# Chapter 4
# Intracranial Pressure: Invasive Methods of Monitoring


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## 4.1 Historical Overview and Basic Concepts

Since the discovery of a small foramen in the floor of the fourth ventricle and its connection to the subarachnoid spaces of the brain and spinal cord by François Magendie in the late eighteenth century, neurologists and neurosurgeons have studied the physiological relationships between the cerebrospinal fluid flow and the problems caused by the raising of its pressure [1], although initially the concept of cannulation of the ventricles was frowned upon, as commented by Robert Whytt in his *Observations on Dropsy in the Brain*, published in 1768: 'any such attempt to draw off the water, could have no other effect than to hasten death' [2].

The first to standardize the technique for lumbar puncture and measurement of the cerebrospinal fluid (CSF) pressure by connecting the lumbar puncture needle to a fine glass pipette open to atmospheric pressure was Quinke, who in 1891 published his studies on the diagnostic and therapeutic applications of diverting the fluid from the subarachnoid space of the spinal cord [1].

Later on, researchers started moving from the lumbar puncture to the direct catheterization of the ventricular system, the first report of an organized method of measuring intracranial pressure took place in France, 1951 by Guillaume and Janny, which consisted in the use of *continuous intracranial manometry*—an electromagnetic transducer to measure ventricular CSF pulses in patients with various types of intracranial lesions [3].

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### *4.1.1 Lundberg's Waves*

It was not until the publications of Nils Lundberg in 1960, though, that foundation for modern intracranial pressure (ICP) monitoring was laid: involving conscious volunteers with a multiplicity of intracranial pathologies, his work aimed to develop a cannulation and measurement method that was minimally traumatic, easy to manipulate and with a low infection and leakage risks [1, 2].

After initial studies with 48 patients, Lundberg classified rhythmic fluctuations in the ICP in 3 graphically measured waves: A, B and C. It is important to point out that Lundberg's method provided a quantitative analysis of the ICP wave, not a qualitative one as the monitors available nowadays.

*'A' waves* (Fig. 4.1)—or *plateau waves*, as they were baptized by Lundberg—are always associated with intracranial pathology, during which is common to observe clinical signs of herniation such as bradycardia and hypertension. Yet without a certain aetiology, it is hypothesized to be related to the loss of cerebral autoregulation, as cerebral perfusion pressure (CPP) becomes inadequately low to meet brain tissue's metabolic demand, causing micro cerebral vasodilation and raise of intracranial blood volume, which then causes further decrease of the CPP in a vicious cycle [1, 4] (Fig. 4.2). They have amplitudes of 50–100 mmHg and last between 5 and 20 minutes, as described my Lundberg:

> The tracing may reveal a number of different spontaneous pressure variations. Among these, one type is of particular interest, because it is closely related to acute cerebral symptoms in patients with intracranial hypertension. This type of pressure variation is characterized by a sudden rapid rise, continuation on a high level for some time, and finally a rapid fall. Because of the typical shape which these pressure variations give to the VFP curve, I call them "plateau waves." [4]

*'B' waves* (Fig. 4.3) are the most frequently observed pattern and less correlated with adverse clinical outcomes as they can be observed in patients with normal mean ICP, being interpreted as non-specific indicators of diminished compliance. They consist of clustered acute peak-shaped upstrokes without plateau waves

