THE MONITORING OF INTRACRANIAL PRESSURE

IN INFANTS AND CHILDREN

by

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DEDICATION

This thesis is dedicated to my wife

Janet Rosemary

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ACKNOWLEDGEMENTS

ABSTRACT

One hundred cases of children in Edinburgh who underwent ventricular pressure monitoring for diverse indications have been discussed in detail and examples of their pressure tracings illustrated. This and other studies have now established that I.C.P. monitoring is a useful clinical tool.

The technique employed has been justified from both a theoretical and practical standpoint and other methods of monitoring I.C.P. reviewed from the medical literature with their relative advantages and disadvantages. There is a place for I.C.P. measurement in suspect neonates by means of fontanometry; in hydrocephalic children by monitoring from ventriculostomy reservoirs; in head injury by way of epidural or subarachnoid space; in childhood encephalopathies and other causes of brain swelling by epidural monitoring and for the continual assessment of the child in whom R.I.C.P. may be an on-going problem by means of a teletransducer.

This work has afforded me the opportunity also to report related areas of interest, firstly the effect of I.C.P. on monoamine metabolites in children's C.S.F. with little correlation being found by a probable maturational increase in one metabolite. Secondly, the effect of various types of seizures increasing the I.C.P. had not been previously reported and thirdly, the fascinating effect of sleep in I.C.P. with cyclical pressure variations occurring in all cases and exaggerated pressure responses and wave forms when there is existing R.I.C.P. I have put forward an hypothesis for this sleep effect based on pressure and polygraphic recordings.

Lastly, the chapter on anaesthesia adds some weight to other studies
that caution the use of ketamine in cases of R.I.C.P., but confirms
its use otherwise. These studies have however raised doubts about its
convulsant action and I have shown the previously overlooked effect
of intubation on I.C.P. and described a momentary pressure reflex
associated with spreading the vocal cords, which together with the
sleep effect, may be related to the Sudden Infant Death Syndrome.

CHAPTER 1

INTRODUCTION

REASONS FOR MONITORING INTRACRANIAL PRESSURE

AIMS OF THESIS

ABBREVIATIONS

CHAPTER 1

INTRODUCTION

"Once a serious study of the cerebrospinal fluid is begun, we find that we have opened a veritable Pandora's box of problems that take us far away from our original questions of how is the fluid formed and where is it drained?"   (Davson 1972)

Raised I.C.P. together with status epilepticus and the meningoencephalopathies constitute the major acute paediatric neurological emergencies. It is of importance therefore to the paediatrician in hospital practice, and until relatively recently, the means of recognising raised I.C.P. were purely clinical. In recent years however, there has been an accelerated interest in the non-invasive and invasive techniques of estimating I.C.P. in childhood.

REASONS FOR MONITORING INTRACRANIAL PRESSURE

I have considered it necessary to monitor I.C.P. principally because it permits better management of the individual patient with intra-cranial hypertension but more specifically because:

(a) R.I.C.P. may be difficult to recognise clinically, i.e. the signs may be non-specific, false localising or even absent in the presence of R.I.C.P.

(b) Detection of those patients with normal I.C.P. will spare them unnecessary and often hazardous costly treatments.

(c) It is important to detect early rises in I.C.P. because identification permits immediate intervention, preventing rapid and often fatal complications.

(d) Evaluating the efficacy of drug regimes or techniques that may lower I.C.P. is made possible on a scientific basis.

(e) It allows avoidance of harmful procedures to the individual patient, e.g. water based enemas, positioning etc.

(f) The initial great improvement in mortality and morbidity resulting from the use of shunts in childhood hydrocephalus has distracted from some fundamental issues, namely more precise criteria for insertion of and removal of particular types of C.S.F. shunting devices (Hagberg & Naglo 1972). C.S.F. shunting systems carry a very significant morbidity and mortality (Hemmer 1971, Forest & Tsingoglou 1968, Nicholas et al 1970, Noble et al 1970) and it is known now that if a child can cope without a shunt his quality of life will be vastly better (Lorber 1972).

(g) All known C.S.F. shunting devices are liable to mechanical failure and it is not always easy to detect malfunction clinically.

To take the first of these reasons for monitoring I.C.P., the difficulty of clinical recognition of R.I.C.P. in some more detail. The signs and symptoms may lack specificity, for example, headache occurs in numerous pathological situations, and in the toddler age group is usually expressed as irritability and anorexia. Vomiting is another non-specific symptom and even when projectile can occur in a number of neonatal and infantile conditions. Combinations of signs and symptoms however are more specific, e.g. the Cushing triad of headache, vomiting and papilloedema is virtually synonymous with R.I.C.P. in the presence of normal systemic pressure.

The anterior fontanelle varies considerably in size in the normal newborn (Popich & Smith 1972, Tan 1976) and can be observed bulging, secondary to increased venous pressure, in neonates with congestive

cardiac failure without any evidence of cerebral disease. It also distends with tetracycline therapy (Raju 1976) occasionally in cystic fibrosis and vitamin A disturbances (Abernethy 1976) and galactosaemia (Vogel et al 1976). The anterior fontanelle may be very small not permitting reliable clinical assessment of the I.C.P.

Of the more chronic symptomatology of R.I.C.P. such as diminished school performance, behavioural problems, emotional lability etc. a myriad of differential causes are possible.

Unusual signs such as pulmonary oedema secondary to excessive autonomic discharge of central origin (Ducker et al 1968, 1969) do not in themselves readily suggest any abnormality of intracranial dynamics.

Papilloedema takes time to develop and although a highly reliable sign when present, it is present in the paediatric situation in less than 50% of cases of R.I.C.P. (Brown et al 1973). Therefore the possibility of R.I.C.P. is not excluded by its absence or the absence of any fundus abnormalities. Also, papilloedema may be confused with:

1. Optic papillitis or neuritis (visual acuity and visual field screening for differentiating is also difficult in young children)

2. Pseudopapilloedema or congenital blurring of the disc is a variation of normal but resembles pathological papilloedema. It may be familial (Fite & Lewis 1966) or occur with other neurological abnormalities with normal I.C.P. e.g. microcephaly, Wildervanek's syndrome, Duane's syndrome (Kirkham 1969)

3. Drusen or hyaline bodies in the optic discs if located in the depths of the nerve head, make differentiation from papill-

oedema difficult and gives rise to congenital blurring of the discs (Walsh & Hoyt 1969)

4. Tilted Disc Syndrome another congenital anomaly which may be confused with papilloedema.

5. Hypertensive Retinopathy — confusion may still arise, despite the knowledge that narrowing and segmental constrictions of the arteries, soft exudate and diffuse haemorrhages are features most likely in this condition.

6. Central retinal vein thrombosis.

There are then the 'false localising signs' which suggest falsely, that there is a focal lesion in a child who has in fact R.I.C.P. The commonest of these is the sixth cranial nerve palsy, but other false signs include anosmia, fifth cranial nerve dysfunction, bitemporal hemianopsia. In fact most of the signs, e.g. hemiparesis, related to internal herniations, i.e. tentorial, cerebellar or cingulate, may appear on presentation and obscure the underlying condition, namely R.I.C.P.

There is a great variation in the expression of signs of pressure from patient to patient and from age group to age group. This is due firstly to the differing aetiologies of R.I.C.P. and secondly to greater buffering or adaptation to raised pressure in the infant compared to the older child, i.e. as well as C.S.F. displacement from the cranium there is also an expansile cranial vault. Traditionally headache, vomiting and papilloedema mean R.I.C.P. as mentioned above and in the adult other signs such as bradycardia, intermittent pupillary dilatation, decerebration, respiratory changes etc. are more related to brain shifts and distortions (Miller & Adams 1972, Thompson & Malina 1959) than to specific pressure levels.

These signs are however very common in children with R.I.C.P., suggesting that children have a much more mobile and maleable brain than adults.

It is obvious from the above that neurological signs are not specific for raised intracranial pressure and that elevated pressure may be present in the absence of such signs (Guillaume & Janny 1951).

Over the last 20 years monitoring of physiological parameters in critically ill children in an intensive care unit has improved the management and outlook for such children. In the same manner, since R.I.C.P. disturbs C.N.S. homeostasis, monitoring of the I.C.P., C.B.F., C.M.R. etc. will help to make management more scientific and successful. It should be stressed that measurement of these physiological parameters is an adjunct to management and should not at any time preclude detailed neurological and system examinations or nursing observations. Any child therefore with a rapidly advancing O.F.C., seizures, coma, decerebration or loss of homeostasis should immediately raise the suspicion in one's mind of R.I.C.P. and although it may not be the underlying cause of the illness, it deserves treatment in its own right.

AIMS

The aims of this thesis are to report and illustrate the observations on ventricular pressure recordings performed on children in Edinburgh since late 1975. It is also my aim to justify the technique employed and to review other methods of measurement of I.C.P. I shall discuss the clinical usefulness of V.P.M. from its use in 100 patients and hypothesise on the results of these monitorings. The influence of I.C.P. on brain amines and the influence of seizures, sleep and anaesthesia on I.C.P. are discussed in the light of original

observations and reports from the medical literature.

ABBREVIATIONS

A.T.N.R.	asymmetrical tonic neck reflex
A.P.H.	antepartum haemorrhage
B.P.	blood pressure
C.P.P.	cerebral perfusion pressure
C.S.A.S.	cortical subarachnoid space
C.S.F.	cerebrospinal fluid
C.S.F.P.	cerebrospinal fluid pressure
C.B.F.	cerebral blood flow
C.V.P.	central venous pressure
C.M.R.	regional cerebral metabolism
C.R./C.R.A.	cardio-respiratory artefact
E.E.G.	electroencephalogram
E.C.G.	electrocardiogram
E.D.D.	expected date of delivery
H^+	hydrogen ion concentration
HCO_3	bicarbonate concentration
I.M.	intramuscular
I.V.	intravenous
I.T.	intrathecal
I.C.H.	intracranial haemorrhage
I.V.H.	intraventricular haemorrhage
I.C.P.	intracranial pressure
L.A.E.G.	lumbar air encephalogram
L	lumbar
L.P.	lumbar puncture
Non-R.E.M.	non-rapid eye movement sleep

O.F.C.	head circumference
P.E.T.	pre-eclamptic toxaemia
pH	partial pressure of hydrogen ions
pCO_2	partial pressure of carbon dioxide
R.E.M.	rapid eye movement sleep
R.I.C.P.	raised intracranial pressure
S	sacral
S.T.N.R.	symmetrical tonic neck reflex
S.A.P.	systemic arterial pressure
S.V.D.	spontaneous vertex delivery
T	thoracic
Torr	unit of pressure (1 torr = 1 mm Hg)
V.F.P.	ventricular fluid pressure
V.P.R.	volume, pressure response
V.A.E.G.	ventricular air encephalogram
V.P.M.	ventricular pressure monitoring

CHAPTER 2

METHODOLOGY

RECORDING EQUIPMENT

A Standard recording equipment

    (i)   Transducer
    (ii)   Transducer control unit
    (iii)   Physiological pen recorder
    (iv)   Head apparatus
    (v)   Calibrating and operating procedure

B (i)   Alternative recording equipment
   (ii)   Example of data notation during case undergoing
              a polygraphic recording

PREPARATION FOR RECORDING

METHODS OF RECORDING VENTRICULAR PRESSURE

SOURCES OF ERROR IN RECORDINGS

CHAPTER 2

METHODOLOGY

RECORDING EQUIPMENT

A   Standard Recording Equipment

(i)   Luer Fitting Pressure Transducer 3EA-a

This unit designed for measuring physiological pressures
outside the body features a Gaeltec 3EA sensor fitted within an
electrically isolated titanium shell tapered to match standard
Luer fittings. It is supplied with a lock nut, also of
titanium.

The robust metal diaphragm is protected by a layer of touch
silicone flush with the end of the tapered shell. The diaphragm
itself is only 1.5 mm in diameter and the volume displacement
under applied pressure is very small. Since this is in general
much less than that of a connecting catheter, the transducer
itself does not usually restrict the overall frequency response
of the system.

The transducer is supplied fitted to a sheathed cable, 3 mm in
diameter and 1.3 m long (Fig. 1). The linear pressure range
is from -30 mm Hg to +300 mm Hg. The sensitivity is
1.2 mv/v/300 mm Hg nominal. The compensated temperature range
is $15^{\circ}C$ to $40^{\circ}C$, that is, it has a wide operational temperature
range with small temperature errors. There is low linearity
and hysteresis error, a good frequency response. A substantial
output, typically 15 mv with a 10 volts supply, is even suitable
for under-water use. The overall length of the Luer body is
2.5 cm, the overall diameter, including the lock nut, is
9.5 mm. The diameter of the Luer taper at the sensor is 4 mm.

Fig. 1
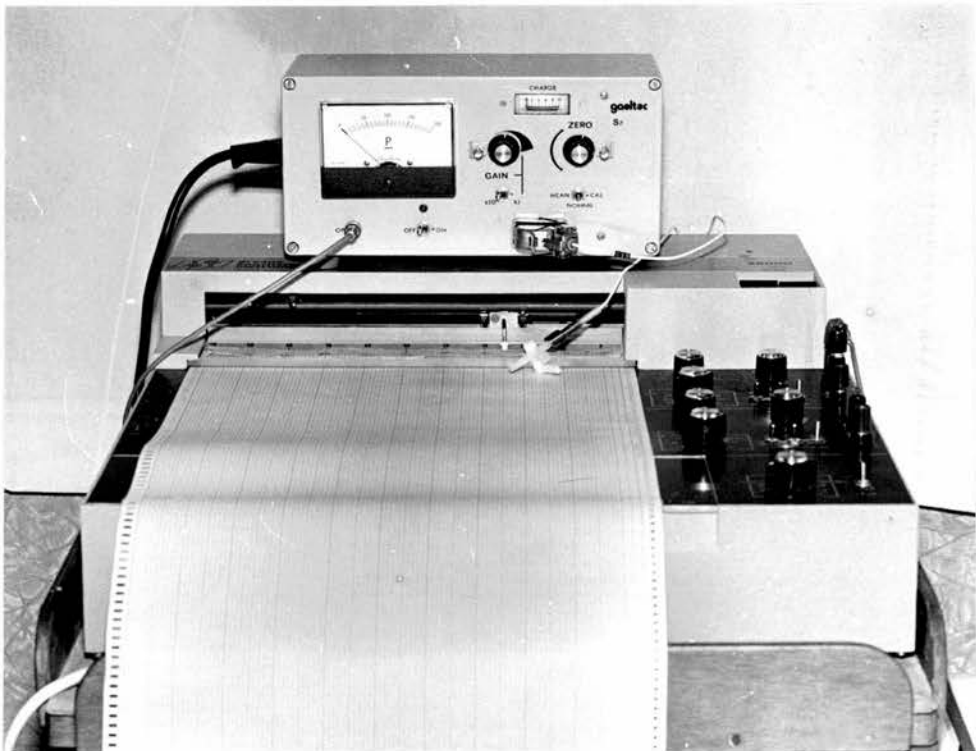
Fig. 2

The transducer is light in weight.

(ii)   <u>Transducer Control Unit 57</u>

This unit features AC excitation of the transducer bridge,
and is used for applications where the best stability of
zero is required over long periods. It is particularly useful
for physiological measurements in conjunction with Gaeltec
transducers. The unit operates from internal batteries for over
a week before it is necessary to recharge by connection to an
AC mains supply and it gives a 1 volt output for a recorder
from a transducer input of as little as 0.07 mv/volt. The
output as well as the state of battery charge is displayed
on separate meters and lockable controls are provided for 'gain'
and 'zero'. The instrument is housed in a moulded polyester case,
9" x 4" x 3" in size and is fully portable. It gives a full
response to pressure changes up to 250 Hz in frequency in its
normal mode, with a heavily damped output also available to give
mean pressure readings when required.

(iii)   <u>Physiological Pen Recorder (Bryan's Southern Instruments,
28000 series)</u>

This is a 2 channel, direct pen recorder and the equipment records
changes in input signal level, against time, by means of a
servo driven pen writing upon a moving chart paper. The pen is
driven by a 'stepper motor' which advances the paper at constant
speed, determined by the 'CHART SPEED' control. (Fig. 2 shows
standard recording equipment with transducer control unit and
pen recorder.)

(iv)   Stabilising Helmet

To facilitate a constant needle position and to allow more
freedom of movement for the older child undergoing pressure
monitoring, a light metal head piece was constructed, under
guidance, by the Bioengineering Unit at the Princess Margaret
Rose Hospital, Edinburgh.

It can be adjusted to suit a variety of head sizes with
expandable side pieces, and maintains the pressure transducer
in a constant position to the Huber needle or cannula. It
protects against needle movement, which could result in damage
to the reservoirs or focal cortical damage and alleviates the
need for a nursing attendant to hold the 'flex' throughout
the period of monitoring. Because of its 'galactic' appearance,
the children seem to accept it readily (Figs. 3 and 4).

(v)   Calibrating and Operating Procedure for Gaeltec
        Transducer, Gaeltec Transducer Control Unit and
        Bryan's 28000 Physiological Recorder

        1.   Set up the transducer control unit (preamplifier)
first - the range switch must be kept at X10, as X1 would
result in a very low reading and would be applicable only for
a different pressure transducer.

The 'mean switch' is only for very fast activity and gives a
readout of the mean level of the signal being received. It is
similar to the 'filter' on the recorder and is therefore not
necessary. For routine use throughout this series, the switch
remains on 'normal', both for calibration and recording. The
'cal' switch is for special Gaeltec transducers only and is
not applicable here.

Fig. 3

Fig. 4

With the switch on 'normal', set the zero, apply pressure to the transducer by means of a standard mercury or aneroid sphygmomanometer, adjust the gain control to a rough setting, release the pressure, readjust the zero and repeat the whole procedure a number of times, thereby obtaining fine control. Seal the zero and gain control.

Set up the recorder

Leave the manometer attached for ready checking of pressures prior to each new patient.

(a) Select the zero point on the chart, with the 'pen offset' control while the 'input' – 'output' switch is 'off'.

(b) Put the 'input' switch to 'on'.

(c) Amplifier and recorder zero should remain at the same point.

(d) Keep the 'range' switch on 2.5 volts to read –20 mm Hg to +80 mm Hg. However, this can be changed to 1 volt range if scale 0–100 mm Hg is required.

(e) Now calibrate the recorder to the amplifier by adjusting the 'range' control to match the amplifier dial (with the 'cal – var' switch on 'var') and, when back to zero, adjust the 'pen offset' (with the 'input/off' switch again to 'off'). It is probably not necessary to re-zero here as it should not move.

(f) Keep the filter in the recorder at all times.

(g) Allow maximum of 1 mm of error when checking the calibration.

(h) When the 'cal - var' switch is set to 'var'
(i.e. a variable range) this switches the range
control to provide variable sensitivity. When set
to 'cal', this switches the range control out of the
circuit to provide parallel calibrated sensitivities.

B (i) Alternative Polygraphic Recording System

The Department of Medical Physics and Medical Engineering at the
Royal Infirmary of Edinburgh, modified a Galileo E.E.G. machine.
They constructed an 'interface unit' and fed the input signals
of respiration, E.E.G., E.C.G. and ventricular pressure into a
tape recorder (Fig. 5). This meant that a child could have an
overnight or 24 hour recording of various physiological
parameters. Later, on an oscilloscope, one could play back all
channels simultaneously at real time or at an advanced speed.
It also provided the possibility of synchronising events
with ventricular pressure recordings. A block diagram is
included to show the connections of this system (Fig. 6). A
further diagram is included which shows the modifications of
the Galileo E.E.G. machine (Fig. 7). The machine has been
modified so that it can be used either entirely as an ordinary
E.E.G. machine or for these polygraphic recordings. Examples
of these polygraphic types of recording can be seen in cases
62 and 68.

(ii) An Example of Data Notation necessary during a
Polygraphic Record measuring Ventricular Pressure
simultaneously with E.C.G. and E.E.G.

NAME: J.D.

AGE: 10 years

DATE OF RECORDING: 20.2.78

PATIENT POSITION: Supine with head and eyes front
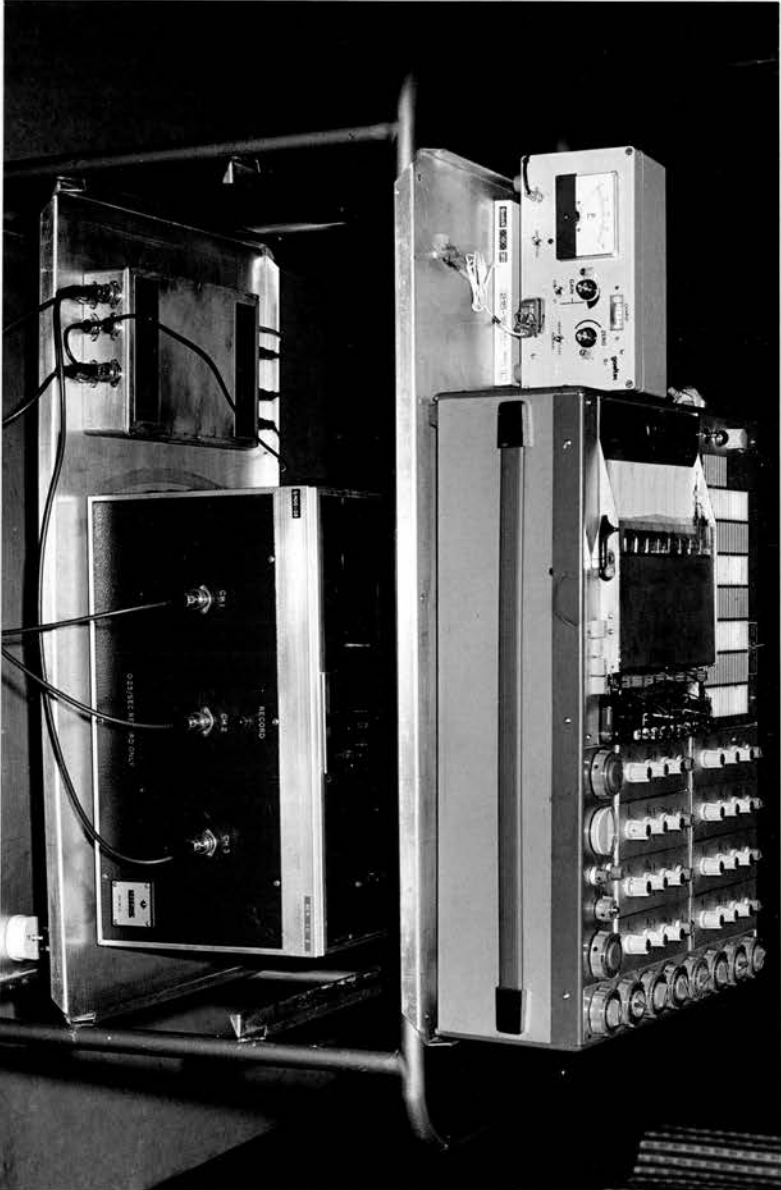
Fig. 5
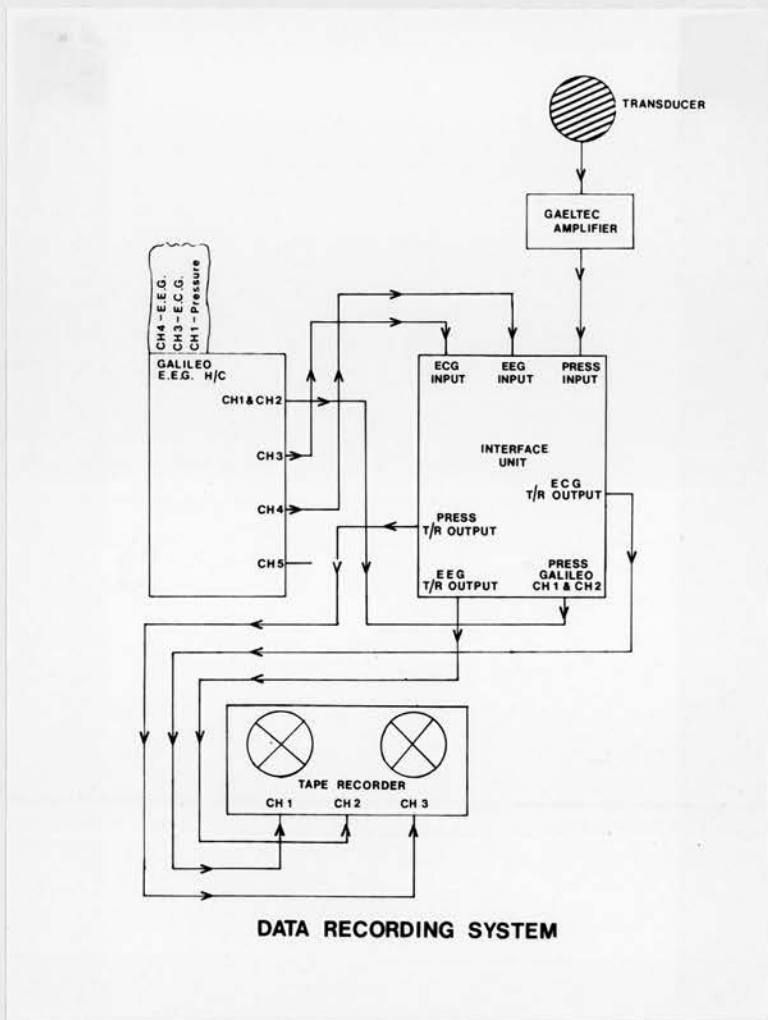
Fig. 6
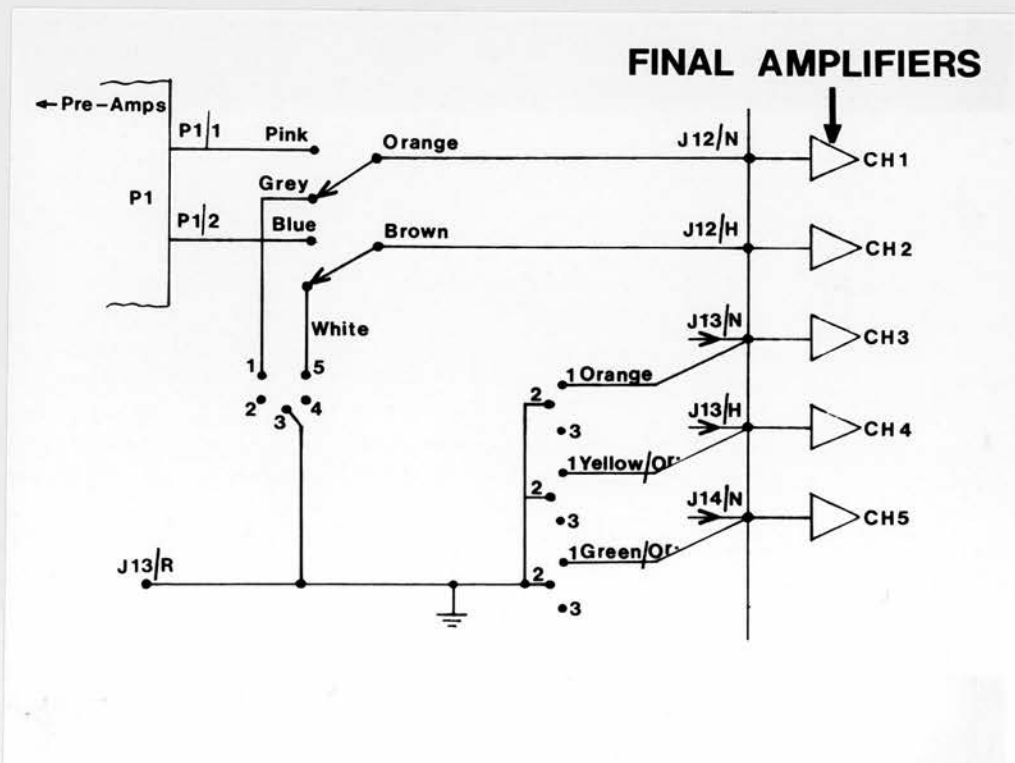
Fig. 7

ZERO PRESSURE: 1½" above upper cortical subarachnoid

space.

Channel 1 — ventricular pressure

Channel 3 — E.C.G.

Channel 4 — E.E.G.

TIME	FOOTAGE	ACTIVITY
3.45 p.m.	0004	Zero pressure
3.47	0007	Pressure 50 mm Hg inserted
3.49	0010	Calibrate E.E.G. with 100 microvolts
3.50	0012	Ventricular pressure record commences
4.06	0034	Valsalva manoeuvre
5.16	0123	Having a meal
5.25	0134	Laughing
6.02	0184	Eating a biscuit
6.17	0203	Talking
7.20	0271	Face washed
7.25	0274	Talking
7.30	0277	Reading
7.40	0283	Child holds breath while Nobecutane applied
9.25	0390	Chatting
9.30	0406	Tidying bed
9.40	0413	Laughing
9.50	0421	Drinking
10.05	0424	Recalibrating pressure transducer
10.07	0429.5	Pressure transducer reconnected
10.16	0437	Beginning to get tired
10.20	0440	Yawn, eyes closed
10.21	0441	Movement of arms with eyes closed

TIME	FOOTAGE	ACTIVITY
10.22	0442	Yawn, eyes closed, movements of face
10.24	0444	Eyes closed, neck flexion movements
10.26	0444.5	Yawn
10.30	0449	Lips smacking in early sleep
10.34	0451	Early sleep and beginning to snore.
10.35	0453	Swings of ventricular pressure at this time from 5-20 mm Hg.
10.36	0455	Snoring
10.37	0455.5	Scratching
10.40	0458	Eye movements
10.40.5	0458.5	Myoclonic movements of the little finger with pressure swings from 4-10 mm Hg and noisy respiration
11.02	0477.5	Deeply asleep now, ventricular pressure from 3-9 mm Hg.
11.13	0487	Scratching his nose, stiffened a little. Head movements and lip movements.
11.17	0491	Rubbed his nose in sleep and stirred slightly.
11.28	0506	Rubbed nose in sleep.
12.00	0533	Rubbed nose and yawned, eyes opened.
12.08 a.m.	0540	Moved legs and hands, yawned.
12.10	0542	Rubbed nose
12.12	0547	Moved legs
12.25	0553	Moved hand
12.35	0562	Moved legs and hand
12.40	0565	Moved legs

TIME	FOOTAGE	ACTIVITY
12.45	0572	Legs jumped
12.50	0574	Moved feet
12.53	0576	Moved legs, feet, head and hands.
12.55	0570	Moved feet
1.05	0584	Moved feet and legs
1.10	0589	Moved arms
1.15	0591	Moving legs
1.18	0595	Yawned
1.30	0605	Scratched ear
1.45	0616	Scratched nose
2.00	0629	Moved foot
2.04	0631	Moved foot and legs
2.05	0632	Moving legs
2.09	0634	Moving lips
2.15	0640	Moved left hand
2.16	0641	Yawned
2.20	0643	Yawned, scratched nose
2.20	0644	Moved feet and legs
2.26	0644	Moved left hand, yawned
2.30	0649	Yawned
2.31	0651	Yawned, moved legs, arms.
2.37	0656	Yawned, moved left hand and feet.
2.40	0656	Moved left hand
2.45	0662	Moved legs
2.47	0664	Yawned, moved head, shoulders and arms
2.55	0669	Stretched
2.57	0670	Moved legs and rubbed eyes

TIME	FOOTAGE	ACTIVITY
3.00	0672	Moved hand
3.10	0680	Moved hands and legs
3.14	0682	Moved hands and legs
3.16	0684	Rubbed nose
3.23	0691	Stretched
3.25	0693	Rubbed nose and cheek
3.45	0706	Stretched
3.48	0708	Rubbed eyes
3.50	0714	Rubbed nose
3.55	0715	Stretched
4.03	0719	Stretched
4.18	0729.5	Moved leg
4.20	0731	Stretched

It is important to accurately record events at the time of
monitoring of ventricular pressure. In the above case,
the physiological parameters are fed immediately onto a
magnetic tape and a separate book with time and footage must be
kept of the events occurring, so that interpretation of the
tracings can be undertaken later. The same is true of simple
ventricular pressure recordings. It is important that close
observation of the child be undertaken so that interpretation
of the tracing is made in the light of such things as movement,
light and deep sleep, yawning etc.

PREPARATION FOR RECORDING

The transducer is sterilised (after calibration has been
carried out) prior to the procedure, by immersion in aqueous
Cidex (2% glutaraldehyde) for 10 minutes.

In most cases no sedation has been employed in these children and where it has been found necessary, mention has been made in the individual case. Furthermore, drugs with a potential to raise the C.S.F. pressure, e.g. morphine etc. have been avoided.

Throughout these recordings, apart from the initial discomfort, the children all settled quickly and remained so throughout. A 'special' nurse was in attendance for the whole period of the investigation in all cases.

METHODS OF RECORDING VENTRICULAR PRESSURE

The method of recording is determined by access to the ventricular system and basically we encountered a number of different clinical situations.

(a) A Rickham or an Ommaya reservoir already in situ.

(b) A Pudenz valve alone in situ, with a patent proximal catheter.

(c) No suitable shunt or reservoir but an open anterior fontanelle, or existing burr hole.

(d) An elective burr hole or C.S.F. reservoir

(e) The cortical subarachnoid space, through an open fontanelle.

The mechanism by which the transducer was connected to the cannula in the ventricle, therefore of necessity varied.

In the first 2 situations, a Huber self-sealing angle tipped hypodermic needle of 21 gauge in Luer mounts and measuring either 1" or $1\frac{1}{2}$" was inserted into the pumping chamber of a Pudenz valve or into the cap of a Rickham or Ommaya reservoir, and this is connected via a 3 way tap to the Luer metal case of the miniature strain gauge pressure transducer. Fig. 8
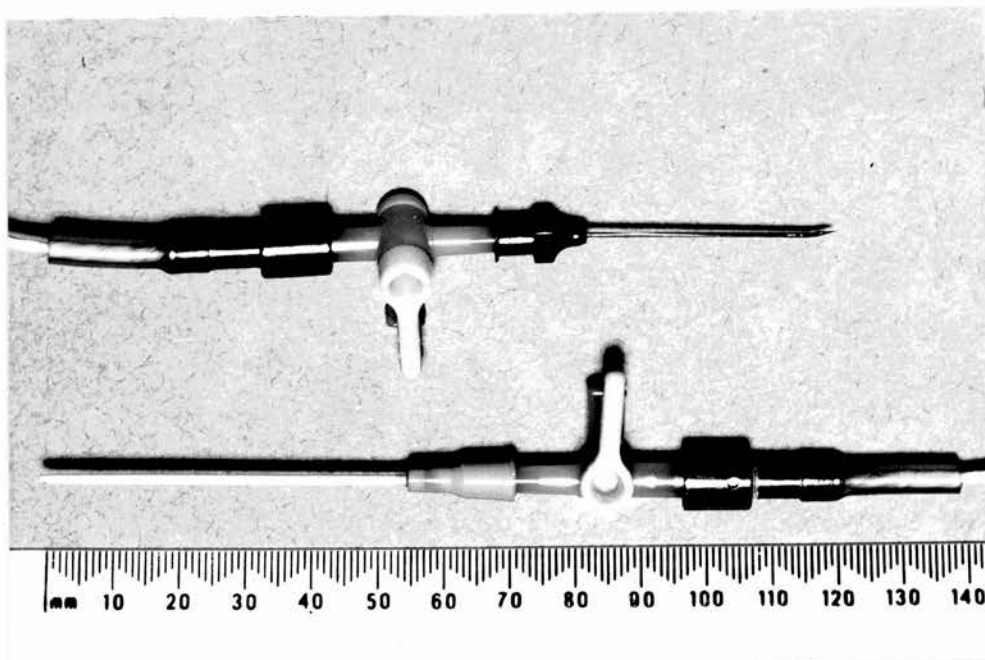
Fig. 8
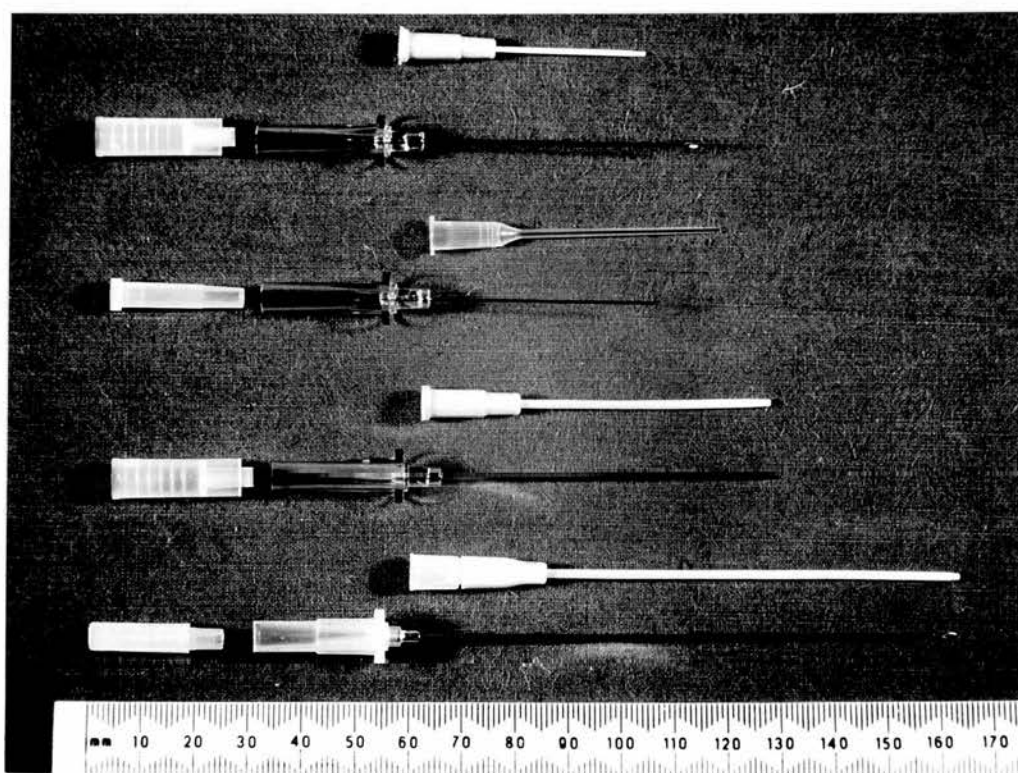
Fig. 9

shows a Huber needle and a teflon cannula attached to the transducer.

In situations (c) and (d) where there is no existing access to the ventricular sustem but an open fontanelle or burr hole, a number of different types of teflon cannulas are used to locate the ventricles in the usual way.

Some of the various cannulas are:

(i)   a luer connection, radio-opaque teflon intravenous cannula (Thackray No. 1823) 23G. 3.2 cm needle or No. 19G. 6.98 cm (No. 1619).

(ii)   alternative cannulas (Fig. 9) are 'Desaret angiocaths' Cat. No. 2878 20G. $1\frac{1}{4}$"

also Cat. No. 2814 16G. 2"

also Cat. No. 873 18G. $1\frac{1}{2}$"

also Cat. No. 2834 16G. $3\frac{1}{4}$"

The only other situation (e) is where a large bore subdural needle is inserted into the cortical subarachnoid space through the anterior fontanelle.

CONSIDERED POSSIBLE SOURCES OF ERROR IN THE RECORDINGS

(a) The equipment has been assessed by the Department of Medical Physics and passes as having an overall accuracy of plus or minus 1 mm Hg. When investigating normal lumbar pressures (Gilland et al 1974), it was found that small bore needles produced modest overdamping of the pressure responses and when using open manometers (which are not used in this series) if the diameter is very small, the capillarity of the tubing gives spuriously high results (Davson 1967).

(b) <u>capillarity</u>

If one considers that the ventricular system, the Rickham
reservoir or shunt and the Huber needle are a completely closed
and completely filled (with C.S.F.) system, then no surface
tension effect can have a bearing on the pressure level or
wave forms.

(c) <u>The amount of C.S.F. displacement</u>

Displacement from a filled system will result in a falsely
recorded lower pressure. However, there is very little
interference with the volume of C.S.F. by this method because
firstly, the reservoir is usually in situ and as such forms
an extension of the existing ventricular system, i.e. it is
isovolumetric. Insertion of the Huber needle will displace
less than one drop of C.S.F. Anything less than a 10% reduction
in the volume from the closed container will have a negligible
effect on the pressure recording.

(d) <u>Turbulence</u> in a small diameter needle as distinct from
'lamina flow' was considered a possible source of error. However,
as mentioned before, this is a non-flowing system and therefore
turbulence will have no effect.

