ANALYSIS OF INTRACRANIAL PRESSURE

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ABSTRACT. Methods for the acquisition and analysis of intracranial pressure (ICP) signals are reviewed from clinical and technical perspectives. The clinical importance of ICP monitoring is presented, and methods for ICP transduction are briefly discussed. These methods include intraventricular catheters, subarachnoid screws, epidural techniques, and the new fiberoptic ICP measurement systems. Approaches to the visual analysis of the ICP waveform are presented, with special emphasis on the relationship between the ICP waveform and the arterial blood pressure signal. Methods of computer-based ICP analysis are also reviewed, including histogram and "systems analysis" methods. Methods to predict ICP pressure rises and to estimate intracranial compliance are also discussed. Finally, ICP monitoring is reviewed from the point of view of patient outcome. It is concluded that advanced ICP waveform analysis methods warrant further clinical evaluation to demonstrate their clinical usefulness.

KEY WORDS. Cerebrospinal fluid: pressure. Brain: intracranial pressure. Monitoring: intracranial pressure. Equipment: computers. Measurement techniques: computer.

The measurement of intracranial pressure (ICP) has for some time been commonplace in the management of the neurosurgical patient. In recent years, however, the analysis of the ICP signal has evolved dramatically from simple measurement of mean ICP and the display of graphic tracings to sophisticated methods based on the Fourier transform analysis. This report presents a brief review of current ICP analytic methods suitable for use by neurosurgeons and neuroanesthesiologists.

It has been estimated that in North America approximately 50,000 patients per year could benefit from ICP monitoring. A paper from Vancouver General Hospital [1] reported 132 patients who underwent ICP monitoring over a 2-year period. Of these, 67 cases involved head injuries, 22 involved cerebrovascular accidents, 16 involved perioperative monitoring of hydrocephalic patients, 10 involved anoxic encephalopathy, 10 involved neoplasms, and 7 involved inflammatory disorders.

A typical candidate for ICP monitoring might be a traffic accident victim who presents with unconsciousness and abnormal decerebrate limb posturing. If the computed tomography scan shows an intracranial hematoma requiring surgical evacuation, an ICP monitoring device will often be implanted at the time of surgery, especially if brain edema is present. At some centers, ICP monitoring is carried out in patients with far less severe clinical circumstances, for example, any multiple trauma patient with possible head injury.

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PATHOPHYSIOLOGY

Changes in intracranial volume can occur with many pathologies by several routes. Intracranial hemorrhages, tumors, abscesses, and parasites may create new structures within the skull; disturbed cerebrospinal fluid (CSF) circulation or brain swelling (cerebral edema) result in enlargement of existing structures. Causes of disturbed CSF circulation include occlusion of the normal pathway of CSF flow (such as by tumor or intraventricular hemorrhage), malabsorption of CSF (e.g., due to inflammation or subarachnoid hemorrhage), or (in principle) excessive CSF formation. Brain swelling can result from direct injury, disturbed blood supply, inflammation, or hepatic failure, resulting in vascular congestion or edema. Of particular importance to the neuroanesthesiologist is the effect of cerebral blood flow and cerebral blood volume on ICP. Decreases in cerebral vascular resistance (e.g., due to volatile anesthetic agents or hypercapnia) may result in increased cerebral blood flow and volume and, consequently, increased ICP.

To cope with such increases in volume, compensatory mechanisms must be brought into action to avoid increased ICP. Because the brain is surrounded by an inelastic skull and compartmentalized by the falx cerebri and the tentorium cerebelli, little space exists for the brain to expand. The efficacy of compensatory mechanisms, for example, CSF redistribution, depends on the rate of volume increase. Acute volume increases, such as those that occur during an intracranial hemorrhage, are accommodated for by intercompartmental and craniospinal shifts of CSF, as well as by intracranial to extracranial blood shifts. This mechanism appears within several minutes of a raised intracranial volume and can buffer up to 15 to 20 ml of CSF. In cases involving a progressive expansion of the brain (as in a tumor), the "effective reserve volume" available can be as high as 150 ml. The responsible mechanism is the resorption of CSF and intercellular fluid in the brain, coupled with brain atrophy in severe cases.