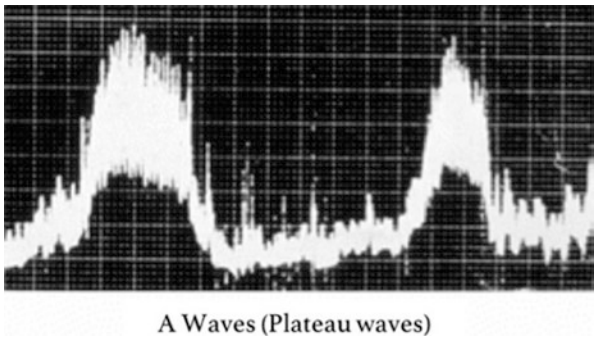



**Fig. 4.1** Lundberg's 'A' waves [5]

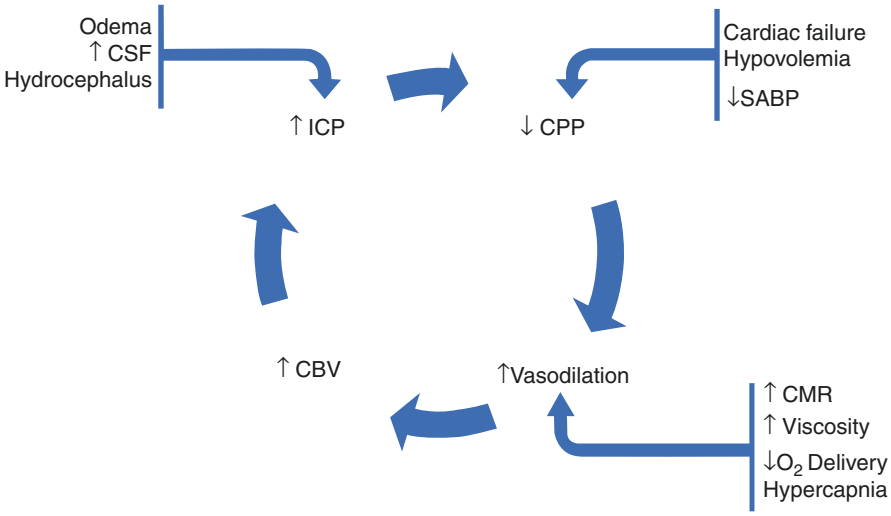
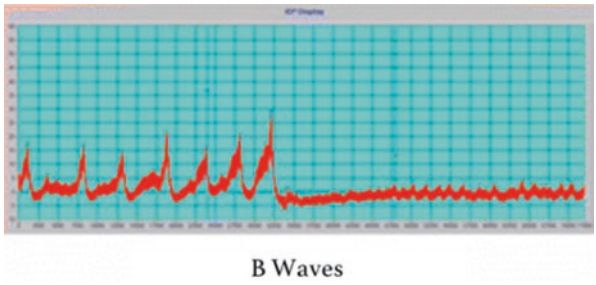



**Fig. 4.2** Cerebral vasodilatory cascade. ICP Intracranial Pressure, CPP Cerebral Perfusion Pressure; CBV Cerebral Blood Volume CSF Cerebrospinal Fluid, SABP Systolic Arterial Blood Pressure, CMR Cerebral Metabolic Rate

**Fig. 4.3** Lundberg's 'B' waves [5]




occurring in a frequency of 1–2 per minute and ranging between 20 and 30 mmHg above the baseline level, up to 50 mmHg. B waves cluster interval duration varies between 5 and 30 minutes [5].

*'C' waves* have been documented in healthy individuals and have little clinical or pathological significance, probably related to the respiratory and cardiac cycles. They are rapid oscillations, in average 4–8 per minute and up to 20 mmHg, thus unrelated to intracranial hypertension [1].

A and B waves require attention and medical intervention in order to maintain CPP and reduce ICP, and even though Lundberg's patterns nowadays are more of historical than clinical value, continuous intracranial pressure monitoring is a useful tool in evaluating the treatment results and feedback.

### *4.1.2 The Monro-Kellie Doctrine*

The principles and rationale of the dynamics between the intracranial elements and pressure are condensed in the doctrine first postulated by professor Alexander Monro Secundus and his pupil George Kellie, which determines that in an intact cranium with closed sutures the volume of the brain plus the CSF plus the intracranial blood volume is a constant; therefore, an increase in one should be compensated by a reduction in the volume of one or both of the remaining two [6].

A similar phenomenon can be observed when a new intracranial volume is added, displacing CSF and/or venous blood volumes, which can be physiological—as in the inflow of arterial blood in systole—or pathological—as the development of a brain tumour or haematoma [1, 6]. Therefore, ICP is a product of the relationship of the alteration in craniospinal volume and the craniospinal axis' ability to accommodate the added volume.