(e) <u>Small Internal Diameter or 'Tolerance' of the Huber needle</u>
(it is slightly variable but generally a 21 gauge needle is
used, that is, an internal bore of 0.021 cms and is $1\frac{1}{2}$" in length).
The internal bore of a Rickham ventriculoscopy tube is 1.4 mm and
the length is variable. The size of the needle will have no
effect on the mean level of pressure recorded. The time taken
for the pressure wave to be transmitted from the ventricle to the
sensor will depend on the velocity of sound going from the
ventricle to the sensor. If this occurred in air, it would be of

the order of 340 metres per second and for a 10 cm distance

between the ventricle and the sensor this would take an impulse

1/3,400 th of a second. Frequencies any faster than this would

produce some false results. In water (or C.S.F.) however, the

velocity of sound is greater than in air and of the order of

1.4×10^5 cm/sec, that is 1,400 metres per second. This means

that with this frequency_response this type of system would only

falsely read frequencies in the kilocycle range, that is

thousandths per second. In my recordings, I am dealing at the

most with frequencies just greater than $\frac{1}{3}$ of a second, and so

as well as not interfering with the mean level of pressure,

the frequency response will not affect the cardio-respiratory

artefact (F.H. Barnes 1979, Personal Communication).

(f) Tracings obtained by way of Pudenz or Rickham

reservoirs or Ommaya reservoirs, are idential to those obtained

from ordinary ventricular cannulation via a burr hole or into

a fontanelle. The very design of a Spitz-Holter system with 2

valves makes it unsuitable for measuring pressure and tracings

obtained from these bear no relation to the actual ventricular

pressure.

(g) It has been found that manipulation of the Luer casing of

the earlier pressure transducers may alter the calibration.

For this reason, the pressure transducers have been connected

to a 3 way tap prior to calibration and this has eliminated

a possible source of error.

(h) The zero point for ventricular pressure in this series

has varied slightly from patient to patient. Generally

speaking, one attempted to arbitrarily fix the zero at the

level of the inter-ventricular foramina. Where this was not the case, an estimation of the zero point has been noted, e.g. upper level of the cortical subarachnoid space, or a measured distance above or below it.

(i) Other factors which might affect the accuracy and be a source of error include the ability to calibrate the machine carefully with a standard mercury or aneroid sphygmomanometer and the accuracy of the system will depend to a degree on how accurate this calibration is carried out.

(j) Variation in recorded pressure with differences in temperature. The temperature of the ward, the patient, the sensor or the metal casing from handling will effect a minor error in the ventricular pressure recorded. Therefore this was investigated.

In a solution of water at different temperatures, the strain gauge transducer was submerged to a given depth, and the various incremental pressure variations were recorded at different temperatures. The results are plotted in Fig. 10 and it can be seen that for a possible patient temperature range of 35°C to 40°C (no child with a temperature had his ventricular pressure monitored) that, with an increase in the temperature of the patient there is a false depression in the level of ventricular pressure recorded. With a decrease in the temperature of the patient over this range, there is a false elevation of the recorded ventricular pressure. Dropping perpendiculars to the abscissa, one can see that the maximum possible error with temperature fluctuation over this range (35°-40°C) is 0.25 mm Hg.
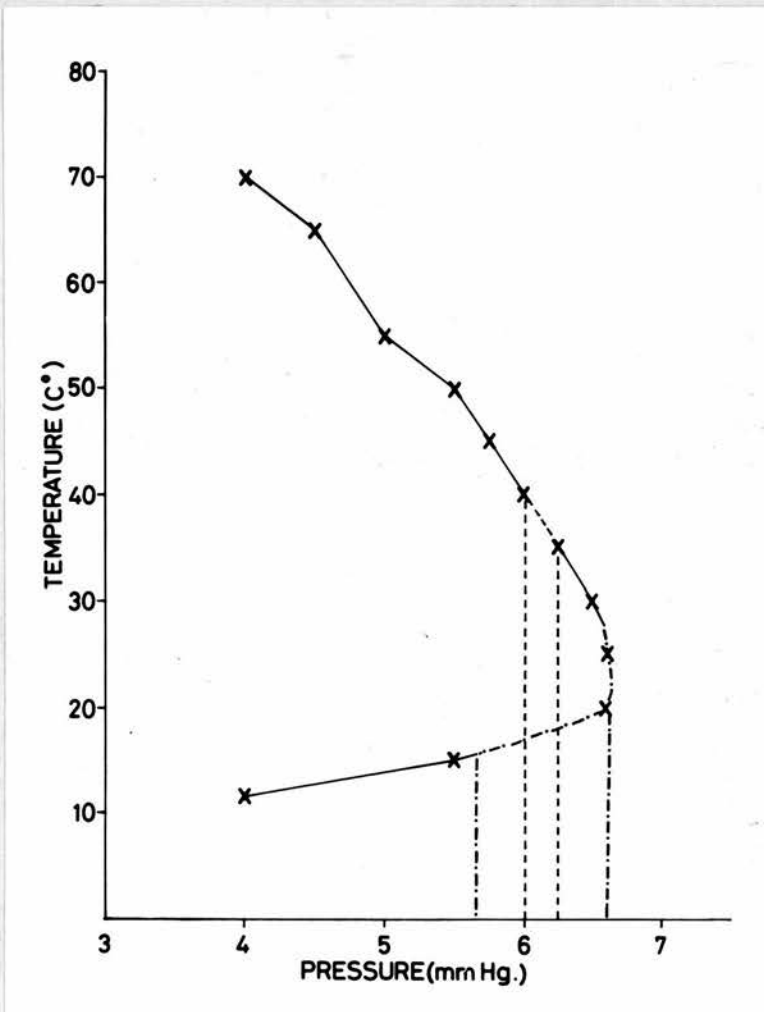
Fig.10

The environmental temperature must also be considered and
in the Neurology Ward at the Royal Hospital for Sick Children
in Edinburgh, the maximum possible temperature fluctuation
over a long period of time was $15.5^{o}C$ to a maximum of $27.5^{o}C$.
Although it is most unlikely that this temperature fluctuation
could occur during the course of any one monitoring, and,
although the sensor itself is not in direct contact with the
environment but with the C.S.F., I have included this range also
in case temperature variations affect the metal casing of the
transducer apparatus. It can be seen on the curve on this
graph that by dropping perpendiculars again to the abscissa,
the maximum possible pressure fluctuation falsely recorded
over this range is 1 mm Hg.

With this method of recording, there is less C.S.F. outside the
skull, so that it cannot cool, and affect the pressure recorded,
nor is it displaced from the ventricular system to any large
degree, thereby recording a falsely low ventricular pressure.
I should mention that this artifactual variation in the
recorded pressure has nothing to do with hypothermia reducing
ventricular pressure. It is known that hypothermia is an
accepted management of some types of raised intracranial pressure
and it would therefore be necessary to consider the various
possible false recordings or error over a much lower range of
temperatures if hypothermia was being used in the management
of raised intracranial pressure and the pressure monitored at the
same time.

In practice, therefore, the maximum possible recorded error
due to differences in temperature affecting the sensor and
recording apparatus, or differences in the patient's temperature

is 0.25 mm Hg. For temperatures above 37°C the recorded ventricular pressure will be falsely (very slightly) low and where the patient's temperature is less than 37°C, the recorded ventricular pressure will be falsely high, also to a small degree. Although I have included the environmental temperature fluctuations, for any one patient undergoing continuous ventricular pressure measurement, the environmental temperature is remarkable constant in the child's immediate environment. If such large temperature excursions did occur, they could account for no more than a false recording of plus or minus 1 mm Hg.

(k) The atmospheric pressure will also affect these pressure transducers, such features as altitude and cloud cover would be expected to have a minor effect on the level of ventricular pressure recorded. However, with the child in a constant environment throughout these recordings, and with frequent re-calibration, this possible source of error is miniscule.

(l) Possible sources of error relate also to the period of monitoring and generally at the outset of measuring pressure, if the pressure is elevated, one has the answer immediately, and a lot of the 'short recordings' have been for this reason. However, where the ventricular pressure is in the normal range, one needs to monitor over some period of time including a cycle of sleep to detect more subtle changes in ventricular pressure under different physiological situations.

(m). Another possible source of error is that of zero drift and this occurs with all miniature strain gauge transducers and the only possible way of minimising this error is to frequently re-calibrate. By far and away the accuracy of this type of

recording apparatus depends on familiarity with the apparatus and careful, frequent calibration.

It has been our policy not to accept tracings of ventricular pressure which have not shown both a respiratory component with superimposed pulse waves and have not changed in the expected fashion to elevation of the head of the bed, Queckenstedt's etc. Although Queckenstedt's have been performed here, it must be remembered that there is immediately available the therapeutic 3 way tap to release pressure if needed. Furthermore, problems are unlikely to occur with relief of C.S.F. pressure with Queckenstedt's above the level of the foramen magnum.

CHAPTER 3

TECHNIQUES OF MEASUREMENT AND MONITORING OF INTRACRANIAL
PRESSURE - REVIEW OF THE LITERATURE

FONTANOMETRY

    (a)   Tambours

    (b)   Schiotz tonometers

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INTRAVENTRICULAR CANNULATION

SUBARACHNOID BOLT

SUBDURAL AND EPIDURAL MEASUREMENTS

TELEMETRY

CHAPTER 3

TECHNIQUES OF MEASUREMENT AND MONITORING OF INTRACRANIAL

PRESSURE — REVIEW OF THE LITERATURE

Techniques for direct measurement and graphic recording of I.C.P.
in animals were first described by Leyden in 1866. A number of
workers have since published reports describing similar methods
for recording either ventricular or lumbar fluid pressures.
Blackfan et al (1929), Carmichael et al (1937), Antoni (1946),
Ryder et al (1951), Guillaume & Janny reported that neurological
signs were unreliable indicators of the presence or absence of
I.C.P. and until Lundberg's monograph on the clinical usefulness of
the measurement of I.C.P. in clinical practice, there had been no
broad clinical application. The progress of V.P.M. has always
proceeded first in adults and subsequently in children.
Two principal types of instrument are now used for continuous
recording of I.C.P.

(i) a catheter or cannula inserted into a C.S.F. space, usually
the lateral ventricle or

(ii) a solid state transducer placed in the epidural or subdural
space or occasionally the brain tissue. There are many different
methods and devices available for monitoring and displaying I.C.P.
and they are discussed below.

FONTANOMETRY

(a) Tambours

In neonates and young infants there is often a sizeable anterior
fontanelle and this, together with the fact that the calverium is

quite thin, precludes the use of many types of I.C.P. transducers. The use of fontanometers has therefore been investigated. It is obvious that there would be enormous advantages if the I.C.P. could be easily estimated without recourse to puncture of the C.N.S., avoiding the possible complications of infection, haemorrhage, local damage from repeated puncture etc. (Brett 1966).

Clinicians have always been able to obtain a rough estimate of I.C.P. by palpating the anterior fontanelle. It is usually patent until 15-18 months of age. The tense bulging fontanelle of an infant is usually indicative of a R.I.C.P. syndrome.

For proper assessment of the anterior fontanelle, the baby should be relaxed and not crying, he should be held in the sitting position with the examiner's hand making an estimate of the degree of elevation and resistance to gentle pressure.

In the normal quiet infant in the upright position, the anterior fontanelle is usually either flat or slightly concave compared with the surrounding scalp. As mentioned previously, there is a great deal of variation in size in the normal infant and it customarily enlarges somewhat in the first few months after birth (Scammon & Adair 1930). Normally pulsations can be felt and they are frequently noted in the full fontanelle with R.I.C.P. but they are usually lost with markedly R.I.C.P.

Other theoretical advantages of this type of pressure recording are that if ventriculitis or intraventricular haemorrhage is present, ventricular taps may be dangerous because of the need to pass a needle through the lateral wall of the ventricle in the region of the germinal plate, an area with a large venous plexus. Also, because it is non-invasive, it can be used on normal children as well as hydrocephalic children.

The disadvantages are that they generally need an anterior
fontanelle of sufficient size, the position of the fontanometer
is critical and there is difficulty in keeping it in position; a nurse
needs to hold it all the time. It is generally not quantitative.

Riechert and Heines (1950) measured the pulsation amplitude in a
tambour applied to the fontanelle and found that as the pressure in
the tambour was increased, the pulsation amplitude of the fontanelle
also increased and seem to reach a maximum.

Purin (1964) showed with an oscillographic technique that as pressure
was applied to the anterior fontanelle the pulsation amplitude in the
tambour increased and then declined. He concluded that the peak
pulsation amplitude occurred when the pressure within the tambour
equalled that within the skull. These findings were confirmed by
Bareshnev & Leontev (1965), also with an oscillographic technique,
who found high fontanometer pressures in infants with cerebral injury
at birth.

The spontaneous variations in pulsation amplitude encountered in
all patients, presumably reflects changes in intracranial pulse
pressure. Alterations in pulse pressure may be caused by changes
in arterial pressure (Sibayan et al 1970), cranial venous pressure
(Guthrie et al 1970), cerebrospinal fluid pressure (Davson 1967)
and the unknown mechanisms which control the pulsation, absorbing and
damping system of the spinal theca and veins (Martins et al 1972).

Lietze et al (1972) found an alteration in wave patterns during
different stages of sleep. They found no difference in healthy or
hydrocephalic infants during sleep and recognised clear waves of pulse,
respiration and 3-8 per minute type waves (similar to the beta waves),
independent of respiration and heart rate in both normal and

hydrocephalic children. The peak of this third type of wave coincided with mid-expiration. They also noted that in post-shunt cases, arterial pulse waves were depressed in some children and in extreme hydrocephalus, only slight pulsations were recorded.

Wealthall & Smallwood (1974) investigated these methods and they found that by averaging pulsation pressures over several 5 second periods, it was possible to identify significant changes in pulsation amplitude, between different applied pressures. The time interval for averaging pulsation amplitudes was critical. Experimental models have suggested that when a tambour occupies a large proportion of the fontanelle, the pressure increase in the tambour results in an increase of intracranial pressure and any increase in intracranial pressure is liable to produce an increased pulsation pressure within the skull which will only stop when auto regulation of cerebral blood flow is lost at very high intracranial pressures (Hussey et al 1970) plus, in infants with normal to moderately enlarged fontanelles, the application of a tambour of comparable size and its inflation is likely to lead to an increase in pulsation amplitude within the physiological pressure range. Thus it would seem that in exceptional patients with large fontanelle, this technique may provide a measure of I.C.P., but it is difficult to apply in the majority of patients, it often upsets the baby, and, because of its unreliability, is no longer used.

(b) Schiotz Tonometer

The underlying principle of this method relies on the tension of the anterior fontanelle. The tension of a membrane containing a fluid is determined by the pressure of the contained fluid and the elasticity of the membrane. Under conditions of constant elasticity, changes of pressure will therefore be reflected by changes in membrane

tension. The elasticity of the structures which make up the anterior fontanelle however, has not been shown to be constant, and it may vary from time to time and from child to child. Davidoff & Chamlin (1959) and Edwards (1974) have reported its use for this purpose, however it requires the infant to be positioned in the vertical and thus allows gravity to act on the plunger; it is unsuitable for continuous measurement and is inaccurate.

(c) Aplanation Transducer

Wealthall & Smallwood (1974) described continuous measurement of I.C.P. in unsedated infants using a modified aplanation principle and they compared the pressures recorded to needle pressures.

The Hewlett Packard APT-16 transducer was evaluated as a fontanometer. If a plain surface is placed against a membrane distended by pressure, the membrane will bulge through any hole in the surface, if a spring-loaded plunger is placed in the hole and a force applied to it so that the end of the plunger is in the same plane as the base plate, the pressure exerted by the plunger will be equal to that within the membrane. In these circumstances the membrane is flat, so there is no effect due to tension in the membrane or compression of it, thus the method would be independent of membrane characteristics.

To evaluate the performance of this technique, pressures by needle and aplanation transducer were recorded simultaneously in a small number of infants. An ileostomy phlange was applied to the scalp over the fontanelle by means of adhesives. The aplanation transducer was secured into the phlange by means of a jubilee clip and the infant allowed to settle. After being placed in a special frame to support the head in the horizontal position, the lateral end of the fontanelle was prepared for a routine ventricle puncture. Five infants were

studied in this manner. The variations of intracranial pressure caused by cardiac systole, respiration and 'sobbing' were displayed equally well by the pressure transducer and the aplanation transducer. Correlating pressures measured by the two methods in 5 patients, indicated that the aplanation transducer faithfully measured intra-cranial pressure over a wide range in each patient. The authors show a graph with a very high degree of significance in the relation-ship between fontanometry and needle pressure in these 5 children. It is obvious that the fontanelle must be of sufficient size and that the circumference of the hole in the base plate must be in contact with the fontanelle and not with its edge. Likewise the fontanelle must protrude above the bony margins. The position of the child is critical in using the aplanation transducer as it is measuring pressure at the level of the fontanelle and so allowances must be made for the distances between the fontanelle and any chosen reference point, such as the right atrium or the lowest point of the head.

These infants were placed in a tilting chair to facilitate nursing. This device was also used for intermittently measuring pressure changes following drugs and they illustrate a decrease in intracranial pressure with the use of isosorbide at 2 gm/Kg/6 hr.

Stethoscope Pick-Up

Blaauw et al (1974) described methods of simultaneously registering E.E.G., eye movement, E.C.G., respiration and fontanelle pulsations achieved with the use of a stethoscope connected to a pulse pick-up. The stethoscope was equipped with a membrane which bulged towards the centre, so that adequate contact with the skin was established. The recording time was at least one hour, but mostly longer, sometimes up to 3 hours. They reported that the pulse waves from the fontanelles of two groups of infants showed comparable patterns during

sleep, and there was no difference between that of healthy and
hydrocephalic infants. They reported that the peaks of the fontanelle
pulse waves coincided with mid-expiration and they too reported a third
wave with a frequency of 3-8/minute which resembled beta waves in both
normal and hydrocephalic children. They noted that in extreme hydro-
cephalus only slight pulsations were recorded.

Haydon et al (1970) observed a direct relationship between the
height of the ventricular wave amplitude and the mean ventricular
fluid pressure and concluded that often in pronounced and progressive
hydrocephalus, V.F.P. was only slightly elevated and sometimes was
even within the normal range. Significantly they found 'plateau' waves
to be rare in neonatal hydrocephalus, if they occurred at all, finding
no indication of them in their recordings.

This then is another qualitative assessment of I.C.P. which presents
a problem of coplanimetry, that is, the relationship of the
stethoscope diaphragm to the anterior fontanelle.

(d) Fibre Optic Sensor

Vidyasagar and Raju (1977) developed a method based on optical
principles and devoid of electrical hazards. The basis of its
mechanism is that pressure acting on the contact surface of a fibre
optic sensor tilts a mirror, uneven reflection of light is then
fed back to a monitor (Ladd Intracranial Pressure Monitoring Device,
Model 1700, Rocke Medical Electronics).

In their clinical application of this device they found the mean
anterior fontanelle pressure in normal infants was 10.14 ± 0.39 cm
H_2O and that the anterior fontanelle pressure was similar in healthy
term and pre-term infants. In attempting to correlate the
pressures recorded with C.S.F. pressures at the lumbar and
ventricular level they compared only 6 values, obtaining an r value

of 0.95 but with significance $p < 0.01$. It is clear that further comparisons need to be performed in this way to establish the accuracy of the anterior fontanelle pressures recorded.

A non-invasive estimate of I.C.P. would be very useful in neonatal units, aiding the management of full term and premature babies with birth trauma, or asphyxia (in particular I.V.H. and I.C.H. in premature babies with respiratory distress syndrome). The R.I.C.P. is subsequent to hypoxia and cerebral oedema and although not all continue to have problems with R.I.C.P., some suffer short and long term effects. Korobkin (1975) reported a delay of 20 to 90 days between clinically suspected I.V.H. and an overt increase in the OFC. For this particular group of infants the modified aplanation device, Hewlett Packard APT-16 and the fibre optic sensor with their direct reading, reduced problems of coplanimetry and non-invasion have shown the most encouraging results so far and the others mentioned are un-likely to have any clinical usefulness.

INTRAVENTRICULAR CANNULATION

Attempts to determine the intracranial pressure by direct measurement and to record the variations graphically were described by Leyden (1866). Key and Retzius (1875) were the first to measure I.C.P. and Knoll (1886) the first to produce a graphic record of the C.S.F. pressure in animals. Several early investigators studied variations of the brain volume graphically; in animals, Roy and Sherrington (1890); Hill (1896) and in humans Mosso (1881), Burckhardt (1881) and Becher (1922).

Since the introduction of the lumbar puncture as a clinical method by Quincke (1891), single or repeated readings of the spinal fluid pressure have been widely used in assessing the intracranial pressure,

Ayer (1929), Merrit and Freemont-Smith (1937), Browder and Meyers (1938) and Cairns (1939).

Lumbar puncture pressures have been used in the investigation of intracranial pressure from various points of view, for example the influence of anaesthetic drugs by Paarnhoj (1949) and Woringer et al (1954); the pressure levels in hypertensive vascular disease and intracranial tumour Jefferson (1955); the spontaneous fluctuation of the elevated intracranial pressure by Ecker (1955) and the influence of urea by Javid and Settlage (1956).

The V.F.P. was measured directly by Hodgson (1928) and by Smythe and Henderson (1938). Graphic records of V.F.P. at this time were only shown in a small number of publications. Blackthan, Carruthers and Ganse (1929) recorded the ventricular and spinal fluid pressures simultaneously in hydrocephalic infants, for differentiating communicating from non-communicating hydrocephalus. Janny (1950) and Guillaume and Janny (1951) used continuous graphic recording of the V.F.P. in the study of intracranial pressure in patients with surgical diseases of the brain.

Curves of the V.F.P. in a hydrocephalic child were presented by Bering (1955) in his work on the arterial pulsations of the C.S.F.

It was not until Lundberg's thesis that measurement of the V.F.P. was put on to a sound clinical routine basis. Lundberg's procedure involved passing a stilette through a ventricular cannula. The ventricular cannula was connected to the recording apparatus by means of a long polyethylene tube with an outer diameter of 2.8 mm. The system was filled with saline after which the connection between the ventricular cannula and the transducer of the recording device was opened. His standard procedure was to puncture the right frontal horn with the patient in the supine position. He summarised the

available data at the time and suggested that the side effects of
ventricular puncture were due

(i) to a fall in intra-ventricular pressure on evacuation of fluid
and the subsequent derangement of the intracranial equilibrium

(ii) direct traumatisation was possibly a contributing factor
whereas haemorrhage from damage to larger intracranial vessels
appeared to be of little significance.

An increase in the brain volume due to hyperaemia, that is an
increased blood flow, or oedema as a consequence of ventricular
tapping had been suggested earlier by Dandy (1932, 1944). Lundberg's
technique made it possible to establish a patent communication from
the lateral ventricle without significane escape of fluid and hence
the risk of deranging the equilibrium. In urgent cases however,
fluid was evacuated successfully under pressure control. Furthermore,
with this closed method, if the ventricles were small and the intra-
cranial pressure high, he did not collapse the ventricles, as would
occur with an open puncture after a few mls of C.S.F. had been
released.

Lundberg emphasised that there must be no leakage through the
burr hole, because it impairs healing and increases the risk of
infections. He chose polyethylene tubing because it is inert to
brain tissue (Ingroham et al 1947).

Prolapse and rupture of the brain within the burr hole was common
when the dura was open to the edge of the burr hole and the intra-
cranial pressure high, and this subsequently did not occur when the
opening of the dura was not much larger than was necessary to permit
passage of the cannula.

The common position of the patient for ventricular puncture according
to the conventional techniques at that time appeared to be supine,

with the head bent forwards (Poppen 1960), but Lundberg discounted
this as being dangerous, as it may cause stasis of the jugular veins
and hence further elevate the intracranial pressure. Consequently
he employed the supine position. In patients with intracranial
hypertension with this method, he found that antiflexion of the head
could cause considerable increase in ventricular fluid pressure from
10-14 mm Hg.

Lundberg's equipment consisted of a strain gauge pressure transducer,
a standard potentiometer, recorder and D.C. supply. Potentiometers
for adjustment of zero and range, and an open bore water manometer
for calibration.

During continuous use, the apparatus was calibrated twice a day
and he found that in 97% of cases the deviation was 1 mm Hg or less.

With regard to reference level, after trials with various alternatives,
the following reference level was chosen - 'a horizontal plane through
a point 1.5 cm below the point of the outer surface of the calotte,
which at the time of measurement is the uppermost one'. This level
corresponds roughly to the uppermost part of the subarachnoid space,
since the total thickness of the skull, calvarium and dura in most
adults is between 1 and 2 cms. The reason for choosing this reference
level was to simplify the adjustment of the transducer during
routine use. He recognised that a horizontal plane through the
approximate centre of the intracranial cavity would perhaps be a
more correct reference level for determination of the intracranial
pressure, in different positions of the head however, such a zero
would be difficult to refer to easily defined landmarks on the
outside of the head and was therefore impractical.

Since the cranial cavity is not a true sphere, turning of the head of
a patient lying in the horizontal position may cause changes of the

distance between the reference level and the centre of the cranial cavity. If the skull is of normal shape these changes would hardly exceed 3 cms, corresponding to a hydrostatically induced change in V.F.P. of somewhat more than 2 mm Hg (Lundberg 1960).

The advantages of this sort of system are that it is a reliable record with good pulsations, with an external zero and easy correction, whether due to temperature or time. One can therapeutically remove C.S.F. and can do volume pressure estimates. The disadvantages are that frequent punctures may cause an intra-ventricular haemorrhage or local cerebral oedema, that the brain may clog the catheter if it is swollen and ventriculitis is a slight possibility. The main disadvantage of this method is that the brain must be punctured and when the ventricles are small and shifted, they may be difficult to locate. Miller (1978) allows no more than 3 attempts at locating the ventricles and if still unsuccessful, he suggests that an alternative method of I.C.P. monitoring is sought.

The obvious indications for this type of measurement are if the ventricles are large. Bruce et al (1977) comments that intra-ventricular haemorrhage is one indication for this method of recording but repeated punctures through a friable ependyma may result in further intraventricular haemorrhage and negates this method. Ideally these cases should have a reservoir electively inserted.

Miller (1978) describes in detail the routine method now commonly used where a fine catheter is implanted, usually in the right frontal horn of the lateral ventricle via a burr hole or a twist drill hole. The drill hole is placed at the coronal suture in line with the pupil and the catheter is directed medially towards the medial canthus of the eye or towards the bridge of the nose, when small ventricles are

suspected. As little as possible C.S.F. is lost during puncture and the catheter is connected by a fluid filled manometer tubing to a stop-cock-manifold system, to which is attached an arterial range pressure transducer. A calibrating water manometer, a fluid filled reservoir, and a drainage bag are required and syringes for flushing the system and filling the manometer. All joints must be water tight and the output is amplified on to a recorder at slow speed.

This system requires common place equipment and can be recalibrated easily. As mentioned before, it allows studies of pressure responses to C.S.F. volume, to assess the intracranial elastance and enables C.S.F. drainage. When open ventricular drainage is used, prevention of ventricular collapse is helped by draining the C.S.F. against a pressure gradient of 15-20 mm Hg.

At the Royal Hospital for Sick Children in Edinburgh, the method for locating the ventricle in infants with sizeable fontanelles is from the lateral angle of the fontanelle, to keep the needle or cannula at right angles to the saggital plane at the surface of the scalp and then direct the needle in a coronal plane towards an imaginary line between the nasion and the internal occipital protuberance.

A strictly aseptic tehcnique is paramount e.g. two solutions of Betadine, a scrub, and a sterilising solution are used after the scalp has been shaved, followed by routine gloving etc.

This method of V.P.M. with an intraventricular cannula is still the most common, and Mickeil et al (1977) reviewed 42 children who had their intracranial pressure measured and of these, 34 cases were measured by means of an intraventricular cannula in the method, suggested by Lundberg, only 4 of their cases had the subarachnoid pressure measured by means of a metal Richmond bolt.

SUBARACHNOID BOLT

This was first described by Vries et al (1973) in adults and in
children by James et al (1975) and Winn et al (1977). This consists
of a hollow bolt which is inserted via a small twist drill hole,
placed just anterior to the coronal suture. The tip of the bolt
resides in the subarachnoid space outside the brain and is connected
via a rigid fluid filled system to a strain gauge transducer and from
there to a recorder.

The bolt can be inserted at the bedside. The other advantages are
that it is securely fixed to the skull, it can be used in cases where
there are small or absent ventricles and is independent of brain
shifts. It has a minimal risk of infection, it avoids brain puncture
and correction for time and temperature drift is easily made. The
disadvantages, however, are that no volume pressure response can be
performed, it cannot be used to withdraw C.S.F., surface bleeding may
occur when the dura and the arachnoid are opened, and the bolt may be
occluded by brain entering the lumen. The system must be absolutely
water-tight. In a review of 80 children over $2\frac{1}{2}$ years Bruce et al
(1977) found no haematomas or infections, but stressed the need to
make a good subarachnoid space-bolt connection. Winn et al (1977)
has successfully used this subarachnoid screw in more than 600
patients and found it reliable and with low risk. This method of
measuring the supratentorial subarachnoid pressure can be used in
children over the age of 9 months and in particular, cases where there
are small ventricles, for example trauma, encephalitis or diffuse
encephalopathies, e.g. Reyes' Syndrome. Its use in cases of Reyes'
Syndrome was reviewed by Bermin et al (1975), Shaywitz et al (1977) and
it is concluded that vigorous supportive therapy including careful
monitoring of the I.C.P. is the basis of management.

SUBDURAL AND EPIDURAL PRESSURE MEASUREMENTS

This can be achieved basically in two ways, firstly by inserting a fluid filled tube connected to an external transducer, or secondly by placing the transducer directly against the dura. In the latter the sensor must be flush with the inner surface of the skull and perpendicular to a line extending from the centre of the brain (Dorsch & Simon 1975). If the sensor distorts the dura, it is subject to forces additional to intracranial pressure. A third type of epidural transducer is possible when the implanted transducer is used with no external connections, that is telemetry where the whole system is completely enclosed within the skull.

The earliest of the devices for measuring subdural pressures was Hoppenstein's balloon (1965) and this was used for recording post-operative intracranial pressures for several years (Rothballer 1963). It is a thin walled, latex tambour constructed for use with an iso-volumetric strain gauge manometer, which can be placed in a subdural or subarachnoid space for measuring and recording both the intracranial pressure and pulsations. It measured 10 mm in diameter and was 3 mm in height when filled with water. Attached to the side of the tambour was a thicker walled latex tube permitting conduction by means of a rigid polyethylene tube to a strain gauge. Since a tambour has its own volume and does not require to be inflated beyond its normal capacity, the elasticity of its walls and the forces necessary to overcome this elasticity do not come into play, therefore the displace-ment of the fluid within the tambour when connected to an isovolumetric strain gauge via a rigid tube reflects the changes within the cranial cavity, when the vault of the skull is resealed. The advantages of it were that it could be placed in the subdural or subarachnoid space through a single small burr hole and did not dislodge with movement

of the head, or during seizures, it recorded changes in intracranial
pressure as adequately as intraventricular cannulae without the
dangers of the latter and it permitted intracranial pulse and
pressure recordings in the wakeful state and could easily be removed
from the patient and be autoclaved.

Hulme and Cooper (1966) decided on the following criteria for an
intracranial pressure recorder. The method must be simple, reliable
and able to function efficiently under a wide variety of conditions
for prolonged periods with only occasional expert supervision, it must
cause no significant additional discomfort or risk to the patient
and must be simple to disconnect the patient from the recording
apparatus so as not to impose unnecessary restriction of activity
or interfere with other investigations, and it must yield maximum
information.

In their technique a pressure sensitive transducer was inserted
directly into the skull. The advantages of it were that it was
not dependent on C.S.F. drainage and could be inserted at any place
in the skull and recorded a pressure exerted by the brain against
the cranium at that point. A lead from the transducer emerged through
the skull wound and the insertion of the pressure transducers combined
conveniently with the placement of subdural gold electrodes for
monitoring phenomena such as cortical E.E.G., oxygen availability
and electrical impedence. Their type of pressure transducer was
manufactured in Massachusetts U.S.A. and is known as the HFD2 which is
a very small transducer, only 3 mm in diameter, with good sensitivity
and base line stability over prolonged periods. In use it is
contained in a stainless steel sleeve, fastened in a stainless steel
strap spanning the burr hole. The position of the sleeve in the strap
is adjusted so that the protected diaphragm of the transducer is in

contact with the cortex ' level with the inner surface of the dura.
The wires are brought out through the scalp, together with a narrow
bore diameter silicone rubber tube open to the atmosphere which
provides the reference pressure to the transducer. They recorded
I.C.P. in 13 patients with this method and were impressed with the
lability and wide range of variation in intracranial pressure
observed.

A pressure indicating bag for monitoring intracranial pressure was
described by Numoto et al (1973) which arose from two earlier
developments of implantable pressure switches (Numoto et al 1969,
1966). Both of these earlier types of apparatus were used in the
clinical situation (Donaghy, Numoto et al 1972; Donaghy, Numoto
1972, Numoto, Donaghy 1970). But they still possessed some
undesirable characteristics, namely the fragility, expense and man-
ufacturing difficulties. The authors therefore designed a pressure
indicating bag, which consisted of two teardrop shaped membranes
with a metal frame between them, and a piece of stainless steel
reinforced elastic tubing inserted between the membranes at their
pointed ends. They used a light mineral oil to fill the balloon and
about 10 cms of tubing. With the pressure indicating bag maintained
in a horizontal position, the level of oil in the tube is marked.
This represents the zero point. This system can be operated manually
or automatically and is inserted into the epidural or the subdural
space at operation. The intracranial pressure is read on a manometer.
With automatic operation the remainder of the tube is filled with a
glycerol water solution and the end of the tube is connected to a
pressure transducer. It showed negligible baseline shift and
variation with temperature. It was implanted in the subdural
space of a dog and when compared with direct C.S.F. measurements
showed good correlation. There are some design features that set it

apart from Hoppenstein's balloon. The two most noticeable ones
are the metal frame and the zero point marking. The metal frame
prevents it from being deformed by adjacent protruding objects and
Hoppenstein's balloon was incapable of being corrected for baseline
shift.

Gobiet et al (1974) reported a new implantable miniature pressure
transducer whose main advantage was the possibility of zero point
calibration in vivo and they compared epidural pressure responses
with that from V.F.P. responses on long term monitoring of 30 patients.
Their main reason for picking the epidural method was that the risk
of infection was smaller as the measurement could be done without
opening the C.S.F. spaces.

A number of other investigators have looked at epidural pressures.
Brock and Diefenthalar (1972), Gobiet, Bock and Liesegang (1972),
Jacobson and Rothballar (1967) and Nornes et al (1970). Since
puncture of the ventricle is often difficult, the epidural space has
obvious advantages. A significant disadvantage of this method was that
it was technically difficult to build a device without a zero point
drift. Therefore only the development of an additional device for
checking the zero point during measurement could solve the problem.
Several authors have described such methods for zero point measurement
during measurement, for example Simon & Dorsch (1972).

Apparatus was developed from a miniature transducer, BW7, manufactured
by Sensotec, Columbus, Ohio, and tested by Gobiet, Bock et al (1972),
on epidural pressure in man.

In preliminary tests they found a zero point drift of approximately
15 mm Hg after 72 hours under a test pressure of 30 mm Hg. The
insertion was made through a special burr hole on the side contra-
lateral to the intracranial lesion and epidural and ventricular

pressures were measured simultaneously in 11 patients. The epidural pressures were always higher than ventricular fluid pressures and this deviation was more marked at increasing pressures. For example, with an epidural pressure of 100 mm Hg, the V.F.P. was approximately 15 mm Hg lower but the form of the pressure wave was similar in both recordings. So far they have applied this method in 45 patients with several types of intracranial operations and the measurement extended from 3-5 days. With regard to epidural pressure levels they determined three easily distinguished stages. In awake patients without signs of brain swelling, epidural pressures did not exceed 25 mm Hg. With pressures up to 50 mm Hg most patients presented slight disturbances. Above this level increasing unconsciousness, abnormal breathing and circulation patterns etc. were observed. They noted the effect of hyperosmotic solutions on the increasing epidural pressure and in another patient, they measured the epidural pressure during a seizure, although there was no discussion of it in the text, one can see on the E.E.G. leads a definite electrical fit. The epidural pressure, although showing signs, does not show any marked increase over that period displayed. The problem with this method was that it was impossible at that time, to build transducers without a zero point drift and the drift differs from one transducer to another and it was not possible to check the zero point after implantation.

A new model BW7Z, apart from its readjustability in vivo incorporated other improvements. This epidural transducer is the type placing the transducer directly against the dura. The transducer is slightly larger than the previous model, its width 8 mm, height 2 mm, and has venting tubes which carry to the exterior. It was tested in about 30 patients with a total running time of 72 days and the longest implantation lasting 11 days.

The authors compared the E.D.P. and V.F.P. in patients with severe head injury, several days after the accident.

There was good correlation between the measurements obtained and those with conventional V.F.P. recordings with a Statham 23DB transducer. The epidural pressure was higher than the ventricular pressure. This difference was only a few mm Hg in pressures up to 50-60 mm Hg and it agrees with earlier results. Above 50 mm Hg the difference between E.D.P. and V.F.P. increased and when the E.D.P. reached 100 mm Hg, the V.F.P.'s were 10 mm Hg lower. They found that up to an intracranial pressure of 80-90 mm Hg, the calibration posed no problem. Beyond these pressures there were difficulties. The main disadvantage of this type of measurement is that it only approximates the C.S.F. pressure and some hours may be needed after implantation of the device, before it records a pressure sufficiently close to C.S.F. pressure (Coroneos 1973).

Beks et al (1977) described the Philips epidural transducer which was used in more than 200 patients for longer than 9,000 hours without complications. It was reported to have very low drift and satisfied most of the requirements for an I.C.P. transducer selected by the National Academy of Engineering, England. There are however no specific reports of its use in children and insertion is recommended under general anaesthesia.

Dietrich et al (1977) described the new miniaturised system for monitoring the epidural pressure in children and adults. The system is as yet the smallest known dimensions. The transducer is implanted automatically coplanar to the dura via a burr hole of 4 mm in diameter, even in the conscious patient without special surgical equipment. Application is possible in adults, children and infants and the authors achieved a good maximal resolution and a very low

zero point drift.

The rationale for using the epidural space is that with space occupying tumours and haematomas, an increased intracranial pressure is produced due to the small reserve volume for pressure compensation (Girke et al 1974). Furthermore disturbed blood supply and severe contusional brain injuries also lead to space narrowing by brain swelling. The resulting damage depends on the extent of the brain oedema and is correlated with the degree of pressure increases (Collice et al 1974, Johnston et al 1970, Vapalahti and Troupp 1971). Children especially are endangered by the tendency to brain oedema (Gaab et al 1975, Kiene 1968, Tenzholz 1972, Rosman 1974). This, plus the fact that measurement of the intracranial pressure with a ventricular catheter has a risk of infection during the long time application (Derougemont et al 1973, Rylinder et al 1976), they caused the development of this method to avoid opening the C.S.F. space, as have others previously (Hulme et al 1966). It was known that a correlation between epidural and ventricular pressure existed (Gobiet et al 1972, 1974, 1975). Dietrich et al (1977) designed this epidural pressure transducer system for implantation coplanar to the dura.

It is applicable to adults and children and the method itself has no zero point drift, only the thermal expansion, and aging of the membrane produces zero point deviation of less than 1 mm Hg/day.

This system consists of a pressure transducer, a holder, a special adjustable screw tap and a mini drill. A mini Trepan (similar to the Umbach drill) is made of two coaxial drills. Both are turned off when the inner one touches the dura. Thus the outer drill prevents a penetration of the skull and hinders damage to the dura. The holder is screwed into the burr hole and automatically touches

the dura, thus implanting the transducer coplanar to the dura.

It is easy to remove the probe for manipulation and transport of the patient and can be replaced by a plastic phantom to prevent contamination of the holder and dura. The small dimensions of the system allow implantation by a point incision.

On personal communication (1978) with Dr. K. Dietrich, I learned that complete insertion of the system could be undertaken in the Intensive Care Unit, without the assistance of a Neurosurgeon, but during all his experiments he had the assistance of a Neurosurgeon, but he was also sure that this assistance was not necessary after short training of a Physician. Unfortunately this system is not commercially available. In the last few years different companies have been interested in its manufacture but, as yet, it has not been mass produced.

Its application in infants, where the skull thickness is less than 3-4 mm, is not practicable, therefore they developed a transducer with a special mechanical design for the same transducer system. In this, the transducer is mounted as a bayonet fitting, and is not completely coplanar with the dura and causes an artificial pressure increase (pressure offset). This area however is small and constant. The implantation in infants with thin skulls demands special surgical equipment. This special infants' pressure transducing system was tested in 35 cats and dogs before and after induced cold injury, and in the thin skull of cats they did not observe any dural damage.

The epidural pressure is several mm Hg smaller than the ventricular pressure measured with this device. The higher values of epidural pressure in prior reports may be due to an artificial pressure offset inherent in a non-coplanar implantation of pressure transducers.

There is, however, a markedly increased epidural pressure in excess of ventricular pressure, when the epidural pressure is measured over the side which had received the parietal cold injury. However, the epidural pressure in the opposite hemisphere remained in the range of the ventricular pressure.