Thus, the body can cope with an elevated intracranial volume to a certain extent. Once this limit is reached, however, a slight increase in intracranial volume leads to a severe rise in intracranial pressure. In these circumstances, the patient is said to be on the "steep" (uncompliant) portion of the pressure-volume curve (Fig 1). If unchecked, further pressure rises may lead to compression of the brain and eventually life-threatening herniation, i.e., movement and compression of brain tissue such as the temporal lobe or cerebellar tonsils. Compression of vital brain centers will lead to unconsciousness and death. A detailed review of the pathophysiology of increased ICP has been provided by Bruce [2].

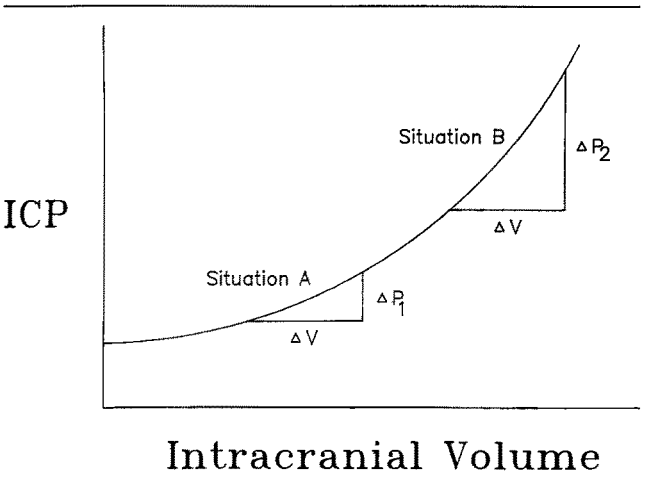

Fig 1. Diagram of the intracranial compliance curve, illustrating regions of high compliance (situation A) and low compliance (situation B).

INTRACRANIAL PRESSURE AND PATIENT MANAGEMENT

Clinical experience has led to knowledge regarding ICP levels: up to 15 mm Hg is normal, 15 to 25 mm Hg is generally tolerable, 25 to 40 mm Hg is severely elevated, and above 40 mm Hg is intolerable for any length of time. (Particular ICP measurements, however, must be interpreted in the context of the position of the patient's head and the transducer position; usually, transducers are positioned at an ear level reference position.) Once ICP equals arterial pressure, the cerebral perfusion pressure (which is equal to arterial pressure minus ICP) falls to zero and no blood flows through the brain. Death follows promptly without treatment, regardless of the extent of compression.

Although a rise in ICP is accompanied by clinical signs such as headache, confusion, and vomiting, they are not specific. Other diagnostic signs such as mydriasis, extension spasms, arterial hypertension, and respiratory paralysis may only occur at the end stage of disease. Furthermore, technical investigations such as electroencephalograms, evoked potentials, or computed tomography scans cannot generally make reliable diagnoses of elevated ICP. Therefore, increases in ICP are not always readily detected by these less traumatic clinical means. Consequently, continuous ICP monitoring can be especially valuable in patients at risk of intracranial hypertension. Such patients include those with severe head injuries, Reye's syndrome, brain tumors, and

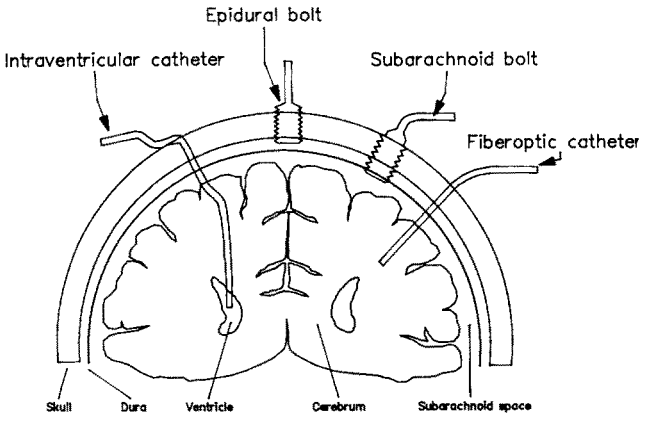

Fig 2. Diagram illustrating intraventricular catheters, epidural bolts, subarachnoid bolts, and fiberoptic catheters for ICP measurement.

acute encephalopathies. When intracranial hypertension is detected, treatment modalities such as hyperventilation, osmotic diuretics (e.g., mannitol), or CSF aspiration through a ventricular catheter may lower ICP. Naturally, any surgical masses must be excluded.