If a new volume is installed, initially there is little change in ICP. Although when cerebral compliance is reduced due to progressive exhaustion of the volumetric compensatory reserve—initially CSF, followed by venous and then arterial blood volume—the continuous inflow of the cerebral component of the cardiac cycle leads to an exponential increase in the mean ICP (and in the amplitude of the ICP wave), which results in the decrease of the CPP until it becomes too great to be overcome by the mean arterial pressure (MAP), resulting in brain death [1].

The aforementioned dynamics, when put in graphical language, take the form of the Langfitt curve (Fig. 4.4), which can be divided in four phases [7]:

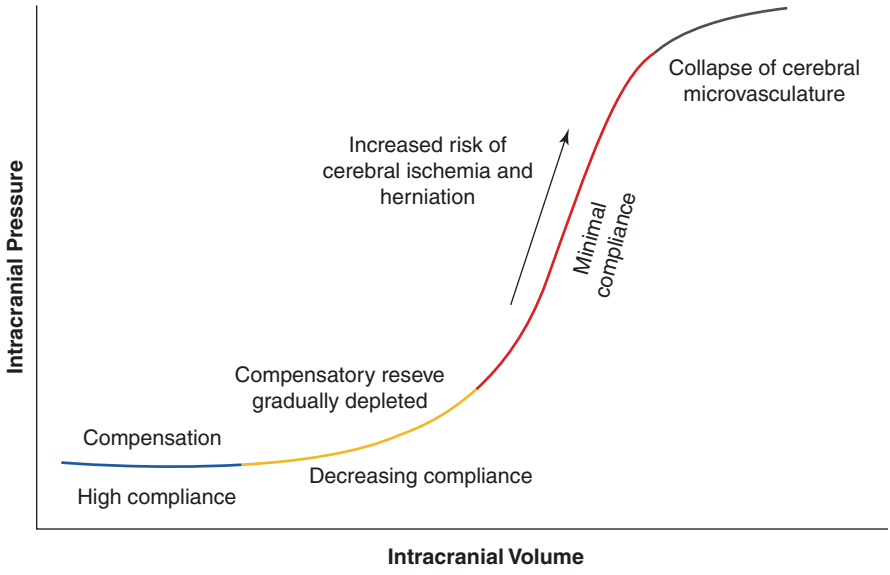



**Fig. 4.4** Langfitt's curve, phases 1–4 [5]

- Phase 1, when, initially, expansive lesions cause proportional dislocation in CSF—from the ventricular system to the spinal subarachnoid space—without increase in ICP.
- Phase 2, when there is virtually no CSF left in the intracranial compartment. There is alteration in the local perfusion, causing lactic acidosis and activation of the vasodilator cascade, increasing blood volume in the cranium.
- Phase 3, when there is an exponential increase in the cerebral blood volume, related to the loss of cerebral autoregulation, with the ICP raising until it equals the MAP and the CPP tends to zero.
- Phase 4, vasoplegia.

## 4.2 Eligibility Criteria for Invasive Monitoring

Intracranial pressure monitoring allows early detection, and therefore early assessment and intervention, in case of expanding focal or diffuse lesion and the calculation of CPP, which are key information in the management of intracranial hypertension.

It is useful in a variety of pathologies such as traumatic brain injury (TBI), hydrocephalus, stroke and encephalopathy. It can be measured using devices inserted into the ventricles, parenchyma, subdural and subarachnoid spaces, being the intraventricular catheter the gold standard [8] (Fig. 4.5).