Due to the small dimensions of the system it is easy to measure pressure gradients simultaneously on both hemispheres in intensive care patients. The small burr hole allows for complete restitution and if the pressure is needed to be measured for a very long time, multiple implantations in one patient could be performed, thus reducing the chances of local infection and errors of measurement by dural fibrosis, which occur within 3 or 4 days (Rylander et al 1976).

Deitrich has used this method in experimental situations and in clinical situations. Other reports of its use in experimental situations include Knoblich, Gaab et al 1978, Gaab, Knoblich et al 1978.

This method has been used for monitoring intracranial pressure in craniocerebral trauma before and after operation. The point being that a continuous monitoring of intracranial pressure after severe craniocerebral trauma allows ominous rises in intracranial pressure to be recognised appreciably earlier than by clinical observation of the patient (McGraw 1976).

For intracranial monitoring after surgical operations he uses a miniature pressure transducer in the form of a thin flexible catheter. It has a flattened pear-shape which prevents rotation around the long axis and the application is in the manner of a drainage tube. It can be inserted epidurally, subdurally or even intracerebrally, e.g. into a resection cavity. In epidural measurements the side of

the pressure transducer membrane is orientated towards the dura
and the cable is then led out like a catheter through the wound
or lateral stab incision. This is the Gaeltec model type 1CTb
with in vivo calibration. After the end of the measurement the
transducers are simply drawn out like a drainage tube.

For pressure monitoring in the non-operative patient, the pressure
transducer with coplanar application, as previously described, is
used. A miniaturised pressure transducer is now commercially
available also by Gaeltec Type 3AE-Special, a drill hole of nearly
5 mm in diameter is necessary. A stab incision is sufficient and
a small guiding funnel is introduced to avoid further skin contact.
A small coaxial drill permits adjustment of the distance between the
inner and outer drill corresponding to the thread length of the
adapter holder. After removing the guiding funnel, the skin over the
miniature transducer is closed completely tension free with one or
two stitches. Only the thin cable is led through the skin to the
measuring instrument. An in vivo calibration with balloon as
described above can be obtained with this pressure transducer.

TELEMETRY

With Telemetric Transducers the transducer is inserted and the skin
closed, so that there is no communication with the exterior. In many
ways it is ideal because it allows free mobility of the patient. The
major problem here is zero drift with time and temperature. Some of
these are undergoing clinical trials at this time. Most of them
tend to be inserted in the epidural space. They are quite expensive,
fragile and have manufacturing difficulties.

The Rotterdam Epidural Teletransducer of Delange et al (1977) is an
epidural telemetric transducer. They mention that the method would

eventually be suitable for outpatient monitoring, which would
be very useful indeed. The first requirement is to eliminate
the necessity for an open connection with the ventricles by
demonstrating that epidural pressure is consistently parallel
though somewhat lower than the V.F.P. (Corneos 1972, Dejong et al
1975).

The second requirement is to adapt the coplanarity principle of
Schettini et al (1971) when monitoring pressure at the brain
surface and other requirements include: it should be easily implanted
into a standard sized burr hole using inert materials and a trans-
ducer which is without significant zero drift over long periods of
time. It should be easy to sterilise, it should have a simple
detection unit and a low construction cost.

With these specifications, they developed a spring-loaded transducer
at the Central Research Laboratory of the Erasmus University.
It consists of a coil capacitor circuit. Alterations in epidural
pressure induce changes in the plate distance of the capacitator
and therefore alter the electrical characteristics of the transducer.
They are sensed by the external detection system, operating on the
transformer principle. Thus the detection system is coupled to the
transducer by electromagnetic waves between the transducer coil and
the detector coil. After a number of animal experiments they implanted
these in 3 patients and obtained encouraging results, the lack of
drift of the zero line and the satisfactory readings obtained after
long periods of time in animal experiments suggested that the devices
might be useful not only in clinically treated patients at risk but
also in patients no longer hospitalised but still at risk regarding
their intracranial pressure.

Olsen and Collins (1972) described a passive radio telemetric device
for insertion in the subdural space so that continuous recording of

pressure variations could be made by means of a surgically <u>implanted</u> <u>glass passive transsensor</u> which caused no tissue reaction and was not subject to long term pressure drift. The cranial vault was sealed and no external connections were necessary. The transsensor can be monitored immediately after implantation and has a milli second response.

Transsensors are passive telemetring devices without batteries or external wires and require one electronic part to perform the functions of a transducer, a modulator and a transmitter. They permit the ultimate in miniaturisation of telemetring sensors and possess unlimited life. The transsensor recognises a physiological pressure, and transmits it by means of its absorption of electromagnetic energy from an external oscillator, at a frequency related to the force acting on the transsensor.

There are other methods of estimating I.C.P. apart from those mentioned above. Brocklehurst (1978) is investigating the possible use of the C.T. scanner to evaluate pressure dynamics, computer cisternography to elucidate C.S.F. flow dynamics (Hindmarsh 1977) etc. but for practical everyday use in paediatrics, intra-ventricular cannulation or the methodology described in this thesis, the implantable epidural transducers and the more sophisticated fontano-meters appear to be of most use. No matter what method is employed, it is now almost universally accepted that a nocturnal record should be obtained as well as an awake record. The possibilities for reviewing 24 hours data are also numerous. It can be collected by a computer or microprocessor and displayed as a condensed record of pressure versus time, or a frequency histogram indicating the time during the 24 hour period when the I.C.P. level lay in a particular pressure domain. Alternatively it can be averaged every

15 minutes and visually displayed at the bedside.

For the future I should like to develop a miniaturised telemetric device which could be incorporated in the proximal tip of a Rickham ventriculostomy reservoir, so that an outpatient determination of I.C.P. using an external loop, could be routinely undertaken, and if raised pressure was detected, the reservoir would already be in situ for immediate pressure relief, and further elucidation.

CHAPTER 4

NORMAL PRESSURE VALUES

ZERO REFERENCE

MEAN PRESSURE .

THE PULSES

TRANSMISSION OF THE PULSE

SHAPE OF THE TRACE

LUMBAR PRESSURE RECORDINGS

   (a)   reliability of lumbar recordings

   (b)   hazards and contra-indications to L.P.

   (c)   the 'careful L.P.'

   (d)   three examples of L.P. recordings

SOME PHYSIOLOGICAL AND PATHOLOGICAL RESPONSES

CHAPTER 4

NORMAL PRESSURE VALUES

When a needle is inserted into the cisterna magna of a dog the
C.S.F. flows out of the needle into the tubing spontaneously,
indicating a pressure within the cranial cavity greater than atmos-
pheric: the actual pressure may be measured by finding the pressure
that must be exerted just to prevent escape of fluid into the needle.
This is the C.S.F. pressure (Davson 1967).

A number of investigators have reported normal pressure values:-

1. Merritt and Fremont-Smith (1937). They examined the lumbar
pressures in 1,033 apparently normal individuals with normal B.P.'s
and no intracranial pathology. They reported pressure ranges of
70-180 mm saline and they stated that pressures up to 180 mm are
normal, that pressures in the range 180-200 mm saline are doubtful
and pressures in excess of 200 mm saline are hypertensive. In a
frequency distribution curve their most frequently observed value
was at 150 mm saline.

2. Masserman (1934) also with a needle in the lumbar sac, averaged
recordings on 284 normal individuals and the pressure suggested was
148 ± 2 mm saline.

3. Longo, Daurado and Donreis (1950) examined 96 normal humans and
found the pressures varied between 55 and 195 mm saline.

4. Lups and Haan (1954) designated the normal range of ventricular
pressure in children as 3 - 7.5 mm Hg.

5. Spinafranca (1963) examined 1,500 patients in the lateral
recumbent position, with a needle in the cisterna magna. The mean
value was 119 mm saline, the range being 41 to 197. He considered
that values outside this were abnormal and that there was no sex

differentiation in the pressure levels. He did however find a slight drift to lower values with older subjects. When Spinafranca examined the lumbar pressures in the lateral recumbent position, values were found which were slightly higher, the mean value then was 167 mm saline.

6. Turner and Macdowall (1976) accepted that the normal range of intracranial pressure was 5 - 13 mm Hg and that the extradural pressure was 1 - 2 mm Hg higher than the ventricular pressure at normal intracranial pressure ranges. They, and others, however found that the extradural pressure was considerably higher than ventricular pressure at high intracranial pressure values (Coroneus 1972, Gobiet 1972, Jorgensen and Rishede 1972, Sundbarg and Normes 1972).

In 60 patients examined in Leeds, no patient with a normal epidural pressure had high ventricular fluid pressure (Gibson et al 1975, Turner et al 1975).

7. Bruce et al (1978) considered the range of normal intracranial pressure from the supratentorial space with a transducer zeroed at the foramen magnum, as 0-15 mm Hg and they considered that _sustained elevations above 20 mm Hg or waves of pressure to above 25 mm Hg were significant elevations of intracranial pressure._

8. Miller (1978) states that a catheter inserted into the lateral ventricle records a pulsatile pressure of 0-10 mm Hg (0-136 mm water) relative to the foramen of Munro when the patient is lying flat.

In normal circumstances however intracranial pressure may transiently increase to 100 mm Hg during coughing and straining.

In experimental animals similar values have been obtained -

1. (Hill 1896) 100 - 130 mm saline in dogs.

2. (Dickson and Halliburton 1914 40 - 70 mm saline in dogs.

3. (Becht 1920) 112 mm.   4. (Bedford 1935) 90 - 150 mm.

5. (Goldensohn et al 1961) 32 - 260 (mean 105). Usually these
recordings in experimental animals have been performed under
anaesthesia and therefore, apart from the species difference, may
not be reliable indicators.

Assuming that there are normal hydrodynamic conditions, the intra-
cranial pressure is equal to the lumbar theca pressure less the
hydrostatic component. This has been calculated by O'Connell 1943
and allowances have been made under those circumstances. However, one
cannot make an exact hydrostatic allowance in the ward if the patient
is at an angle to the horizontal, e.g. on a pillow, but Lundberg
suggests that in the steady state this does not exceed 2 mm Hg
usually.

ZERO REFERENCE

Even though when there is free communication the same pressure is
recorded from the ventricle, cisterna magna and lumbar subarachnoid
space when the same reference point is used (Langiftt 1969) the
lumbar zero is of little reference value as one needs to know what
effects are occurring in the head.

Ideally therefore, the choice of reference level should be at the
centre of the head, however, there is no clear consistent surface
mark to relate to clinically, so in practice the usual choice is
the upper cortical subarachnoid space (i.e. level with the forehead
with the patient supine) or arbitrarily at the level of the inter-
ventricular foramina (approximately half the distance between the
forehead and occiput with the patient supine).

The pressures obtained should be expressed as torr or mm Hg as
both arterial and venous pressures are expressed in this way.

The pressure curve gives information about wave forms and the level

of ventricular pressure is all that is usually needed in the
clinical situation.

MEAN PRESSURE

This has been defined variously,(Guillaume & Janny 1951) denoted
this to mean the height of pressure which included the pulse and
respiratory waves and small cyclical variations with a frequency
of 0.5 to 12 per minute, but no fluctuations of larger duration
or amplitude.
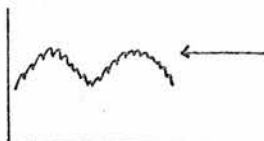
Goldensohn 1951 and Ryder et al 1953 discuss their mean as being
midway between the deflections of respiratory and cardiac origin
and by repeated manual measurements without the aid of wave
processors, this is the level which I have employed in these
recordings as the mean pressure level.
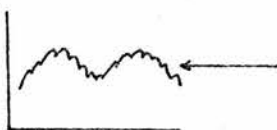
More correctly one should calculate the mean I.C.P. in the same
way as arterial pressure i.e. diastolic pressure + one third
(systolic-diastolic) because the respiratory wave becomes less
significant with increasing I.C.P. For the stress pressure values
and sleep values in this series, unlike the mean pressure estimates,
I have measured from the peak of the cardio-respiratory oscillations.

The width of these cardiac and respiratory wave forms depends on
their own original amplitude, and also any damping within the system.
Lundberg could, by compressing the tubing in front of the transducer,
affect the damping of his recording system and he also noted that on
sub-maximal constriction of the lumen he obtained a line readout,

which was the mean of all variations ironed out by this damping
effect. Lundberg used this to calculate his mean pressures, reading
them every $2\frac{1}{2}$ minutes for 1-2 hours. That is, his mean pressure is
that which included all the fluctuations of the ventricular fluid
pressure.

THE PULSES

Two wave forms are visible on the ventricular pressure record, a
rapid oscillation corresponding to arterial pulsation and a slower
wave form synchronous with respiration. They are produced by changes
in intrathoracic pressure (Bradley 1970).

In cats (Weed and McKibben 1919) found a cardiac pulse of 1 mm saline
with a respiratory variation of 4 - 5 mm saline. They noted that there
was a rise towards the end of expiration and a fall during inspiration.
In man (Howe 1928) reported a cardiac pulse of 2 - 3 mm saline and
(Fremont-Smith and Forbes 1927) stated that generally the respiratory
variation was five times greater than the cardiac.

(O'Connell 1943) found that the variations were considerably greater
by using a wide bore needle thus reducing the damping effects. From a
cardiac variation of 15 mm saline and respiratory variation of 35 mm
saline, he calculated that the true variations in the absence of damping
would be larger, namely 20 and 60 mm saline. (Goldensohn et al 1951)
using a wide bore needle in the cisterna magna of dogs with a strain
gauge found a cardiac pulse of 20 mm saline and a respiratory
variation of 35 mm saline. (Bering 1955) found a cardiac variation
in the ventricle of a dog of 50 mm saline and he showed that damping
of an open ended manometer reduced it to 28 mm saline.

A rise in the fluid pressure results in an increase in the amplitude
of the cardiac and respiratory variations. Goldensohn found average

variations of 70 mm saline cardiac and 75 mm saline respiratory
(when the fluid pressure was the order of 300 mm saline), compared
with values of 20 and 35 mm saline when the fluid pressure had its
normal average value of 100 mm saline. The reason for this is that
when the fluid pressure is raised, the venous pressure is high, and
a pulsatile increase in the diameter of the blood vessels cannot now
be compensated for, by a collapse of the veins. The arterial pressure
changes are thus not strongly damped and are reflected in a high
fluid pulse pressure (Davson 1967).

TRANSMISSION OF THE PULSE

(Hering 1955) concluded that the pulse probably was transmitted from
the choroid plexus. From experiments he did involving plexectomies
he suggested that the cerebral arteries contributed little to the
cardiac pulse. In similar experiments he confirmed that the pulses
of 62 mm, 49 mm and 29 mm saline respectively from the ventricle,
cisterna magna and lumbar sac, resulted in mixing of the fluid. The
time course of the cardiac pulse from the ventricle was measured and
he found that the pulse followed immediately after systole, and was
unconnected with the central venous pulse. More recently (Dunbar,
Guthrie and Carpell 1976) have confirmed the close relationship between
the arterial and fluid pulses. With other experiments they concluded
that the transmission of the pulses to the cord was not merely by
means of the C.S.F. and they concluded that the arteries within the
skull and spinal column contributed to the pulse directly,e.g.with regard
to the spinal pulse they proved by blocking the aorta above the take
off of the subclavian artery, the pulse was abolished in the spinal
theca. Also the time relations of the lumbar pulses indicated
transmission from the lower aorta, rather than from the choroid

plexus. Therefore the spinal pulse originated mainly from transmission from the thoracic and lumbo sacral arterial connections.

SHAPE OF THE TRACE

I have stated in an earlier publication, and it has been said more recently by 'Bruce et al 1977) that if one either cannot undertake infusion studies for compliance and elastance (because of using a subarachnoid screw etc.) or if infusions or withdrawal of C.S.F. methods have not been undertaken as suggested by (Marmarou and Shulman 1976), then an estimate of the intracranial elastance can be obtained by observing the responses to a given physiological stress or small changes in PCO_2, central venous pressure or systemic arterial pressure etc. The importance of these concepts to patient management is that intracranial pressure waves may occur without causing any alteration in the patient's state or cerebral haemodynamics, however, with progression, these small changes in volume will not be readily compensated for, and may lead to a sudden large pressure wave with clinical deterioration or cessation of cerebral flow, and therefore one wants to know what the residual compliance is left within the cranium.

The particular responses to given physiological stresses in children are remarkably constant in the same child. Also, in young infants and neonates, the ratio of maximum stress pressure to the mean resting pressure is larger than the ratio in older children.

These observations reflect a single dynamic state which is fairly constant at any particular time, but which changes with growth.

If the pressure does not return to the pre-stress level in a certain time then one can conclude that there is diminished compliance.

Compliance and elastance estimations are obviously of more importance

in states of head injury and gross brain swelling than in the bulk
of the cases seen in this department, namely hydrocephalics.

LUMBAR PRESSURE RECORDINGS

The value and preciseness of objective measurements from the
lumbar theca done in the past is open to question, mostly because
open-ended manometry with C.S.F. displacement from an otherwise
closed system must have recorded falsely a slightly lower pressure.

It is obvious that controls are unethical for the measurement of intra-
ventricular pressure and cisternal pressure and we are left with the
only other possibility, that of a non-displacement method using a
pressure transducer for measuring the lumbar theca pressure. The
paediatric situation, where a lumbar puncture is indicated, (e.g.
suspected meningitis) where the C.S.F. ultimately proves to be clear,
is one example of how a collection of pressure recordings, over the
different childhood age groups could be compiled thus defining more
accurately normal pressure ranges with growth.

The use of a miniature pressure transducer, such as the one in use
during this study, has obvious advantages for routine use in the
treatment rooms of paediatric units, as it can be applied directly
to the spinal needle, without displacement of C.S.F., and a short
recording of about 5-10 minutes obtained.

Two main areas however need to be investigated: firstly the
reliability of lumbar recordings in some paediatric neurological
conditions and secondly the possible hazards of employing a lumbar
recording.

(a) <u>Reliability of Lumbar Recordings</u>

(Smyth and Henderson 1938) and (Kaufman and Clark 1971) observed that <u>internal herniations</u> with <u>aqueduct obstruction</u> or other form of <u>non-communication</u> cause a normal or falsely low pressure to be recorded despite raised pressure above the level of the foramen magnum.

We do not know how reliable the lumbar recording is when there are states of <u>mild generalised or localised brain swelling either occurring de novo or associated with hydrocephalus</u> and such a study would provide valuable information as to its usefulness in these states. We do know however that the recorded lumbar pressure is a true reflection of I.C.P. when the 'mean pressure' is within normal limits, provided the reference point is correct.

(b) <u>Hazards and Absolute Contra-Indications to Lumbar Recordings.</u>

Not only will known <u>internal herniations</u> (tentorial or cerebellar) record a false pressure but they are distinctly hazardous, as coning and medullary compression may occur with fatal results. Other situations in which L.P.'s are frankly contra-indicated include signs suggestive of a <u>posterior fossa lesion</u>, or indeed a suspected <u>tumour</u> or <u>abscess</u> anywhere within the cranium. Any suspicion of an <u>acute epidural haematoma</u> or other <u>external collection</u> is a further contra-indication.

The consternation of clinical signs contra-indicating L.P. is therefore quite diverse but in general includes: a combination of long tract and cerebellar signs with papilloedema, severe papilloedema alone, i.e. with haemorrhages, acute sutural splaying in children of the toddler age group and above, a combination of diminished conscious state with severe papilloedema, focal neurological signs in combination with papilloedema, investigative evidence of internal herniations, tumour or abscess.

The use of an L.P. in any condition with a suggestion of R.I.C.P. is contra-indicated for junior medical staff but may be advanced in certain other conditions where there is clinical evidence of R.I.C.P.

(c) The 'Careful L.P.'

There are clinical situations when R.I.C.P. exists, but it is imperative that C.S.F. is obtained, e.g. suspected C.N.S. infections or suspected subarachnoid haemorrhage. In haemophilus meningitis, which is quite common in childhood, the signs of R.I.C.P. are likely to be due to a combination of brain swelling (from associated cerebritis) and diminished C.S.F. absorption through inflamed meninges.

Tuberculous meningitis and cryptococcal meningitis may present with a multiplicity of neurological signs associated with papilloedema and again it is imperative that C.S.F. is obtained and on some occasions intrathecal therapy commenced. With tuberculous meningitis the argument could be advanced that with an early developing hydrocephalus, the danger of L.P. precludes this approach to the C.S.F. Obviously where entry to the ventricular C.S.F. is available in these conditions (through a burr hole or fontanelle) C.S.F. should be obtained from here first. This does not give complete peace of mind, even if clear C.S.F. is obtained, as localised spinal meningitis may be present.

Summarily, therefore, the careful L.P. may be undertaken and pressure recording obtained when there is a suspicion of subarachnoid haemorrhage, meningitis or encephalitis. Its use in tuberculous and cryptococcal meningitis is still doubtful.

For a 'careful L.P.' one should use the smallest possible bore needle, and regulate slowly the removal of not more than 10 drops of C.S.F. (this should be sufficient for staining, glucose and protein estimations and culture). These should be adequate cardio-respiratory resuscitation

equipment close at hand as well as a loaded syringe to replace fluid if coning appears likely. It is also worthwhile giving an infusion of Mannitol prior to these 'careful L.P.'s' and although this will upset the pressure recording, the aim in this situation is to obtain C.S.F. and not primarily to record the pressure.

At the Royal Hospital for Sick Children we are in the process of collecting a number of recordings during routine L.P's in children. Routine antiseptic precautions are taken and the skin dried thoroughly prior to insertion of the spinal needle. In general the child's legs are extended, there is no abdominal compression and the head is in a 'neutral' position. As posture is obviously important for the interpretation of these recordings, it has to be balanced against the safety and comfort of the patient.

(d) Three Examples of L.P. Recordings

Example 1

In this case (Fig. 11) is seen the effect of deflexing the head (b) and the effect of flexing the head (c) and again deflexing the head at (e). The other points (a) indicate a sigh and (d) a cry. This was in a child admitted febrile and irritable at the age of 8 months who, prior to admission, was treated with penicillin and ampicillin. A 20 gauge $1\frac{1}{2}$" needle was used to obtain this record. The earlier part of the record with a wide cardio-respiratory pulse was associated with the child being sleepy but she was awakened and disturbed by the head positioning. The effect of flexing the head causes an increase in pressure, no doubt due to jugular compression and so the level of intracranial pressure in this child is of the order of 7 mm Hg. C.S.F. was then sampled and was shown to have 50 polymorphs microscopically but no growth and no organisms and she was treated as a case of partially treated meningitis.
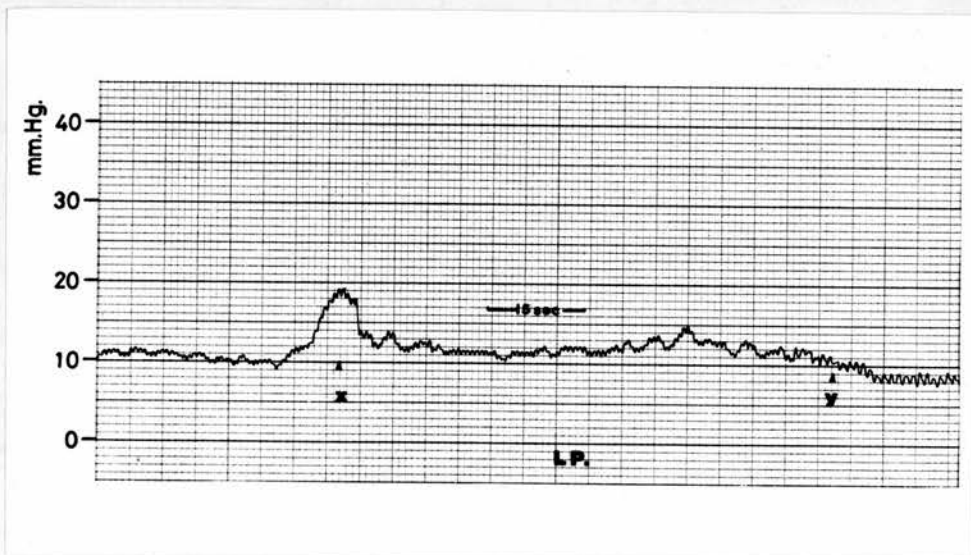
Fig.11

Fig.12

Example 2

In this case (Fig. 12) is shown the lumbar recording from a child
aged 14 with a past history of seizures and syncopal attacks and
with persistent right hemiplegic signs. She was positioned in the
left recumbent position and a 20 gauge 3" needle was inserted. It can
be seen from the record that the level of pressure is within normal
limits and for demonstration purposes at point (x) a left sided
Queckenstedt's test was performed showing an appropriate elevation
in pressure. I think that Queckenstedt's testing has little value
in the paediatric situation and although it is said to be diagnostic
if there is complete spinal block, resulting in no elevation in the
lumbar pressure, this block must be complete and this situation must
be extremely unusual as any condition leading to even a minute
communication, results in a negative Queckenstedt's. It is routinely
done on monitoring pressures above the level of the foramen magnum
where there is no problem with releasing C.S.F. Similarly a Quecken-
stedt's variant the 'tobey-ayer' test looking for unilateral failure
of rise of the lumbar pressure with transverse sinus obstruction is
of little value. At point (y) the child was asked to overbreathe and
one can see a depression of the ventricular pressure level for a short
period.

Example 3

In Fig. 13 and Fig. 14 an infant with brain stem fits, severe
opisthotonous, inappropriate A.D.H. due to a viral encephalitis, is
seen to have a lumbar pressure level of 15 mm Hg with stress response
deflections. This would indicate that this child who had no intra-
cranial space occupation or advanced hydrocephalus but a degree of
brain swelling due to encephalitis (evidenced further by swelling of
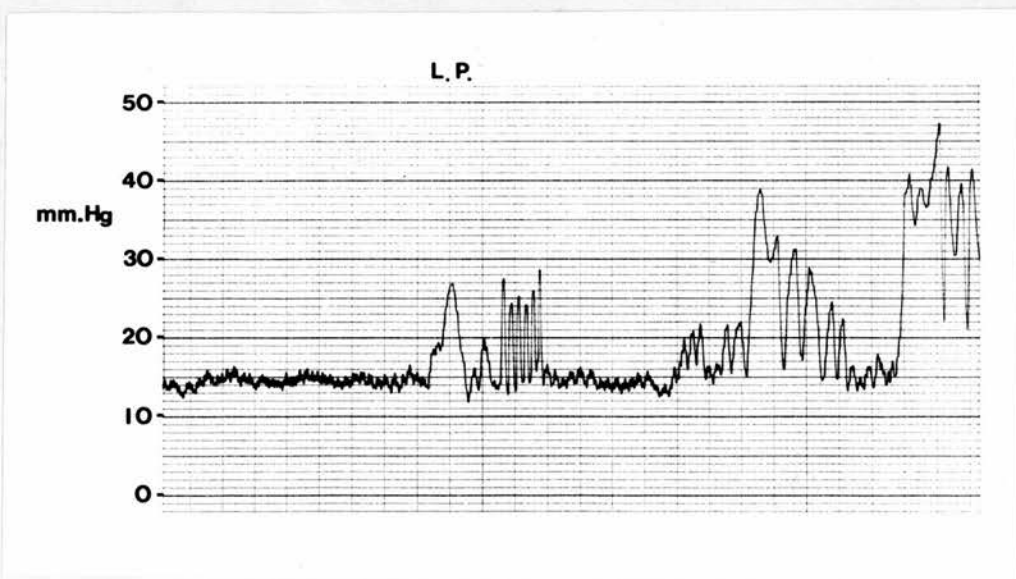
Fig.13

Fig.14

the face and neck) records a pressure compatible with his clinical
state. Further experience with this situation is needed.

In neonates it is my impression that the recorded lumbar pressures
(Fig.15) are marginally lower than older infants or toddlers.
Positioning of these children in the neonatal period is critical and
one should be careful to avoid the mechanical restriction of ventilation
in infants with respiratory distress syndrome (Margolis and Cook 1973).
In the past it has not been thought necessary to measure lumbar theca
pressures when L.P's were done for reasons not mentioned earlier,
such as during a lumbar air encephalogram, lumbar myelography,
installation of therapeutic agents for meningeal leukaemia, Guillian
Barré syndrome, subacute sclerosing panencephalitis, pseudo-tumour
cerebri etc. However, now, with non-displacement methods of safe
measurement, there is no reason why a pressure line should not be
recorded for 5-10 minutes as part of a routine collection of normal
data.

We do not know what effect a raised C.S.F. protein has on the level
of C.S.F. pressure, but there may be a minimal correction factor. In
premature infants with a volume of C.S.F. of 10-30 mls (Otila 1948)
the C.S.F. protein is greater than that of full term infants with a
total C.S.F. volume of 40 mls. This is also greater than the C.S.F.
protein in older children and adults who have a C.S.F. volume of
110-140 mls. At any age the protein in the ventricular fluid is less
than that in the lumbar fluid (Bell and McCormick 1978) and the C.S.F.
protein is increased by 1.5 mgm/100 ml for every 1,000 red cells in
the C.S.F. (Taurtellotte et al 1958). The mean level of recorded
pressure will not be influenced by C.S.F. protein changes or blood
stained C.S.F. However, the frequency response to the transmitted
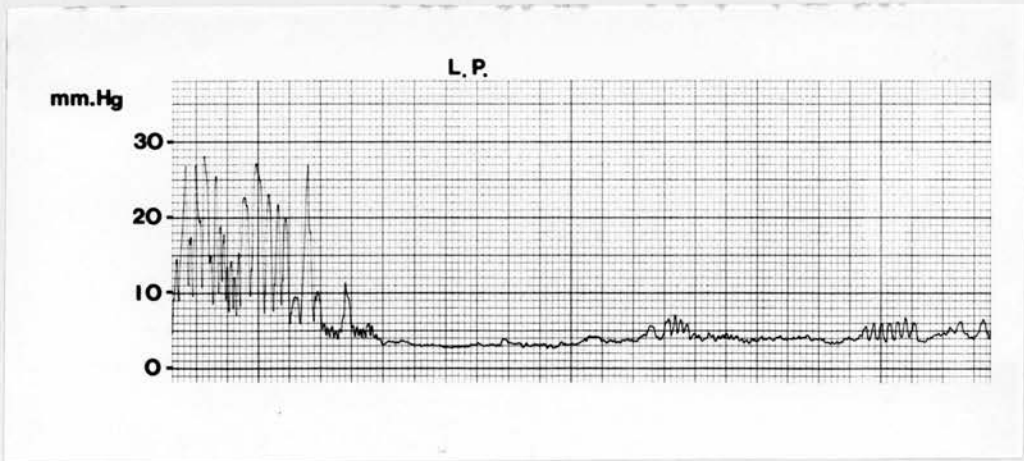cardio-respiratory waves will vary slightly in states of extremely
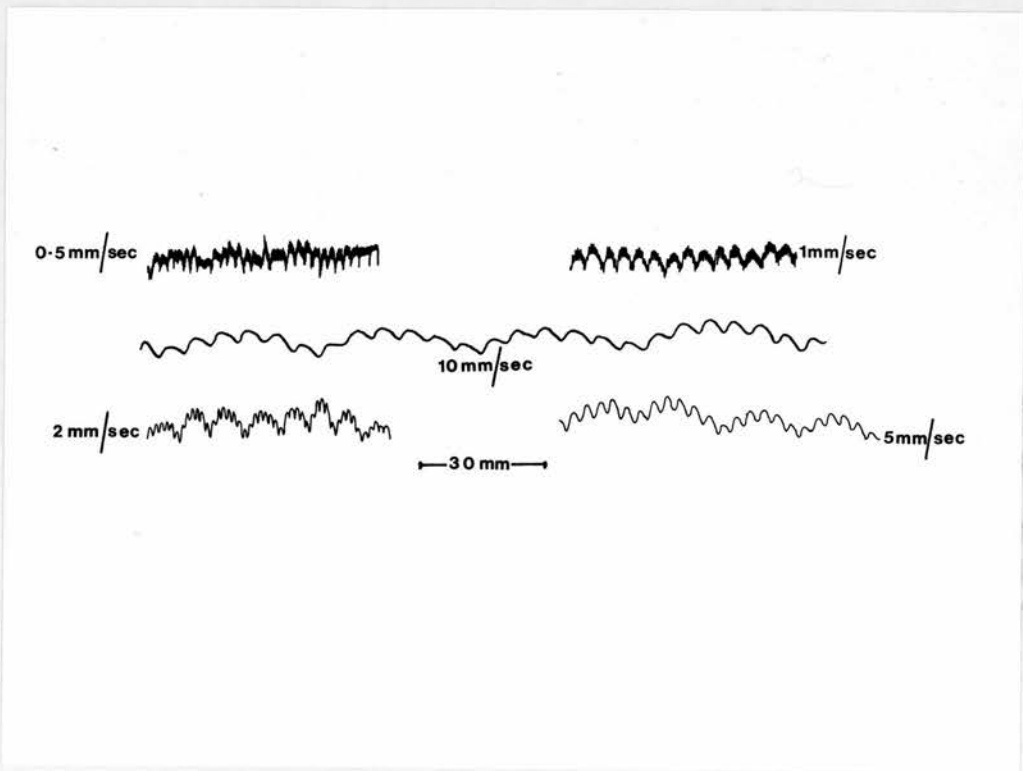
Fig.15

Fig.16

raised protein and possibly lead to damping of the cardio-respiratory band, but this should not greatly influence its practical application.

SOME PHYSIOLOGICAL AND PATHOLOGICAL RESPONSES

The absolute pressure values of these responses are not important as they will vary from patient to patient. They are illustrated here predominantly to show the pattern or distortion of the pressure recording which occurs with a given stimulus.

Recognition of the different wave forms which occur with different recorder speeds is seen in Fig. 16 with compression of the cardiac and respiratory waves into a band at slow recorder speeds and expansion of it at faster speeds. The variability of the fluid pressure in the same subject, from one moment to the next, was observed by (Riser 1929) with his observations that a cough in one human subject, produced a rise from 25 to 200 mm saline while a sneeze raised it to 280 mm. There are numerous accounts of the effects of psychic stimuli (Ayala 1923). See Fig. 17 the pointers indicating substantial rises in I.C.P., when an emotional welling-up occurred, without tears, in a child with a mean pressure level of 20 mm Hg, when the child's mother left the ward.

Figure

18 -   shows a complete bowel movement.

19 -   shows an unusual inspiratory type of cry with negative
         deflections being inspiration.

20 -   shows crying in an older child.

21 -   'girning'

22 -   crying and cessation of crying at two different recorder speeds.

23 -   crying at fast recorder speeds.

Figure

24 – shows 'retching' at the points indicated on the graph.

25 – shows 'possetting' of 15 mls clear fluid after feeding.

26 – vomiting.

27 – shows the passage of a nasogastric tube at the first
      arrow head inducing a 'sneeze' at the second arrow.

28 – at the first arrow a left sided Queckenstedt's response,
      and at the second arrow a bilateral Queckenstedt's response.

29 – shows a right and left Queckenstedt's with superimposed
      coughing responses.

30 – shows responses with head positions to the right and left
      with similar responses in amplitudes of pressure, with right
      and left Queckenstedt's.

31 – the arrow indicates shouting and there are two respiratory
      components per deflection.

32 – shows hiccoughs in a $4\frac{1}{2}$ month old child with normal pressure.

33 – the arrows indicate vigorous sucking on a bottle, in a
      6 week old infant.

34 – shows coughing responses.

35 – laughing.

36 – a rectal examination.

37 – an air ventriculogram showing removal of 24 ccs of C.S.F.
      at the first arrow and insertion of 24 ccs of air at the
      second arrow. The points on the graph indicated 'C' are
      cough responses.

38 – shows the pressure changes during an I.M. injection.

39 – shows the responses in a child blowing up a balloon.

40 – sneezing.

Figure

41 –   abdominal pressure 'A', in the right upper quadrant,
         'B' in the epigastrium, 'C' in the left upper quadrant.

42 –   shows micturition (between the arrows) in a 12 year old
         boy.

43 –   shows micturition in a pre-school child.

44 –   passage of flatus.

45 –   yawning twice at the positive deflections shown.

46 –   shows continuous head movement effects on elevated
         ventricular pressure.

47 –   shows changes in body position, at the first arrow the
         child is sitting up, at the second arrow the child is lying
         horizontal with eyes front.

48 –   shows the effect of struggling.

49 –   in a child with a non-functioning valve, the first arrow
         indicates abdominal palpation resulting in a hepato-
         jugular reflex and elevation of the ventricular pressure,
         the second arrow shows the effects of pumping the valve
         twenty times.

A number of other physiological phenomena affect the ventricular
pressure level, e.g. feeding, nappy changing, other nursing
activities and the important effect of tracheal suction and toilet
(James et al 1977). Various other pathological situations which
influence the I.C.P. are depicted throughout Chapters 12–15, e.g.
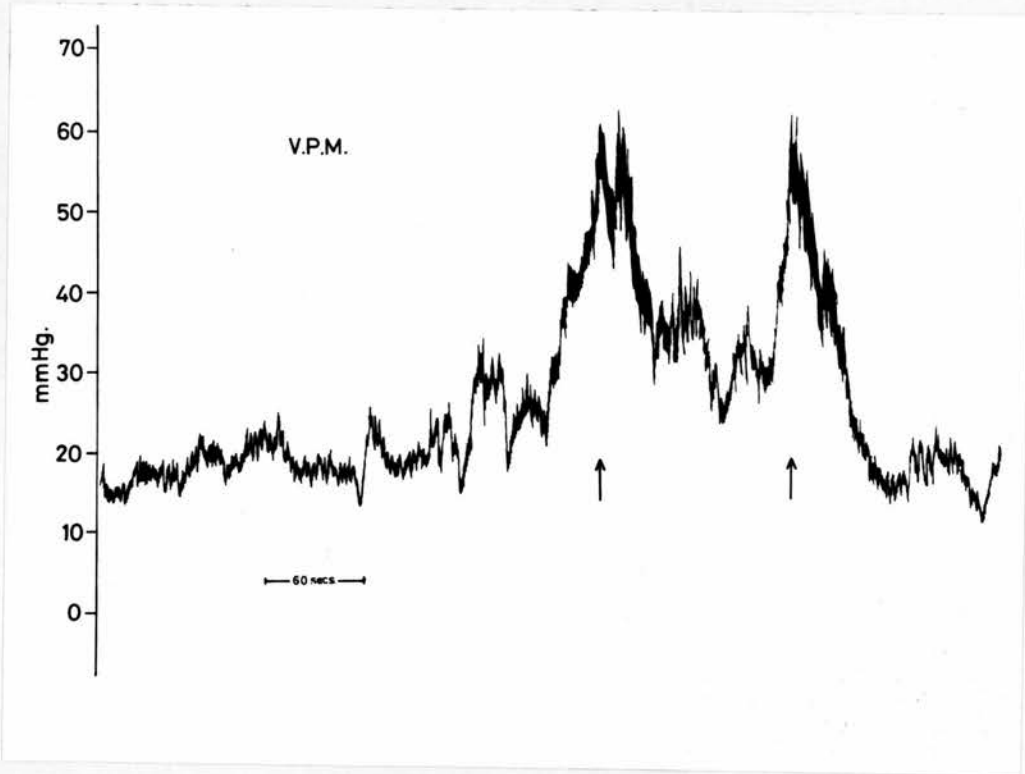the effect of C.S.F. removal, plateau waves, sleep, decadron,
applied pressure over the fontanelle etc.