INTRACRANIAL PRESSURE MONITORING METHODS

Traditional Methods

A number of ICP monitoring methods exist. Perhaps the easiest to perform is a lumbar puncture, in which a catheter is placed in the spinal subarachnoid space. However, this method suffers from technical and clinical limitations that make it less suitable for chronic clinical use (for example, spinal CSF pressures often do not reflect intracranial pressure, and the method presents practical nursing problems). Clinically, the most common methods of ICP monitoring are intraventricular catheters and subarachnoid screws (Fig 2).

The intraventricular catheter is a more direct method frequently used to monitor ICP; of the methods in common use, it gives the most accurate pressure measurements and the most satisfactory waveforms for study [3]. In addition, small amounts of CSF can be removed from the catheter to reduce ICP or for analysis. Similarly, small amounts of artificial CSF may be infused with the catheter to determine the pressure-volume relationship of the brain (i.e., brain compliance). Because of the risk of infection, intraventricular catheters are generally removed as soon as is practical.

Another common ICP monitoring device is the subarachnoid screw (Richmond bolt). The opening of this device is placed just under the arachnoid membrane through a small hole through the skull and dura. It may require occasional irrigation or adjustment to prevent blockage of the opening under the dura.

Finally, epidural methods are also in common use in some centers; for more information on this and other traditional methods of ICP monitoring, see the review by Gaab and Heissler [3].

Fiberoptic Intracranial Pressure Monitors

In recent years, fiberoptic transducer-tipped catheters (FTCs) have been introduced into clinical service for ICP measurement. The principal component in these devices is a monitor that senses variations in the amount of light reflected from a pressure-sensitive diaphragm located at the catheter tip. Such devices appear to offer advantages over conventional ICP monitors, especially in their ability to measure brain parenchymal pressures.

One of the earliest reports on fiberoptic ICP monitoring comes from Ostrup et al [4], who studied a system from Camino Laboratories (San Diego, CA), initially in animals, and later in 15 adult and 5 pediatric patients. They noted that the system "appears to offer substantial advantages over other monitors currently in use."

A later evaluation by Crutchfield et al [5] also identified advantages over conventional ICP measurement systems. They noted, however, that the devices have a maximal cumulative drift of ± 6 mm Hg over a 5-day period, a level that may be clinically significant. Since these devices, unlike conventional units, cannot be recalibrated once inserted, Crutchfield et al recommend replacement if monitoring is to exceed 5 days.

In a report by Hollingsworth-Fridlund et al [6], the advantages and drawbacks of fiberoptic ICP monitoring were discussed; some key considerations are presented here. Fiberoptic transducer-tipped catheters can be placed into intraventricular, subarachnoid, or intraparenchymal sites, thus making them more versatile than conventional systems, which work poorly in measuring brain parenchymal pressures. Present-day FTC systems require dedicated equipment for waveform display and alarm management, whereas conventional ICP systems use existing bedside monitoring equipment to achieve this purpose, thereby reducing capital equipment costs. Both systems allow ventricular drainage; however, in the case of the FTC system, drainage and monitoring can occur simultaneously. The FTC systems are potentially less susceptible to artifacts caused by patient movement or from fluid column sources. They also eliminate the need to standardize transducer height.

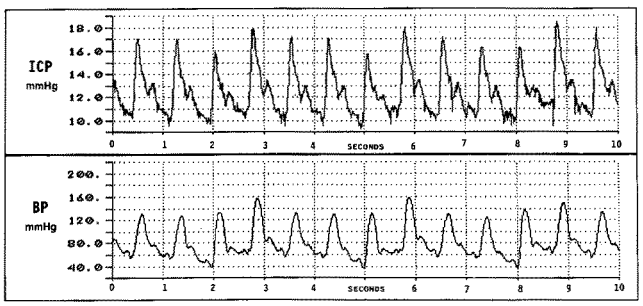

Fig 3. Typical recordings of ICP from an intraventricular catheter (top) and from arterial blood pressure (bottom) obtained from a ventilated patient in our neurosurgical intensive care unit. This patient suffered from cerebral edema after a craniotomy for a leaking cerebral aneurysm. Notice the variation in both signals related to mechanical ventilation.