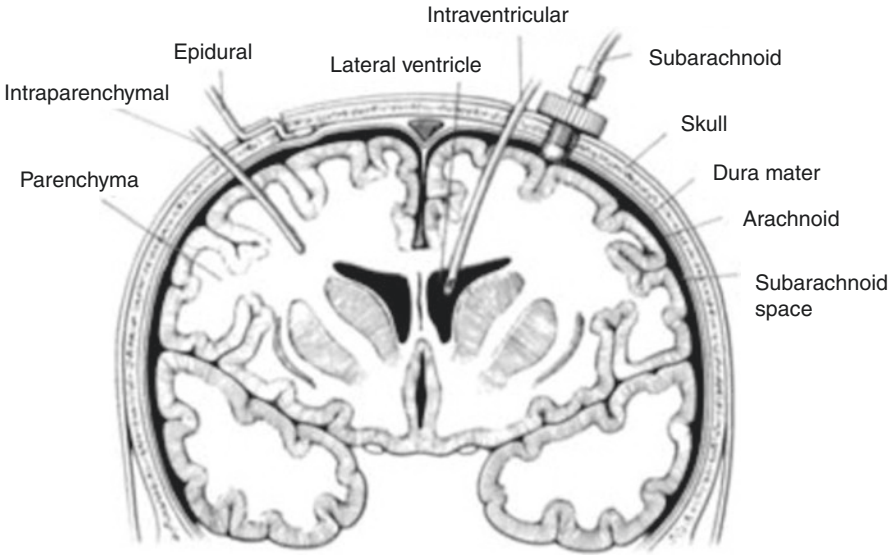



**Fig. 4.5** Different positioning for ICP catheters [5]

In the context of TBI management, invasive intracranial monitoring has become standard of care, as the Brain Trauma Foundation (BTF) recommends invasive ICP monitoring in all salvageable patients with severe TBI—that is, Glasgow Coma Scale (GCS) 3–8 after neurological resuscitation—and an abnormal computed tomography (CT) scan in order to reduce in-hospital mortality and in the initial 2 weeks (Recommendation level II B). In patients with severe TBI but normal CT, ICP monitoring is indicated if two or more of the following criteria are met at admission: age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure < 90 mmHg [9]. The authors of this chapter do not recommend the installation of invasive ICP in patients with admission GCS 3 associated with severe brainstem disfunction after neurological resuscitation, due to limited prognostic and therapeutic possibilities.

Recently, a multicentre randomized trial has been published comparing invasive ICP monitoring to serial CT imaging in patients with severe TBI. It found no advantage in the former when compared to the latter [10]. This study had great international repercussion and has been widely questioned, mainly about its external validity—once it was conducted exclusively in lower-income countries with less routine access to ICP monitoring—and methods [11].

In patients with ischemic stroke, it is not recommended routine use of ICP monitoring, either in malignant cerebral or cerebellar infarction [12]. In case of cerebellar stroke with oedema and compression of more than 50% of the fourth ventricle, due to the high risk of hydrocephalus and direct brainstem compression, a suboccipital decompressive craniectomy immediately preceded by an external ventricular drainage (EVD) associated with intraventricular ICP monitoring is indicated (class I, level of evidence C) [13].

There are two main scenarios in which ICP monitoring is used for malignant cerebral infarction: (a) patients in intensive care for whom medical (non-operative) management is the primary line of care and (b) patients awaiting secondary decompressive craniectomy (DC). ICP values may be used to guide medical treatment intended to reduce ICP or to indicate DC, but solid ICP thresholds for neither of those interventions have been settled, rendering invasive monitoring uncommon in practice. In these patients, transtentorial herniation and clinical deterioration can be observed without any ICP rise [9, 12].

The authors, with this last information in mind, in case of clinical and radiological evidence of malignant thrombosis of the middle cerebral artery (MCA), prefer a primary decompressive craniectomy instead of exclusive ICP monitoring. In the primary DC scenario, ICP monitoring can be used to observe post-operative complications or secondary injuries—for example, epidural haematoma, haemorrhagic transformation.

Patients presenting with mass lesions or ischemia on the temporal lobe should be submitted to routine neuroimaging once they are also prone, due to anatomic reasons, to uncal herniation without great variations in the intracranial pressure.