Fig.17
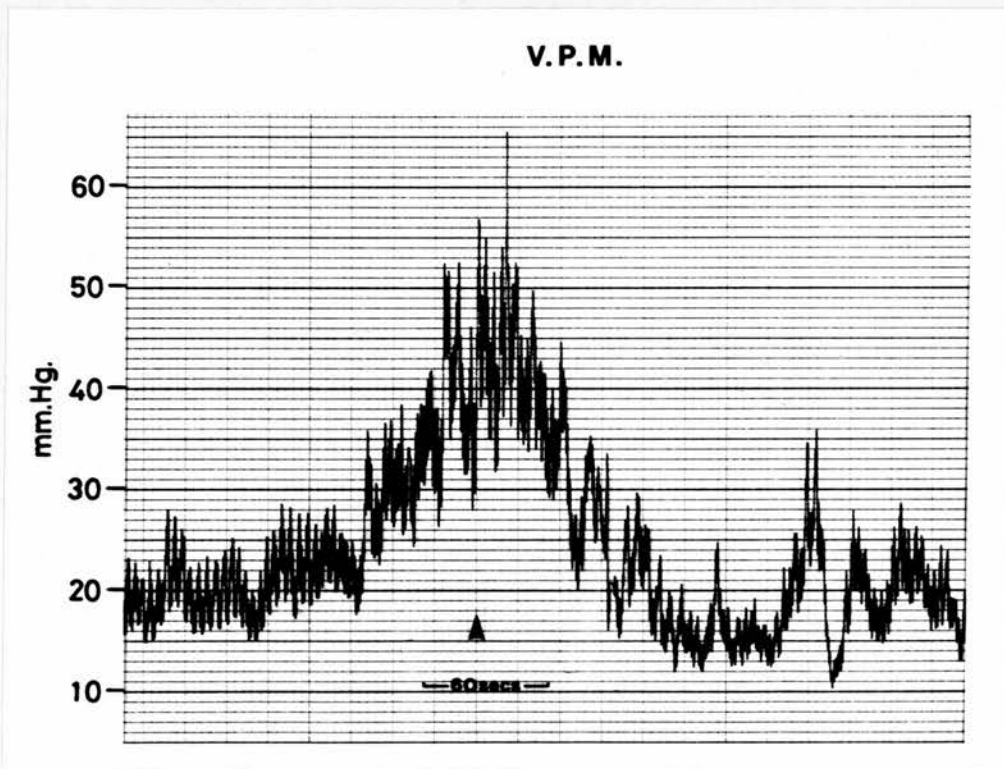
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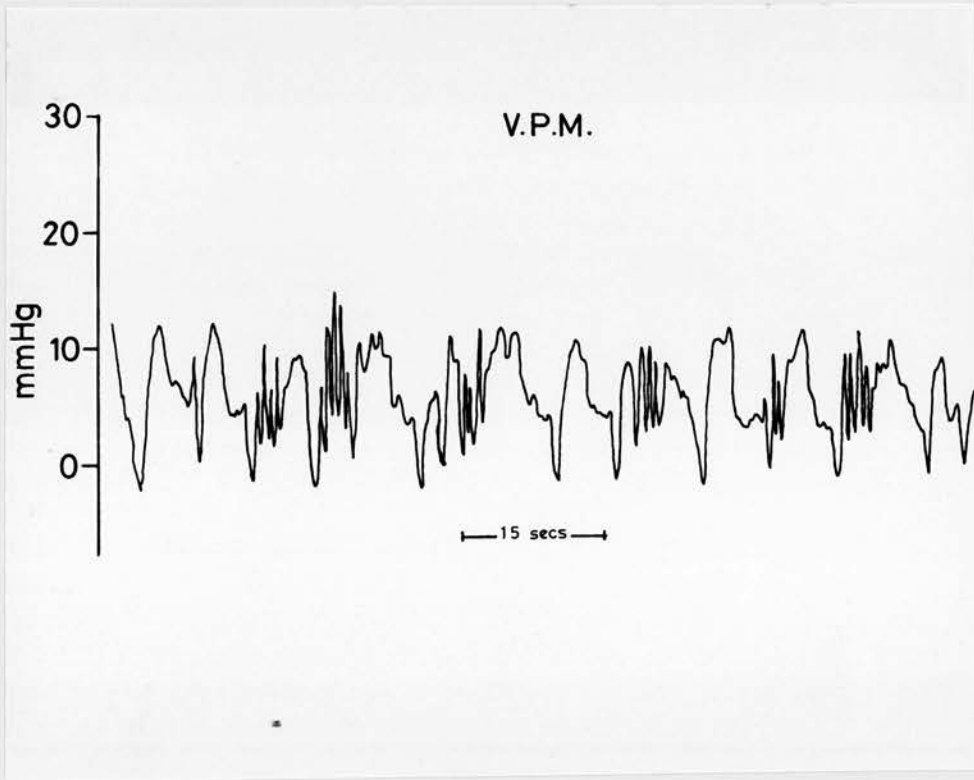
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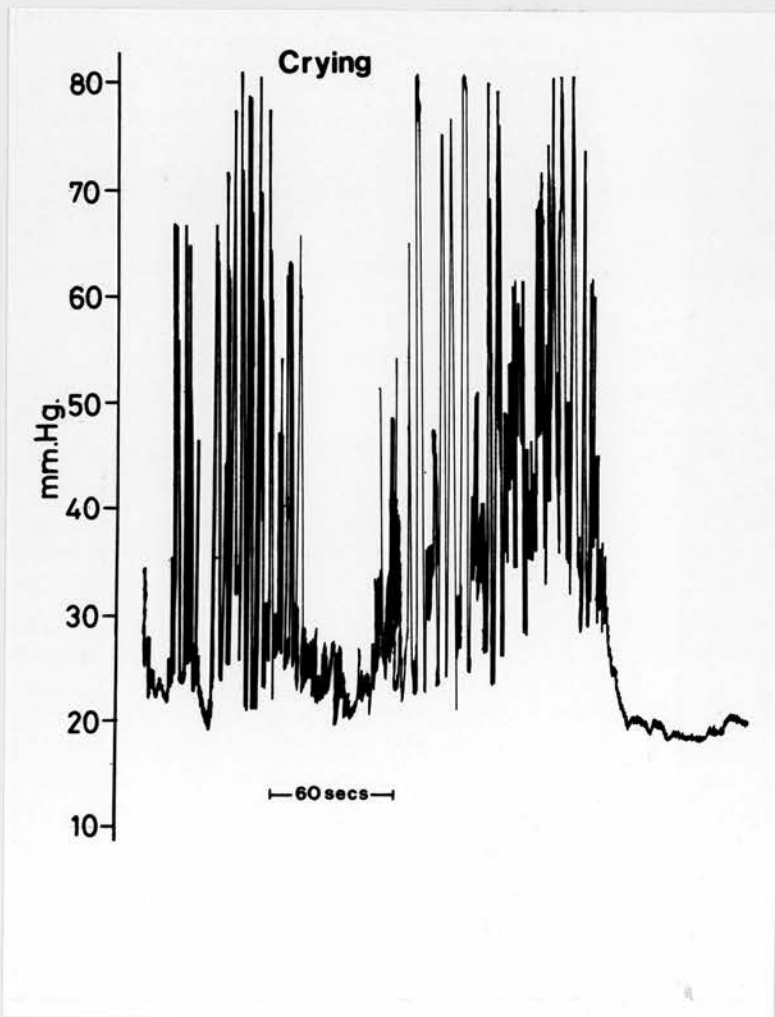
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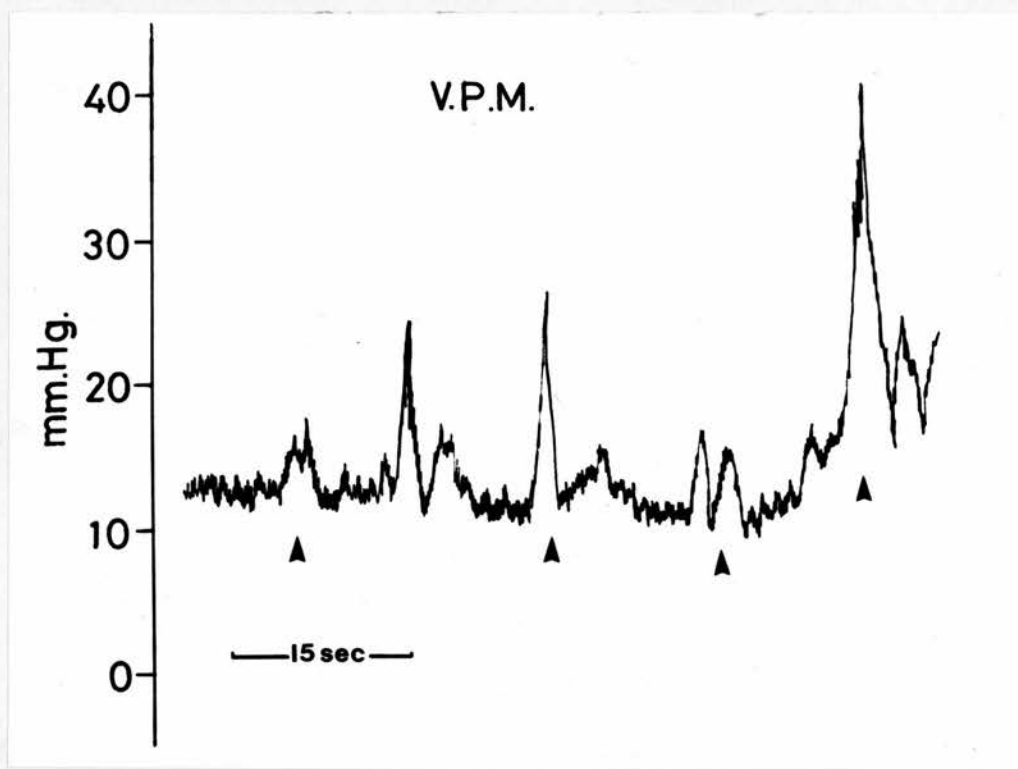
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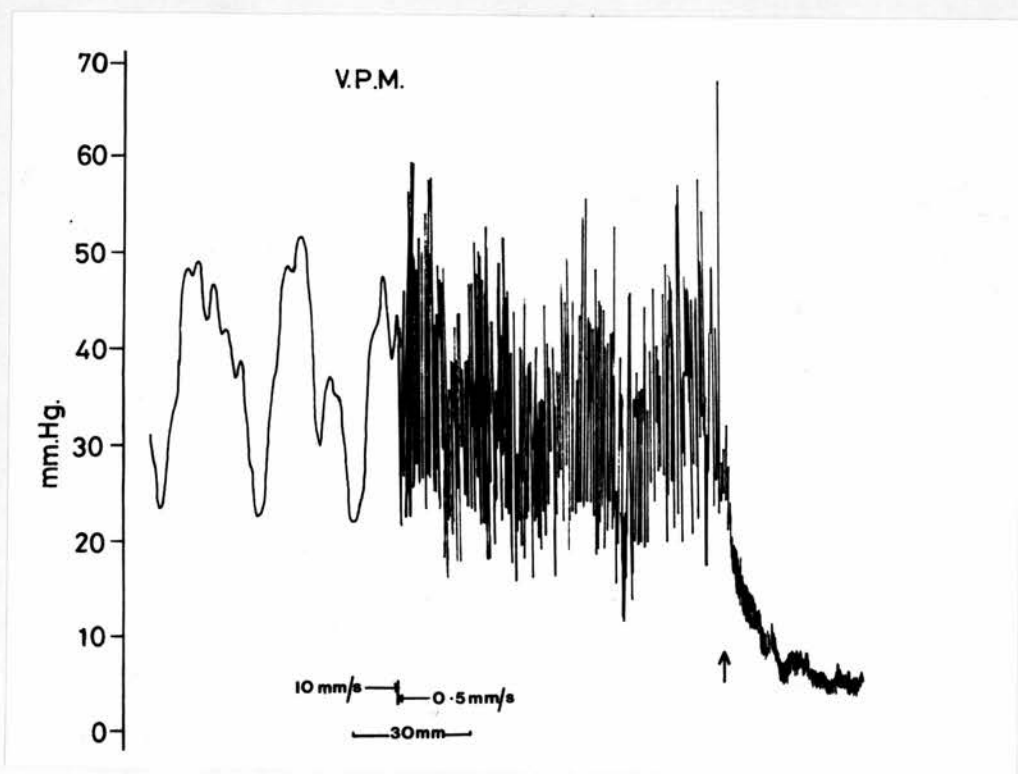
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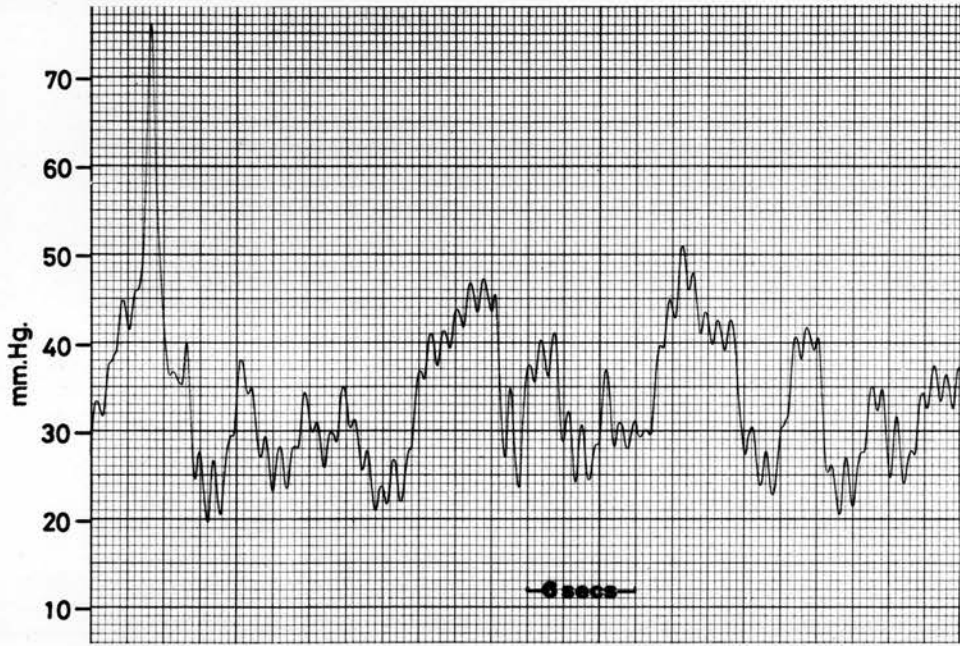
Fig. 23

Fig. 24

Fig. 25
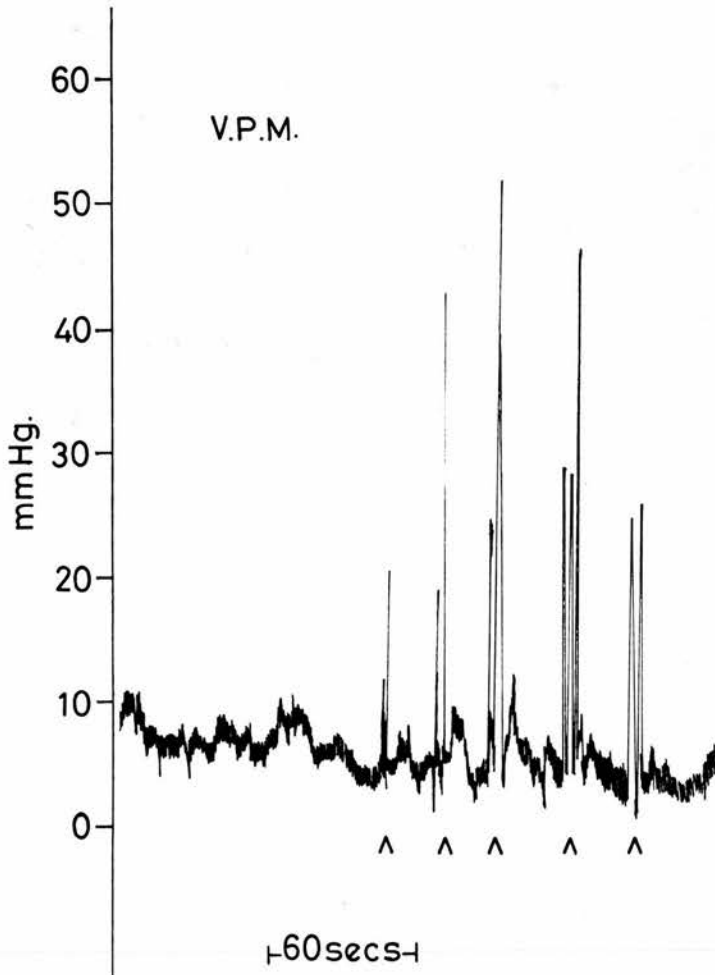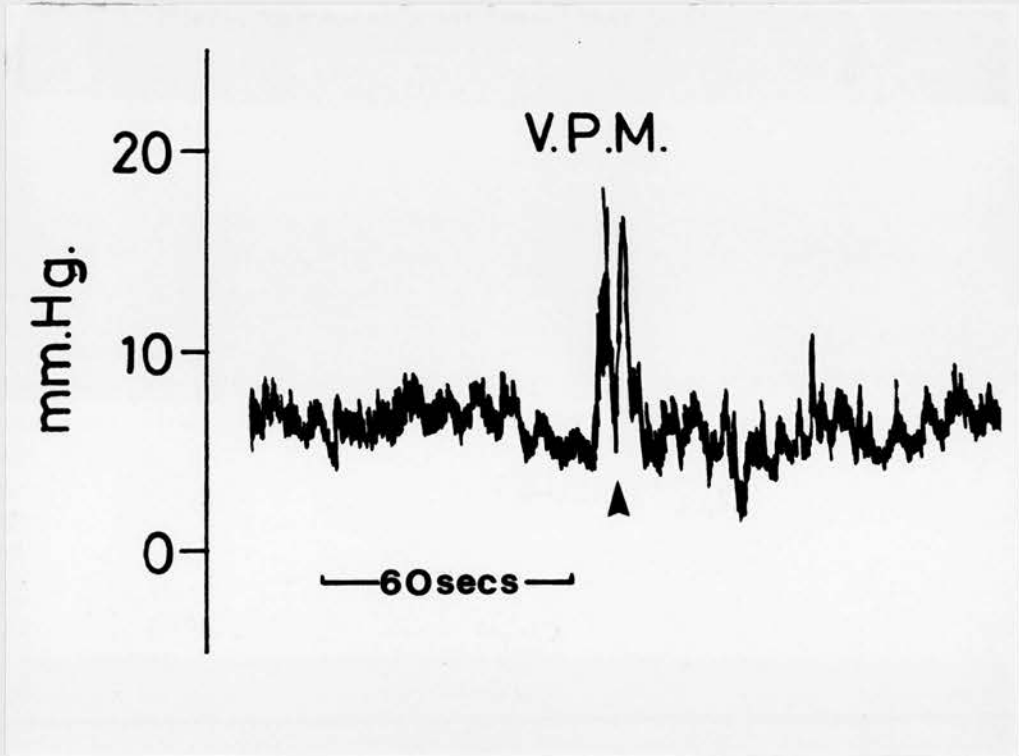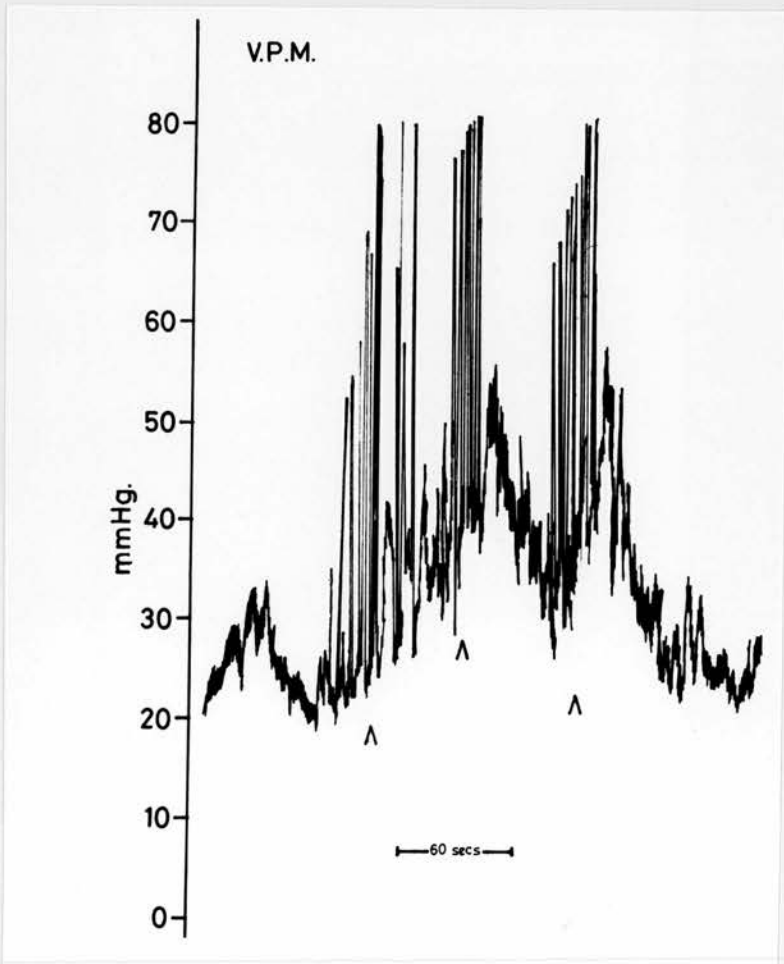

