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Significance of the Ventricular Fluid Pressure Wave Form in the Diagnosis of Cerebral Circulatory Arrest and Brain Death

By

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With 7 Figures

Summary

The fluctuations in the absolute value of the ventricular fluid pressure (VFP) with simultaneous changes in the amplitude and frequency of the oscillations of the ventricular fluid wave form are described in seven patients who developed brain death following either a head injury or a cerebrovascular accident, and are compared with those observed in nineteen patients who survived similar brain pathology. The findings in the two groups were significantly different. It is suggested that VFP monitoring does provide reliable evidence of brain death even while the patient is on artificial respiration.

Key words: Intracranial pressure recording—Ventricular fluid pressure wave form—brain death—diagnosis.

Introduction

The accepted medical definition of brain death applies to patients with arrested respiration due to irreversible brain damage but with intact cardiac function, the latter being preserved only by the use of mechanical ventilation. The clinical criteria for establishing brain death have been formulated and generally accepted as follows (Mohandas *et al.* 1971). Over a period of at least 12 hours there should be no spontaneous respiration for periods of 4 minutes at a time, and absence of brain stem reflexes. Provided that hypothermia, metabolic disturbances, and depressant drugs, including alcohol, have been excluded then brain death may be pronounced. Several laboratory tests have been

used, more to reinforce the clinical criteria of brain death than to supplant them; a summary of those most commonly used is presented in Table 1. The use of the EEG has been stressed by investigators, but there are examples of cases with isoelectric (flat) EEG tracings with very good recovery (Levin *et al.* 1966). Also a 24 hours EEG service is not

Table 1

No.	Investigation	Results
1	Electroencephalography (EEG)	Isoelectric tracing
2	Echoencephalography	Isoelectric tracing
3	Arteriovenous Oxygen differences	Absence of gradient
4	Bilateral carotid and vertebral angiogram	Non-filling of the intracranial vessels
5	Isotope measurements:	
	a) Isotope scintiphotography	Cold area
	b) Beta or gamma-emitting isotopes	Cold area
	c) Cerebral blood flow	Very low flow
	d) Rihsa (spinal subarachnoid space)	Absence of CSF flow
6	Neuropathological findings	Autolytic changes (Respirator brain)

available to most Hospitals. Angiographic and isotope investigations present problems in use as they require removal of the patient from the intensive care unit and are of limited value for making repeated measurements.

Intracranial pressure monitoring may be of value in the prognosis of brain damage. High pressures have been found in the few cases of brain death in which the ICP has been measured (Quaknine *et al.* 1973). However, there are a few examples in the literature of patients with severe head injuries who have survived in spite of high ICP. In other words the absolute level of ICP may be indicative of severe brain injury but cannot alone be used to establish the diagnosis of brain death. Our experience suggests that the wave pattern of the recording may correlate with changes in intracranial cerebrovascular dynamics and could be used as a reliable criterion of arrested cerebral circulation. This could be of great value in those patients with brain injuries in whom although there are signs of severe damage, there may still be a good chance of survival. At present these patients are put on long term assisted respiration either because controlled ventilation is used for their better management, or because they cannot maintain adequate spontaneous ventilation. Accurate clinical examination is impossible as long as the patient remains paralysed, and since there are thus no clinical signs of brain

death other laboratory tests are not applicable. It is suggested that monitoring of the ventricular fluid pressure wave form could provide reliable evidence of arrested cerebral circulation and therefore of brain death.

Material and Methods

Five males and two females, aged 12 to 48 years, sustained severe brain damage, five from closed head injury and two following cerebrovascular surgery. Their neurological conditions before being put on ventilation, duration of ventilation, the times of monitoring, and fluctuations of the main ventricular fluid pressure (VFP), and the necropsy findings are summarized in Table 2. The VFP was measured through a rigid transparent polyethylene ventricular catheter with a Bell & Howell physiological pressure transducer (type 4-422-0001/2) and recorded at constant amplification by a two-channel felt pen recorder (type 2800, Bryans Southern Instruments Ltd.) for a period of three to eight days. The hydraulic zero and the liquid continuity of the fluid in the tubing system to the transducer's pressure chamber were regularly checked. Postmortem examination was done in five cases.