## 4.3 Definitions in Intracranial Dynamics and Monitoring Methods

### *4.3.1 Definitions*

#### 4.3.1.1 Intracranial Pressure (ICP)

ICP is the pressure exerted by the intracranial components on the inside of the cranium and on each other. It can be measured by a variety of devices and its normal value varies with age: 1.5–6 mmHg in term infants, 3–7 mmHg in young children and up to 15 mmHg in older children and adults. In newborns, it can be subatmospheric [14].

Almir Ferreira de Andrade, Brazilian neurosurgeon and researcher, in his works regarding traumatic brain injury and intracranial pressure, defines values between 15 and 20 mmHg as *altered ICP* without intracranial hypertension [15]. ICP values between 20 and 40 mmHg represent mild intracranial hypertension, which already requires medical attention and intervention. Sustained ICP values greater than 40 mmHg represent severe, life-threatening intracranial hypertension [13–15].

The most recent BTF guidelines indicate intervention in patients with ICP greater than 22 mmHg [9]. The authors recommend stricter attention to patients showing altered intracranial pressure (15–20 mmHg) in addition to (a) reversal of the P1/P2 amplitude ratio (P2 > P1) or (b) difficulty in keeping CPP > 60 mmHg.

Some studies demonstrate lesser mortality in patients who underwent intervention with ICP > 15 mmHg than in those with ICP > 20 mmHg. The authors strongly recommend, whenever available, multimodality monitoring of the ICP (absolute values and waveform) and $PtiO_2$, once severe brain tissue hypoxia can be found in patients with normal ICP (Fig. 4.6) [16].


**Fig. 4.6** Intracranial multimodality monitoring—ICP, temperature and $PtiO_2$

#### 4.3.1.2 Cerebral Perfusion Pressure (CPP)

CPP is the net pressure gradient that drives oxygen and glucose delivery to brain tissue, is measured in millimetres of mercury (mmHg) and is calculated by subtracting the ICP from the mean arterial pressure (MAP), *ergo*, **CPP = MAP – ICP**.

Normal CPP varies between 60 and 80 mmHg, but these values can change depending on the patient's pathology [17].

#### 4.3.1.3 Cerebral Blood Flow (CBF)

The brain receives 15–25% of the cardiac output and the cerebral blood flow ranges from 40 to 50 mL/min for each 100 mg of brain. As there is no invasive method of directly measuring the CBF, it is estimated by the cerebral metabolic rate of oxygen consumption ($CMRO_2$), which in turn varies according to the cerebral vascular resistance [18].

### *4.3.2 ICP Monitoring Methods*

The gold standard method for invasive intracranial pressure monitoring is the insertion of an intraventricular catheter, which enables not only the measurement of the ICP but also the drainage of the CSF in order to treat intracranial hypertension. With that in perspective, there are possibilities of fluid-filled systems and transducer-tipped catheters [18, 19]. The intraventricular catheter is the most cost-efficient method. After Kocher's point trepanation, the device is placed on the hemisphere with most lesions in the imaging studies in order to avoid complications due to a possible interhemispheric pressure gradient. In patients without focal lesions, the surgeon should prefer placing the device in the right hemisphere, due to the greater prevalence of left hemisphere dominance.

After trichotomy, proper asepsis and draping of the patient, a 3-cm straight skin incision is made centred over Kocher's point—11 cm posterior to the glabella, 2–3 cm lateral to the midline, in order to avoid posterior lesions to the superior sagittal sinus or the primary motor cortex (Fig. 4.7a, b). Upon exposition of the frontal bone (Fig. 4.7c) and after identification of the coronal suture, a burr-hole is placed at Kocher's point with a twist drill (Fig. 4.7d), which varies in diameter depending on the type of catheter. Then, cauterization and incision of the now exposed dura with a no. 11 blade are followed by bipolar coagulation of the frontal underlying cortex and opening of the pia mater (Fig. 4.7e). After that, the ventricular catheter is inserted no more than 7 cm into the frontal horn of the ipsilateral ventricle aimed in the coronal plane to the nasion and in the sagittal plane to the tragus' line, toward the foramen of Monro (Fig. 4.7f)—in case of an intraparenchymal catheter, catheter introduction should not exceed 3 cm. After visualization of the CSF through the