Fig. 26
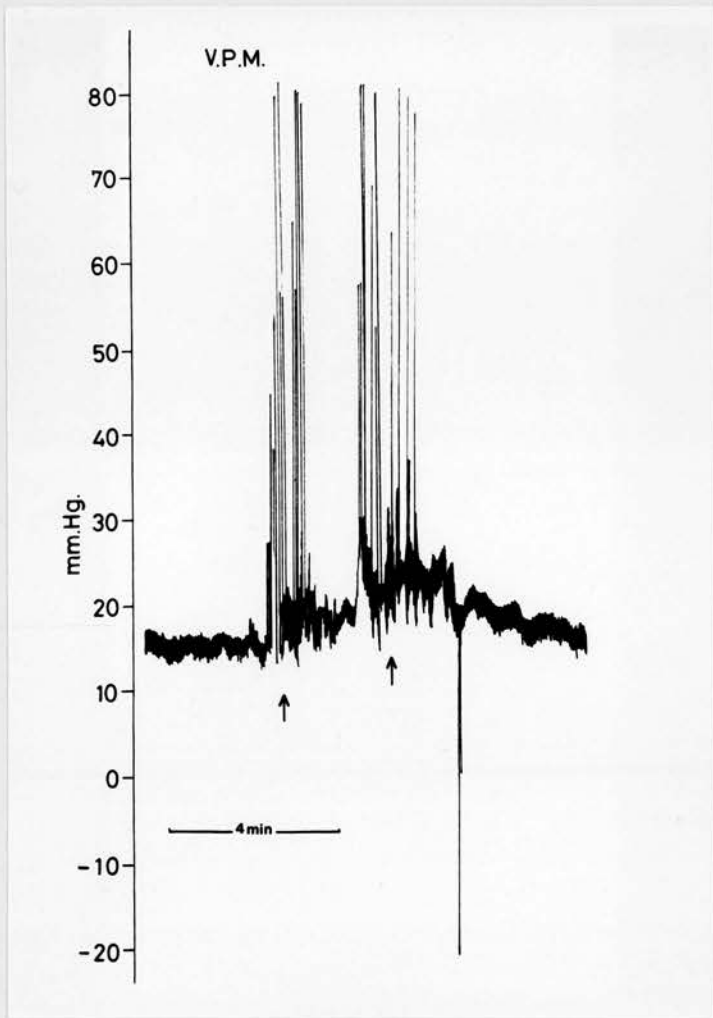
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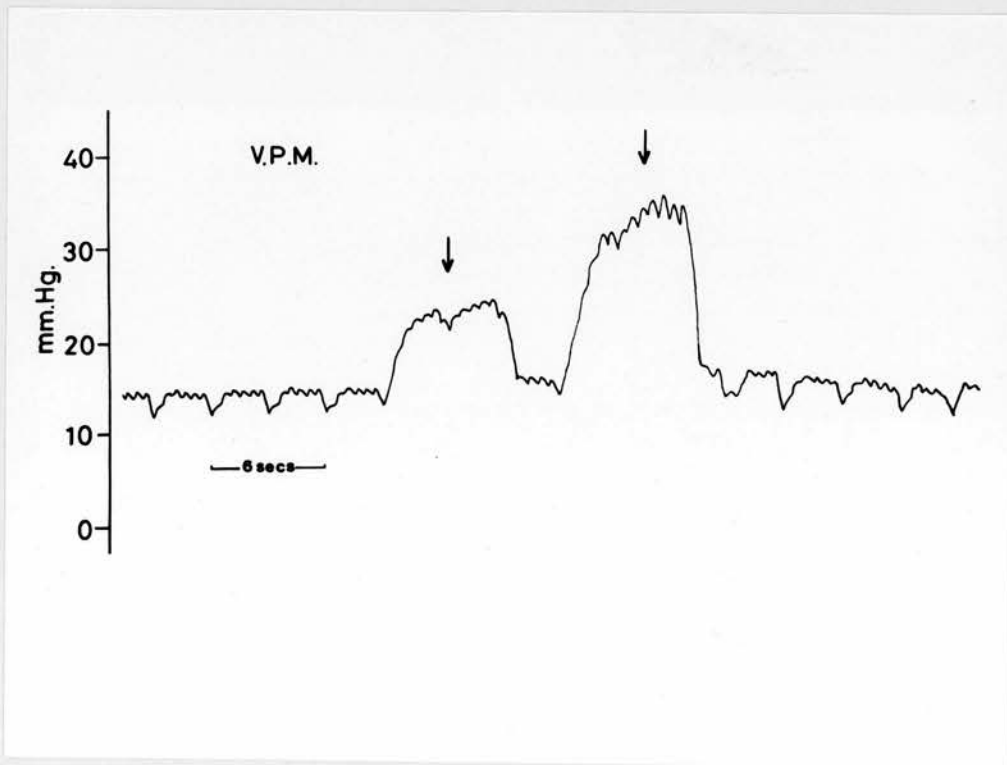
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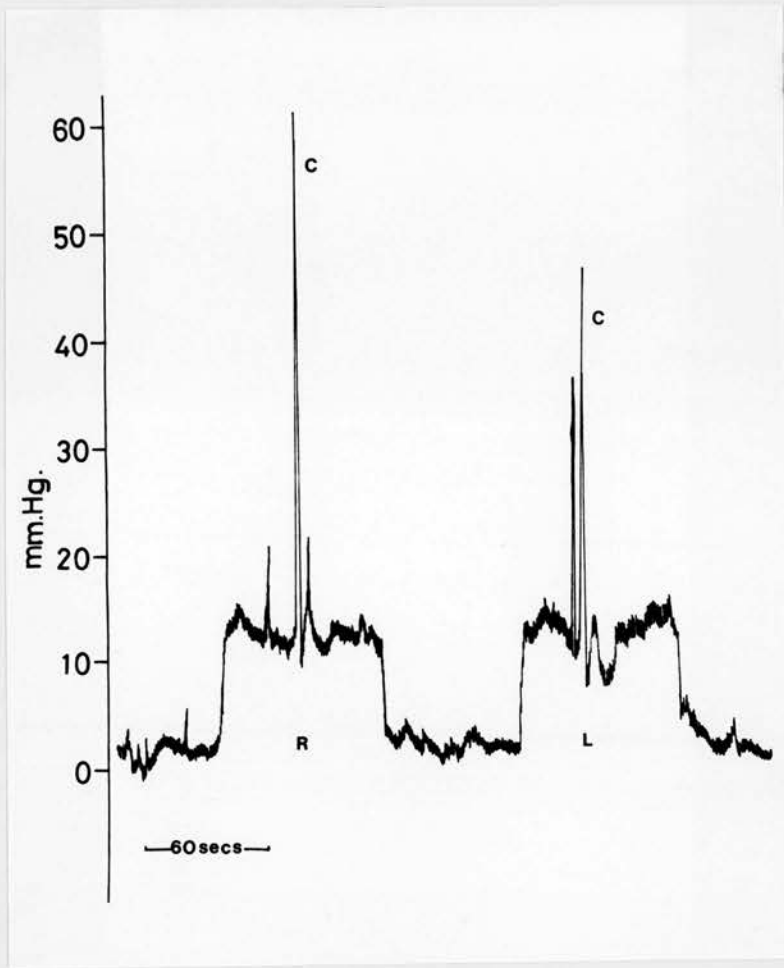
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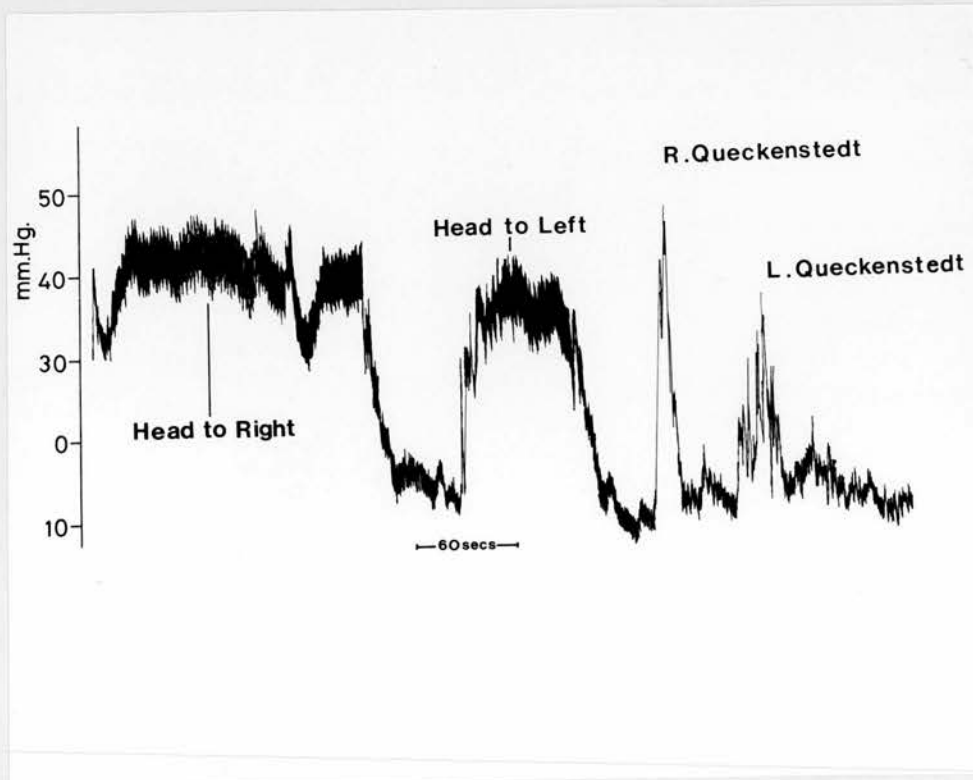
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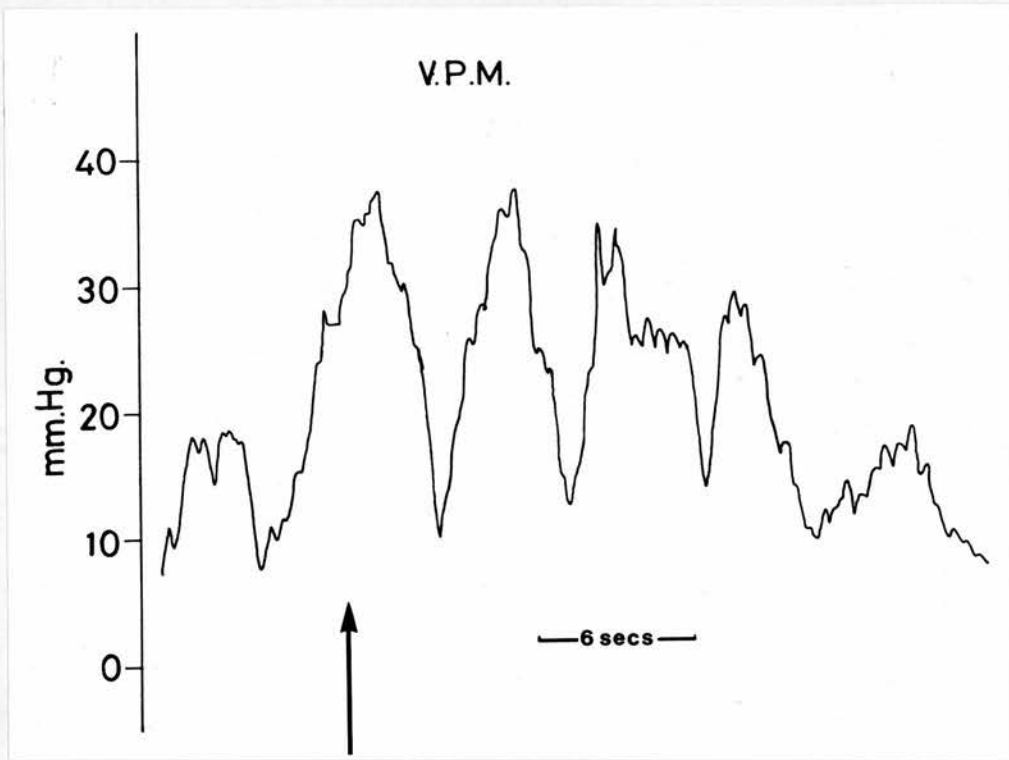
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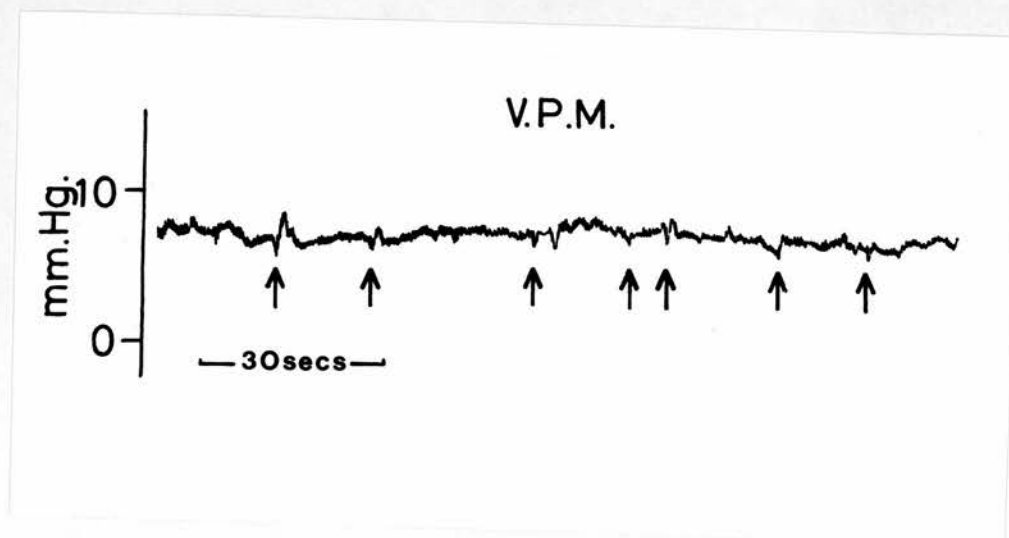
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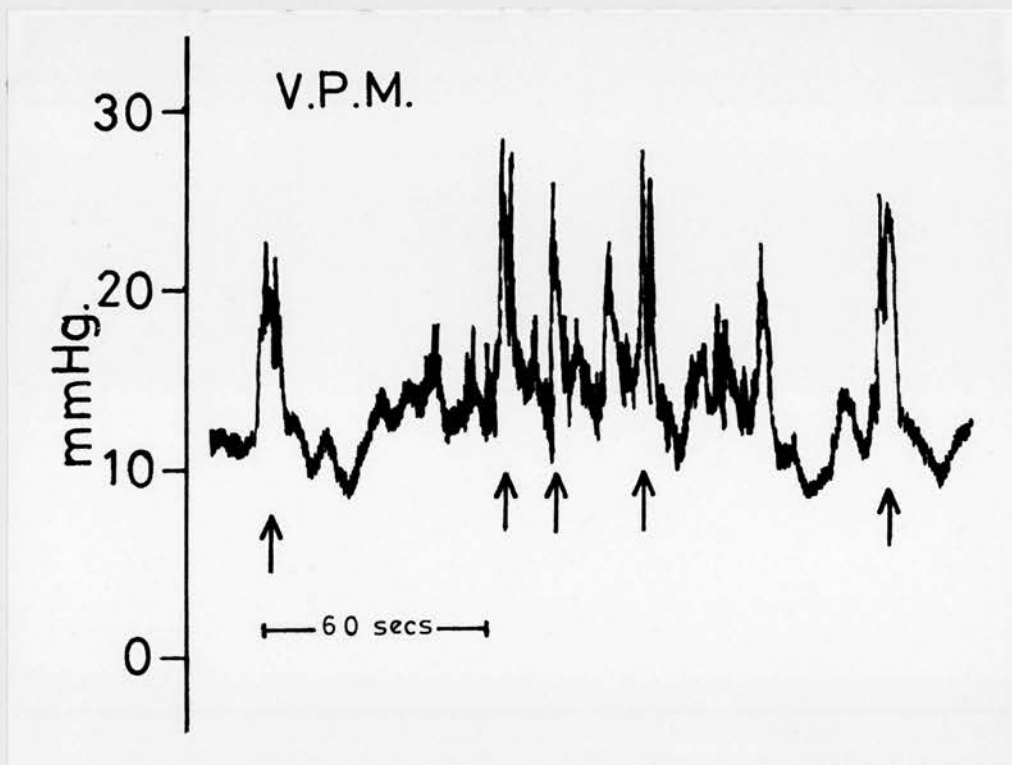
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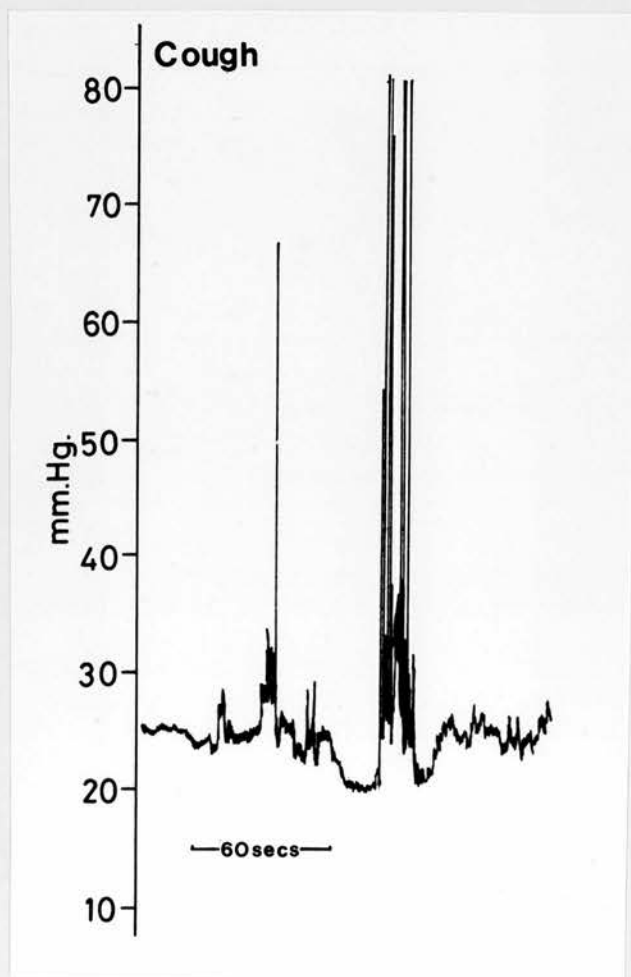
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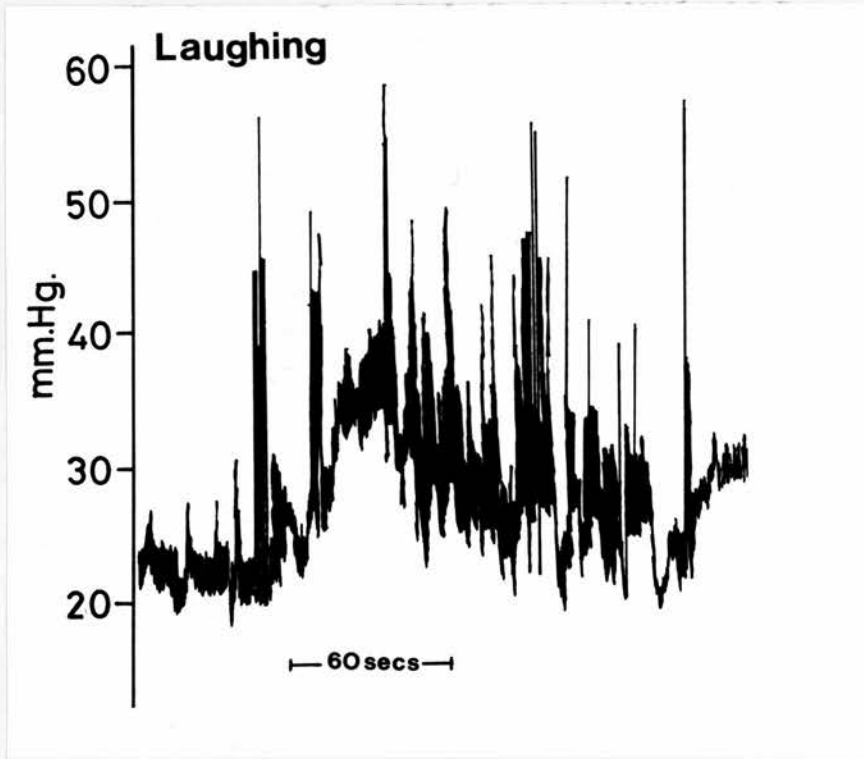
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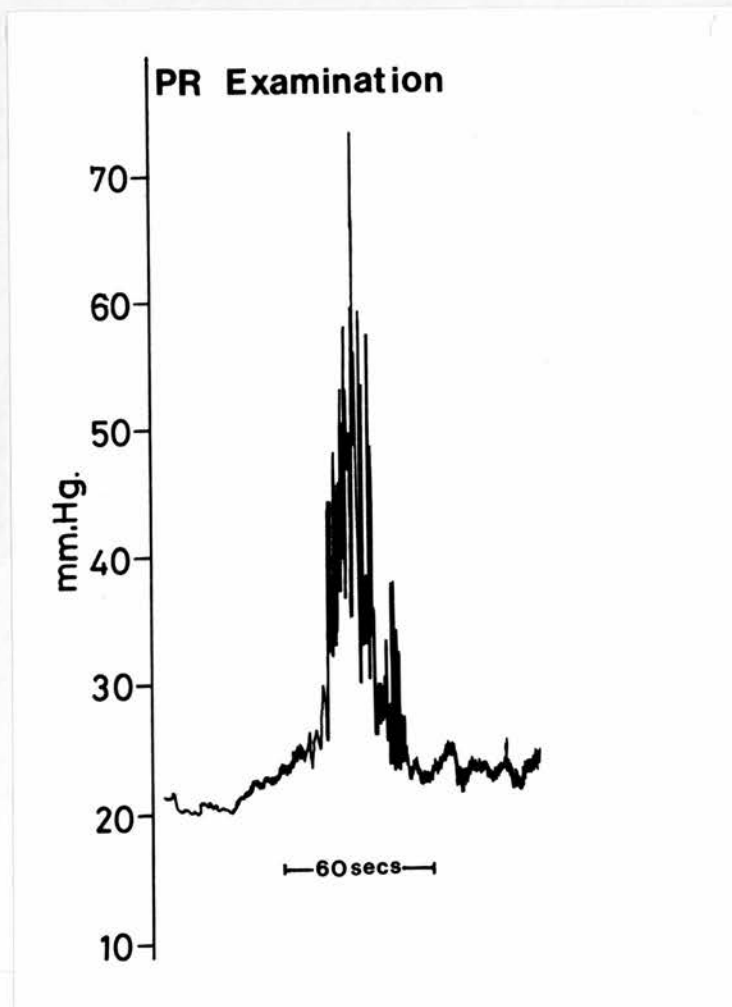
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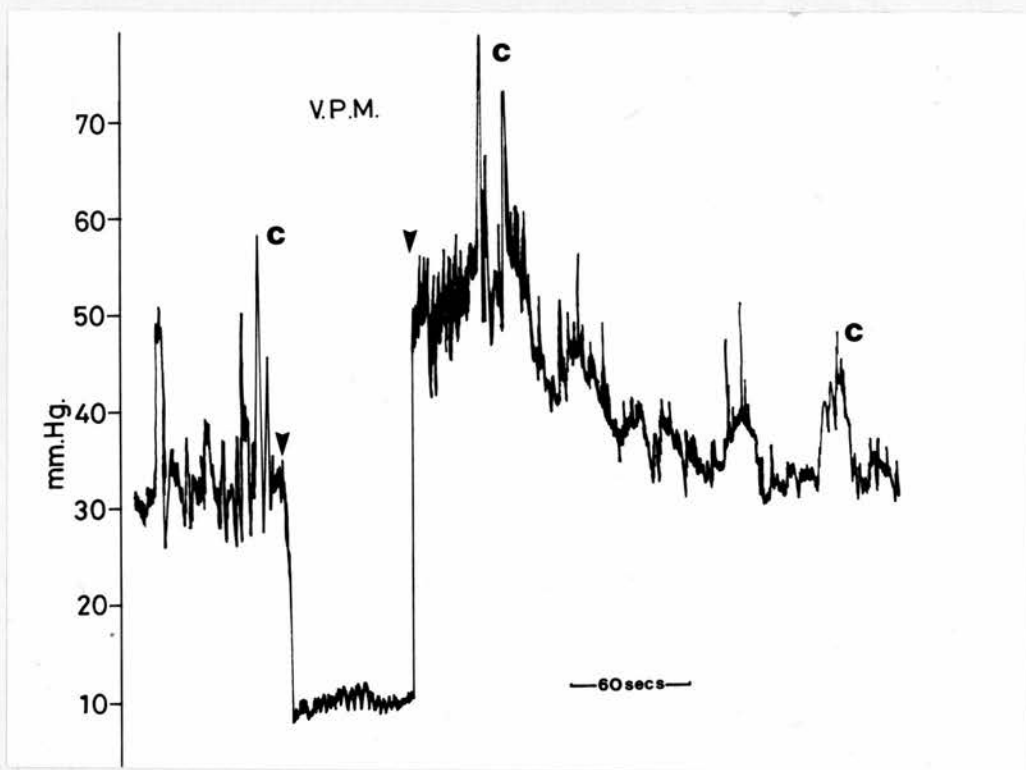
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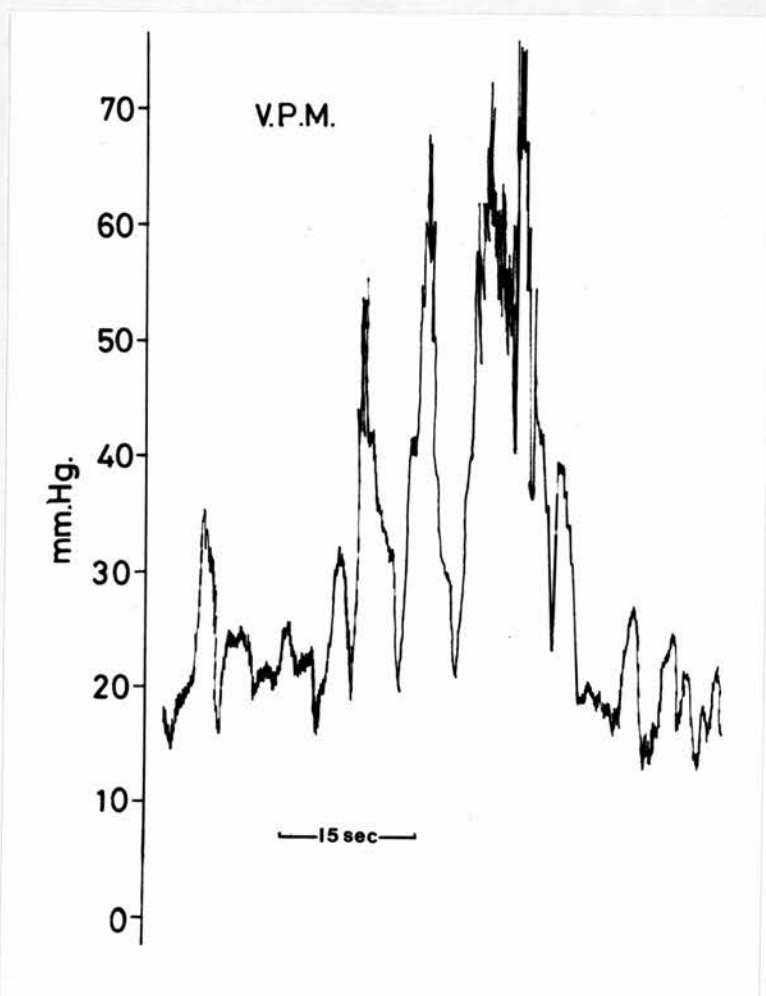
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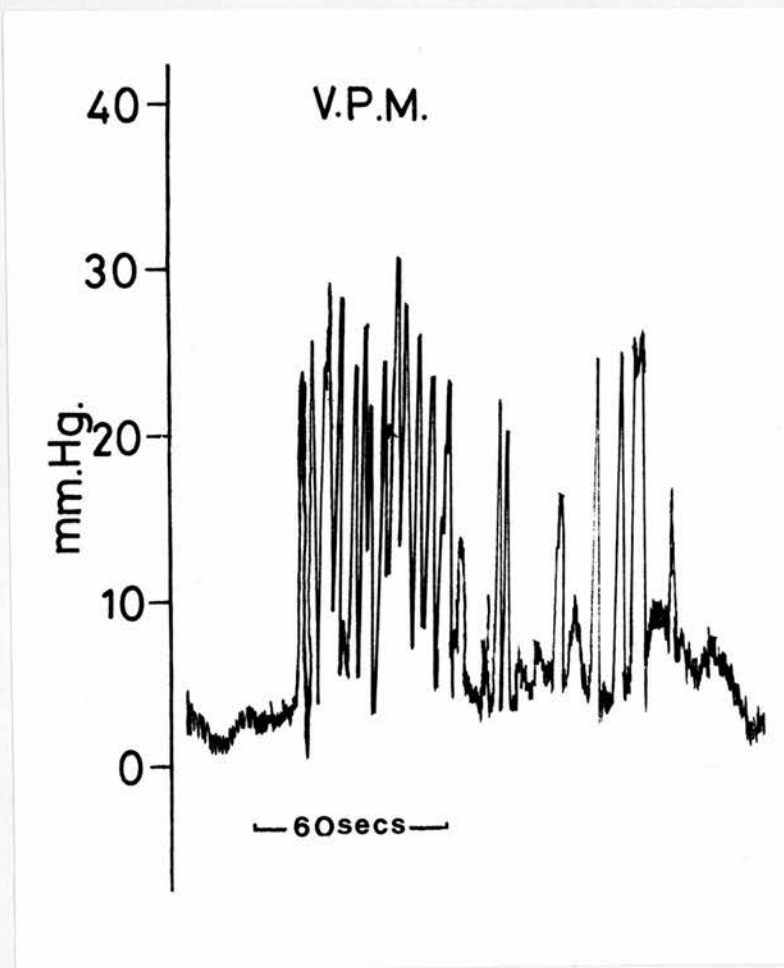
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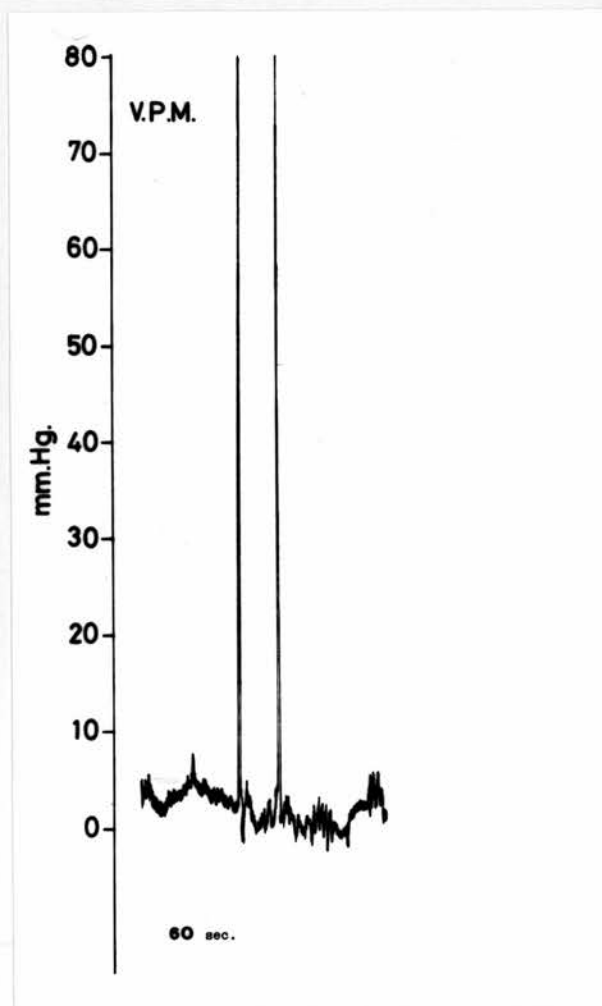
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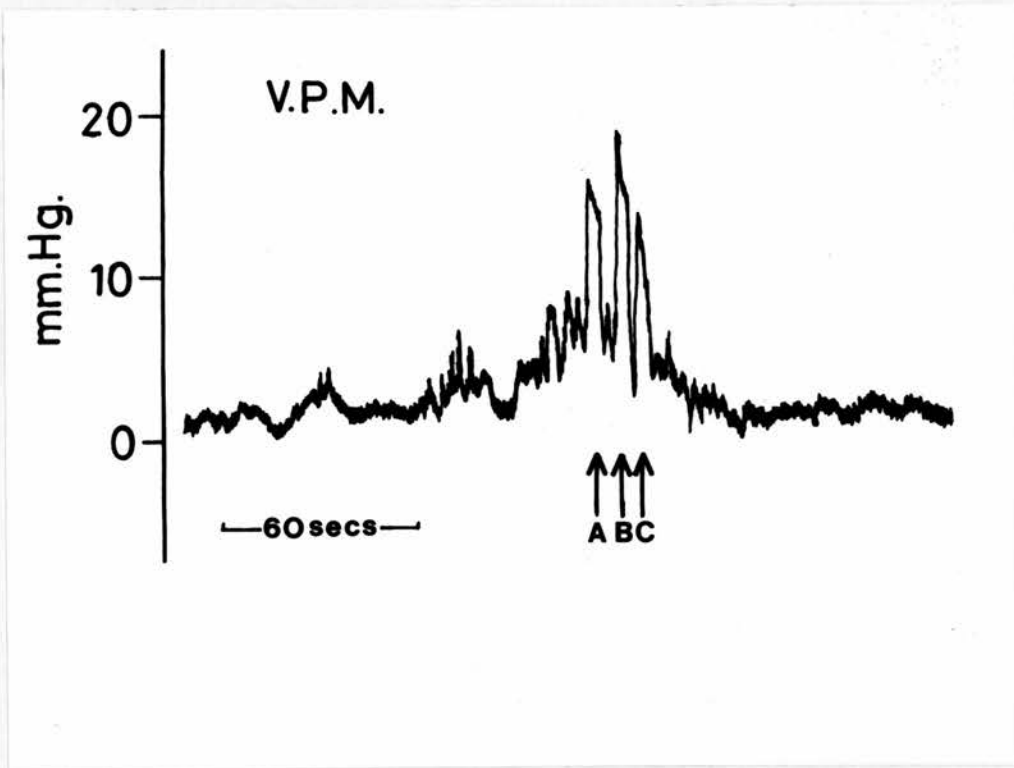
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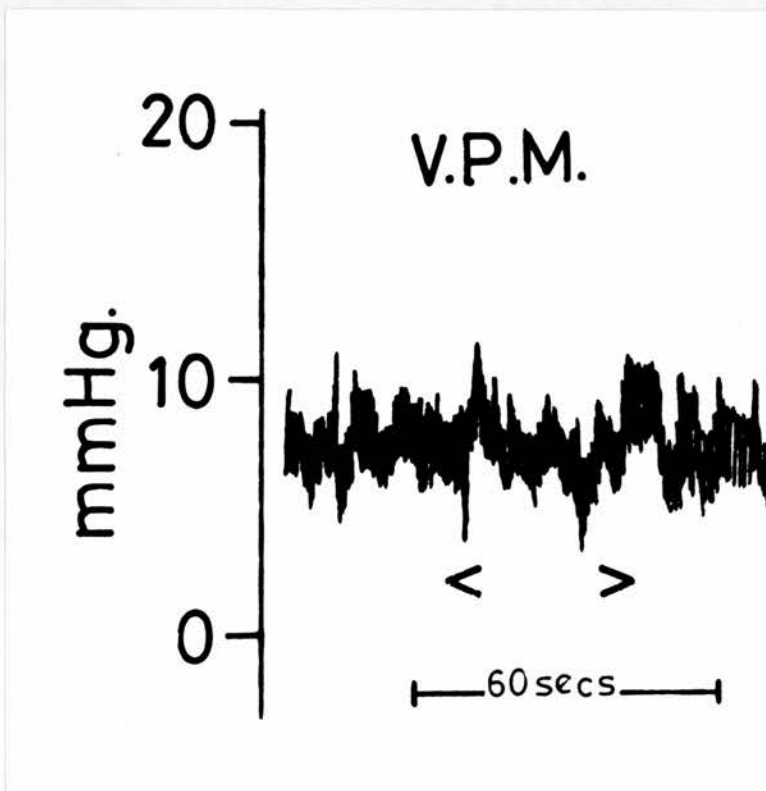
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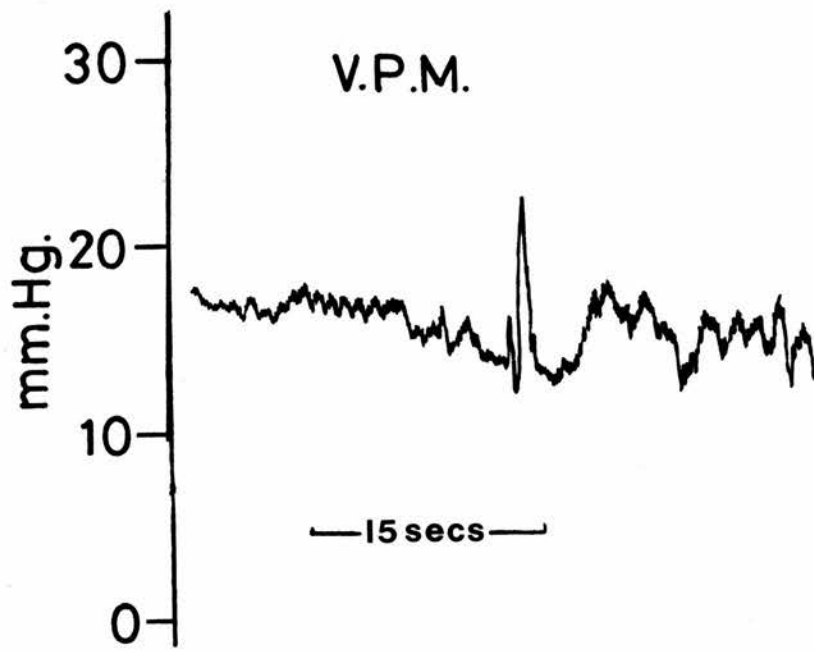
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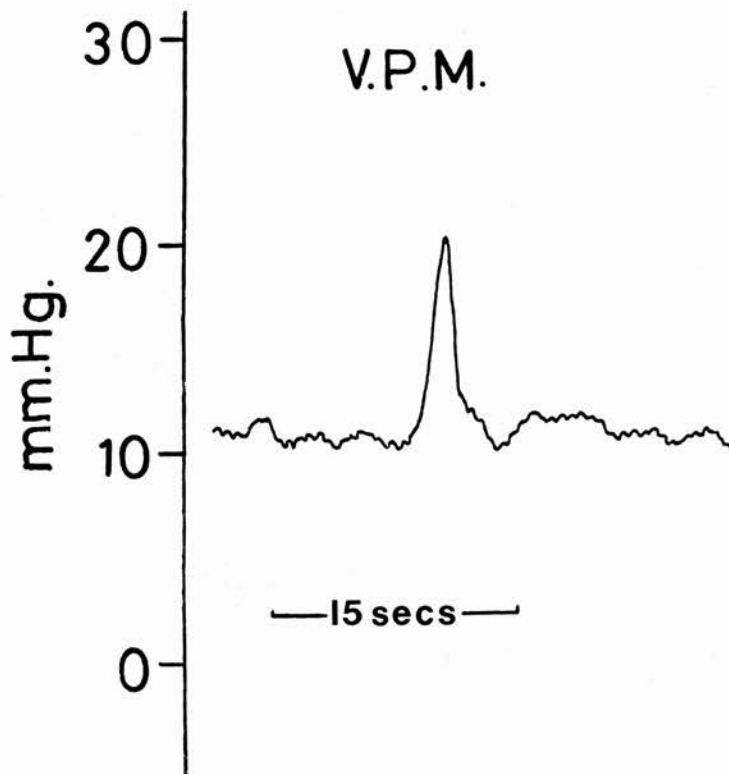
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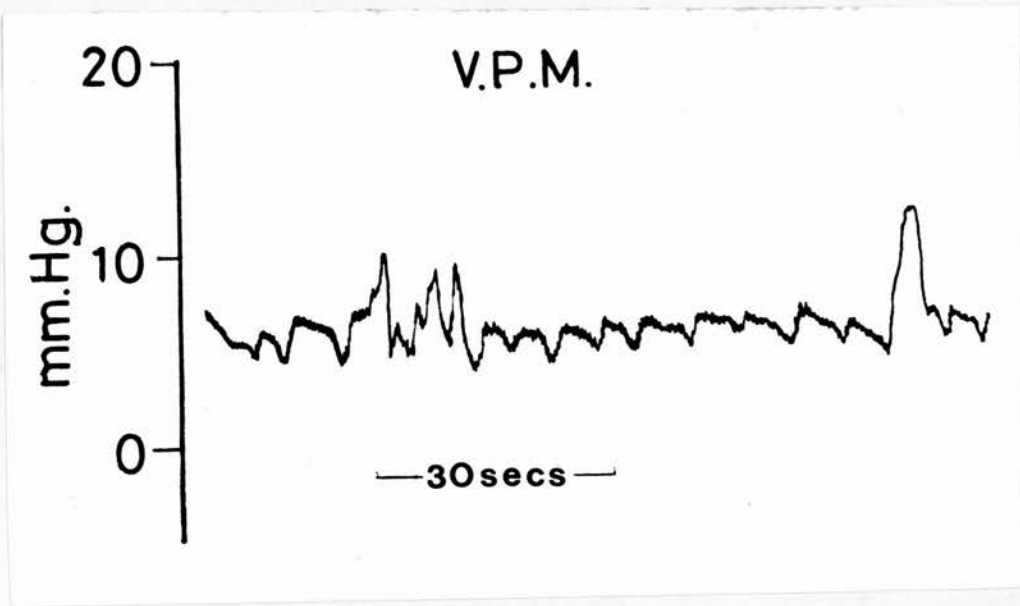
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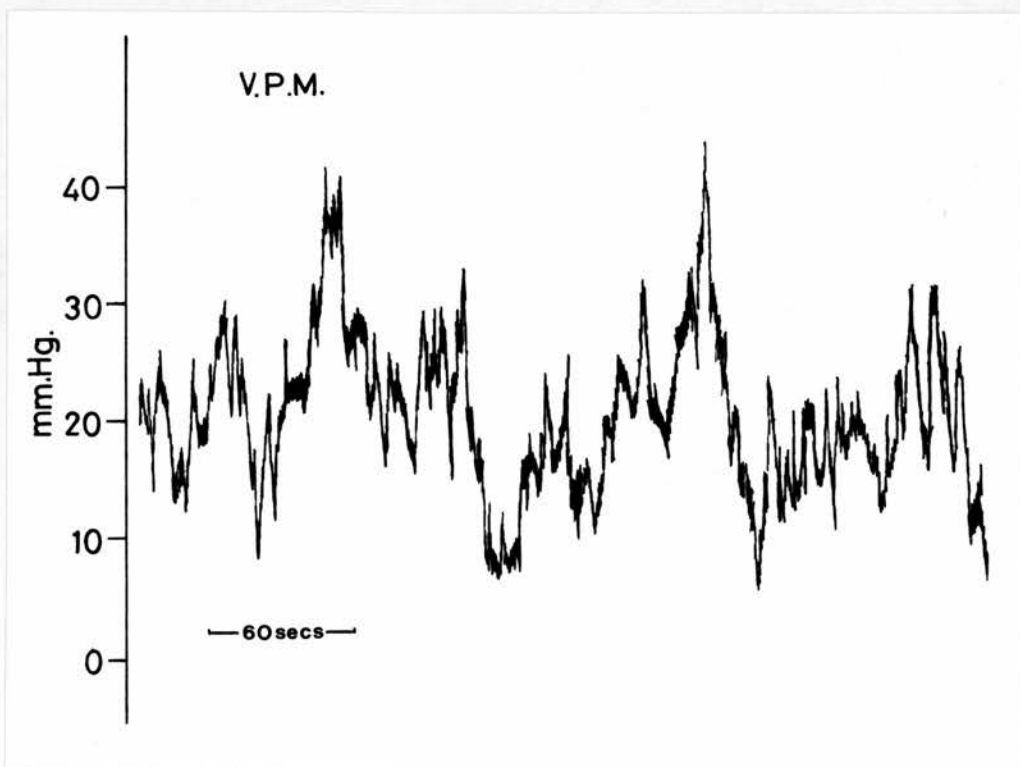
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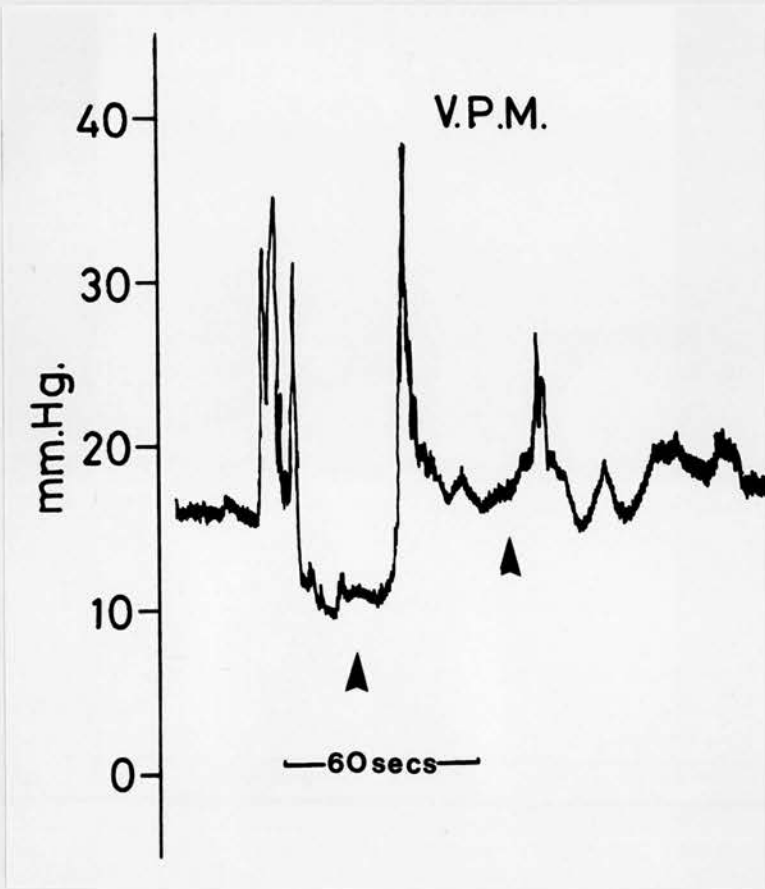
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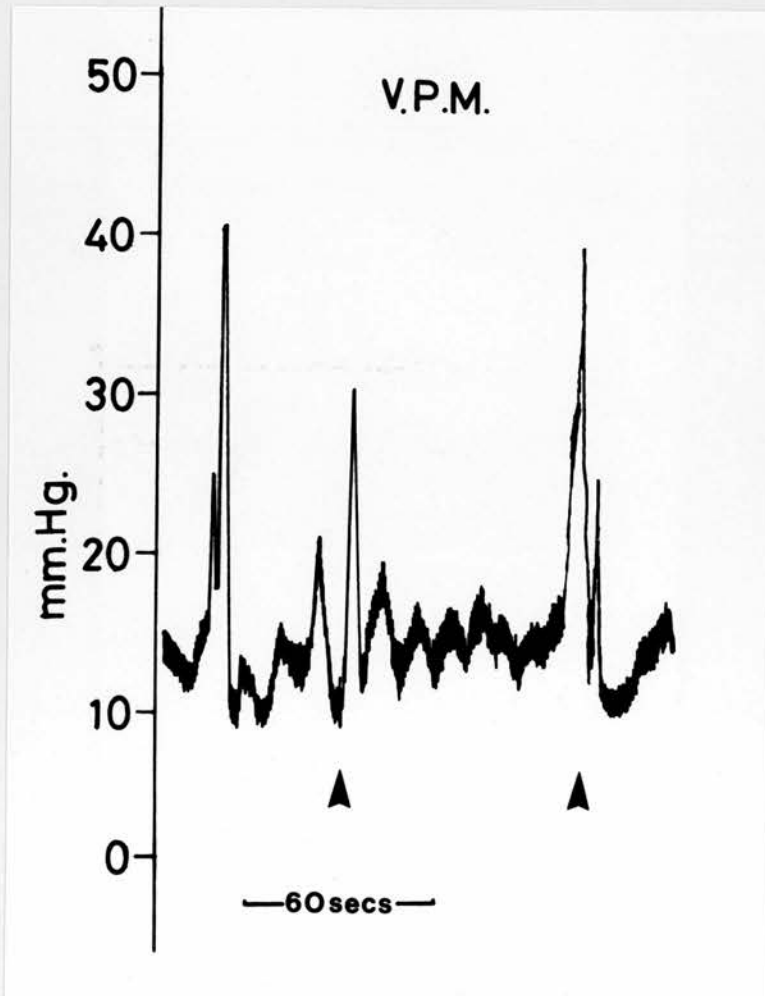
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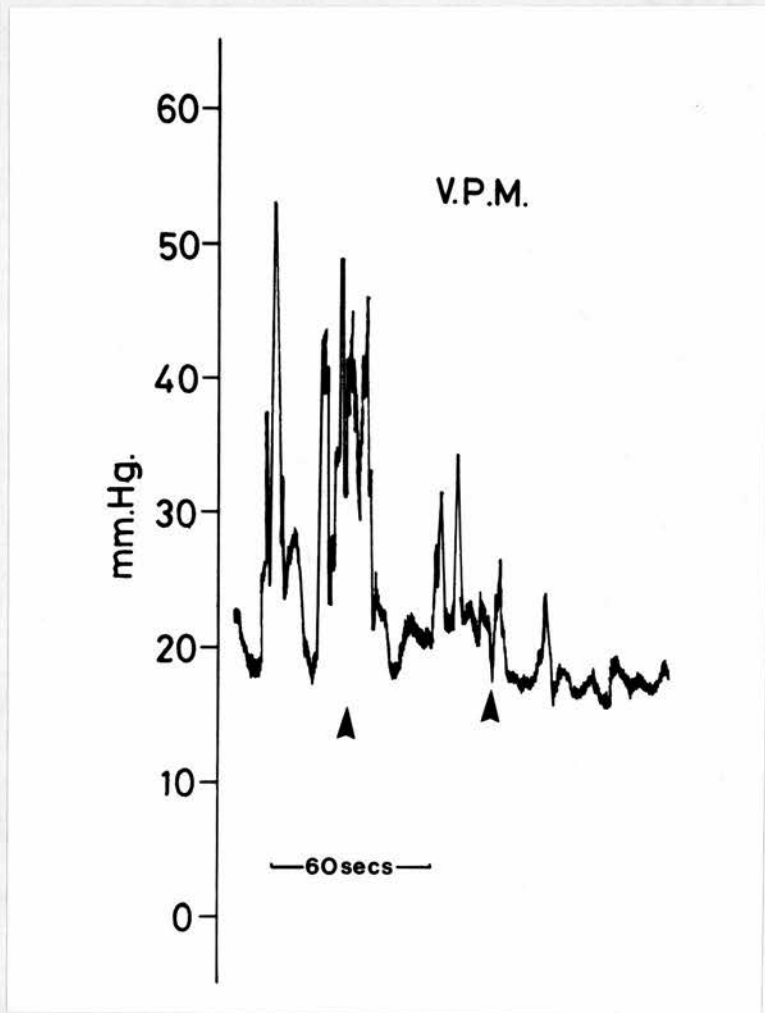
Fig. 49

CHAPTER 5

RESULTS

INDICATIONS FOR VENTRICULAR PRESSURE MONITORING IN THIS SERIES

OTHER INDICATIONS

GROUPING OF PATIENTS

FORMAT OF CASE REPORTS

CHAPTER 5

RESULTS

The results are presented of cases monitored since the latter half
of 1975. In all, 100 cases of ventricular pressure monitoring are
discussed. The procedure was carried out at the Royal Hospital for
Sick Children, Edinburgh, the Children's Unit of the Astley Ainslie
Hospital, Edinburgh, Western General Hospital Department of Neuro-
surgery, Edinburgh and the Neurosurgical Department, Royal Infirmary
of Edinburgh.

INDICATIONS FOR VENTRICULAR PRESSURE MONITORING

The indications for pursuing monitoring of intracranial pressure were
quite diverse:

1. With a C.S.F. shunt in situ:

(a) a child asymptomatic, with evidence of a blocked shunt clinically,
that is a sluggish valve, or the valve definitely not working with
investigative evidence from valvograms or skull x-rays etc. There may
be suspected valvular dysfunction due to time taken for the valve to
refill or the inability to depress it. The child may present with
definite shunt damage due to trauma, with an asymptomatic swelling
about the pumping chamber or along the distal limb. The pressure
estimation is done in these situations to determine the effectiveness
of the valve in reducing the intracranial pressure.

(b) a child symptomatic with or without evidence of a clinical
blockage of a shunt, that is, the child is symptomatic with either
acute pressure signs such as loss of consciousness, neck stiffness,
headache, increasing ataxia, fits or decompensated swellings (e.g.
in the subcostal region or over the lumbar spine in the region of the

original myelomeningocoele). Or the child may present with <u>chronic</u> symptomatology such as deterioration in school performance, behaviour, emotional ability, listlessness and vague tiredness, an increased incidence of fits and chronic headahces etc. These acute and chronic symptoms may occur with or without evidence of shunt blockage clinically. The child may also present with two in situ blocked shunts.

(c) When the question arises of <u>converting a ventricular atrial to a ventriculo-peritoneal shunt or removal</u> of the entire shunting device in a child whose linear growth has resulted in ascension of the valve tip from the atria into the superior vena cava or other major venous vessels where thromboembolic complications are a possibility.

(d) In the <u>post-operative state</u> where there are persisting signs of raised intracranial pressure, such as unresolving papilloedema after a shunt has been inserted; that is, the competence of the single valve to cope with a level of intracranial pressure.

(e) Evidence of <u>brain stem dysfunction</u> in the absence of infection.

(f) To establish if there is a need to <u>modify an existing shunt system</u> or if an additional cysto-peritoneal shunt is required in different pathological states, for example, within a cyst of the ventricles, asymmetrical ventricles etc.

(g) To decide if <u>recent neurological deterioration</u> in the lower limbs or bladder is due to intracranial pressure transmitted through the obex, after other spinal pathology has been excluded.

(h) To <u>distinguish if symptoms are due to a primary head injury or due to shunt malfunction.</u>

2. <u>The assessment of suspected active neonatal or infantile hydrocephalus</u> of whatever cause, primary, e.g. sex linked hydrocephalus or secondary,

e.g. hydrocephalus following subdural haematoma.

In situations where the clinical signs are unreliable or inconclusive:

(a) Prior to a decision re. C.S.F. shunting, ventricular pressure monitoring is carried out, as is an estimate of ventricular size on C.T. scan or pneumo-encephalogram and an estimate of the width of the cortex to increase the number of clinical parameters for assessing the child with hydrocephalus, in an attempt to make stricter indications for the insertion of shunting systems.

(b) Prior to a decision for 'selection' of spina bifida children for treatment, where the remainder of the neurological assessment is equivocal.

(c) To determine the choice of a high, medium or low pressure system where it is decided to insert a C.S.F. shunting system.

It is important to point out that the timing of V.P.M. in these cases where neonatal or infantile hydrocephalus is assessed, can be crucial and should not be undertaken too early in neonates with an expanding O.F.C., that is, when there are minimal signs, a normal pressure result may be obtained and pathology which initiates the hydro-cephalus may continue thereafter, and the pressure rise some days or weeks later, necessitating a second period of pressure monitoring.

3. The assessment of ventricular pressure at the time of pneumo-encephalogram.

4. Estimation of ventricular pressure at the time of 'tapping' of C.S.F. to evaluate how much C.S.F. to remove, to make the child asymptomatic, but not to remove excessive quantities and induce excessive production of C.S.F.

5. Non-active hydrocephalus.

(a) To assess if hydrocephalus is 'arrested' and if a shunting system can be safely removed.

(b) The child whose hydrocephalus is initially conservatively treated, who later becomes symptomatic with irritability, sleep disturbance etc.

(c) The child with a shunt in situ but thought to have an inactive C.S.F. dynamics but who becomes symptomatic (a wide range of possible symptoms).

(d) The shunted hydrocephalic child who develops craniostenosis requiring relaxing craniotomies and subsequently develops active hydrocephalus, a further assessment of ventricular pressure is necessary.

(e) To follow the course of post-meningitis hydrocephalus where one hopes for an early arrest.

6. Following resection of post-infective adhesions in the posterior fossa, it is necessary to know if ventricular pressure remains elevated sufficiently to warrant C.S.F. shunting.

7. To distinguish if fundal changes are due to intracranial causes or due to renal hypertension.

8. A case of mixed sutural synostosis with an O.F.C. which is not increasing.

(The other indications for looking at the intracranial pressure in these cases of Crouzon's disease and acroscaphocephaly are where there is increasing deafness or developing papilloedema.

9. Decerebration and loss of consciousness (a post-cardiac-catheter) but any cause of decerebration or coma could be included in this indication. Without the aid of intracranial pressure monitoring, patients who show decerebrate responses to pain may deteriorate and lose all motor responses without any indication clinically of what is happening.

10. The intermittent brain swelling syndromes (with loss of consciousness and cardio-respiratory arrest). Included in this indication are other causes of brain swelling either exogenous or endogenous.

11. Recurrent meningitis.

12. During intrathecal chemotherapy for cerebral tumours and during later complications of cerebral tumours, 80% of patients who die with cerebral tumours have tentorial herniation and suffer severe terminal R.I.C.P. (Finney & Walker 1961). V.P.M. is also useful in tumour patients awaiting surgery (Kullberg & West 1965).

13. An expanding skull fracture where a decision has to be made if a second shunt would be required or if compartmentalisation has occurred.

14. To monitor post-operatively any form of decompressant craniotomy.

15. Any child who requires open ventricular drainage.

16. During anaesthesia in children with known raised intracranial pressure or conditions likely to cause raised intracranial pressure.

17. Intractable status epilepsy.

18. 'Failure to thrive' with suspected intermittent pressure symptoms.

19 In experimental situations, e.g. hydranencephaly.

OTHER INDICATIONS

The indications outlined above are those applicable to the paediatric neurological service in Edinburgh and obviously will vary from centre to centre and depend on the nature of the unit and the type of patient that is seen. Other obvious indications which are not dealt with in this unit include severe comatose head injuries. These have been reviewed (Miller et al 1977) in 160 patient monitorings and divided into those with intracranial mass lesions (40%), mostly acute subdural haematomas, almost all of whom had markedly elevated I.C.P. and those with no intracranial mass lesions (60%), three quarters of whom had less elevated I.C.P. which was still a problem after treatment in 30%. Patients with combined head and chest injuries who are ventilated and paralysed rely on I.C.P. monitoring as the sole guide to intracranial events.

R.I.C.P. therefore is common after severe head injury, is closely
linked to the presence of intracranial haematomas or brain swelling
and influences the outcome of the injury. Other reports of the use
of V.P.M. in head injury include (Lundberg 1965, Johnston 1970,
Vapalahti & Troupp 1971),

> viral encephalitis and encephalopathies, e.g. Reyes syndrome,
> are further indications (Bruce 1976) also post-hypoxic brain
> damage, intraventricular haemorrhage with or without intra-
> cerebral haemorrhage, ischaemic 'strokes' with infarcts,
> pseudo-tumour cerebri (Johnston & Patterson 1974) and ruptured
> arterio-venous malformations.

GROUPING OF PATIENTS

Patho-physiological states which determine raised intracranial
pressure include:-

1. Hydrocephalus
2. Space occupation due to (tumour, clot or abscess)
3. Brain swelling (either cerebral oedema or cerebral congestion)

Accordingly the patients investigated have been grouped into:

Group A - those who were investigated on account of hydro-
cephalus,

Group B - those investigated on account of space occupation,

Group C - those investigated on account of brain swelling, and

Group D - those investigated for miscellaneous reasons, e.g.
failure to thrive.

Of the total of 100 cases monitored, 87 fell into Group A, 3 into
Group B, 8 into Group C and 2 into Group D. These 100 cases are
dealt with fully in Chapters 12 - 15.

FORMAT OF CASE REPORTS

In each case a number of salient points are made under the following headings:-

Case Number

Initials

<u>Age</u> at the time of monitoring the intracranial pressure

<u>Method</u> - that is whether via the anterior fontanelle, via Rickham reservoir etc.

Medical Diagnostic Background

<u>Temperature</u> at the time of monitoring

<u>Zero</u> reference point for the ventricular pressure level

<u>Duration</u> of monitoring in hours

Specific <u>Indication</u> in each case

<u>Resting Ventricular Pressure</u> - this was obtained by the average of three measurements taken from the tracing when the child was quiet in a resting state, but not asleep.

<u>Stress Ventricular Pressure</u> - a number of stressful situations may occur during the course of any one monitoring but the maximum peak which occurred is the recorded value here.

<u>Cerebral Perfusion Pressure</u> - because we had no continuous 'write out' of systemic arterial pressure, sequential B.P. measurements were obtained from a cuff and the cerebral perfusion pressure estimated by the relationship C.P.P. = systemic arterial pressure minus the intracranial pressure. The systemic arterial pressure was taken as the mid point between the systolic and diastolic B.P. recordings.

<u>Result</u> - that is, whether the level of intracranial pressure was raised, normal, whether there was a functioning or malfunctioning valve etc.

<u>Action</u> - whether conservative or surgical treatment or other
form of management was employed.

<u>Cardiac/Respiratory Artefact</u> - manual measurements of the width
of the cardiac and respiratory pulse superimposed on the ventricular
pressure level, has been estimated in most cases. In cases where the
alternative polygraphic recording apparatus has been employed, there
would be no comparibility with the routine method, hence no estimate
is given for these cases.