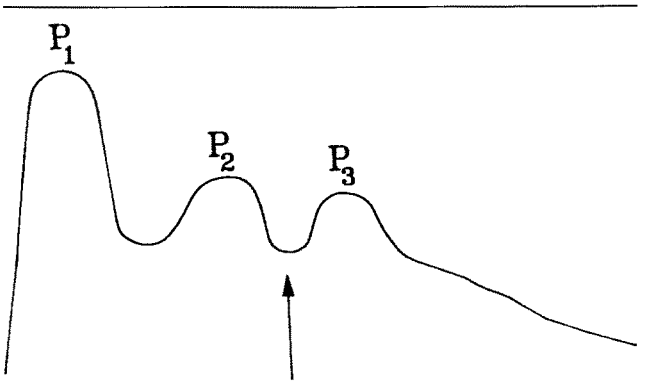

Fig 4. Diagram of the ICP waveform components according to the nomenclature of Gega et al [17] and Cardoso et al [18]. P_1, percussion wave; P_2, tidal wave; P_3, dicrotic wave. The arrow *indicates the position of the dicrotic notch in the arterial pressure wave.*

Conventional systems are easily re-zeroed following insertion. Finally, disposable fiberoptic catheters are considerably more expensive than conventional units.

Further discussion on FTC is presented by Piek [7] and Barin et al [8].

Other Intracranial Pressure Measurement Methods

Numerous other methods of ICP measurement have been reported. In neonates, who have an open anterior fontanelle, many techniques of noninvasive ICP monitoring have been described. Recent reports, which provide references to earlier methods, suggest that these techniques have promise [9–11]. Many of the limitations of measuring neonatal ICP using applanation fontanometers are discussed by Kaiser and Whitelaw [12]. Applanation fontanometers estimate ICP in infants with an open anterior fontanelle by quantifying the degree of depression that results from a given applied force.

Gucer et al [13] have reported the long-term performance of an implantable telemetric intracranial pressure sensor, while Leung et al [14] have described the use of semicustom integrated circuits for this purpose.

THE INTRACRANIAL PRESSURE WAVEFORM

The normal fluctuations in ICP create a characteristic waveform. A clear correlation exists between peaks in the ICP wave and the arterial pulse wave (Fig 3). Changes in the ICP due to ventilation are also present, but may be subtle. Investigators have documented specific variations in the ICP waveform, changes that correspond to certain alterations in the cerebral vascular system, CSF circulation, and respiration, and that sometimes can be used to measure the progression of disease. The early work of Lundberg [15] is especially important in this regard. Portnoy and Chopp [16] commented on their experiences with ICP waveform morphology. They noted "in patients who do not have an expanding mass or cerebral edema, the waveform shows an initial sharp rise and subsequent downward slope similar to the arterial pulse . . . as an expanding mass or edema develops, the waveform becomes more rounded . . . this can frequently be noted several hours before there are significant changes in the ICP."

In another approach to the analysis of the ICP waveform, Gega et al [17] recorded simultaneous ICP waves and carotid pulsations and subjected them to a number of measurements. Waves on the ICP tracing were related to carotid waves of similar morphology and timing (Fig 4). The ICP wave was described as "a sawtooth wave composed of small peaks from P1 to P5," the first three of which were "universally present" and designated as "component waves." These waves were said to correspond to the "percussion wave," "tidal wave," and "dicrotic wave" of the carotid recordings. No comment was made on the possible clinical significance of their findings.

Cardoso et al [18] extended the work of Gega et al in an attempt to establish relationships between anatomic substrate and particular wave components. It was noted the ICP waveform shape was related to mean ICP; at low ICP levels, the P1 component was clearly distinct, but as the mean ICP rose spontaneously, there was a progressive elevation in P2, while P1 changed only slightly, resulting in rounding of the waveform. Other studies were carried out using head elevation, CSF withdrawal, and hyperventilation. It was concluded

that "the P1 component could result from pulsations originating at the choroid plexus and large intracranial conductive vessels, while the P2 component may reflect variations in the cerebral bulk compliance . . . and could be used to indicate microcirculatory vasoparalysis, cerebral swelling and edema."

INTRACRANIAL PRESSURE HISTOGRAM ANALYSIS

Early attempts at statistical analysis of the ICP waveform centered around histogram analysis, a method whereby pressure measurements taken at regular intervals are grouped into pressure classes at standard intervals, e.g., 5 mm Hg.

Kullberg [19] reported one of the first pieces of specialized equipment for ICP analysis. This was a system of 13 electronic counters for producing ICP histograms. Histogram intervals were 10 mm Hg in width, with a sampling rate from 0.1 to 1 sample per second. The maximum counting capacity was 10,000 counts, which defined the maximum recording duration at any given sampling rate.