A further seventeen cases of brain injuries who survived have been studied during the last 13 months. Recordings of their VFPs were used for comparison with those of the above seven patients.

Results

During the course of the ventricular fluid pressure recording our seven patients developed moderate to high resting pressure and several A-waves, the plateau levels of which were between 80–110 mmHg. Usually, and as we observed in the recordings of the survival group, following an A-wave the wave form returned to its pre-plateau appearance with regard to amplitude, frequency, and usually the pressure level (Fig. 1). However, in our cases following some A-waves the VFP fell below the abnormal resting level; the oscillations were less discrete and the amplitude showed a slow but definite decrease (Fig. 2). In Figure 3 we can see the continuous decline of the VFP curve until it reaches a low level and after a period, which ranges from one to six hours, the VFP starts rising again to a level above the abnormal pre-plateau resting value. It then maintains this level (except in case No. 2, where the VFP soon fell below the resting pressure and remained there without any change in the following three hours of monitoring), and *no more A waves occur* (Fig. 4). Figs. 2, 3, and 4 are taken from the same patient. Towards the end of the last A wave we were unable to detect the diastolic pressure with the sphygmomanometer, whereas the systolic pressure was easily registered. A low diastolic pressure was detected only when the VFP started to fall (Fig. 6). Fig. 5 shows a recording made during the second day after the last A wave. Compression of the

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Table 2

No.	Age, Sex	Cause of injury	Clinical condition after the injury and before artificial ventilation	Days on respirator	ICP monitoring mmHg	days	Autopsy findings
1	33, M	Cerebrovascular surgery (ruptured aneurysm)	Postoperatively responding to simple commands and moving all four limbs. Reflexes present, with brisk reaction of pupils to light. Respiration and circulation within normal limits	6	50—80	6	PM not permitted
2	13, F	Head injury	Unconscious. Responding to painful stimuli in a decerebrate fashion, with extensor spasms in all four limbs. Pupils not reacting to light	4	30—110	4	Ventilator brain
3	21, M	Head injury	Unconscious. Upper limb extensor rigidity. Lt pupil reacting to light sluggishly	5	70—100	5	Ventilatory changes
4	26, M	Head injury	Unconscious. Responding to painful stimuli with extensor spasms in all limbs. Rt pupil reacting to light. Laboured respiration. On the 6th day of assisted ventilation drugs were reversed and patient was able to maintain spontaneous respiration. Blood gases within normal limits. Because of facial twitching, which was observed when the patient was ready for discharge from the intensive care unit, the patient was reventilated for another two days	8	50—85	8	Early ventilatory changes