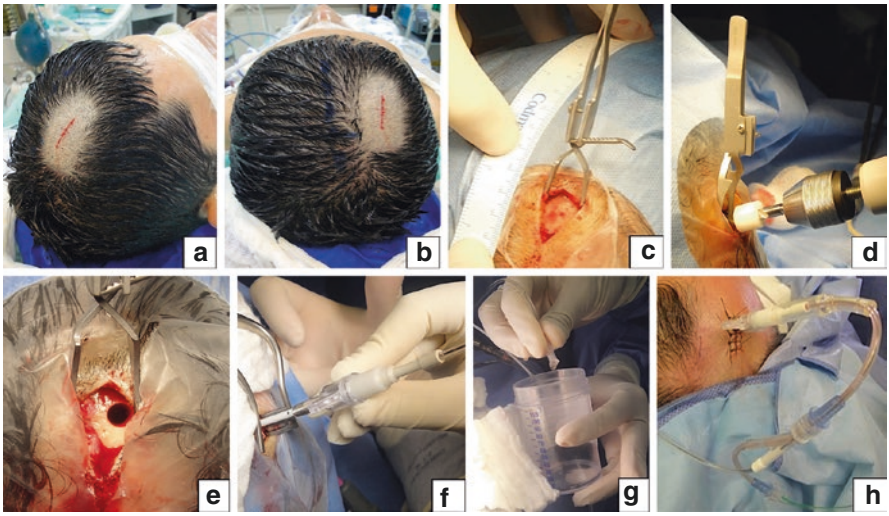



**Fig. 4.7** ICP catheter insertion. (**a**–**b**) Skin incition. (**c**–**e**) Burr role drill in Skull. (**f**) Catheter insertion. (**g**) Catheter CSF check. (**h**) ICP system conected

catheter lumen, some can be collected to laboratorial analysis if necessary (Fig. 4.7g), with posterior fixation and connection of the catheter to the ICP monitoring system and suture of the skin (Fig. 4.7h) and dressing.

Complications secondary to intraventricular catheterization include infection, especially if the catheter stays in place for more than 5 days. Tunnelling the catheter as far as possible from the incision site and strict aseptic conditions in the moment of catheter placement—as well as in any manipulation of the EVD system—are known to reduce the infection rates. Intravenous antibiotic prophylaxis is not recommended [18]. Other possible device placement sites include intraparenchymal, subdural, epidural and lumbar; although the only one frequently used in practice nowadays is the intraparenchymal.

Fluid-filled systems are composed of a catheterized fluid line that connects with an externally placed transducer fixed at the level of the tragus—same level of the foramen of Monro and, ideally, the tip of the catheter. As there is a patent communication between the intraventricular space and an outer system, these systems have the advantages of enabling CSF drainage and, eventually, administration of therapeutic agents such as antibiotics and fibrinolytics [20].

Transducers for measuring pressure are based on strain gauges, originally designed to measure effects of tension and compression in beams, adapted to transmitting the pressure into a pen recorder or an oscilloscope. The preferred device for ICP recording is the *catheter-tip transducer*, which consists of a flexible diaphragm at the tip of a fibreoptic catheter. The diaphragm reflects light and alterations in its intensity are translated into pressure variation.

Another possible device is the *implanted microchip transducer*, which is a very small titanium or ceramic case containing a pressure sensor-microchip system

connected by wires into a nylon tube to complete a Wheatstone bridge type circuit. It can be inserted directly into the parenchyma, but can also be associated with an intraventricular catheter [19, 21].

## 4.4 Interpretation of ICP Monitoring

Besides analysing the absolute ICP value, monitoring gives relevant information regarding the cerebral compliance (curve morphology) and, consequently, autoregulatory disorders. This allows us to plan early treatment for ICP raise before irreversible lesions to the brain parenchyma take place [15].