Later, Barnes and McGraw [20] developed a somewhat more refined ICP histogram machine using 16 histogram intervals. The unit offered the following features: reduced size, a choice of interval ranges, and a histogram display using a 16-column bar graph. A rather similar system was also described by Brock et al [21].

Janny et al [22] refined the analysis of ICP histograms to study dyssymmetry coefficients and flattening coefficients, as well as the gaussian distribution of the histogram data (Fig 5). Mean, modal, and median ICP measurements were also computed. In sixteen 24-hour histograms from 13 patients, it was found that the data did not fit an underlying gaussian distribution, that the histograms were generally skewed to the right (possibly as a result of "plateau waves" [15]), and that increases in ICP were paralleled by increases in ICP fluctuation, but asymmetrically so, the fluctuation increases above the histogram mode being more strongly related to ICP levels than those below the mode.

Brock et al [23] examined the reliability and reproducibility of ICP histograms in two groups of neurosurgical patients. They concluded that lengthy 24-hour data collections are not always necessary to produce clinically useful histograms, and that periods as short as 1 or 2 hours "reliably reflect the overall pressure distribution of longer time intervals (up to 1 day) under conditions of clinical stability."

Despite all the effort expended in studying ICP histograms, clinical application of histogram analysis has been met with limited enthusiasm because, aside from the mean value of the histogram, clinical significance has yet to be ascribed to ICP histograms.

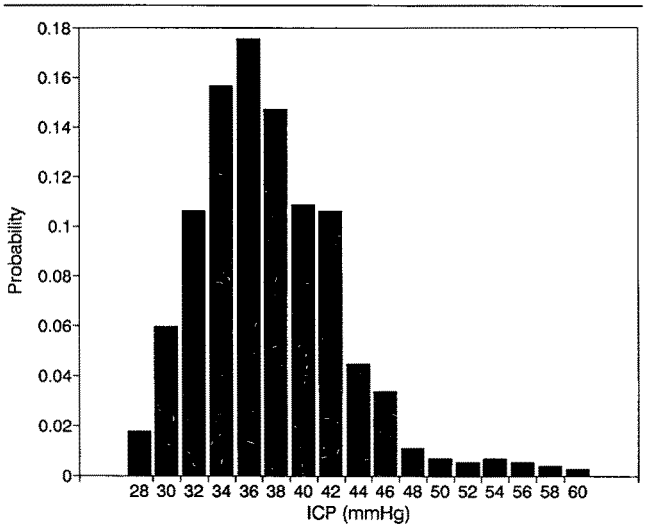

Fig 5. Sample ICP histogram based on data from Janny et al [22] recorded over a 24-hour period.

PREDICTION OF INTRACRANIAL PRESSURE RISES

One goal of ICP analysis is to look for apparently normal ICP patterns that may herald subsequent increases in ICP. Szewczykowski et al [24] analyzed ICP data via a minicomputer system and computed mean ICP, ICP standard deviation, and other statistical data for consecutive 10- or 30-second periods. The relationship between ICP mean and standard deviation values was studied and used to classify ICP data in two zones, a normal zone where ICP variation (standard deviation) was independent of mean ICP and a "warning zone" where ICP variation was linearly related to mean ICP. It was hypothesized that the observed relationship between ICP variation and mean ICP signified impaired compensating ability of the brain such that "at ICP levels above some critical value, there appear disturbances in venous blood outflow with a resulting change in the dynamic properties of the intracranial system." This appears to be one of the first attempts to detect ominous ICP patterns by statistical analysis.

In similar work by Turner et al [25], the ICP was sampled at 4-second intervals for 500 samples to obtain histograms every 33 minutes. Four of the 13 patients studied developed elevated ICP levels heralded by widening of the ICP histogram, i.e., increased ICP variance. It was suggested that the increased variation in the histograms may be due to increased ICP pulse pressure, as evidenced by an increase in the ratio of ICP pulse

pressure to mean ICP prior to a worsening of (increase in) the ICP.

In studies using continuous saline infusion to artificially raise ICP, Price et al [26] observed an increase in the ICP pulse pressure ("cardiac component" in their terms) preceded by a rise in mean ICP, and concluded that "an increase in standard deviation of the ICP often provides a warning of an impending rise in pressure."