| 5 | 27, M | Head injury | Unconscious. Lt pupil reacting sluggishly. Slight increase of muscle tone in the upper extremities after stimulation. Adequate respiration | 3 | 50—80 | 3 | PM not permitted |
| 6 | 48, F | Cerebrovascular surgery (ruptured aneurysm) | Postoperatively confused, pupils reacting to light. No movements on the right side observed. Spontaneous respiration | 6 | 70—80 | 6 | Respirator brain |
| 7 | 12, F | Head injury | Decerebrate extensor movements in all four limbs. Both pupils reacting sluggishly. Ventilated. On the 3rd day she was allowed to breathe spontaneously. Accidental blockage of ventricular drainage with concomitant raised ICP and laboured respiration. Reventilated. Never recovered | 6 | 70—100 | 6 | Ventilatory changes |
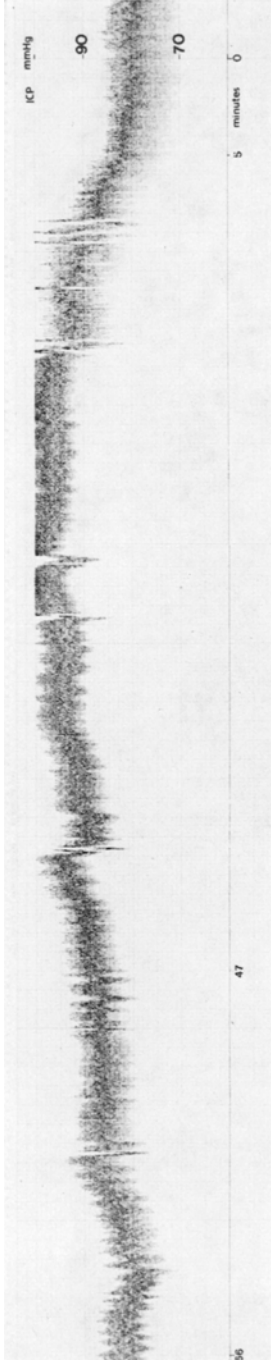
Fig. 1
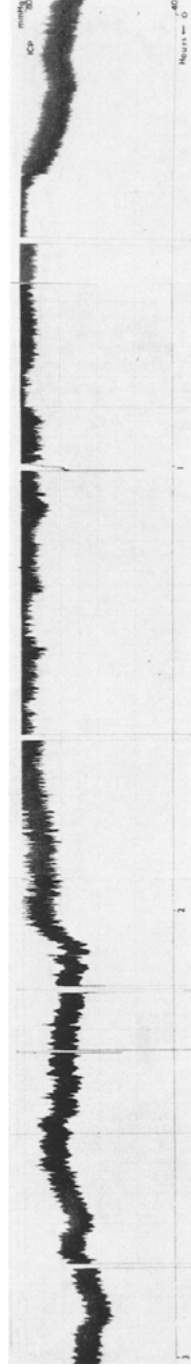
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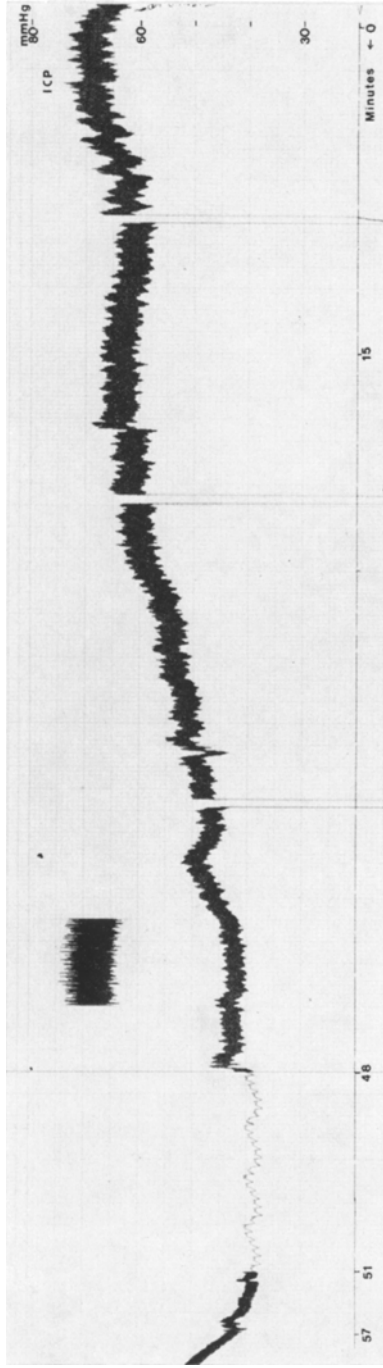
Fig. 3. Arrows represent times during which the ventricular tube was open to free drainage. Above the tracing a preplateau sample of the VFP wave form

external jugular veins does not alter the VFP. The injection of a minimal amount of saline into the ventricle immediately raised the VFP enormously, a clear indication of the critical volume/pressure relationship within the cranium in these circumstances. This procedure did not alter either the amplitude or the frequency of the oscillations, in complete contrast to the VFP recordings in the group of patients who survived. The VFP is not altered by compression of the external jugular veins or by changes in the position of the head from the resting to the upright position (an alteration of about 30°), provided that the hydraulic zero remains unchanged.

B waves of a constant frequency of 1½ per minute are observed in Fig. 5. When the respirator was disconnected B waves disappeared,
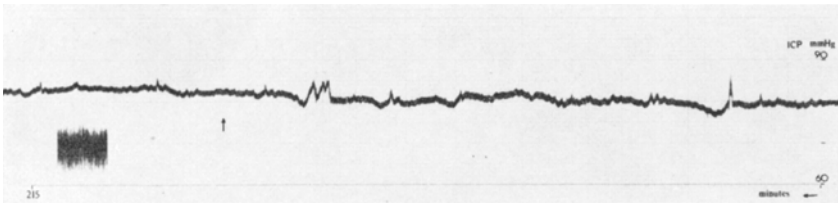

Fig. 4. Arrow below trace shows time of compression of the external jugular veins. A pre-plateau sample of the VFP wave form below tracing on the left. (We had to alter the zero level to — 20 mmHg on the paper chart in order to increase the upper limit of the recording from 80 to 100 mmHg)

and the patient made no respiratory effort. We are also able to see the fine and shallow Hering–Traube–Mayer waves at a constant subnormal frequency of 13–14 per minute.

