- The brain parenchyma is nearly incompressible;
- The volume of the blood in the cranial cavity is therefore nearly constant and
- A continuous outflow of venous blood from the cranial cavity is required to make room for continuous incoming arterial blood.

The importance of these observations is that the skull cannot easily accommodate any additional volume. The craniospinal axis is essentially a partially closed box with container properties including both viscous and elastic elements. The elastic or, its inverse, the compliant properties of the container will determine what added volume can be absorbed before intracranial pressure begins to rise. In its original form the Monro-Kellie doctrine did not take into account the CSF as a component of the cranial

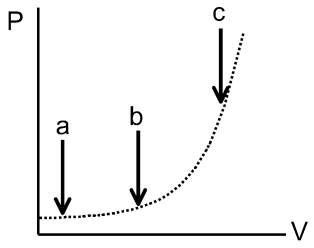


**Fig. 2** Pressure-volume curve of the craniospinal compartment. This figure illustrates the principle that in the physiological range, i.e. near the origin of the x-axis on the graph (*point a*), intracranial pressure remains normal in spite of small additions of volume until a point of decompensation (*point b*), after which each subsequent increment in total volume results in an ever larger increment in intracranial pressure (*point c*)

compartment. The concept of reciprocal volume changes between blood and CSF was introduced in 1846 by Burrows and, later, extended in the early twentieth century by Weed to allow for reciprocal changes in all the craniospinal constituents.

An understanding of raised ICP encompasses an analysis of both intracranial volume and craniospinal compliance. Therefore, ICP is a reflection of the relationship between alterations in craniospinal volume and the ability of the craniospinal axis to accommodate added volume (Fig. 2).

If a new intracranial volume displaces venous blood and CSF, for example haematoma, tumour, oedema or hydrocephalus, initially there is little change in ICP. However, the ability to accept the cerebral blood flow component of the cardiac cycle is decreased and, provided the volume of each cerebral component of the cardiac cycle remains constant, close observation will recognise an increase in the ICP wave amplitude [6]. This is because intracranial compliance is reduced. Further exhaustion of the volumetric compensatory reserve leads to an increase in mean ICP and a further increase in ICP wave amplitude. At very high ICP the amplitude of the ICP wave decreases as cerebral blood flow (CBF) is reduced by a reduction in compliance and perfusion pressure. Avezaat and Van Eijdhoven were some of the original researchers to study the changing shape of the ICP wave as the patient moves along the volume-pressure curve. They developed a model showing that the ICP pulse amplitude (ΔP) was linearly proportional to the ICP and the elastic coefficient (E1). They used this method roughly to estimate the intracranial compliance of the patient.

## Intracranial compliance

Intracranial compliance is the change in volume (ΔV) per unit change in pressure (ΔP). Compliance is the inverse of elastance (ΔP/ΔV), sometimes known as the volume-pressure response (VPR).

$$\text{Compliance}(C = \Delta V/\Delta P) = 1/\text{Elastance} = 1/VPR$$

Marmarou, interested in CSF dynamics, was the first to provide a full mathematical description of the craniospinal volume-pressure relationship. He developed a mathematical model of the CSF system which produced a general solution for the CSF pressure. The model parameters were subsequently verified experimentally in an animal model of hydrocephalus. As a corollary of this study, Marmarou demonstrated that the non-linear craniospinal volume-pressure relationship could be described as a straight line segment relating the logarithm of pressure to volume, which implies a mono-exponential relationship between volume and pressure. Marmarou termed the slope of this relationship the pressure-volume index (PVI), which is the notional volume required to raise ICP tenfold. PVI is expressed by the formula:

$$PVI = \Delta V/(\log_{10} P_o/P_m)$$

Where ΔV expresses the volume, in millilitres, added or withdrawn from the ventricular system, $P_o$ is the initial pressure and $P_m$ the final pressure.

Unlike elastance or its inverse, compliance, the PVI characterises the craniospinal volume-pressure relationship over the whole physiological range of ICP. The PVI is calculated from the pressure change resulting from a rapid injection or withdrawal of fluid from the CSF space and was utilised both clinically and experimentally as a measure of summed craniospinal compliance. In the clinical setting, PVI measures are obtained by first removing 2 ml and recording the reduction in pressure [7]. By this technique, the PVI can be estimated and, after deciding upon a peak pressure that should not be exceeded, a maximum volume injection can be calculated. Ordinarily, the PVI measures are obtained by repeated withdrawal and injections of 2 ml and the average PVI is calculated from multiple injections. Injection of fluid into the CSF space is not performed when ICP is high. In those cases, PVI is obtained only from withdrawal of known quantities of fluid.

However, the use of the PVI method is not without disadvantages:

- Variability exists between measurements due to the difficulty in manually injecting consistent volumes of fluid at a constant rate. As a result an average of repeated measures is usually required.
- There is an increased risk of infection with this technique. Aetiologies include: manipulation of the CSF access system to test the PVI, CSF sampling and recalibration of the pressure transducer, all of which potentially expose the patient to a higher risk of infection.
- Moreover, the procedure is time consuming and requires highly trained personal.

As a consequence of these limitations, the PVI tests are not routinely used in the clinical situation.

## Conclusion

Intracranial pressure is a reflection of the relationship between alterations in craniospinal volume and the ability of the craniospinal axis to accommodate added volume. It can not be estimated without directly measuring it. In 1972, Mario Brock realised the interest in ICP monitoring and organised the first International Symposium on Intracranial Pressure in Hanover. This was the start of a very successful series of meetings and continues in Hong Kong this year, as ICP XII.

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