ESTIMATION OF INTRACRANIAL COMPLIANCE

With excessive increases in intracranial volume, the effectiveness of the body's compensating mechanisms is reduced, and further increases in volume produce progressively larger increases in ICP. This characteristic is reflected in the exponential shape of the intracranial pressure-volume relationship, or compliance curve [27] (Fig 1). Clinically, it is important to monitor for evidence of any decrease in the volume-buffering capacity of the intracranial system. Unfortunately, intracranial volume reserve cannot be estimated from resting ICP level; furthermore, the shape of the intracranial compliance curve is known to vary among individuals as a result of disease or trauma.

One class of methods used to quantify intracranial compliance involves adding or withdrawing a standard amount of CSF and studying the resulting change in ICP [28–30]. However, these methods do have drawbacks. For example, there is no general agreement on how to administer the bolus infusions (if this method is chosen), exactly how to interpret the results, or even under what conditions the methods are valid. Further drawbacks include an increased risk of infection, the possibility of provoking an increased ICP, and the method's inability to be used with epidural recordings. In addition, the method cannot be used continuously in an online manner [31]. In fact, Chopp and Portnoy [32] believe that the clinical success of these methods in patients with increased ICP appears "negligible."

These difficulties have led some investigators to attempt to develop a continuous index of brain compliance from ICP waveform tracings and related measurements. These methods attempt to extract information from the natural pulsations that exist in the ICP waveform due to the increased cerebral blood volume with each heart beat. In contrast to injection methods, these methods are based on an unknown volume change resulting in a known (measured) pressure change. This class of methods has been termed "CSF pulse amplitude analysis" by Chopp and Portnoy [32], and will be discussed below.

INTRACRANIAL PRESSURE PULSE ANALYSIS METHODS FOR COMPLIANCE ESTIMATION

It is well-known that the ICP waveform undergoes variations of arterial origin and is composed of diastolic and systolic components in phase with the systemic arterial pressure waveform. The ICP waveform may be viewed as resulting from a rhythmic injection of blood into the cranial vault; the pressure response from this systolic volume increment will depend to some degree on the overall compliance of the intracranial contents. Thus, it is hoped that the study of relationships between the ICP waveform and the arterial pressure waveform may provide insight into intracranial compliance without resorting to more invasive means.

Castel and Cohadon [33] studied the ICP waveform in three groups of neurosurgical patients with a view to obtaining information regarding brain compliance. They noted that a linear relationship exists between the ICP waveform amplitude and the mean ICP level, but obtained no clear indicators of decreased brain compliance from their data.

Avezatt et al [34] investigated whether the relationship of ICP pulse pressure to mean ICP could yield information on brain compliance. In all 31 patients studied, a linear relationship was found between the ICP pulse pressure and mean ICP. They also investigated the use of a variable known as the "pulse pressure/pressure relationship" as an index of brain compliance. They suggested that "a change in this relationship during patient monitoring means a change either in the volume-pressure relationship or in the net volume change per cardiac cycle." To differentiate between these two possibilities, a formal volume-pressure test using artificial CSF is needed, but "this has the advantage that the volume-pressure test can be applied more rationally."

In further studies by Van Eijndhoven et al [35], the interrelationship between the ICP pulse pressure and the volume-pressure relationship was examined during cerebral compression in dogs. Below a certain breakpoint ICP, the ICP pulse pressure "was shown to be a useful parameter (sic) to monitor changes in intracranial elastance," but "above this pressure, . . . pulse pressure is a more reliable guide of a patient's clinical condition than the volume-pressure response." The breakpoint was attributed to vasoparesis and failure of autoregulation.

Price et al [26] used a computer with a high sampling rate to follow the ICP waveform with higher than usual resolution to measure the "pulse wave amplitude" and the "pulse wave mean," variables used in calculating a "pulse wave index" during a jugular compression ma-

neuver. This index is equivalent to the elasticity measure described by Szewczyknowski et al [36] and is based on the ratio of pulse wave amplitude and pulse wave mean over a series of ICP waves.

In a review by Van Eijndhoven and Avezatt [31], the theoretical relationships between ICP pulse pressure and volume-pressure response were discussed. While formal assessment of the volume-pressure response involves injection of a known amount of artificial CSF (ΔV) into the ventricles with subsequent measurement of the resulting pressure change, the change in craniospinal volume with the arterial pulse is not known, so that changes in ICP pulse pressure may be due either to changes in brain stiffness or to changes of patient hemodynamics resulting in variations in the change in cerebral blood volume during systole.