All the five patients who underwent necropsy showed early to advanced ventilator brain changes (Walker, E. et al., 1975).

Discussion

Cerebrospinal fluid pulsations are thought by most investigators to originate from the arterial pulse and to contain a respiratory component. The main site of transfer of the arterial pulsation is regarded by some as being the basal and spinal arteries (Antoni 1946, Dunbar et al. 1966) and by others as the ventricular choroid plexuses (Bering 1955). The amplitude of the ventricular CSF wave form may be altered by variations in intracranial pressure (Ryder et al. 1952). CSF pulsation in thy presence of normal or slightly raised ICP is reduced by compensatory mechanisms such as CSF displacements, reduction or cessation of CSF production, alteration in cerebrovascular tone and blood volume, and by dural distention. Progressive elevation of the ICP results in sequential
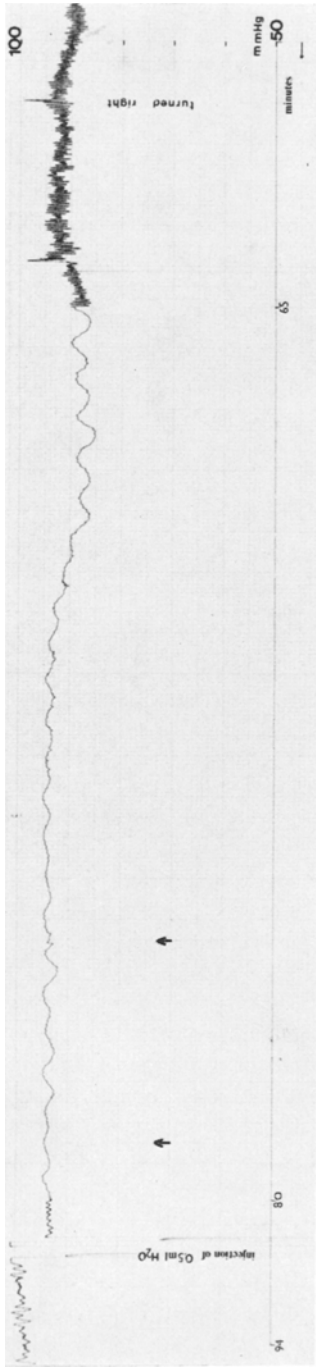
Fig. 5. Arrows below VFP tracing show a momentary compression of the external jugular veins. (The speed of the paper was increased from 5 mm/min to 0.5 mm/sec in order to clarify all the waves in the tracing)
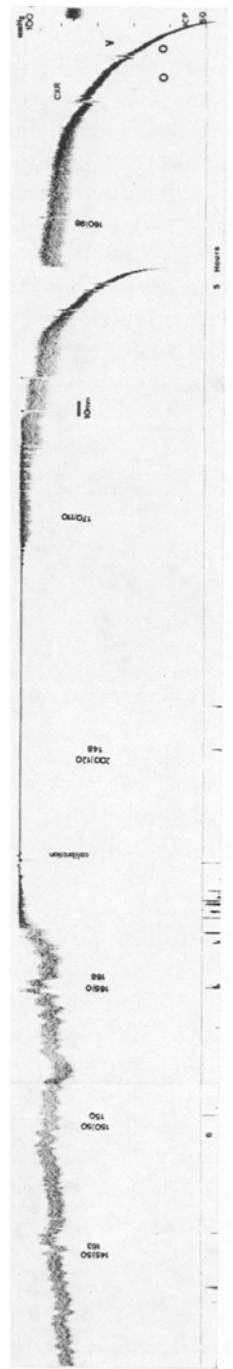

Fig. 6

exhaustion of these compensatory mechanisms, thus abolishing their damping effects on CSF pulsations. The amplitude increases and the wave form resemble more the arterial pulse.