<u>OFC</u>

<u>Ventricular Dilatation/Cortical Mantle</u> - where possible an estimate
of the ventricular size and width of the cortical mantle is included
from the results of either computerised tomography or pneumo-
ventriculogram.

<u>Points of Interest</u>

<u>Pressure Recordings</u>

<u>Follow Up</u>

CHAPTER 6

RESULTS

PATIENTS

MEAN AGE OF CHILDREN MONITORED

DURATION OF VENTRICULAR PRESSURE MONITORING

METHOD

NORMAL, RAISED AND EQUIVOCAL PRESSURE RESULTS

CASES OF VENTRICULAR DILATATION WITH NORMAL RESTING AND SLEEP PRESSURES

EQUIVOCAL CASES

CLINICAL VENTRICULAR PRESSURE CORRELATES

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OTHER ABNORMAL WAVE FORMS

SEQUENTIAL SIGNS AND PRESSURE LEVELS IN INFANTILE HYDROCEPHALUS

CARDIO-RESPIRATORY ARTEFACT

HYDROCEPHALUS

CHAPTER 6

RESULTS

PATIENTS

Total number of cases = 100

Total number of patients monitored = 76

Males = 45

Females = 31

MEAN AGE OF CHILDREN MONITORED = 3.9 years

DURATION OF V.P.M.

Total duration of V.P.M. in 100 cases = 481.05 hours

Average duration of V.P.M. = 4.81 hours (includes 'short duration'
V.P.M's.

Number of 'short duration' V.P.M's (i.e. 1 hour or less) = 31

METHOD

1. Via Rickham vventriculostomy reservoir = 51

   (this includes 19 cases where a Rickham reservoir was electively
   inserted for V.P.M. and 1 case of a Rickham attached to a
   Spitz–Holter valve.)

2. Ommaya reservoir = 2

3. Pudenz flushing chamber = 5

4. Anterior fontanelle or 'burr hole' = 39

5. Cortical subarachnoid space = 1

6. Pudenz and brain surface simultaneously = 1

7. Ommaya and Pudenz simultaneously = 1

NORMAL, RAISED & EQUIVOCAL PRESSURE RESULTS

In all 100 cases, 30 cases were considered to have normal intra-
cranial pressure. The remainder consisted of 51 patients with
definite raised pressure and 19 patients with equivocal pressure.
This last group of patients constituted the most difficulty clinically,
in that their resting awake pressures were within normal limits but the
levels of their pressure elevations in sleep were unacceptable. In some
cases these have been treated as having R.I.C.P., while others have
been managed conservatively and hence have been closely followed up.

CASES OF VENTRICULAR DILATATION WITH NORMAL RESTING AND SLEEP PRESSURES

Five cases fell into this category (Case Numbers 67,26,65,74 and 75)
and a positive decision was taken not to insert a shunt in all.
Case 67 with a mean resting pressure of 6 mm Hg and a sleep maximum
of 10 mm Hg in the presence of moderate ventricular enlargement has
maintained excellent progress without a shunt.
Case 26 with normal pressures and a pallium of 20.5 mm, measured at the
vertex, has likewise shown no signs of R.I.C.P. and has shown
acceptable developmental progress.
Case 65 with gross ventricular dilatation has similarly been well at
follow up.
Cases 74 and 75 with gross hydrocephalus, has not been prejudiced by
the lack of a shunt, although physically handicapped and mentally
retarded as a result of the original pathology.
All of these above cases are from Group A, i.e. hydrocephalus and
could be considered as having a spontaneous arrest of hydrocephalus.
I am defining 'arrested hydrocephalus' as a stable clinical and
developmental state with normal resting and sleep ventricular pressures
and no progression of ventricular dilatation: or more correctly,
progression of the developmental state commensurate with growth.

It follows therefore from these observations and others (Schick & Matson 1961) that spontaneous arrest of hydrocephalus in early life is not very common and that close observation of these cases is warranted. Assessment without a shunt in situ is relatively free of pitfalls but assessment with a shunt in situ poses a number of problems: firstly if the shunt is working or not, and if so how much is it contributing to the control of the pressure? Secondly evaluating if the patient could manage without the shunt and thirdly, if the shunting system is removed (even though it may be non-functional) will this upset the delicate C.S.F. dynamic balance that has been achieved, by altering surface area and minute changes in volume availability etc.

EQUIVOCAL CASES

In all, 19 cases presented equivocal results which posed a problem in management. In these the resting pressures were normal but levels in sleep were elevated to varying degrees. All of these cases with the exception of the last one, belong to Group A. Eight of these 19 cases subsequently had a C.S.F. shunting device inserted (Table 1). There were a further 11 children with equivocal pressure values where a decision was made not to insert a C.S.F. shunt. This group listed below affords a good comparison and should be followed up in considerable detail in the future with more subtle tests of neurological function.

Case 23 - resting pressure 7.5 mm Hg, sleep maximum 21 mm Hg. At follow up her OFC began paralleling the percentiles and there has been no further evidence of raised intracranial pressure.

Case 27 - resting pressure 9 mm Hg, sleep maximum 19 mm Hg. At follow up there are no further signs of raised intracranial pressure, a slight developmental speech delay, but a C.T. scan, from being grossly hydro-

TABLE 1

Case Number	Resting Pressure	Sleep Pressure	Course
81	9 mm Hg	40 mm Hg	Shunt inserted but little follow up.
72	12 mm Hg	32 mm Hg	Revision planned instead of removal. However, at operation, technical difficulties meant V.A. shunt was left in situ. Little follow up.
69,70	3-5 mm Hg	27 mm Hg	Clinically well with large OFC. V.P. shunt inserted. Similar pressure values obtained later when asymptomatic.
8	8 mm Hg	23 mm Hg	Symptomatic: sleepy and vomiting and at operation an occlusive stitch was removed from the distal tubing.
49	10 mm Hg	28 mm Hg	No reduction of pressure on pumping the valve. Therefore revision which confirmed a proximal end blockage.
57	5 mm Hg	22 mm Hg	V.P. inserted but since then has had a number of shunt revisions and one attack of ventriculitis.
62	4 mm Hg	42 mm Hg	V.P. shunt inserted

cephalic with enlarged sulci initially is now completely normal.

Case 16 - resting pressure 3 mm Hg, sleep maximum 28 mm Hg.
Normal progress at follow up.

Case 28 - resting pressure 7.5 mm Hg, sleep maximum 32 mm Hg. There
has been no deterioration or further signs of raised intracranial
pressure up to 2 years later.

Case 42 - resting pressure 4 mm Hg, sleep maximum 36 mm Hg for 5-10
minute periods. No abnormalities noted on follow up.

Case 73 - resting pressure 6 mm Hg, sleep maximum 15 mm Hg. With a
communicating hydrocephalus no shunt was inserted and at follow up the
OFC became nearer the percentiles and he remained asymptomatic.

Case 76 - resting pressure 10 mm Hg, sleep maximum 30 mm Hg for 4
minutes. No shunt inserted and good progress at follow up, with serial
hand function assessments, psychometric assessments, speech assess-
ments and C.T. scans.

Case 77 - resting pressure 8 mm Hg (unfortunately no sleep pressure
obtained on this occasion). The child was discharged with a non-
functioning shunt in situ at the age of 16 months, to be readmitted
at 2 years 8 months of age with raised intracranial pressure.
Presumably sleep pressures would have been elevated on the first
recording but one cannot be sure. The readmission was associated with
Haemophilus upper respiratory infection. He had been very well in the
interim, without a functioning shunt. It is also worth noting that
the shunt probably should have been removed at the time, as it would
constitute a future focus for a bacteraemic infection.

Case 79 - resting pressure 7.5 mm Hg, sleep maximum 20 mm Hg and no
shunt was inserted.

Case 84 - resting pressure 5 mm Hg, sleep maximum 38 mm Hg for 6
minutes. Close follow up with physiotherapy, hand function tests,

psychometric assessments, C.T. scans and occupational therapy assessments have shown no deterioration.

Age is a further variable in the quest for levels of sleep pressure which are acceptable in the long term.

All of the above 18 cases are part of Group A, that is hydrocephalics, proven by one or other method.

Case 91 - resting pressure 10 mm Hg, sleep maximum 17.5 mm Hg. Both brain and ventricular system were normal on C.T. scan. Follow up has been satisfactory so far. However this case is obviously different with craniosynostosis which, with growth, may hamper brain expansion. Of this 19 patients with equivocal pressures therefore, two groups have emerged, one where a C.S.F. shunt has been inserted, which will obviously not provide much information about acceptable sleep levels in the long term, and a second group where a C.S.F. shunt has not been inserted and so far of these 11 cases only one case (which did not have a sleep pressure recorded) has been readmitted with R.I.C.P. due to an upper respiratory infection. However, at the time of writing this, a further patient in this group has developed a sub-galeal extravisation of C.S.F. about the site of a non-functioning in-situ valve.

When a shunt is not inserted, close observation of such parameters as ventricular size, neurological state, school performance, psychometric assessments etc. need to be assessed to ensure that subtle deterioration is not insidiously occurring.

Three cases were considered 'too severe' for active treatment after V.P.M. and clinical assessment. In only one case was the results of the V.P.M. not acted upon and this was a decision to insert a C.S.F. shunt where on assessment of the ventricular pressure, it did not appear necessary. This child was readmitted a short time later with

irritability, and a further V.P.M. again confirmed normal pressure, but now with a functioning shunt and an alternative cause was sought.

CLINICAL VENTRICULAR PRESSURE CORRELATES

1. Six children out of a total number with raised pressure (51) presented with R.I.C.P. in the absence of any clinical signs (11.7%). A further 2 children presented with pressures in the equivocal range also without any clinical signs. No child with R.I.C.P. presented without signs and symptoms.

2. Seven children with R.I.C.P. presented with evidence of an upper respiratory infection. On one occasion this was due to a proven Haemophilus infection, and, on another occasion, chicken-pox.

3. Eight children with R.I.C.P. suffered from a possible pressure induced seizure. In 2 cases pressure problems had resulted in an increased incidence and frequency of fits, particularly the 'akinetic' variety. In one case the seizure may have been the result of infection plus or minus pressure, in a child with a blocked infected shunt. In another case, a child with equivocal range pressure, presented with a fit and in a further case fits may have been due to a brain stem haemorrhage as they were clinically of the 'brain stem' variety and there was associated R.I.C.P. Excluding the last three we have 5 definite cases of pressure induced seizures from a total number of 51 cases of R.I.C.P. I stress that these 5 were not children who were epileptic or who had any other reason for fitting (e.g. brain damage from birth asphyxia). They are different from the cases mentioned in Chapter 7.

4. Eight cases with R.I.C.P. presented with papilloedema. One case was most likely hypertensive retinopathy although the sleep pressure

was elevated. One case belonged to Group B (i.e. space occupation)and 2 cases to Group C. There were no cases of papilloedema under the age of 5 years. Papilloedema associated with intracranial hypertension may be subdivided into:

(a) incipient or early papilloedema

(b) fully developed papilloedema

(c) chronic papilloedema

(d) chronic atrophic papilloedema   (Lubow 1973)

In the cases listed here there was more than retinal venous distension, mostly complete blurring of the disc margins (not just on the medial edge), or the absence of retinal vein pulsations before these cases were designated as having papilloedema.

5. There were 7 cases of optic atrophy (2 patients being recorded in this number twice). The existence of atrophic changes in the disc influences the subsequent development of papilloedema in that it tends to develop much slower. Children who have had preceding pressure problems may now have the chronic atrophic papilloedema, which can cause clinical difficulties. Furthermore, young children in particular neonates and infants normally have very pale discs and in the 7 cases mentioned above, two were infants.

INFREQUENT OR UNUSUAL SIGNS OF R.I.C.P.

1. Gross sweating. This occurred repeatedly in two patients in this series, usually when the ventricular pressure was of the order of 60 mm Hg. In one child it was associated with papilloedema and poorly responsive dilated pupils.

2. An erythemato-macular rash on the extensor surfaces of the upper and lower limbs. At a ventricular pressure level of 20-25 mm Hg

consistently, this proceeded to loss of consciousness. Prior to the rash, the vital signs were normal.

3. <u>Jitteriness</u> in an 8 day old baby with normal calcium, glucose etc, but associated with an increasing OFC, irritability and scalp vein distension. It was also noted in the child with hydranencephaly.

4. <u>Dysarthria</u>, tachycardia and hypertension in a child with a 'kinked stem' at a pressure of 12.5 mm Hg.

5. <u>Apnoea</u>

6. <u>Poor appetite and weight loss</u> associated with vomiting, headache and <u>neck pain</u> over a 10 day period (case 45) with a resting pressure of 15 mm Hg.

7. <u>Disturbed sleep</u> occurring for example in a child with a rapidly expanding OFC, vomiting, scalp vein distension, 'sunsetting' and tense fontanelle at 6 months of age and with a resting ventricular pressure of 27 mm Hg.

8. <u>Normal breast feeding</u> It is sometimes surprising that a child of say 2 months (case 61) with a resting ventricular pressure of 22 mm Hg, should continue to feed very well, without vomiting and increase weight and appear quite contented and happy.

9. <u>Leakage of C.S.F.</u> through an umbilical fistula, or through a repaired or unrepaired myelomeningocoele lesion.

10. <u>Neurogenic stridor</u> occurring in a 6 week old child with a resting ventricular pressure of 25 mm Hg.

11. <u>Hippus</u>, bradycardia and diminished conscious state with a tense fontanelle in a child of $10\frac{1}{2}$ months of age with a resting ventricular pressure of 22 mm Hg.

No distinct relationship between the mean V.F.P. and the occurrence of clinical pressure symptoms could be established in 64 cases of Lundberg and the same holds true in this series. There is however

an often remarkably consistent display of pressure signs at the
same pressure level in each individual case whether monitored weeks
or months apart.

MORE CLINICAL CORRELATES

The commonest signs of R.I.C.P. which occurred in all cases in this
series, and covering all age groups were: an increasing OFC, scalp
venous distension, tension of the anterior fontanelle, sutural
separation, loss of upward conjugate gaze, drowsiness, nystagmus,
bradycardia, decerebration and loss of or variations in the conscious
state, hypotonia, neck stiffness and neck retraction and 'sluggish
or sticky valves'.

'SLUGGISH VALVES' where parents have been told to test daily, or
test when the child appears 'off colour' or frankly ill, the
flushing device of a C.S.F. shunt, they often present with the
complaint that the 'valve does not feel normal'. With the Pudenz
shunt one can often clinically diagnose proximal or distal
obstruction by feeling the flushing device's reluctance to return
to the pre-pumping state or its inability to be depressed,
respectively. With the Spitz Holter system, because of the twin
valve arrangement, the intermediate plastic section appears 'sticky'
or 'sluggish'. It is my opinion that parents sometimes report a
'sticky' valve when the climatic conditions vary, e.g. during hot
weather plastic material is generally more pliable, and less so and
more difficult to depress during cold weather!

INSIDIOUS PRESENTATION OF I.C.P.

Six children presented with symptomatology extending beyond one
month, and in one case symptoms for up to 2 years prior to admission.
In all these cases there was R.I.C.P. on direct measurement. In
retrospect the symptomatology consisted of:

(a) a change in personality, becoming withdrawn and non-communicative (1 child)

(b) headache, hyperacusis and drowsiness for a few months in another child.

(c) headache and intermittent vomiting for 3 months

(d) headache and vomiting for 6 weeks, absent signs and resting ventricular pressure of 28 mm Hg.

(e) deterioration in school performance (from a teacher's report), diminished mobility, and behavioural deterioration for 1 month before admission. The resting pressure was 20 mm Hg.

(f) Two years history of increasing temper tantrums in a 10 year old boy, cyclical vomiting and headaches every 2 weeks and a minimal worsening of an existing hemiplegia. The pressure was considerably elevated at night.

ABNORMAL WAVE FORMS

These can be thought of as either (a) steady or (b) periodic and the importance of them is the identification of the physio-pathological cause which gives rise to their initiation and cessation.

(Lundberg 1960) described three basic wave forms A (or plateau waves), B and C waves, since then other descriptions have appeared of prolonged plateaus (lasting $\frac{1}{2}$ - 1 hour), pre-plateau waves (pressures of less than 50 mm Hg) ramp waves and scallop waves. One could also add to this list the normal rhythmic sleep oscillations, exaggerated pressure responses to sleep (Canyon-like waves), post-meningitis sleep changes (saw-tooth waves) etc.

A or Plateau Waves

These have rises to greater than 50 mm Hg for 5 - 20 minutes and then fall spontaneously. Often they occur without precursory symptoms or precipitating factors and Lundberg considered them due to 'intrinsic

vasomotor control of the cerebral circulation' and causally
related to tonic fits.

Plateau waves were observed in 5 patients in this series (Case 54
N.B.), (Case 1 Y.P.), (Case 89 S.D.), (Case 41 A.W.) and (Case 93
G.H.) and it is significant that in all these cases there was an
absence of the normal spinal adaptive accommodating mechanism for
C.S.F., i.e. there is low or absent compliance which results in a
shift of the pressure-volume curve (Chapter 11) to the left so that
a small increase in volume results in an inappropriately large
increase in pressure. This then is the reason plateau waves are
not commonly seen in neonates as they have the additional buffer of
being able to increase the size of their heads.

The cases listed above had (a) a theco-peritoneal shunt that was of
no use, as the child had converted from a communicating to a non-
communicating hydrocephalus. (Incidentally it may be that shunting
from the lumbar theca induces non-communicating in the same way as
shunting from the ventricles induces closure of the aqueduct described
by Shellshear & Emery 1976.)

(b) Case 54 had had a severe episode of 'coning' at 2 years of age
with brain shift demonstrable on investigations.

(c) Aqueduct stenosis.

(d) A posterior fossa tumour in strategic position from a point of
view of interfering with spinal compensation. When this girl was
experiencing an ascending plateau disturbance she awoke and became
dysarthric, irritable and complained of a headache.

Some minor or pre-plateaus were observed in Case 87, a child with
hydrocephalus, and in Case 16 while a child complained of a headache.
These were self-limiting and of short duration.

'B' Waves

These are described by Lundberg as sharp peaked waves of variable height, at an average rate of 1 per minute, extending from 0-50 mm Hg. There are examples of these in this thesis, in Chapter 10. They were thought to be related to periodic breathing, the peaks of the waves corresponding with hyperpnoea and associated with depressed wakefulness. They disappear with artificial ventilation and with 'tapping C.S.F.' and are thought to be due to variations of blood pressure within the cerebrovascular bed. Periodic breathing, which can occur in advanced renal and cardiac disease, pulmonary disease and poisoning with morphine and chloral, is thought to be an intrinsic slow brain stem rhythm of respiration , i.e. the respiratory system is functioning at its lowest level, without higher level regulation and as mentioned in Chapter 10, it is during R.E.M. or paradoxical sleep that they have been observed, in this series, which is in fact the deepest level of sleep and no integration with other levels occurs.

'C' Waves

These are rhythmic oscillations of small amplitude from 0-20 mm Hg corresponding to the Traube-Hering plots of systemic blood pressure and occur on average 6/minute (5-8/min). They have been observed on the plateau wave and tend to dominate the wave when the V.F.P. is high.

OTHER ABNORMAL WAVE FORMS

A further wave form, not previously described, is seen in Case 1 where short bursts or 'spindles' appeared, not altering the mean pressure level and occurring while the patient was quiet, motionless and relaxed with no vital sign dysfunction. This pattern occurred after the first tap of C.S.F. performed for more severe plateau

waves. Their origin is obscure. Other wave forms seen frequently in sleep are discussed in detail in Chapter 10 on sleep.

SEQUENTIAL SIGNS AND PRESSURE LEVELS IN INFANTILE HYDROCEPHALUS

In my opinion it is not uncommon that signs of R.I.C.P. and recorded pressure values in infantile hydrocephalus often follow a step-wise pattern, i.e. scalp vein distension, fontanelle tense etc. may occur for a few days (decompensation) followed by one or more days when there is no increase in the OFC and the child is asymptomatic (compensation). This is again followed by a period of decompensation then compensation etc. e.g. Cases 26 and 61. This pattern is not invariable, in some children there is an undeterred progression of signs and symptoms.

The age 15-18 months (19 months in one case) appears a critical time for the appearance of signs of transient R.I.C.P. to develop. At this age pressure accommodation, by an increase in the OFC, is no longer easy (except in very gross untreated hydrocephalics) and consequently symptoms appear.

It would appear that children with internal brain herniations do not necessarily record excessively high pressures after compensation has occurred, i.e. with residual clinical and investigative evidence of brain shifts, the recorded pressures are not excessively high, e.g. Cases 30 and 51.

CARDIO-RESPIRATORY ARTEFACT

Cardio-respiratory artefact has been plotted against the ventricular pressure level at its mean resting value and against the maximum asleep measurement. 122 observations are plotted on graph shown and the mean cardio-respiratory measurement overall was 9.06 mms.

RESTING
r = 0.5744

SLEEP
r = 0.6779

TOTAL
r = 0.799

V.F.P. mm Hg
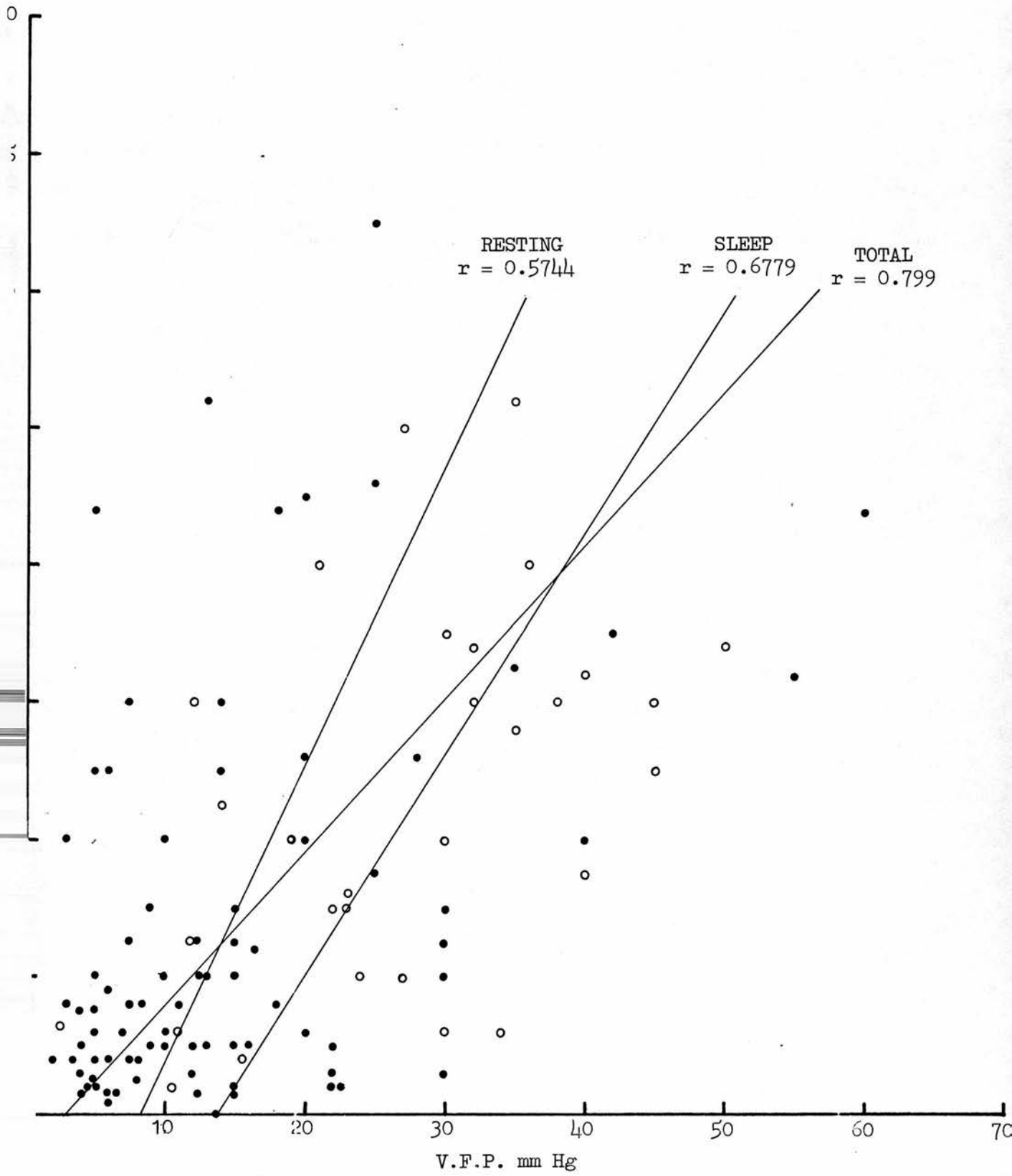
The mean cardio-respiratory artefact when the ventricular pressure is in the normal range, that is 10 mm Hg or less, was 4.52 mm. Between 10, up to and including 20 mm Hg, pressure was 8.42 mms. Between 20 up to and including 30 mm Hg, pressure was 9.64 mms. Between 30 up to and including 40 mm Hg, pressure was 15.5 mms. Between 50 up to and including 60 mm Hg, pressure was 19 mms and between 70 and 80 mm Hg, it was 35.38mms. As is expected the CR pulses increase in width with increasing pressure.

When 34 sleep values were extracted from this graph, their mean value was 9.51 mms, i.e. greater than the value in the normal range and greater than the overall mean value of 9.06 mms.

In the graph three regression lines have been shown for resting pressure C.R.A. values $(r = 0.5744)$, sleep pressure C.R.A. values $(r = 0.6779)$ and total number (i.e. resting and sleep C.R.A. values) $(r = 0.7990)$. Each is related to the ventricular pressure level at $p < 0.001$, ie. highly significant.

HYDROCEPHALUS

Values were extracted for all neonates and infants with hydrocephalus and Tables 2 and 3 show the results of monitoring neonates (i.e. less than 1 month of age) and infants (up to 1 year of age).

Firstly in the neonatal group, all the values for the mean resting pressure and mean stress pressure were tabulated and both ratios of stress/resting pressures and the degree of rise (stress pressure - resting pressure) tested for significance within each sub-group (i.e. those with R.I.C.P., normal pressure or equivocal pressure) and the group as a whole, against the degree of ventricular dilatation. These values were also tested against similar values in children aged between 1 month and 1 year and against the whole infant age group.

TABLE 2

HYDROCEPHALUS

	No.	Mean R.P.	Mean S.P.	Mild Vent.Dilt.	Moderate Vent.Dilt.	Severe Vent.Dilt.
RICP	20 76.9%	$+15.85$ -3.71	$+40.89$ -13.86	0	6	4
NICP	2 25.38%	$+7$ -4.24	$+25.5$ -2.12	0	1	1
EQUIV.	1 7.69%	13	58	0	1	0
T	13	14.27	39.75	0	8	5
		(range 4-22 mm Hg)	(range 23-64 mm Hg)			

NEONATAL GROUP (i.e. < 1 month)

TABLE 3

	No.	Mean R.P.	Mean S.P.	Mild Vent.Dilt.	Moderate Vent.Dilt.	Severe Vent.Dilt.
RICP	23 60.5%	$+17.33$ -4.65	$+51.07$ -16.82	0	12	11
NICP	9 23.6%	$+6.39$ -2.1	$+49.5$ -22.44	0	2	6
EQUIV.	6 15.9%	$+7.33$ -3.39	$+44.17$ -17.77	1	3	2
T	38	13.16	49.5	1	17	19
		(range 3.5-27 mm Hg)	(range 15-80 mm Hg)			

ALL INFANTS (i.e. <1 year)

No significance was found, in fact in all cases the observations were
so identical that no statistical test even approached significance.
One can say therefore that the amplitude of the stress response bears
no relation to the volume of C.S.F. or degree of ventricular
dilatation.

In the neonatal group as a whole, the signs and symptoms are
listed in Table 4 in decreasing frequency with which they occurred.

In the infantile group again mean values and standard deviations
are shown in Table 5 for the mean resting and stress pressures and the
degree of ventricular dilatation. Again within this group, no
statistical significance accrued when the resting/stress ratios
and the actual elevation (stress-resting pressure) were tested within
each sub-group and the group as a whole, against the degree of
ventricular dilatation.

It is interesting that the mean resting pressure in states of R.I.C.P.
in neonates is 15.85 mm Hg, and very little higher than 17.33 mm Hg
over all infants monitored. This is not a high level of ventricular
pressure when compared to values in later years. Again, the signs
and symptoms are displayed in decreasing frequency for this group
of infants.

TABLE 4

SIGNS AND SYMPTOMS OF R.I.C.P.

Neonates i.e. 1 month

1. ↑OFC (13) i.e. in all cases

2. ↑AF (10)

3. ↑Scalp veins (5)

4. Sutural separation (4)

5. 'Sunsetting' (3)

6. Poor feeding (2)

7. Neck retraction (1)

8. Brain stem fit (1)

9. Jitteriness (1)

10. Irritability (1)

11. C.S.F. blow-outs (1)

12. Optic atrophy (1)

TABLE 5

SIGNS AND SYMPTOMS OF R.I.C.P.

Infants i.e. 1 year

1. ↑OFC (28)

2. ↑AF (17)

3. ↑Scalp veins (13)

4. 'Sunsetting' (9)

5. Sutural separation (7)

6. Lethargy and irritability (7)

7. Anorexia, vomiting, poor feeding (4)

8. Brain stem fits (3)

9. Head lag (2)

10. Opisthotonus (2)

11. Optic atrophy (2)

12. Remainder were (1) each, jitteriness, C.S.F. blow-out, course, bradycardia, hippus, squint, early papilloedema, pallor, loss fix and following, poor sleeping, sluggish pupils, stridor (hypertonic with occular anom.(hydran-encephaly).

CHAPTER 7

INTRACRANIAL PRESSURE CHANGES ASSOCIATED WITH CHILDHOOD SEIZURES

CHAPTER 7

INTRACRANIAL PRESSURE CHANGES ASSOCIATED WITH CHILDHOOD SEIZURES

INTRODUCTION

It is generally accepted that a rise in intracranial pressure
occurs during induced fits in the experimental animal, but it has
also been shown that a rise in intracranial pressure occurs in
experimentally induced seizures in paralysed human volunteers
(White et al 1961). Cronqvist et al 1967 found in one case a rise
in V.F.P. from 15 to 20 mm Hg at the same time as the E.E.G. indicated
a seizure. This patient did not have permanently raised I.C.P. and
was curarized and ventilated at the time.

Clinical experience suggests that raised intracranial pressure may
occur following prolonged epileptic seizures in children, as witnessed
by the dramatic improvement in conscious level that occurs after the
administration of mannitol and the direct observation of brain
swelling at operation (Gordon 1972).

Reported here are the recorded intracranial pressure changes in
several children known to be epileptic, who had co-incidental fits
during monitoring or their intracranial pressure.

During the study of more than 100 recordings, five children – all
known to have had previous seizures – had a clinical fit whilst
pressure was being monitored. Two other children also are included
to illustrate the changes in intracranial pressure that occur
normally with myoclonic jerks, presumed to be hypnagogic myoclonus.

The children had a variety of different disease entities: two had
post-meningitic hydrocephalus, one child had brain damage as a result
of non-accidental injury, one had spina bifida and hydrocephalus,
one had brain damage after perinatal asphyxia, one was probably

a case of tuberose sclerosis, and one young boy had subacute
sclerosing panencephalitis.

RESULTS

Major Convulsions

Two children suffered major tonic-clonic grand mal seizures
(Figs. 50 & 51). It can be seen in Fig. 50 from a boy with post-
meningitic hydrocephalus, that the resting ventricular pressure was
normal with a mean of 12 mm Hg prior to the onset of the convulsion.
The pressure rose to about 24 mm Hg shortly after the onset of the
seizure, appeared to fall and then showed a second rise to a peak
of 30 mm Hg. This fit occurred after a period of deep sleep, when
ordinarily the pressure is less than in the awake state. The
seizure resulted in an acute rise in pressure, with subsequent
elevation of the mean level for some time. The rise in pressure may
persist for as long as 20 minutes after cessation of a single clinical
ictus. Fig. 51 from a boy with hydrocephalus associated with
spina bifida, illustrates this point. Both these children had had
Rickham reservoirs inserted at previous surgery, therefore the
tracing reflected direct measurement of ventricular pressure.

Absence Seizure

Fig. 52 shows the pressure tracing in a mentally retarded boy who
had arrested hydrocephalus, with a Rickham reservoir in situ for
many years. His epilepsy was of a mixed type, with grand mal
seizures and also the Lennox Gaustaut type of myoclonic epilepsy
with stare, jerk and fall seizures. During monitoring of his
intracranial pressure he had several absence seizures without any
generalised motor component and without any clinical interference
with respiration. It can be seen that each seizure was accompanied

V.P.M.

mm.Hg

Fig. 50

V.P.M.

CHART SPEED 10mm./MIN.
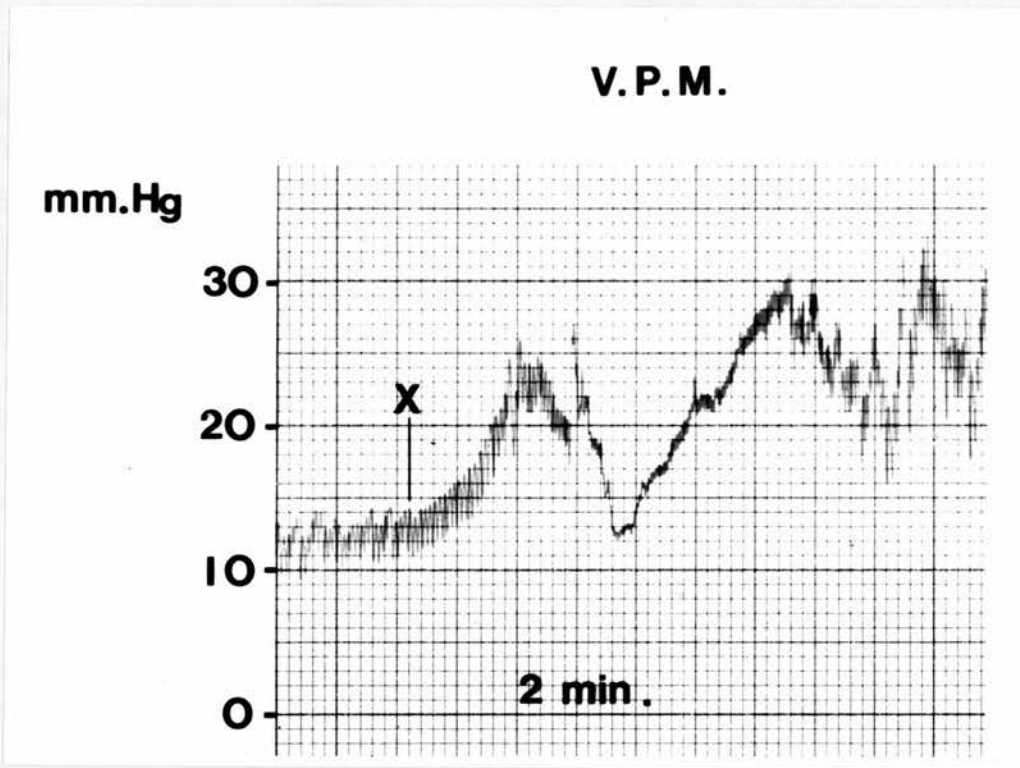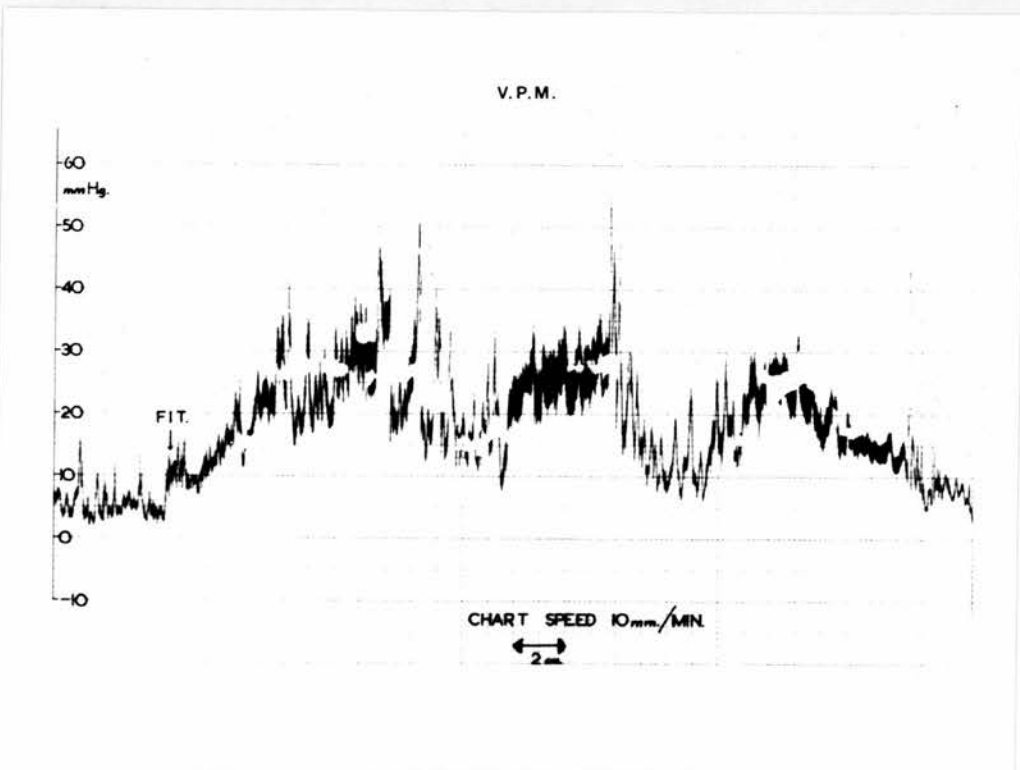
Fig. 51
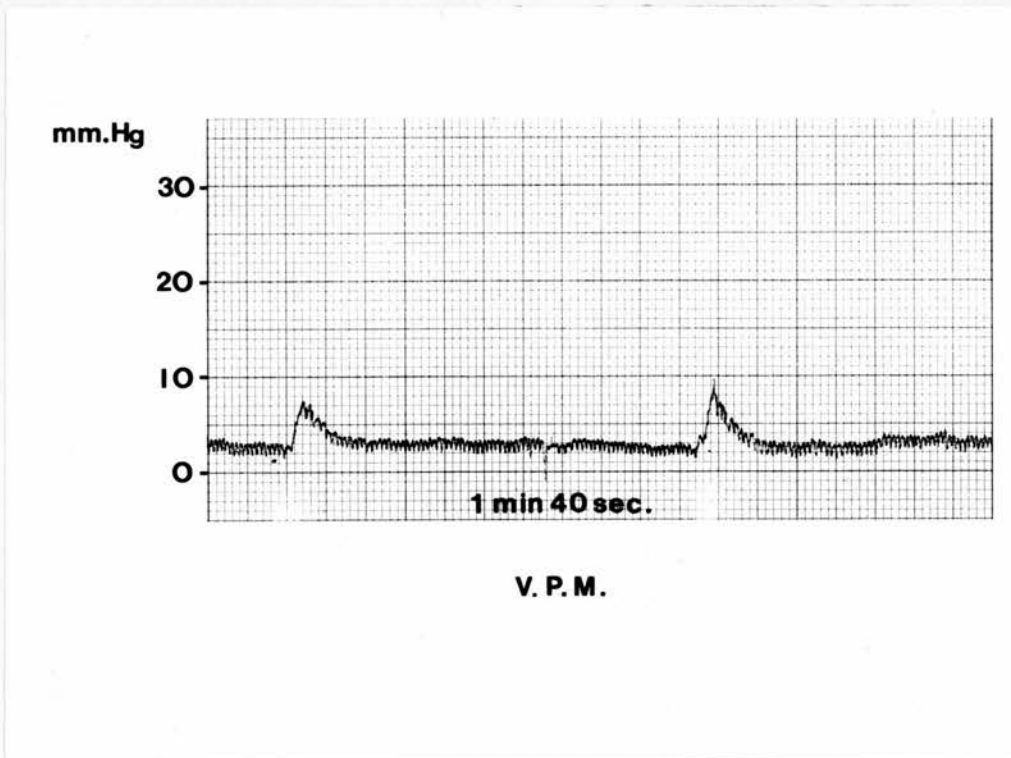
Fig.52

Fig.53

by a demonstrable change in intracranial pressure, from a baseline
of 3 mm Hg to a peak of 10 mm Hg.

Adversive Seizure

This child was a mentally handicapped, brain-damaged girl of 12
years who had mixed epilepsy but was particularly prone to
'grouping' of adversive seizures. A typical one is illustrated in
Fig. 53. Intracranial pressure was recorded via lumbar puncture.
An E.E.G. recording was taken at the same time and is shown super-
imposed in Fig. 54 . Again it can be seen that the pressure does not
rise prior to the seizure and starts to decline after the major
tonic component of the clinical fit, but it does not return to the
baseline immediately, even when the electrical activity ceases.