Various upsweeps can be distinguished in the ICP curve morphology, mainly *cardiac waves* and *respiratory waves* [18], aside from the aforementioned Lundberg's waves:

Cardiac waves (Fig. 4.8) are the intracranial repercussion of elements in the cardiac cycle and its reflex in the cerebral blood vessels, and are composed of three upstrokes:

- P1 (Percussion wave): the first upstroke and the one of greater amplitude in the average patient. Reflects the arterial input during systole and its echo in the vessels on the choroid plexi [18].
- P2 (Tidal wave): the second upstroke and of lesser amplitude than P1. In the average patient, P2 corresponds to 80% of P1 in amplitude. Reflects the fluid (venous blood/ CSF) output in response to the brain's volume intake after systole. It is pertinent to note that a reversal of the P1/P2 amplitude ratio reflects a state of low cerebral compliance, once intracranial inflow and outflow seems to be out of synchrony [18, 22].

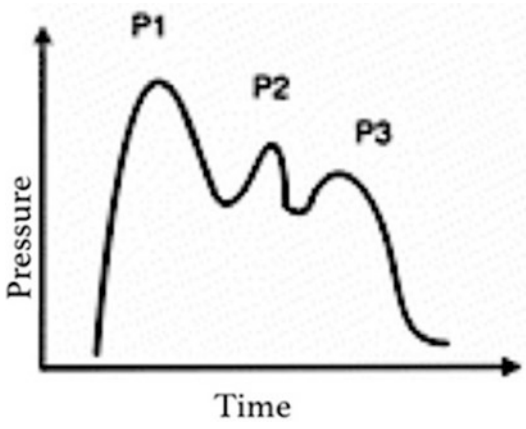



**Fig. 4.8** ICP cardiac wave: percussion (P1), tidal (P2) and dicrotic (P3) waves [5]

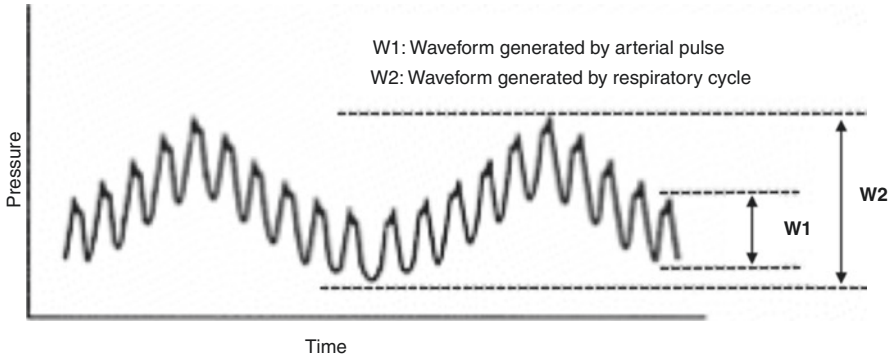



**Fig. 4.9** ICP respiratory waves [5]

- P3 (Dicrotic wave): the third and last upstroke is also the one with less amplitude. Immediately follows the dicrotic notch on the arterial waveform, reflecting the closure of the aortic valve. It has no clinical or pathological value.

Respiratory waves (Fig. 4.9) are synchronous with variations in central venous pressure due to changes in intrathoracic pressure along the respiratory cycle.

## 4.5 Complications

As any other invasive procedure, installation and maintenance of an intracranial catheter are not free of complication, although most of the times the management of these is not of surgical nature. The most common is infection, with an incidence of 5–14%—colonization of the catheter is more incident than clinical infection. There is no association between infection reduction and prophylactic substitution of the system.

Some factors not associated with infection are insertion in neurologic ICU, previous catheter insertion, CSF drainage and use of steroids. The use of antibiotic-coated intraventricular catheter reduced the risk of infection from 9.4 to 1.3% [14].

Other complications are haemorrhage (with an overall incidence of 1.4%), which rarely has indication for surgical evacuation, malfunction, obstruction and malposition. The authors recommend that in suspicion of ventricular catheter obstruction, the physician does not proceed to blindly irrigate the system before a brain CT may eliminate the possibility of ventricular collapse—in which case irrigation will be fruitless and may elevate the infection risk.