Lundberg suggested that the plateau or A waves might be produced by rapid and reversible changes in the cerebral blood volume or by intermittent blockage of the CSF pathways, but he did not exclude the possibility that they might be due to water exchange into the brain tissue. Our patients with prolonged intracranial hypertension must have lost their buffering mechanisms. It has been reported that the CSF volume is the first to be lost, initially by displacement into the spinal subarachnoid space and later by diminished production, since this is pressure dependent (Shapiro, H. 1974). Sudden blockage of the CSF pathways raises the VFP gradually. The sudden and large changes in
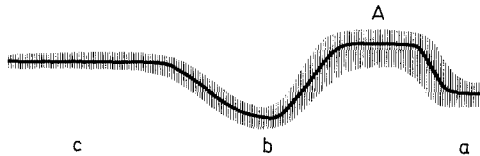

Fig. 7. *a* pre-plateau recording. *A* plateau-like wave. Main fluctuations of the VFP curve with regard the mean VFP and its amplitude

the VFP during an A wave could not be explained on the ground of increased vascular permeability and subsequent fluid exchange with the brain substance. The development of A waves in our cases, is thought to be due to changes in the cerebrovascular blood volume. This is reinforced by the changes in the wave form of the VFP, which occur following a plateau-like wave. With these changes CSF pulsation is diminished, and it is postulated that this reflects such a severe slowing of the cerebral circulation that brain death is inevitable. These changes in the VFP are summarized in Fig. 7.

What is the reason for the decrease in the VFP below the abnormal resting pressure level? Langfitt, T. W., *et al.* in 1969 have shown a diffuse collapse of the cerebral vessels, including the sinuses and the large arteries, when intracranial pressure equalled the mean arterial pressure (MAP). It is suggested that, during the increase in the VFP of an A wave, the pressure may exceed the MAP and by compression of the vessels blood would be displaced into extracranial channels. Since the volume/pressure relationship is so critical, a small amount of displaced blood could produce a significant fall in the VFP.

The subsequent rise of the VFP, which occurs over the following one to six hours, is, we suggest, due to fluid exchange within the brain tissue. Increase of the tissue osmotic pressure could conceivably occur

as a result of wide spread leakage of particles from damaged cells of all types (Gordon 1976). Fluid would migrate from the intravascular to the extravascular space until the hydrostatic equals the osmotic pressure when the flow would stop. The rigid low amplitude VFP tracing, which is observed in Fig. 4 and 7 (c), may be explained by the established equilibrium between the hydrostatic and osmotic pressures.

We are not able to explain the disappearance of the diastolic radial pressure when measured by a sphygmomanometer towards the end of the last A wave, or its reappearance soon after the A wave is over. Direct measurement of the arterial pressure may be of value in interpreting these events.

In general, it would be particularly interesting to relate the changes in the VFP wave form to cerebral blood flow and perfusion pressure.

The occurance of B waves appears to be due to ventilation and is of no value in the diagnosis of brain death.

The shallowness and slowness of Traube-Hering-Mayer waves offer further evidence of the failure of the cerebral circulation.

The presence of advanced autolytic changes in the brain at necropsy suggests that brain death occured long before its clinical diagnosis. Neuropathologists are unable to time precisely the onset of those autolytic changes; it is thus not possible to correlate the onset of autolysis with our VFP recordings. Case 4, which had an eight day course of VFP monitoring, was shown to be alive by reversing the relaxants and sedatives during the sixth day of ventilation. He was reventilated for another two days in which he presented the above-mentioned changes in the VFP recording. Early ventilatory changes were noted at necropsy.

Neurosurgeons often disagree about how long one should persist with mechanical ventilation of cases with severe brain damage. In Edinburgh the minimum duration is 48–72 hours, which is increased in young patients who show evidence of very severe brain injury; but the more severe the brain injury the more likely the patient is to succumb soon after the injury. As soon as the changes in the VFP described above are observed, we suggest that patients on the respirator should have their drugs reversed and be given a thorough neurological examination, in which the clinical criteria of brain death should be applied. This would lessen the strain on medical and nursing staff, would release facilities which might be better used, would reduce the suffering of the relatives, and would also speed up kidney donation.

Acknowledgement

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