An important observation is shown in Fig. 55, which is a recording
taken from the same child as above but during a clinical abortive
seizure when she simply showed elevation of the eyes and a very
brief lifting of one arm, lasting only a fraction of a second
(i.e. the tonic phase was missing). It can be seen, however, that a
rise in intracranial pressure of 10 mm Hg occurred, with persistent
elevation for two minutes.

Myoclonic Seizures

Fig. 56 shows the pressure recording from the young boy with
subacute sclerosing panencephalitis. The recording was a direct
measurement of ventricular pressure through a Rickham reservoir which
had been inserted at the time of 'brain biopsy' because the cerebrum
had been found to be tense, and also to facilitate intraventricular
therapy with adenine arabinoside.

It can be seen in Fig. 57 that he has the classical 'metronomic
myoclonus' of subacute sclerosing panencephalitis. The pressure
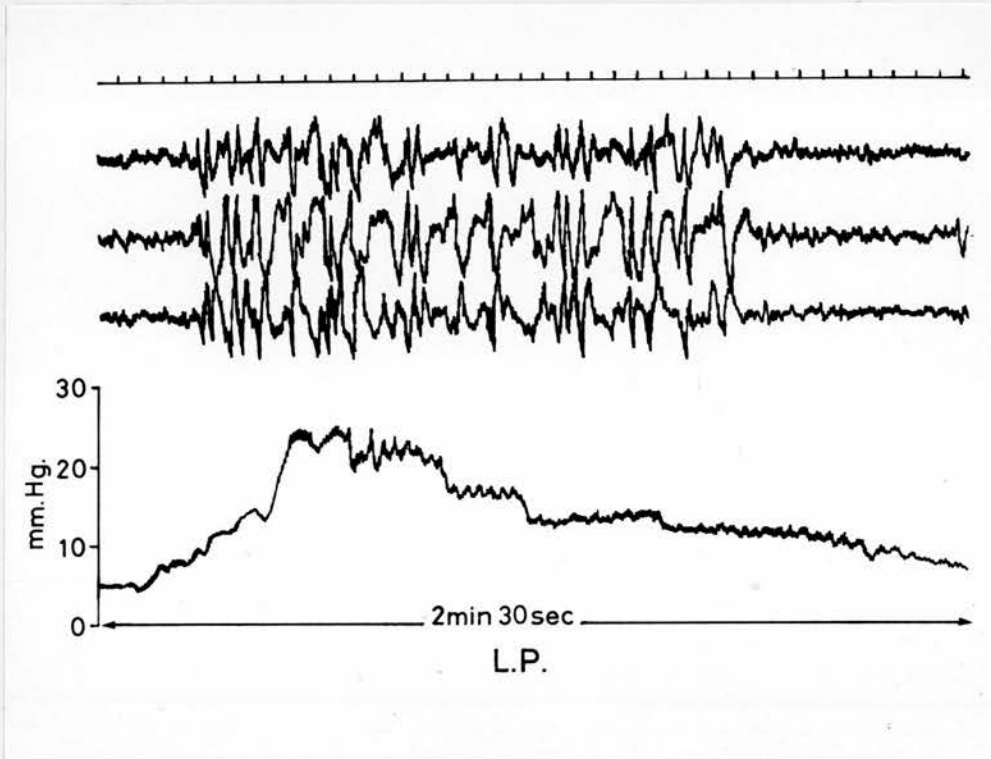
Fig.54
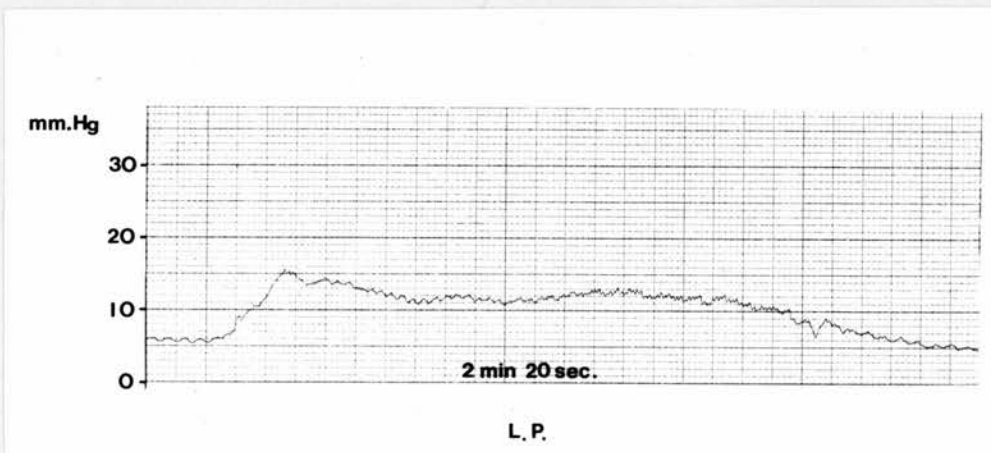
Fig.55
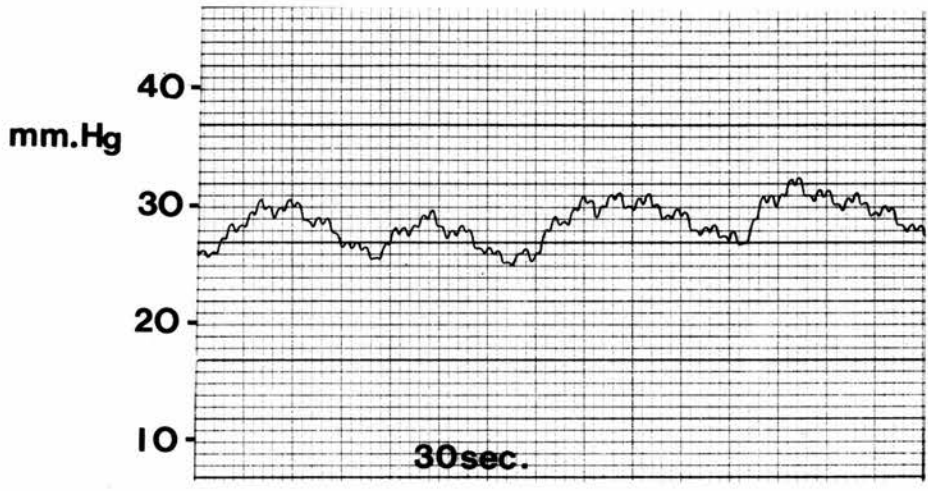
V. P. M.

Fig. 56
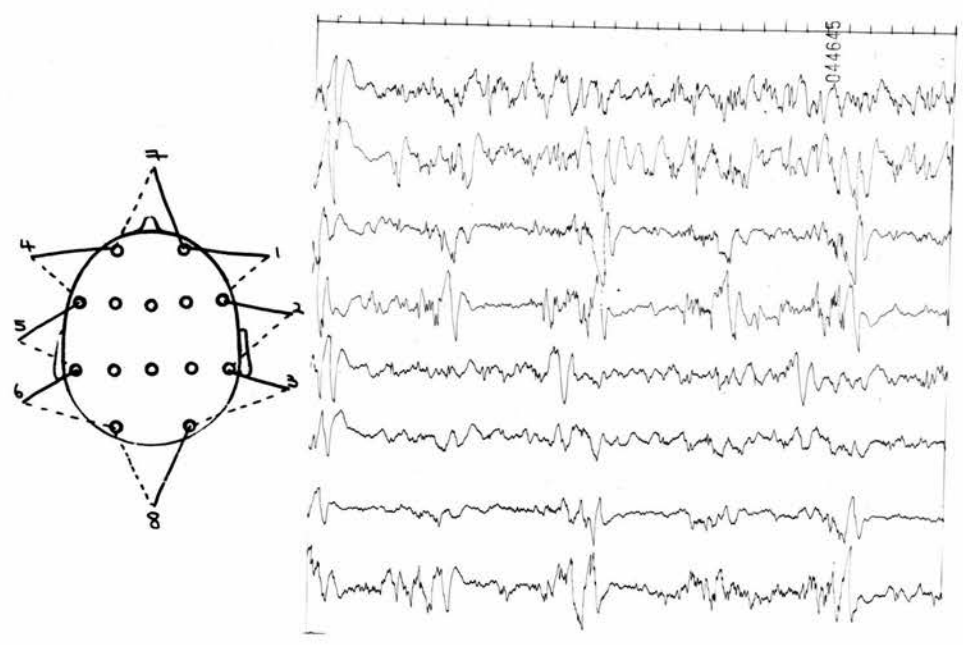
Fig. 57

recording shows that he has a very high resting intracranial pressure, above 25 mm Hg, but there is a fluctuation in the elevated baseline associated with each myoclonic jerk of the arms.

Hypnagogic Myoclonus

Figs. 58 and 59 are recordings from different children. The first is from a child with post-meningitic hydrocephalus who showed marked hypnagogic myoclonus associated with a 'saw-tooth' pattern of ventricular pressure. This tracing was obtained during R.E.M. sleep, and children with post-meningitic hydrocephalus appear to show prolongation of this phase of active sleep with exaggerated nocturnal myoclonus.

Fig. 59 is a pressure recording from a boy who suffered brain damage as a result of cerebral haemorrhage at birth and subsequently developed hydrocephalus. During monitoring in deep sleep the ventricular pressure fell from the initial rise in early sleep. As happened in all cases monitored, the period prior to awakening is associated with spiked pressure waves, with an increase in the baseline, and myoclonus occurs with these 'spikes'. Although it is well known that fits are likely to occur on awakening, in both these cases the myoclonus was not thought to be primarily epileptic in origin. Their E.E.G.'s were normal.

DISCUSSION

The results demonstrate that a rise in intracranial pressure usually accompanies any type of clinical fit. The major tonic-clonic consulsive seizures, as would be expected, were accompanied by a much greater rise in intracranial pressure than the absence of myoclonic types. It was surprising to find that a rise also accompanied the very minor absence seizure. Most normal children have a resting pressure of less than 12 mm Hg, so a rise of 7-10 mm Hg during an absence seizure
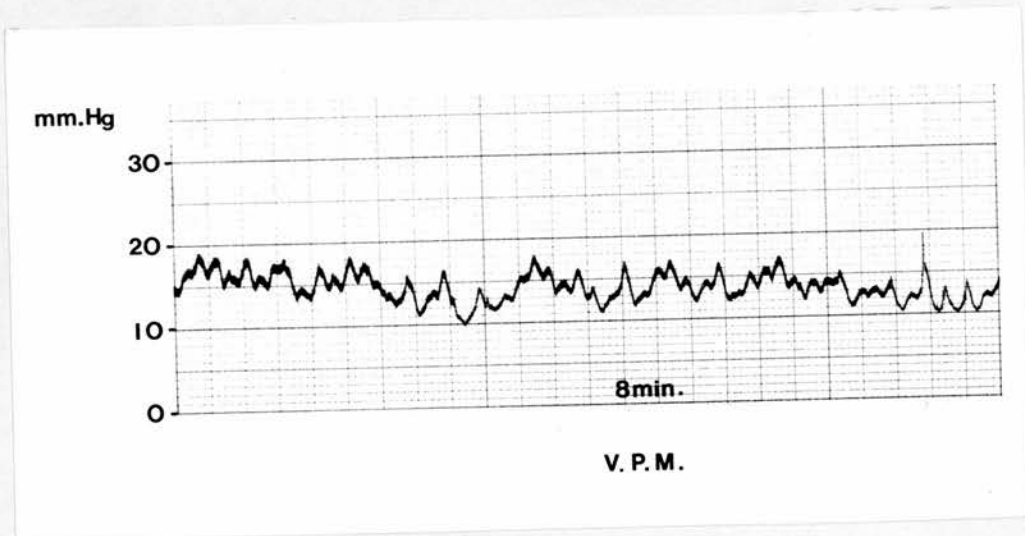
Fig. 58
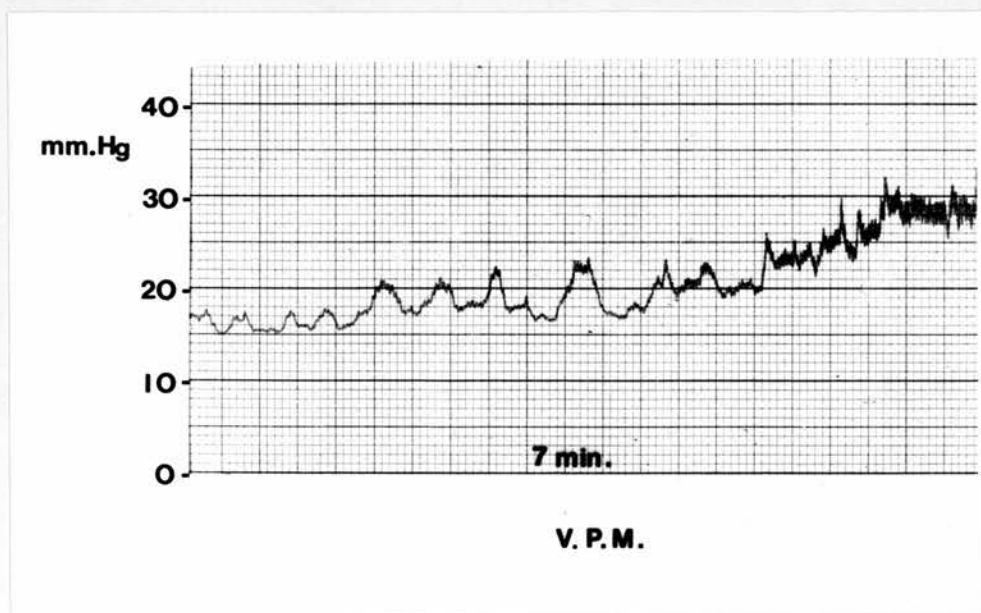
Fig. 59

represents double the resting pressure. Pressures of over 50 mm Hg have been recorded with major seizures, and the rise in pressure definitely lasts longer than the clinical fit or the abnormal E.E.G. discharge.

Very high intracranial pressure may of itself cause tonic fits, which are in effect episodes of decerebrate rigidity. These may come and go as intracranial pressure fluctuates, the so-called mesencephalic seizures occurring during plateau or A waves (Lundberg 1960), but more often they are the result of handling or stimulating the child. Sustained pressure over the trigeminal or perineal area will often reproduce the seizure and is a valuable clinical sign. An alternative explanation for these tonic fits is that raised ventricular pressure in association with hydrocephalus may alter endocrine or metabolic stability, resulting in a fit and a further increase in ventricular pressure.

Intracranial pressure may rise dramatically on going off to sleep, Case Number 96, and one must be aware that this is the time when fits are also most likely to occur; the unwary may not realise that the fits are the result and not the cause of pressure. In this series, only one child (with subacute sclerosing panencephalitis) had raised pressure prior to the onset of the seizure. One has witnessed episodes of decerebration association with sudden pressure rises, as well as focal seizures due to distension of porencephalic cysts, but these cases have been excluded from this series. Each of these five children had had prior seizures and all at some time had an epileptic E.E.G., all were brain-damaged and it was felt that their seizures were spontaneous ones. Very many more of the children in the total series were epileptic; most were monitored for several hours, if possible through one sleep cycle, so it is not surprising

that a few 'obligingly' had a seizure.

In tonic-clonic seizures there is a rise in central venous pressure
and retention of carbon dioxide and hypoxaemia because of the tonic
muscle spasm and arrest of respiration. The muscle spasm may also
cause metabolic acidosis as a result of lactic acid production.
However, these are not the only mechanisms whereby intracranial
pressure rises, since we must also explain the rise in pressure
associated with very minor clinical fits, the long duration
(e.g. 20 minutes after the seizure is finished), together with
evidence from human volunteers and experimental animal work showing
that the rise in pressure still occurred when the motor component
of the fit was prevented by curare and ventilation was maintained at
normal pCO_2. It was not possible to monitor end tidal CO_2 concen-
trations in these children. The rise in pressure is too rapid for
any change in C.S.F. production to be contributory. It is known that
in generalised seizures there is a massive sympathetic discharge,
with hypertension, tachycardia and hyperglycaemia, which is in part
to increase the cerebral blood-flow. This may rise by as much as
500 per cent.

The blood cannot increase its oxygen-carrying capacity, so the brain
can only either increase total blood flow or take more oxygen out of
what blood flow exists. The arteriovenous oxygen difference is
maintained unless decompensation occurs in severe status epilepticus
(Plum 1971), i.e. the great increase in oxygen demand by the
convulsing neurons is normally met by an increase in blood flow,
which is known to increase within 1 to 2 seconds of the onset of the
fit and to be accompanied by an increase in cerebral metabolic rate.
This is the most likely cause of the rise in intracranial pressure,
together with interference with ventilation in tonic-clonic seizures.
If this mechanism fails, for example by lowering blood pressure

with drugs such as diazepam or barbiturates, then cerebral blood-flow will fall. It has been demonstrated in primates that diazepam itself can lower cerebral blood-flow by 23 per cent, with resultant increase in cerebral anoxia (Papy and Naquet 1971). Those authors also found no relationship between cerebral blood-flow and electrical activity in the E.E.G. in the post-ictal period.

The convulsing neurons will be the ones most easily damaged by consumptive asphyxia should the amount of oxygen in the blood or the cerebral blood-flow fail. The latter is more important, as anaerobic metabolism is possible if blood flow is maintained to remove organic acids, but not if they then accumulate and inhibit enzyme systems. The result then is brain damage, for example a hemiplegia or cerebral atrophy. In most cases, therefore, the rise in intra-cranial pressure, like the rise in cardiac output and cerebral blood-flow, represents a compensatory mechanism.

In some cases a further mechanism, brain swelling, may complicate the picture. This is more likely to occur in status epilepticus than after a solitary fit. Acute brain swelling has been demonstrated at operation on children who have had repeated seizures (Gordon 1972) and, as mentioned in the introduction, one sees children who regain consciousness with great speed following the use of osmotic diuretics such as mannitol. It is possible that the prolonged rise in intracranial pressure following a group of seizures is due to cerebral oedema, but the acute rise at the onset of a seizure must relate to primary vascular change. Intracranial pressure of over 30 mm Hg may interfere with cerebral blood-flow, so pressures of 50 mm Hg, especially if the blood pressure was lowered by over-enthusiastic use of anti-convulsants, could set the scene for brain damage.

It is hoped that with the advent of non-invasive methods of determining cerebral blood-flow we shall be able to determine (a) if the rise in pressure is compensatory and due to a rise in cerebral blood-flow, with little risk of brain damage, or (b) whether sustained rises, e.g. for 20 minutes or more, are associated with falls in cerebral blood-flow due to brain oedema, when brain damage could result. A further fascinating aspect of the study is the relationship of the rises in intracranial pressure which occur on going to sleep and on awakening. In pathological states these can be quite tremendous and obviously could contribute to early morning headaches or even progressive intellectual deterioration in so-called 'normotensive' hydrocephalus. Although rises in intracranial pressure occur in normal individuals when going to sleep, these are of small amplitude and short duration – for example, 10 mm Hg for 15 seconds. Abnormal sleep patterns have been described in the E.E.G.'s of patients with epilepsy (Jovanovic 1967) and it is well known that fits, especially when originating from the temporal lobes, are likely to occur just after falling asleep or in the early morning sleep period (Janz 1974), at the same time as these paroxysmal rises in pressure described. The changes in intracranial pressure which occur on going to sleep and awakening from sleep are again very acute and paroxysmal and could not be explained by a change in C.S.F. production rate or by cerebral oedema, and again must be related to a sudden change in the regulation of cerebral blood-flow.

CHAPTER 8

MONOAMINE METABOLITES RELATED TO V.F.P. IN CHILDREN

INTRODUCTION

PATIENTS AND METHODS

      Patients

      Intracranial pressure measurement

      Collection of C.S.F.

      Biochemical Estimation

RESULTS AND DISCUSSION

SUMMARY

CHAPTER 8

MONOAMINE METABOLITES RELATED TO V.F.P. IN CHILDREN

INTRODUCTION

Children with hydrocephalus have been reported to have higher
5-hydroxyindolylacetic acid (5-HIAA), the metabolite of
5-hydroxytryptamine, in their ventricular C.S.F. than age-matched
controls (Anderson and Roos, 1969). Increased concentrations of
5-HIAA and of homovanillic acid (HVA), the metabolite of dopamine,
in ventricular C.S.F. have also been found in patients aged 3-65
years with raised intracranial pressure and impaired C.S.F. drainage
associated with a tumour (West et al 1972). Children with raised
intracranial pressure of a sub-acute or chronic nature often present
with affective disorders and occasionally 'pseudodementia'. Since
the monamines have been implicated in affective diseases (Ashcroft
et al 1972). It was considered appropriate to investigate the
relationship of intracranial pressure to the monamine metabolites
in ventricular C.S.F. The concentrations of 5-HIAA, HVA, total and
free 3-methoxy, 4-hydroxyphenylglycol (MHPG), the metabolite of
noradrenaline, were therefore measured in samples of ventricular
C.S.F. taken from children who were undergoing continuous ventricular
pressure recording.

PATIENTS AND METHOD

Patients

Twenty-one children aged 1 day to 11 years were investigated. There
were 8 females and 13 males. Ten of the children had hydrocephalus
associated with spina bifida complex, eight had congenital hydro-
cephalus, one had a cystic astrocytoma in the posterior fossa and
one was suffering from post-meningitic hydrocephalus. One further

child not included in the tabulated results had an expanded
cortical subarachnoid space and was investigated because of
'failure to thrive'. Of the 8 patients with congenital hydro-
cephalus, two had associated craniostenosis, one had an inversion
of number nine chromosome with secondary hypothyroidism and a hemi-
syndrome and one was mentally retarded, mute and had optic atrophy.
The ventricular fluid pressure was monitored for a number of
reasons including the assessment of active neonatal hydrocephalus
and the adequacy of C.S.F. shunting devices (Minns 1977). At the
time of monitoring the children were on no medication, had no active
infection of their central nervous system and had temperatures within
the normal range.

Intracranial Pressure Measurement

This was monitored (Minns 1977) by one of the following routes:

1. ventricular cannulation using an existing burr hole or open
   fontanelle,

2. using an existing Pudenz shunt with a patent proximal limb or
   a C.S.F. reservoir selectively inserted or already in situ

3. from the cortical subarachnoid space (one patient only).

Collection of Ventricular C.S.F.

The ventricular fluid pressure was recorded continuously prior to
removal of C.S.F. The first few drops of C.S.F. were discarded and
the next 2-3 ml collected into a glass test-tube. Coloured samples
were excluded. The sample was frozen and stored at $-20^{o}C$ until the
biochemical analyses were performed. One child had two ventricular
pressure recordings, one year apart. The results of the first
recording are shown in Table 6 , patient number 10. During the
second recording, three consecutive samples of 1-1.5 ml C.S.F. were
taken at intervals of 50 minutes. The first sample was taken

immediately after 20 minutes of non-R.E.M. sleep, the second
immediately after 50 minutes of R.E.M. sleep and the third after the
child had been awake for 2.5 minutes. The V.F.P. was monitored
throughout.

Biochemical Estimations

HVA and 5-HIAA were measured fluorimetrically and MHPG by gas-liquid
chromatography (Davidson et al 1977). Total MHPG was determined
after hydrolysis of the conjugate with a sulphatase preparation
('Helicase') and free MHPG was measured in the absence of the
sulphatase. Protein was determined by the method of Lowry et al 1951.

RESULTS AND DISCUSSION

In Table 6 the patients have been grouped according to their basal
ventricular pressure at the time of sampling of the C.S.F. Patients
12 and 17 had C.S.F. removed from the temporal horn of the lateral
ventricle. In all other patients C.S.F. was sampled from the body
of the lateral ventricle. Two children (patient Nos. 6 and 12) were
sampled on two separate occasions. Within the three groups comprising
normal pressure (<11 mm Hg), moderately raised pressure (11-20 mm Hg)
and very high pressure (>20 mm Hg), the patients have been arranged
according to age. The mean concentrations of each metabolite did
not differ significantly between the three pressure groups; nor was
there a significant correlation between the concentration of each
metabolite and the basal or stress pressures. In a 1 month old
boy (patient No. 10), the levels stayed remarkably constant, with the
exception of 5-HIAA in the final sample, in samples of C.S.F. obtained
during periods of different ventricular pressure levels of 7.5 mm Hg,
10 mm Hg and 12 mm Hg (Fig.60). The ventricular fluid pressure
is relatively lower in neonates and infants, because of an unfused
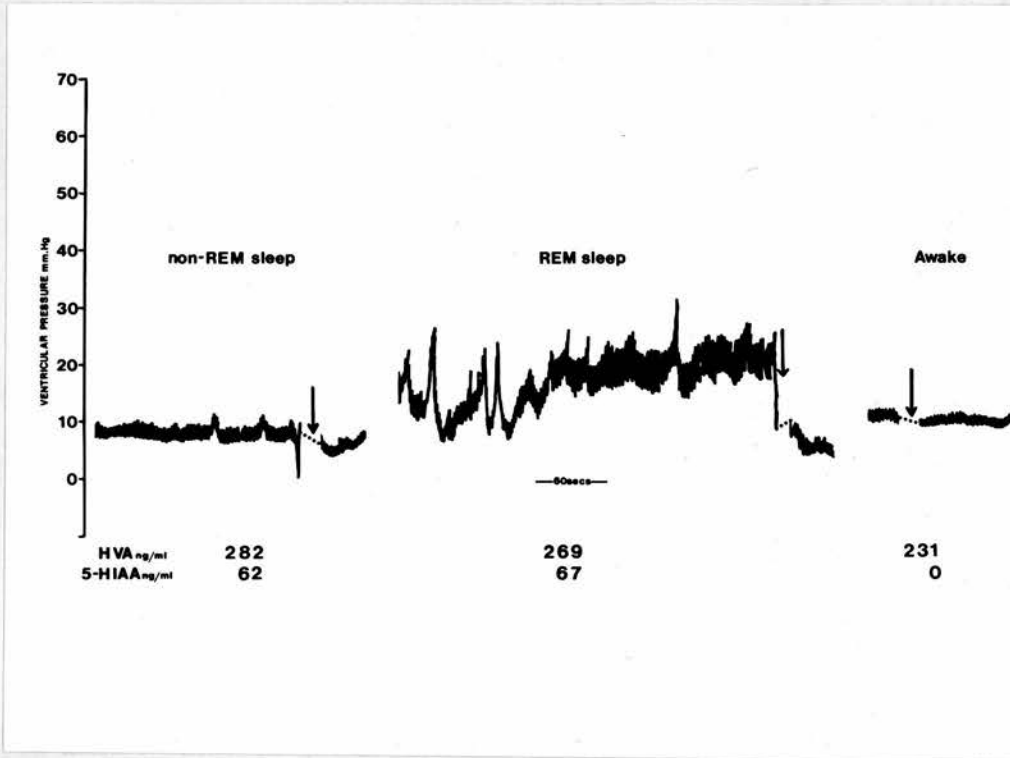
Fig.60

craniovertebral axis, than in older children (>18 months) in whom fontanelles and sutures are closed. No significant correlations were however obtained between the metabolite levels and pressure in children aged below or above 18 months. West et al 1972 found that the levels of HVA and 5-HIAA in ventricular C.S.F. were increased in patients with raised intracranial pressure secondary to a posterior fossa tumour in whom there was obstruction of C.S.F. drainage. Two patients (numbers 9 and 11) who had blocked aqueducts and moderately raised intraventricular pressured did not, however, have elevated metabolite levels. Indeed, patient number 9, who had extreme ventricular dilatation with a palium of 2-3 mm, had the lowest HVA and nearly lowest 5-HIAA of the samples measured, possibly due to dilution by the excessive volume of C.S.F. The highest HVA concentration appeared in patient 19 who had continuously elevated intracranial pressure with excursions ranging from 20-70 mm Hg. However, patient 20 who also had a very high pressure without any such excursions did not have elevated metabolite levels. It would therefore appear that there is no simple relationship between intra-ventricular fluid pressure and the concentrations of the amine metabolites in ventricular C.S.F.

The concentrations of HVA and MHPG in ventricular C.S.F. in the present age group (1 day to 11 years) were similar to values reported for non-hydrocephalic adults (West et al 1972, Porta et al 1975). The mean concentration of 5-HIAA was higher than in control adults (West et al 1972) and similar to reported levels in adult and childhood hydrocephalus (Ashcroft and Sharman 1960, Anderson and Roos 1969). The metabolite levels in a sample of C.S.F. from the subarachnoid space of a 7 month old female child were HVA 100 ng/ml, 5-HIAA 43 ng/ml, total MHPG 19 ng/ml and free MHPG 9 ng/ml. The

very high metabolite levels in patient 12 aged 1 month could be
attributed to a recent meningitis, since much lower levels were
obtained 5 months later when this patient was fully recovered.
Bakke et al 1974 related the increased HVA concentration which they
found in lumbar C.S.F. in bacterial infections to inflammation
of the choroid plexus and consequent reduction of metabolite
transport.

Total MHPG was negatively correlated with age ($r = 0.47$, $p < 0.05$).
There were no significant correlations between age and the levels of
HVA, 5-HIAA and free MHPG or between the different metabolites.
The increased concentration of total MHPG found in children aged
less than 6 months ($p < 0.01$) must be due to an increase in conjugated
MHPG since the level of free MHPG was unaltered (Table 7). An
increase in conjugated MHPG could be due to a decrease in the
elimination of MHPG or to an increased turnover of noradrenaline
in the brain. The latter could be related to proliferation of
noradrenaline-containing terminals which continues for up to
5 weeks after birth in the rat (Loizou 1969). Andersson and Roos
1969 found increased levels of 5-HIAA in ventricular C.S.F. in children
aged less than 12 weeks. In the present study no increase in
5-HIAA was detected in six patients within this age group or in
9 patients aged 6 months and less (Table 7). Since conjugated
MHPG appears to use the same transport mechanism as 5-HIAA and
HVA (Ashcroft et al 1976), an age-related increase in the activity
of this system could explain the increased levels of 5-HIAA
(Andersson and Roos 1969) and conjugated MHPG found in ventricular
C.S.F. of children aged less than 6 months.

SUMMARY

Samples of ventricular C.S.F. were obtained from 20 children in
whom ventricular fluid pressure was continuously monitored. The
concentrations of the monoamine metabolites, 5-hydroxy-indolylacetic
acid, homovanillic acid and 3-methoxy, 4-hydrocyphenylglycol, in the
C.S.F. were not related to the ventricular fluid pressure. The
mean concentration of 3-methoxy, 4-hydroxyphenylglycol was higher
(p<0.01) in 9 children aged 1 day to 6 months than in 12 children
aged 8 months to 11 years.

TABLE 6

VENTRICULAR C.S.F. PRESSURE AND CONCENTRATIONS OF HVA, 5-HIAA AND MHPG (ng/ml) IN VENTRICULAR C.S.F. IN CHILDREN

Patient No.	Sex	Age	Ventricular C.S.F. Pressure (mm Hg)		HVA	5-HIAA	MHPG		Protein mg/ml
			Basal	Stress			Total	Free	
Normal pressure (<11 mm Hg)									
1	M	4 days	5	25	241	308	16	–	1.10
2	M	2½ weeks	9	27	169	141	19	16	0.58
3	F	8 months	8	85	181	222	6	–	0.21
4	M	15 months	<10	–	–	413	20	14	0.34
5	F	22 months	8	–	142	251	–	–	0.19
6	F	4 yrs 10 mths	5	36	–	92	12	8	0.33
7	M	7 years	10	60	271	91	7	–	0.30
8	M	8 years	3	80	163	126	9	–	–
mean ± S.D.					+194 −46	+206 −107	+13 −5	+13 −3	
Raised pressure (11 − 20 mm Hg)									
9	M	1 day	17	45	105	93	24	23	1.26
10	M	2 weeks	15	45	154	54	26	9	1.02
11	M	5 weeks	12	45	220	201	12	7	0.22
12*	F	1 month	15	40	517	335	31	8	1.50

12	F	6 months	15	77	257	167	7	–	0.33
13	M	5 months	13	75	292	248	23	8	0.37
14	M	4 years	15–20	45	324	106	10	6	0.11
6	F	5 years	16	76	131	113	7	5	0.72
15	M	8 years	15	26	245	92	6	–	–
16	F	11 years	20	53	289	182	19	13	0.72
17	M	11 years	15–20	60–70	140	98	13	6	0.28
mean ± S.D.					$+243 \atop -112$	$+154 \atop -79$	$+16 \atop -8$	$+9 \atop -5$	
Very high pressure (>20 mm Hg)									
18	F	4 months	21	55	181	164	23	12	1.11
19	M	6 years	25	70	698	199	12	–	0.28
20	F	10 years	>30	45	190	272	10	8	0.21
mean ± S.D.					$+356 \atop -242$	$+212 \atop -45$	$+15 \atop -6$		
mean ± S.D. (n)					$+246 \atop -137$ (20)	$+180 \atop -91$ (22)	$+15 \atop -7$ (21)	$10 \atop 5$ (14)	

– no estimate

* recently recovered from meningitis

TABLE 7

EFFECT OF AGE ON THE CONCENTRATIONS OF HVA, 5-HIAA AND MHPG IN VENTRICULAR C.S.F. IN CHILDREN

Age Range	ng/ml		mean $\pm$ S.D. (n)	
	HVA	5-HIAA	MHPG	
			Total	Free
1 day – 6 months	237 $\pm$113(9)	190 $\pm$88(9)	20 $\pm$7(9)	12 $\pm$5(7)
8 months – 11 years	252 $\pm$154(11)	173 $\pm$91(13)	11* $\pm$4(12)	8 $\pm$3(7)

* $p < 0.01$

CHAPTER 9

VENTRICULAR PRESSURE RESPONSES DURING ANAESTHESIA IN CHILDREN WITH SPECIAL REFERENCE TO KETAMINE

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CHAPTER 9

VENTRICULAR PRESSURE RESPONSES DURING ANAESTHESIA IN CHILDREN, WITH

SPECIAL REFERENCE TO KETAMINE

INTRODUCTION

Ketamine is a non-barbiturate hypnotic drug which causes a
dissociative anaesthesia and is used for its analgesic and anaesthetic
action with minimal depression of the laryngeal reflexes. It is used
as an induction agent for general anaesthesia and commonly as a means
of sedating children for minor procedures, such as burns dressings,
bone marrow aspiration, muscle biopsies, lumbar punctures, computerised
tomography scans and other neuro-radiological procedures.
Various reports have appeared in the literature concerning the
effects of ketamine on intracranial pressure. Gardner, Olsen and
Lichtiger (1971) found an increase in the C.S.F. pressure in healthy
adult volunteers to values as high as 600 mm of water. Shapiro,
Wyte and Harris (1972) in 5 patients on 9 occasions, found a mean
increase in the intracranial pressure in patients with cerebral con-
ditions to the extent of 41.5 mm Hg (standard deviation 16.6).
List, Crumrine, Cascorbi and Weiss (1972) investigated two patients
and in one, who had normal pressure initially, there was a rise from
40 to 18.2 cms water. The other patient who had raised pressure
initially showed a marked increase from 42 to 112.5 cms water.
Sari, Okuda and Takeshita (1972) investigated 8 patients and in the
first four there was an increase in intracranial pressure in response
to ketamine, 153% of the control value. In the second four there was
an increase in intracranial pressure and this was controlled by
hyperventilation. Gibbs (1972) investigated 20 patients, 11 of these
with normal C.S.F. pressure produced no significant change but 6 out

of 9 with an intracranial space occupying lesion produced

a substantial rise in response to ketamine. Crumrine, Nulsen and

Weiss (1975) investigated 26 hydrocephalic children and found that

there was a rise in intracranial pressure from two to eight times

the control value. Mennella, Bracali, Schiavello (1976) gave

ketamine to 4 patients with normotensive hydrocephalus and found

increases in the pressure from 270% to 918% of the resting value.

They reached a maximum pressure in 3-4 minutes, remained so for

about 2 minutes and returned to normal limits in 12-15 minutes.

Most of these investigators have concluded that there was an increase

or no change at all in the C.S.F. pressure but the most recent report

by Kaul et al (1976) showed that in 10 patients studied, the

intracranial pressure rose in 3 cases and fell in 7 cases in response

to ketamine.

The importance of a knowledge of the expected response in the

clinical situation is obvious from an example: the case of a 12 year

old boy admitted with a bitemporal headache 3 weeks after a head

injury. Despite a normal skull x-ray, a midline echoencephalogram and

no abnormal neurological signs, a lumbar theca pressure was recorded

in excess of 20 mm Hg. Ketamine however had been used as a sedation

prior to the procedure and this resulted in a clinical dilemma which

necessitated a C.T. scan which was normal with no evidence of sub or

epidural collections.

The aim of this chapter therefore is to report the effect of ketamine

and other anaesthetic procedures on the intracranial pressure trace in

children.

The commonest cause of blindness now is hydrocephalus and it has been

reported that post-operative shunt surgery often results in dramatic

visual loss, presumed largely to be due to relative upward 'coning'.

It seems likely however, that raised intracranial pressure per se, due to events at the time of operation may also be responsible and hence these events during anaesthesia were monitored more closely.

PATIENTS

All the patients were studied in the Royal Hospital for Sick Children, Edinburgh. All were given ketamine, 9 by the intravenous route and 3 by the intramuscular route. The dose was 2-4 mgms per kilogram. All cases were given Atropine in a dose of 0.02 mgms per kilogram, a half to one hour prior to the ketamine injection. A total of 12 patients were studied. Their ages range from the neonatal period to 15 years, there were 7 males and 5 females. Five of the children had Rickham reservoirs in situ and so it was possible to monitor their ventricular pressure before and during anaesthesia in which ketamine was used as an induction agent. Seven of the children had their pressure monitored for minor procedures, three by the lumbar route and four by ventricular puncture.

All the patients studied had neurological diseases which included such conditions as hydrocephalus, herpes simplex encephalitis, intracranial haemorrhage, meningeal leukaemia etc.

In the 5 cases which were undergoing an operative procedure, ketamine was injected intravenously and where the clinical situation allowed it, a delay of 4-5 minutes was allowed before applying a face mask and continuing with the remainder of the anaesthetic, that is intubation, nitrous oxide etc. The operative procedures were all shunt revisions although one of these involved a thoracotomy to replace a calcified atrial positioned C.S.F. shunt. These five children all had hydrocephalus associated with the spina bifida complex.

RESULTS

No complications of the procedure were encountered. The results of
the intracranial pressure responses are tabulated in Table 8
and it can be seen that the mean resting pressure in which the
pressure was normal at the outset, was 7.2 mm Hg. The mean
increase in pressure from ketamine when the C.S.F. pressure was
elevated beforehand was 59 mm Hg. This is arrived at by excluding
the values for Case 10 and by arbitrarily picking a resting level in
Case 6 of 20 mm Hg. The mean time for the C.S.F. pressure to begin
to rise in response to ketamine given I.V. was 38.5 seconds and in
the 3 cases given I.M. 3 minutes, 12 seconds. The duration of the
pressure responses could not be calculated as other anaesthetic
manoeuvres supervened in most cases. However, in Case 2 the
duration of the response was 7 minutes 40 seconds, in Case 4 the
duration of the pressure response was 7 minutes. The shortest
period for a response to occur, occurred in Case 3 when the pressure
began elevation 15 seconds after the I.V. ketamine.