## References


1. Andrews PJS, Citerio G. Intracranial pressure – part one: historical overview and basic concepts. Intensive Care Med. 2004;30:1730–3.
2. Srinivasan VM, O'Neill BR, Jho D, Whiting DM, Oh MY. The history of external ventricular drainage. J Neurosurg. 2014;120:228–36.
3. Padayachy LC, Figaji AA, Bullock MR. Intracranial pressure monitoring for traumatic brain injury in the modern era. Childs Nerv Syst. 2010;26:441–52.
4. Lundberg N. Continuous recording and control of ventricular fluid pressure in neurosurgical practice. Acta Psychiatr Scand Suppl. 1960;36:1–193.
5. Harary M, Dolmans RGF, Gormeley WB. Intracranial pressure monitoring — review and avenues for development. Sensors. 2018;2:465–80. (Open access, distributed under CC BY 4.0 - https://creativecommons.org/licenses/by/4.0/).
6. Mokri B. The Monro-Kellie hypothesis: applications in CSF volume depletion. Neurology. 2001;56:1746–8.
7. Hoz SS, Pinilla-Monsalve GD, Padilla-Zambrano HS, Rubiano AM, Moscote-Salazar LR. Langfitt curve: importance in the management of patients with neurotrauma. J Neuroanaesthesiol Crit Care. 2018;5:121–2.
8. Elwishi M, Dinsmore J. Monitoring the brain. BJ A Educ. 2018;19:54–9.
9. Brain Trauma Foundation. Guidelines for the management of severe traumatic brain injury. 4th ed. USA; 2016.
10. Chestnut RM, et al. A trial of intracranial-pressure monitoring in traumatic brain injury. N Engl J Med. 2012;367(26):2471–81.
11. Chestnut RM, et al. A consensus-based interpretation of the benchmark evidence from south American trials: treatment of intracranial pressure trial. J Neurotrauma. 2015;32:1722–4.
12. Simard JM, Sahuquillo J, Sheth KN, Kahle KT, Walcott BP. Managing malignant cerebral infarction. Curr Treat Options Neurol. 2011;13:217–29.
13. American Heart Association/American Stroke Association. Recommendations for the management of cerebral and cerebellar Infarction with swelling. USA; 2014.
14. Rangel-Castillo L, Gopinath S, Robertson CS. Management of intracranial hypertension. Neurol Clin. 2008;26:521–41.
15. Andrade AF, et al. Mecanismos de lesão cerebral no traumatismo cranioencefálico. Rev Assoc Med Bras. 2009;55(1):75–81.
16. Ghajar JB, et al. Survey of critical care management of comatose, head-injured patients in the United States. Crit Care Med. 1995;23:560–7.
17. Mount CA, Das JM. Cerebral perfusion pressure. [Updated 2020 Feb 21]. In: StatPearls [Internet]. Treasure Island: StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537271/.
18. Rodriguez-Boto G, Rivero-Garvia M, Gutiérrez-González R, Márquez-Rivas J. Basic concepts about brain pathophysiology and intracranial pressure monitoring. Neurologia. 2015;30:16–22.
19. North B. Intracranial pressure monitoring. In: Reilly P, Bullock RS, editors. Head injury: Chapman & Hall; 1997. p. 209–16.
20. Kawoos U, McCarron RM, Auker CR, Chavko M. Advances in intracranial pressure monitoring and its significance in managing traumatic brain injury. Int J Mol Sci. 2015;16:28979–97.
21. Baral B, Agrawal A, Cincu R. Intracranial pressure monitoring: concepts in evaluation and measurement. Pak J Med Sci. 2007;23:798–804.
22. Raboel PH, Bartek J Jr, Andresen M, Bellander BM, Romner B. Intracranial pressure monitoring: invasive versus non-invasive methods – a review. Crit Care Res Prac. 2012;2012:1–14.