Chopp and Portnoy [32] identify further difficulties with using the ICP waveform to estimate intracranial compliance. They point out that the method requires a range of data points so that "if there is no spontaneous elevation of ICP, ICP must be artificially elevated, usually by jugular compression, which may be hazardous." They also point out that the theoretical basis of the method may be flawed in that the volume change ΔV is not an instantaneous change, but "results from an arterial inflow and venous outflow, and the temporal relationship between the two." They advocate the use of "systems analysis" of the ICP to circumvent these problems.

SYSTEMS ANALYSIS OF INTRACRANIAL PRESSURE

In the "systems analysis" method of Chopp and Portnoy [32], the intracranial contents are modeled as an unknown system (the so-called black box) in which the arterial pulse acts as input signal and the ICP waveform as output signal. Digital signal processing methods were used to compute the transfer function (XFR) between the blood pressure and ICP waveforms under conditions of epidural balloon inflation and intraventricular infusion in rats. The systems analysis method was able to distinguish between epidural balloon inflation and intraventricular infusion above 30 mm Hg, while the volume-pressure test failed at all ICP levels. Chopp and Portnoy point out that "as ICP increases, there is a concomitant cerebral vasodilation, which decreases the ability of the arterioles to attenuate the pulse . . . this results in an increased transmission of the pulse to the compliance vessels, with enhancement of the transmural pulse pressure." Under these conditions, enhanced transfer function between the blood pressure and ICP waveforms is to be expected. A somewhat similar analysis method was also reported by Kasaga et al [37].

Later, Portnoy and Chopp [38] looked at the effects of hypoxia and hypercapnia (both cerebral vasodilators) in cats using the methods of ICP systems analysis. These interventions produced an increase in ICP pulse pressure, an increase in the fundamental harmonic of the XFR, and rounding of the ICP waveform. The results were attributed to cerebral arteriolar vasodilation. In a related study in dogs [39], ICP systems analysis methodology was used to establish evidence for loss of vasomotor tone during hypercapnia.

As promising as this new methodology appears, it should be pointed out that it is intended primarily as an investigational tool and its use for clinical ICP monitoring has met very limited application, at least so far. A serious drawback to the method is that its application generally requires a specialized, expensive digital signal processing system. As further reports concerning the method become available, its clinical value will become more apparent.

EXISTING INTRACRANIAL PRESSURE MONITORING COMPUTER SYSTEMS

Several computer systems have been developed for the specific purpose of clinical ICP monitoring. Takizawa et al [40] used a microcomputer to do ICP histogram analysis using a sampling rate of 3 samples per second and a histogram accumulation time of 5 minutes to 24 hours. The histogram was shown on an oscilloscope during the accumulation of data and recorded on paper when all the data were collected. A rudimentary form of ICP trending was also performed by running an analog chart recorder at slow speed. The system incorporated no alarm features. The slow sampling rate (3 samples per second) precluded any form of ICP morphology analysis.

Corbin et al [41] reported on the use of a Hewlett-Packard 9845 desk-top computer as an ICP monitoring/display station. Technical limitations precluded continuous sampling; therefore, as a compromise, the ICP was sampled at 100 samples per second for 14 seconds every minute. Both mean ICP and the ICP waveform could be displayed using keyboard control. Simple trends also could be detected by visual analysis of compressed ICP data. No alarm features were present.

Graham et al [42] described a specialized ICP computer system based on the Z80 microprocessor. A unique feature of the system is its ability to perform signal averaging using the QRS complex of the electrocardiogram as a trigger. The system was used to study

ICP waveform changes during hemorrhagic hypotension in animals and was not designed for clinical purposes.

Mason et al [43] describe an ICP monitoring system that integrates the ICP with other physiologic signals. Intracranial pressure, electroencephalogram, and capnogram (end-tidal CO_2) signals were entered into a computer via analog-to-digital conversion and transferred every 15 minutes into a database on disk along with a number of variables from the patient clinical examination. The system used thumbwheel switches rather than a keyboard to facilitate data input in a computer-naive environment. The investigators report that the system dispensed with almost all hand-written charting and saved significantly on nursing time. As this system was primarily intended for a database application, alarms, trending, and ICP waveform analysis were not provided.

Allen [44,45] describes the use of a PDP-11/34 minicomputer for online determination of ICP histograms. Data are sampled at 5 Hz, and histograms are constructed and presented automatically every half hour. The mean ICP and the percentage of readings exceeding 30 mm Hg for the period are also displayed. A special feature of this system is that those histograms obtained during patient physiotherapy are appended with a "physio flag" to indicate the possibility that artifacts may be present. No alarms were supported.

Anderson et al [1] describe a prototype, portable microcomputer-based system for monitoring and displaying ICP and related data. Intracranial pressure and arterial pressure data are followed digitally and processed to produce hardcopy graphs every 2 hours and up to 12 different ICP histograms, which can be displayed both graphically and in tabular form. Intracranial pressure alarms are supported and occur when the ICP exceeds an adjustable level for a specified duration. Trend data can be displayed on a CRT display. A special feature of the system is online determination of the cerebral perfusion pressure (the difference between the systemic blood pressure and the ICP) to provide a rough guide to the adequacy of cerebral blood flow.

COMPLICATIONS OF INTRACRANIAL PRESSURE MONITORING

While monitoring ICP has its advantages, these benefits are not without cost. As with any invasive procedure, the use of an ICP measurement device may be associated with an increased risk of infection. Aucoin et al [46] have found a higher incidence of ventriculomeningitis in patients with an ICP monitor compared with those with only a craniotomy. The intraventricular catheter was associated with the highest incidence of infection. The subarachnoid screw was reported to have the lowest infection rate. Aucoin et al also found that no infections developed in patients whose ICP monitor was left in place for less than 5 days. Those monitors used for 5 or more days were associated with a significantly higher risk of infection, regardless of the type of device. Similarly, Clark et al [47] observed a lower incidence of infection in patients if ICP was monitored for less than 72 hours. In contrast, however, they found no association between the type of device used and the risk of infection. Should prolonged monitoring be required, it is recommended that the original ICP monitor be removed and another inserted at a different site. Mayhall et al [48] found that this practice was not associated with an increased complication rate.

The effect of prophylactic antibiotics on the risk of infection is still uncertain. Some investigators have found them to be beneficial [49,50], while others have not [46,48].

Other complications of ICP monitor use are less common and include subdural empyema, brain abscess, superficial wound infection, CSF leak, hematoma, and cortical laceration [47].

EFFECT OF INTRACRANIAL PRESSURE MONITORING ON PATIENT OUTCOME

Many studies have shown an association between elevated ICP and outcome. Ponten [51] found a high ICP to correlate with poor outcome and suggested the use of ICP recording for guiding nonsurgical and surgical treatment of intracranial hypertension. Similar recommendations were made by Johnston et al [52] and Galbraith and Teasdale [53], who also stated that clinical and radiologic signs are not as reliable as ICP monitoring in determining ICP. Levene et al [54], working with a pediatric population, reported that the higher the ICP, the more likely that the patient would die or be severely handicapped. They found that monitoring ICP allowed appropriate attempts to treat intracranial hypertension, although only a few patients responded to treatment in their study. It was concluded that a knowledge of ICP was an additional factor in deciding on prognosis and further treatment plans.

Barnett et all [55[similarly found an association between lower ICP and ultimate survival, but stated that ICP control did not predict the quality of recovery. In contrast, Marshall et al [56] reported that ICP monitoring permitted early identification and treatment of raised ICP, resulting in a decrease in mortality and morbidity. Uzzell et al [57] also described that an elevated ICP in head-injured patients ultimately resulted in a

greater memory deficit than in those patients with normal ICP.

CONCLUSION

Although clinical ICP monitoring has been in use for over 30 years, its role in the management of the neurosurgical patient still elicits controversy. However, there is abundant anecdotal and scientific evidence supporting its clinical use, especially in guiding therapy in the head-injured patient. Therapeutic nihilists may argue that ICP monitoring does not always affect outcome; we are, however, more impressed by the fact that ICP monitoring may facilitate prompt intervention in situations in which aggressive early treatment favorably influences outcome.

Few of the ICP wave analysis methods described here are in common clinical use. Although part of the reason may be their complexity, an additional problem lies in the interpretation of the results that they present. We suspect that some time will be needed before any of these techniques will prove to be unequivocally useful, confining them largely to investigational settings. As computing hardware costs continue to drop, however, we expect further clinical evaluation of these methods.

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