In Case 10 a marked elevation of ventricular pressure was recorded
in the anaesthetic pre-operative room, 2 and 20 mls C.S.F. were
removed. This reduced the pressure from 40 mm Hg to 5 mm Hg
(Fig. 61). Ketamine subsequently initiated a pressure rise to
60 mm Hg (Fig. 62). Although the pressure was within normal limits
at the time of the ketamine injection, with her unstable C.S.F.
dynamics, a rise was not surprising. This can be compared to Case 8,
who was a boy admitted with a substantial pressure problem
necessitating open ventricular drainage for a 24 hour period prior
to operation. On ceasing the drainage immediately pre-operatively,
the ventricular pressure was measured as normal (2.5 mm Hg)
and this rose to 7.5 mm Hg after ketamine. One could make a point of

TABLE 8

SEX	INITIAL	CASE	DIAGNOSIS	INDICATION	LP/VP	KETAMINE DOSE	ROUTE	KETAMINE RESPONSE	AGE	PROCEDURE
M	B.B.	1	Spina Bif.& Hydroc.	Pneumoventiculogram	V.P.	6 mgms	IV	5-10 mm Hg	1 day	Minor
F	T.B.	2	Herp.Simp.Encephal.	Infection screen	L.P.	25 mgms	IV	3-10 mm Hg	2½ yr	Minor
F	I.C.	3	Occipit.Meningocoel.	Shunt exploration	V.P.	100 mgms	IV	15-64 mm Hg	11 yr	G.A.-Oper
F	I.C.	4	Occipit.Meningocoel.	Thoracotomy	V.P.	120 mgms	IM	16-110 mm Hg	11 yr	G.A.-Oper
M	D.L.	5	Intracran.Haemorr.	Pneumoventriculogram	V.P.	20 mgms	IV	6-12 mm Hg	11 mt	Minor
M	A.B.	*6	Ac.Lymphoblas.Leuk.	Rais.Intrac.Press.	L.P.	90 mgms	IV	R-100 mm Hg	10 yr	Minor
M	C.S.	7	Hydroc. Men.Handicap	Investigations	L.P.	50 mgms	IV	3-3 mm Hg	12 yr	Minor
M	G.S.	8	Spina Bif.& Hydroc.	Shunt revision	V.P.	50 mgms	IV	2.5-7.5 mm Hg	5 yr	G.A.-Oper.
M	O.D.	9	Spina Bif.& Hydroc.	Shunt revision	V.P.	16 mgms	IV	5-15 mm Hg	14 mt	G.A.-Oper.
F	J.P.	**10	Spina Bif.& Hydroc.	Shunt revision	V.P.	50 mgms	IV	40,5-60 mm Hg	6y2mt	G.A.-Oper.
M	J.McM.	11	Spina Bif.& Hydroc.	Sedation procedure	V.P.	50 mgms	IM	5-23 mm Hg	11 yr	Minor
F	G.M.	12	Spina Bif.& Hydroc.	Sedation for VPM	V.P.	25 mgms	IM	12-25 mm Hg	4 yr	Minor

* Child with meningeal leukaemia and papilloedema

** Pre-operative pressure of 40 mm Hg and 20 ml. of C.S.F. removal reduced pressure to 5 mm Hg before ketamine was administered
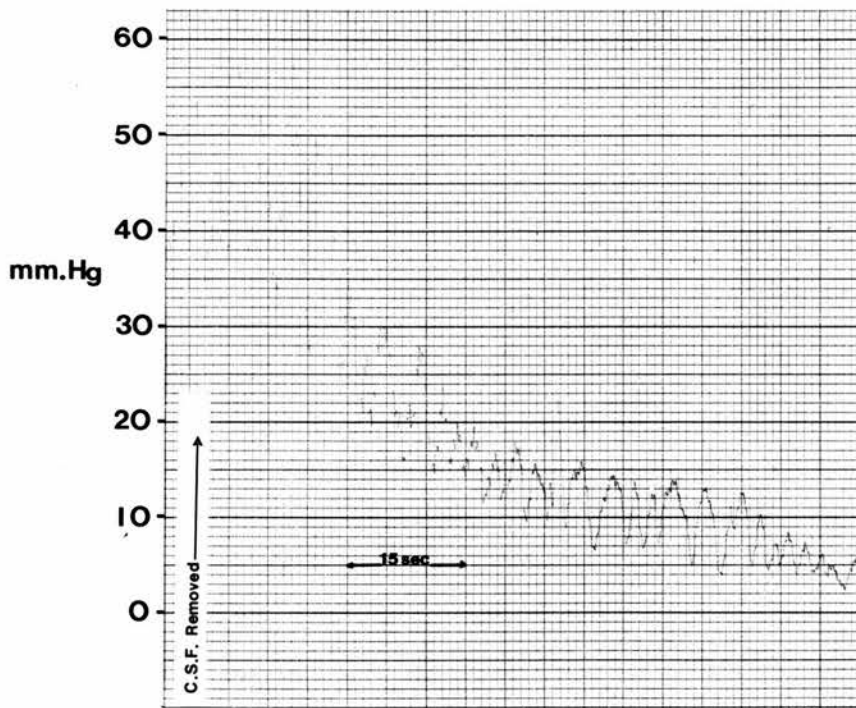
Fig. 61
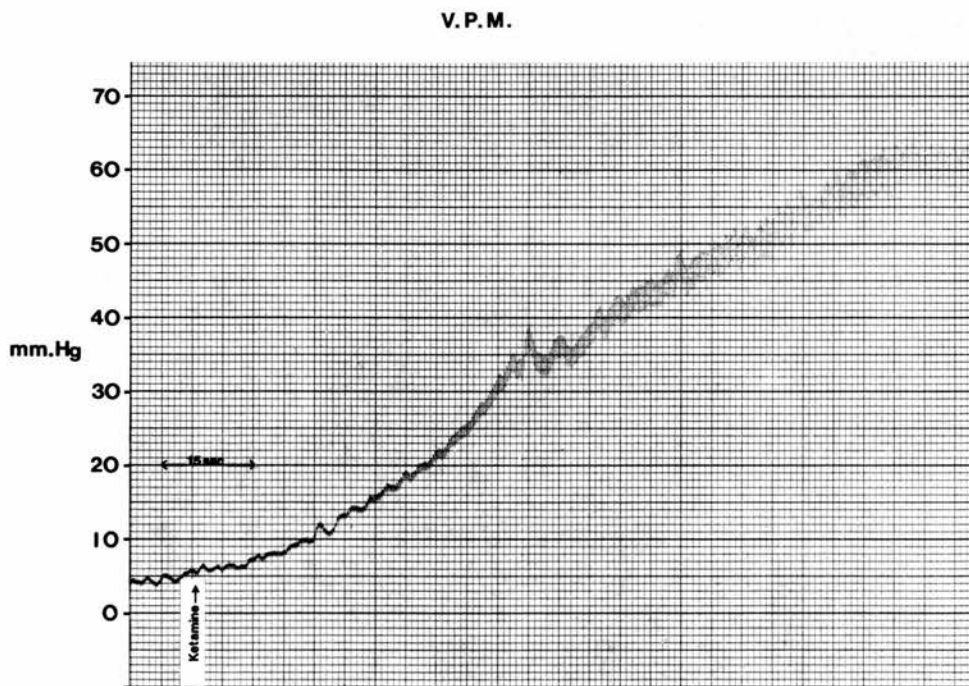
Fig.62

comparison here that open ventricular drainage improved the C.S.F. dynamics more than regular tapping of C.S.F. to control pressure pre-operatively. Laryngoscopy and endotracheal intubation however more than trebled this child's pressure level (Fig. 63). The previously given short acting paralytic suxamethonium did not have any effect on the ventricular pressure and this is in accord with other observers. In this child the anaesthetic manipulation was potentially more of a risk to him than the ketamine. It is remarkable that in all cases at the time that the vocal chords are touched by the laryngoscope or endotracheal tube, a momentary reflex pressure response occurs. The point should be made that because of this pressure response to endotracheal intubation or laryngoscopy that it is probably a good prophylactic practice to hyperventilate with a mask prior to inserting an endotracheal tube and a laryngoscope, in all cases undergoing anaesthesia but in particular those where the intracranial pressure dynamics are suspect. Although systemic blood pressures were recorded clinically, it was felt that without continuous direct reading intra-arterial monitoring, an estimate of the C.P.P. would not be sufficiently reliable to comment on the possible tissue hypoxia which might occur in this series.

In Case 6 a boy 10 years of age with an acute lymphoblastic leukaemia in remission, who had previously had a testicular relapse, presented with a 3 week history of headache, vomiting, malaise and a florid papilloedema. Ketamine was given prior to the lumbar puncture and bone marrow aspiration. Consequently no pre-ketamine level is recorded here but there is evidence that it was raised clinically. The use of the lumbar puncture in this situation finds justification in the literature (Pochedly 1973). Meningeal
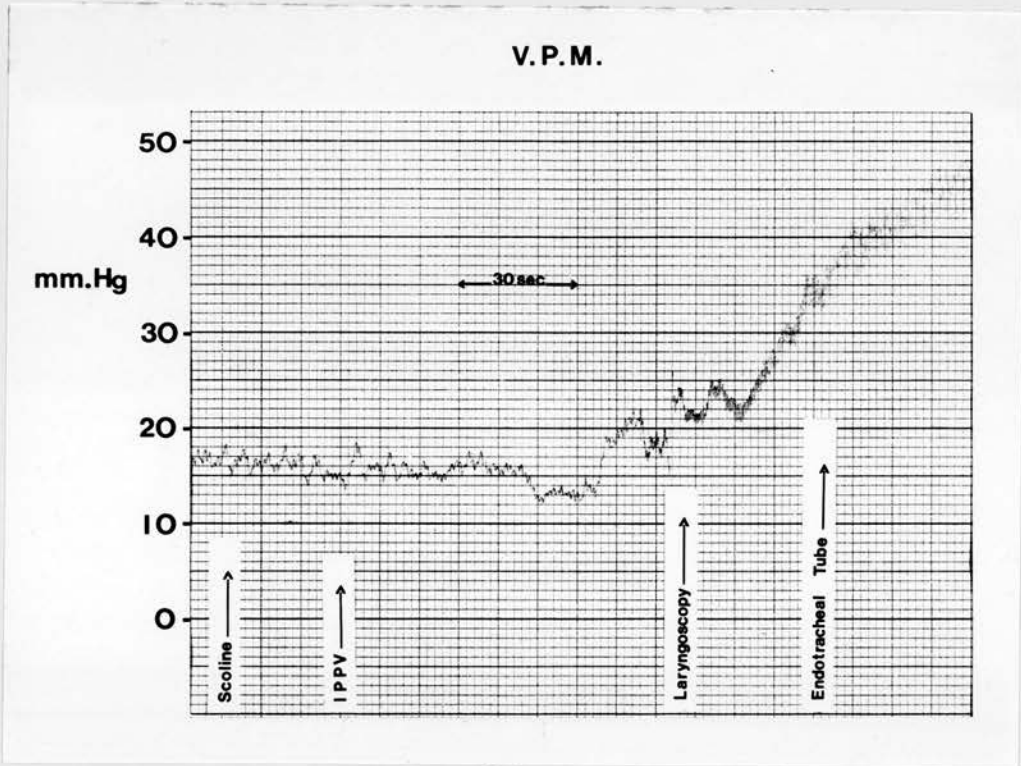
Fig. 63
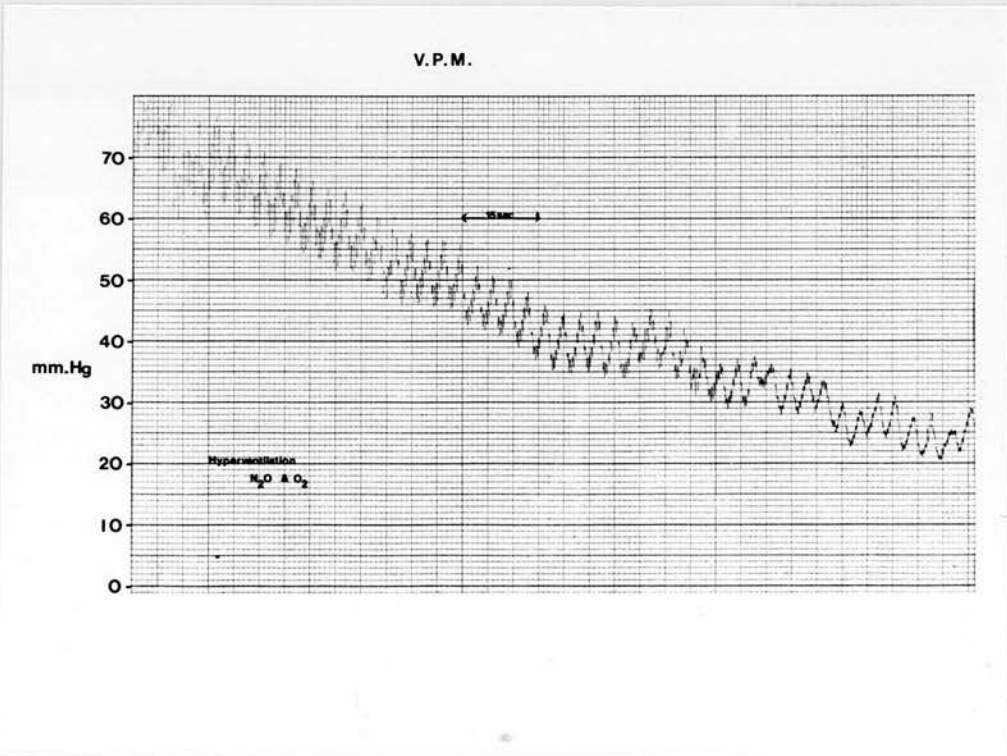
Fig. 64

leukaemia was suspected here and cases of meningeal leukaemia without space occupation do not 'cone'. In fact lumbar puncture may be therapeutic even without the use of intrathecal cytotoxic drugs. This is similar to the use of L.P. in cases of benign intracranial hypertension in childhood. Ketamine was considered a necessary adjunct to prevent movement during the L.P. to reduce the risk of a dural tear with subsequent leukaemic spread.

In Case 4 the routine for anaesthetic had been ketamine, oxygen, intubation and not until hyperventilation with nitrous oxide and oxygen of 5 litres each, did the pressure return to normal limits (Fig. 64). Excessively high pressures were encountered during induction for 7 minutes in this case and it is significant that the mean duration of raised pressure in excess of 20 mm Hg for any event at the time of induction of anaesthesia in this series was $7\frac{1}{2}$ minutes, mostly due to ketamine, laryngoscopy, and inserting the endotracheal tube.

A girl with a previous occipital meningocoele was admitted in a state of 'pseudo-dementia' with chronically elevated ventricular pressure of the order of 15 mm Hg in the awake state (Case 3). A substantial rise is seen with ketamine (Fig. 65) accentuated slightly by pancuronium until paralysis was complete and then hyperventilation with a 3 + 3 litre mixture, of nitrous oxide and oxygen at a ventilation rate of 20 per minute again controlled the pressure. Later in the course of this operation ketamine was given in a dose of 2 mm per kilogram IV while ventilation was controlled and presumably blood gases stable. On this occasion no rise in ventricular pressure occurred (Fig. 66). Incidentally the narcotic analgesic Fentanyl given during this anaesthetic resulted in no change in the ventricular pressure level. At the conclusion of this operative procedure, the
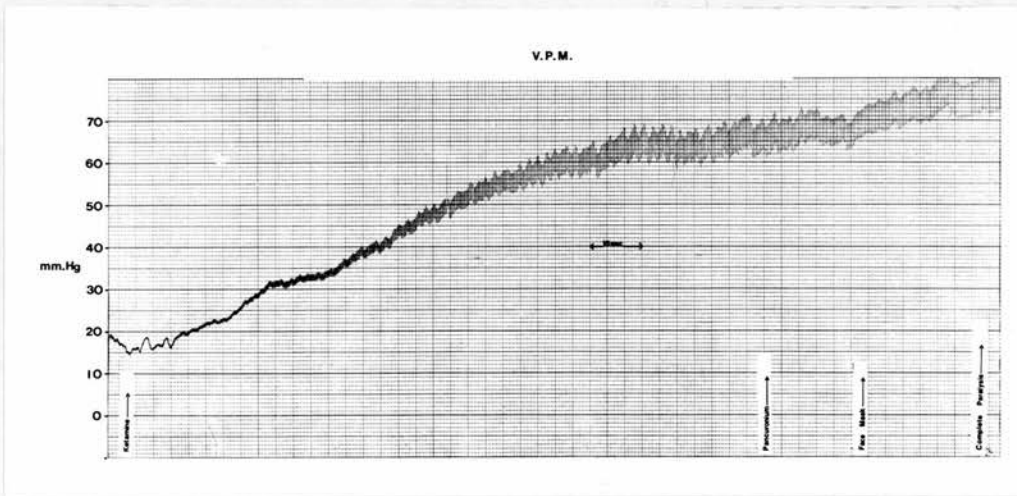
Fig.65
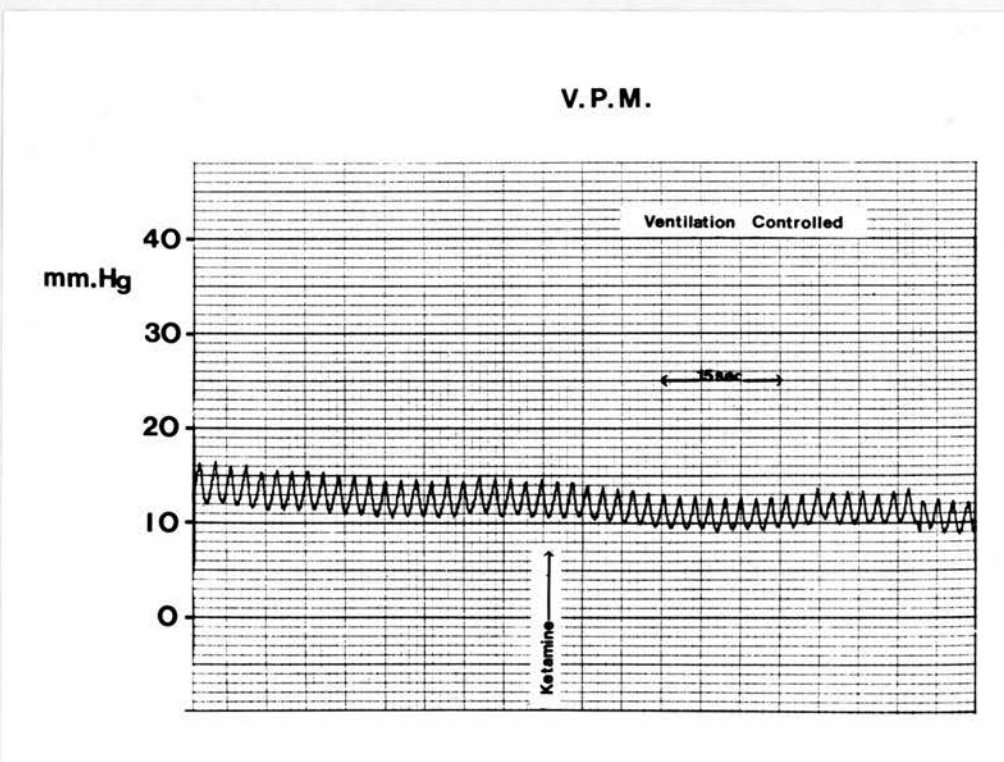
Fig.66

respirator was switched off and spontaneous breathing allowed.

It is not an uncommon practice at this stage following muscle paralysis to stimulate the respiratory centre transiently with 2 litres of carbon dioxide and 4 litres of oxygen admixture before 100% oxygen alone is used (Fig. 67). The reason the ventricular pressure is elevated at the conclusion of this operation is that the calcified distal portion of the atrically placed shunt could not be removed at this operation and required a more radical approach.

In Case 5 a mentally handicapped child with 'infantile spasms', hydrocephalus and a C.S.F. shunt in situ, required ventricular C.S.F. and pneumoventriculography. During the course of the pressure responses to ketamine (a positive rise of 6 mm Hg) an oscillating pattern of ventricular pressure recording was seen, not unlike those noted during active sleep with hypnagogic myoclonus. It is tempting therefore to reason that since the changes in sleep may be related to CO_2 responsiveness and that ketamine reduces sleep which allows the cerebrovascular tree to remain sensitive to the effects of CO_2, that ketamine may have a place in identifying the child with labile C.S.F. dynamics, thus producing an early rise in pressure and reducing the period of ventricular pressure monitoring necessary.

In Case 7 no change occurred in the C.S.F. pressure after I.V. ketamine, and since this was a child with mixed epilepsy who was prone to severe grouping of seizures, E.E.G. control was obtained throughout. Reports in the literature of fit precipitation in children by ketamine (Bennett 1973 and Walker 1972) have necessitated a manufacturer's warning of this possible adverse reaction. (Schwarz 1974) however has shown that the E.E.G. with
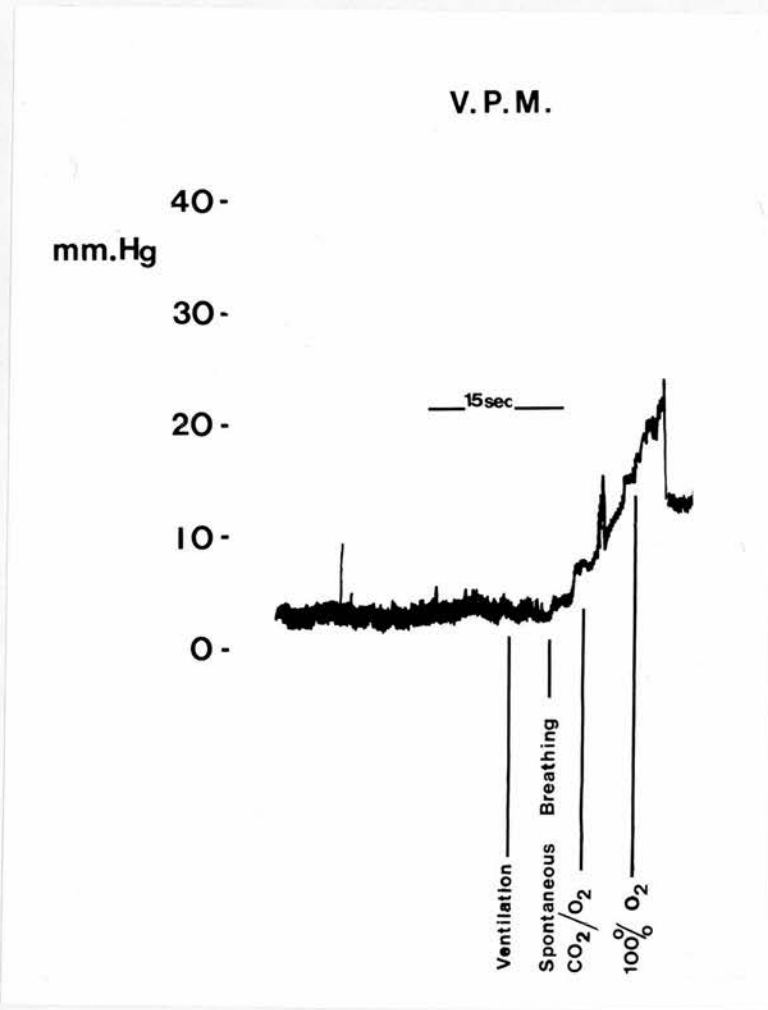
Fig.67
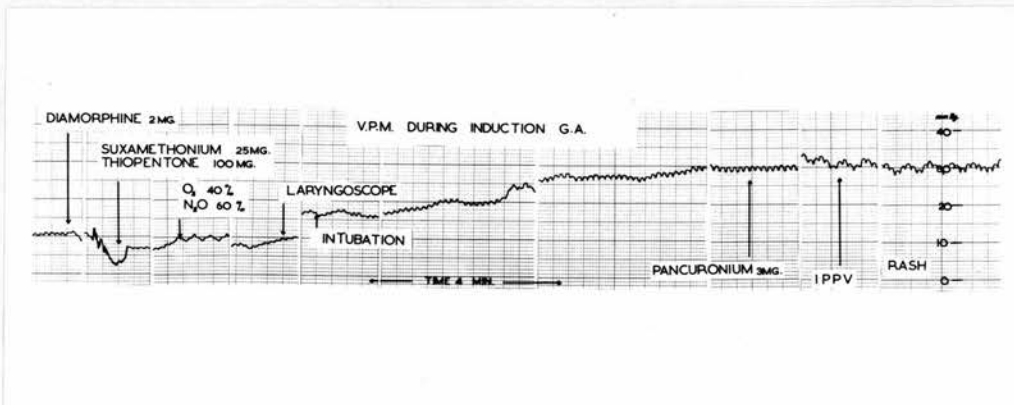
Fig.68

ketamine is similar to that occurring during anaesthesia or
sleep with no increase in spike activity. In our case the rhythmical
activity with periodic epileptiform discharges prior to the
injection changed immediately on injection to an irregular slow
wave pattern with superimposed faster activity and no epileptiform
discharges occurred. This remained so for about 20 minutes until
the drug effects wore off when the previous pattern recurred.

The effects of induction of anaesthesia without the use of ketamine
(Fig. 68) can be seen. The effect of diamorphine with a minimal
reduction in ventricular pressure, sodium thiopentone with its
cerebral vaso-constrictive effect and marked drop in ventricular
pressure. The nitrous oxide and oxygen mixture, which in this case
did not significantly alter pressure (although it is claimed to be
by some a mild cerebral vaso-dilator). Laryngoscopy and intubation
again trebled the resting ventricular pressure and pancuronium in this
instance had no effect.

A complete operation for shunt revision during which ventricular
pressure was monitored is shown in Fig. 69 . The various sections
of the tracing labelled alphabetically will be identified. The
induction of this anaesthetic with gas oxygen and halothane was not
included in the monitoring.

(A) indicates the effect of halothane in which the respiratory
component has been largely obliterated. Between points (B) and (C)
the child was breathing air by herself and at point (D) the ventilator
was commenced. At (E) 1 in 400,000 Lignocaine was injected locally
into the scalp. At point (F) a local anaesthetic was injected into
the abdominal wall. At point (G) pancuronium 1 mgm was given and
halothane turned down to 0.5%. The child continued with 4 litres of
nitrous oxide and 2 litres of oxygen. At point (H) the first surgical
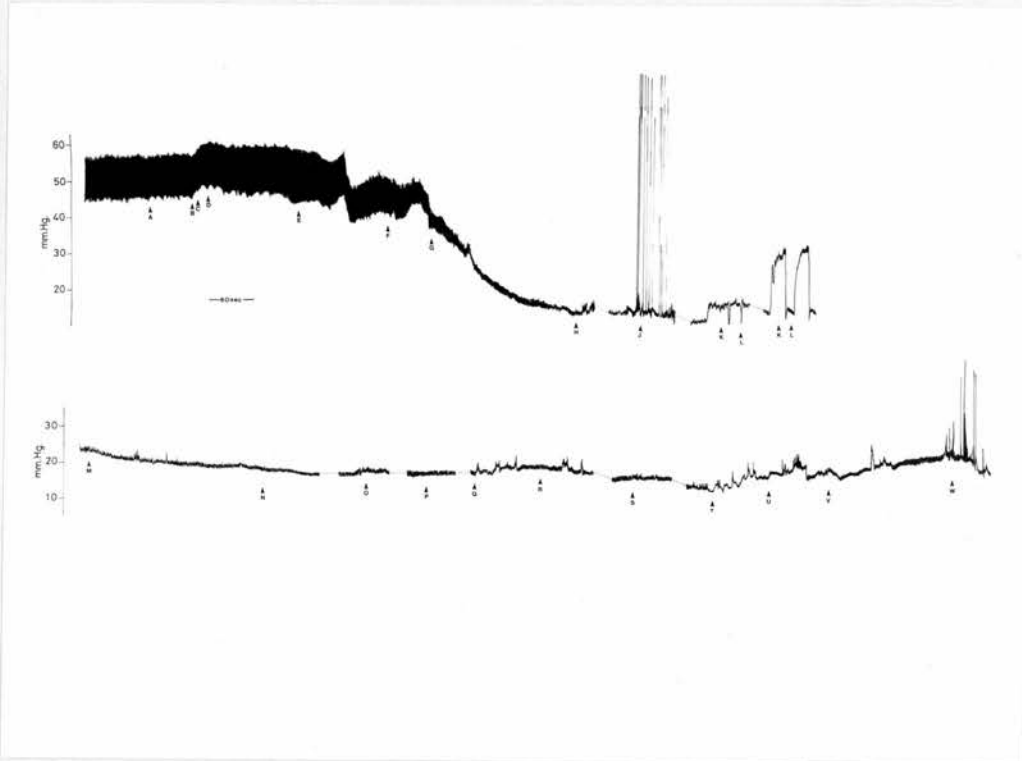
Fig.69

incision was undertaken. At (J) the artefact is due to the use of the diathermy. At point (K) the drill is used on the skull and (L) is cessation of drilling. At point (M) is a stellate incision of the dura with some C.S.F. oozing and a slow drop in C.S.F. pressure to (N). Shortly after this C.S.F. was obtained at 6 cms from the ventricles, that is, the brain was swollen and the ventricles small. At point (O) the ventricular end of the shunt was completely inserted. Between (O) and (P) there was the abdominal incision and dissection and at point (P) insertion of the abdominal end of the ventriculo-peritoneal shunt. Shortly after point (P) the operation had been in progress for 1 hour. Then suturing of the abdominal wound was commenced and introduction of the subcutaneous tube. From point (Q) with an increase in pressure of just over $1\frac{1}{2}$ mm Hg to point (R) at which point diamorphine 1 mgm was given I.V. and the halothane switched off. This was followed by pumping of the valve 3 or 4 times as a trial and then suturing of the wound commenced just before point (S). At point (S) a further 1 mgm of diamorphine was given. This was repeated again and then at point (T) the ventilator was turned off. At point (U) dressings were required and at point (V) an airway inserted. At point (W) the child is straining against the airway. This child was aged 11 years and had been undergoing pre-operative monitoring of his intracranial pressure for 25 hours via his Rickham reservoir. Throughout all these monitorings the children's temperatures were normal.

As mentioned at the outset, there is a clinical dilemma when one does not know the effect of a particular sedative or anaesthetic drug on the C.S.F. dynamics; a similar situation arises whenever one uses an anaesthetic drug which has a known elevating effect on C.S.F. pressure when it is used as a sedative or pre-lumbar puncutre analgesia, for e.g. the case of a child aged 10 years with a lymphomosarcoma of the

tonsil was given treatment with Vincristine, other cytotoxics and radiotherapy. During the course of one lumbar puncture he developed a 6th cranial nerve weakness and C.T. scan showed a mild ventricular dilatation. He subsequently developed 3rd, 6th, 7th and 12th cranial nerve palsies and therefore was given a careful lumbar puncture under general anaesthetic with nitrous oxide and oxygen and 'a touch' of halothane. Prior to the L.P. he was given sedation with diamorphine 2.5 mgms. Obviously one needs C.S.F. to establish whether there is a recurrence of his leukaemia with malignant cells in the spinal fluid, but when these drugs are used prior to the procedure it is difficult to interpret the pressure response. In this case an L.P. was done with 5 litres of nitrous oxide and 25% oxygen using a 22 gauge $2\frac{1}{2}$" needle. The mean pressure response is 18 mm Hg and at the end of the procedure one is still unsure whether his equivocally raised C.S.F. pressure is due to intrathecal cytotoxics or whether it is due to meningeal leukaemia, or whether it is due to the anaesthetic drug etc. Hence the importance of using something which has no effect on the C.S.F. pressure before doing the L.P. Certainly this child's pressure response looks like a typical halothane effect but at a much reduced level.

DISCUSSION

It is said that 'fully half of the operations in neurosurgery will either be rendered impossible or be performed at considerable prejudice to the patient's health and complete recovery, if the activities of the anaesthetist raise the intracranial pressure' (A.R. Hunter 1975).

Ketamine

Ketamine was first advocated for use as an anaesthetic agent during
diagnostic procedures in neurological patients by Corssen et al (1969),
Wilson Fotias and Dillon (1969). Its hypertensive action made some
authors issue a caution in its use in patients with intracerebral
vascular lesions (Brown, Col and Murray 1970) but not until Evans
et al (1971) was it suggested that the administration of ketamine
might be associated with undesirable levels of C.S.F. pressure.
Gardner, Olsen and Lichtiger (1971) showed a significant elevation of
C.S.F. pressure in normal man, measured at the level of the lumbar
subarachnoid space. This was confirmed by Evans, Rosen and Weeks (1971).

Gardner, Dannemiller and Dean (1972) reported two cases in which a
rapid rise in intracranial pressure occurred during ketamine anaes-
thesia. In one patient who had normal intracranial pressure initially,
the magnitude of the rise was similar to that noted at the lumbar
subarachnoid space and in the patient with elevated intracranial
pressure the ventricular C.S.F. pressure rose precariously during
ketamine.

They postulated that the mechanism responsible for the increase in
C.S.F. pressure in man may be similar to that responsible for the same
phenomena in dogs, namely the increase in cerebral blood flow.
Since ketamine causes an increase in cardiac output (Virtus, Alanis,
Mori et al (1967), Stanley, Hunt and Willis (1968) and Traber, Wilson
et al (1968) and since there is no change or a decrease in peripheral
resistance, it must increase blood flow to some organs. If the brain
were such an organ a rise in C.S.F. pressure would be expected. The
relationship between C.S.F. pressure and cerebral blood flow and a
arterior pCO_2 was already well established (Reivich 1964).

Ketamine affects the cerebral arterial tree more when used alone
than when it is used in combination with other anaesthetic agents,
e.g. nitrous exode (Bovill 1971).

Ketamine resembles halothane in its ability to increase cerebral
blood flow. Dawson, Michenfelder and Theye (1971) found an increase
in cerebral blood flow of 80% within 5 minutes and Takeshita, Okuda
and Sari (1972) found an increase of 62% in the cerebral blood flow
within the 5 minutes. Macdowell (1969) also found an increase in
the cerebral blood flow. In all situations it returned to normal
limits within 20 minutes. Therefore ketamine is a pronounced cerebral
vaso-dilator and increases the CMRO of 12% during normocapnia
(Takeshita et al 1972).

Whyte et al (1972) studied the effects of ketamine in two patients,
one with and one eithout raised intracranial pressure. They were able
to demonstrate a marked rise in intra-ventricular pressure following
I.V. injection of ketamine in one patient with raised intracranial
pressure. They also noted reduction of this response with thiopentone
and comment that the short duration of this modification is related
to the time taken for distribution of the thiopentone.

Gibbs (1972) found that the magnitudes of the changes in both C.S.F.
and arterial pressures were less than those reported by Gardner,
Olsen and Lichtiger (1971). They found that the rise in C.S.F. pressure
associated with ketamine was minimal in the normal anaesthetised subjects
but considerably greater in those patients with disturbed C.S.F.
pathways as a consequence of intracerebral lesions and they found
that the mean values of pCO_2 were similar in both groups of patients
and they felt it unlikely that ventilation played a part in the
different C.S.F. responses in the two groups. They also noted a rise
in arterial pressure associated with ketamine, a fact which is well

known because ketamine is a cardiovascular stimulant, increasing the cardiac output and the arterial pressure but with little effect on the total peripheral resistance (Johnston 1976). Gibbs (1972) noted that the rise in arterial pressure was greater in patients with space occupying lesions. He found that the C.P.P. was in general maintained, although in the patients who had marked rises in C.S.F. pressure with ketamine, the C.P.P. did fall. The question whether the rise in arterial pressure would protect areas of marginally ischaemic brain against the adverse effects of a further rise in intracerebral pressure was not answered.

Tekeshita et al (1972) suggested that ketamine increases cerebral blood flow and C.P.P. but does not significantly alter cerebral metabolism in normal man. By contrast, halothane causes a fall in arterial pressure and a rise in cerebral blood flow in normocapnic patients (Freeman 1969).

Jennett et al (1969) showed the effect of inhalational anaesthetic agents in raising the C.S.F. pressure was more marked with intra-cranial space occupying lesions. So although halothane and ketamine are similar in that they increase C.S.F. pressure, they are different in that halothane causes a fall in systemic blood pressure and ketamine causes a rise.

Heart rates tended to increase but not consistently, and despite C.S.F. elevations, no patient developed apnoea or convulsive movements. This was in 7 hydrocephalic patients and during insertion or revision of ventriculo-peritoneal or ventriculo-venous shunts.

Shapiro, Whyte and Harris (1972) showed that I.V. injection of thiopentone may terminate the intracranial pressure rises from ketamine and manual hyperventilation with an anaesthetic bag and face piece reduced the ketamine induced intracranial hypertension.

They report that the thiopentone action was rapid, taking less than 1 minute to reduce the intracranial pressure to pre-induction levels but report that its effect was transient and subsequent hyperventilation may be required. Arterial blood gas analysis in one patient revealed a pre-induction pCO_2 of 37.5 mm Hg and a slight decrease to 33.5 mm Hg during a peak I.C.P. of 27 mm Hg. The arterial pO_2 remained above 100 mm Hg during the entire anaesthetic course. It is postulated that the possible reasons for the increased pressure when the C.S.F. dynamics are abnormal is due to the variable loss of intracranial pressure buffering mechanisms or due to abnormal cerebro-vascular responses.

Most general anaesthetics depress cerebral metabolic rates and may thereby offer some protection to the brain during ischaemic hypoxic episodes. However, no evidence for a reduction after ketamine in cerebral metabolic rate was found in studies in dogs or in humans. They demonstrate however, ketamine can reduce C.P.P. below the critical cerebral blood flow auto-regulatory limit of 60-30 mm Hg (Langford 1969, Heilbrum, Balslev and Boysen 1972). C.P.P. reductions of this magnitude due to increased intracranial pressure have been associated with biochemical evidence of cerebral hypoxia (Zwetnow 1970), therefore intracranial hypertension caused by ketamine may potentially be more hazardous metabolically, than increased intracranial pressure due to volatile anaesthetic agents which decrease oxygen uptake. Thiopentone decreases cerebral blood flow and metabolism in man (Pierce et al 1962). Pre-treatment with thiopentone blocked both the cerebral blood flow and metabolic responses to ketamine in dogs (Dawson et al 1971). They again feel that the risk of acute repetitive episodes of intracranial hypertension associated with the induction and maintenance of ketamine

anaesthesia is high in patients with intracranial pathology. They also mention that this situation may be manifest not only in neurological patients but also in recently traumatised children with possible head injuries undergoing minor orthopaedic procedures with ketamine as an anaesthetic. List and co-workers (1972) felt that monitoring the I.C.P. through an external ventriculostomy provided the conditions necessary for the safe conduct of ketamine anaesthesia so as to control pressure by removal of C.S.F. However, they caution that expansion of the brain during ketamine may collapse the ventricles and interfere with the effective removal of C.S.F.

Crumline, Nolsen and Weiss (1975) stated that factors that are known to increase the V.F.P. include hypercapnia, hypoxia and increased arterial pressure. In their study, these variables did not change enough to explain the increases in V.F.P. The end expired CO_2 tensions remained fairly constant at levels below 35 torr, arterial blood pressure did not rise significantly, and PAO_2 did not fall below 60 torr. It is known that arterial oxygen tensions above 50 torr when the $PACO_2$ is constant do not affect cerebral blood flow (Smith and Wollman 1972). They also found that the root of administration of ketamine did not change the time to peak the ventricular fluid pressure, but they did find that those anaesthetised by I.M. administration had higher V.F.P. changes than those given I.V. In this paper they used Broperidol, Seconal and Valium in an attempt to block the ketamine induced rises in V.F.P. They failed to demonstrate a decrease in V.F.P. after ketamine with Secobarbital premedication. They used Broperidol in 5 children because ketamine had been found to augment alpha adrenergic receptor properties in dogs and these effects had been blocked by Phentolamine (Trabler, Wilson et al 1971). Broperidol has alpha adrenergic blocking

properties (Yelnosky et al 1964) and has been reported to antagonise ketamine induced tachycardia and hypertension (Becsey et al 1972). However the use of Broperidol in clinical doses did not prevent ketamine induced rises of V.F.P. Neither did Diazepam prevent these rises.

Haugaard, Hansen and Brodersen (1974) have suggested that ketamine is not a direct cerebro-vasodilator but that it might affect regional blood flow secondary to drug induced changes in regional neuronal activity. More latterly (Kaul et al 1976) reported a fall in intra-cranial pressure following ketamine. However a number of features in their paper are questionable, firstly they used ventricular manometry and not a non-displacement method, secondly the children were given Promethaxine as a premedication, and lastly the children went to sleep; the three variables which affect comparability. Not only did the pressure not rise in 7 out of 10, but it actually fell and they attempted to augment their case by quoting Dawson, Michfelder and Fay (1971) who found that ketamine preceded by Thiopentone caused a drop in C.B.F. and C.S.F. pressure!

In summary therefore gaseous or volatile anaesthetic agents are cerebral vaso-dilators and by increasing cerebral blood volume cause an increase in intracranial pressure. I.V. anaesthetic drugs, on the other hand, are cerebral vaso-constrictors and have the reverse effect, with the exception of ketamine (Lassen and Christiansen 1976).

My findings confirm that ketamine can aggravate pre-existing raised intracranial pressure to levels which are potentially dangerous. If the intracranial pressure is normal prior to the ketamine, then the ketamine may have little or no effect on it and lastly in no case did ketamine precipitate fits and the E.E.G. actually improved in one severely epileptic boy monitored throughout the procedure.

With regard to the epileptic properties to this drug, it seems based on fairly flimsy evidence. Walker (1972) reported that ketamine precipitated convulsions in a child with a previous history of fits and Bennett (1973)   confirmed that ketamine is a cerebral stimulant and could induce seizure activity in epileptics.

Some of our measurements throughout these anaesthetics have shown that the ventricular pressure was in excess of 20 mm Hg for $7\frac{1}{2}$ minutes. This is considerable in the unwell child. His ability to compensate, for example by a rise in B.P., is impaired as a result of raised intracranial pressure and may result in local or generalised hypoxia in areas of marginal cerebral blood flow. This could be one reason for blindness in the post-operative period. In the course of one anaesthetic in our series, ketamine produced no effect on ventricular pressure while ventilation and blood gases were controlled suggesting that the cerebro vascular tree remained sensitive to CO_2 during ketamine anaesthesia.

In the assessment of ventricular pressure, resting pressure, stress pressure and sleep induced pressure are recorded and it has been demonstrated that marked excursions can occur in sleep, and in one case in this series, the resting pre-ketamine pressure was normal and rhythmical, sleep-type oscillations occurred, which prompts the question of the possible use of this drug in identifying the labile pressure problem. Although ketamine induces changes in ventricular pressure which are fairly easily reversed, I feel that prolonged anaesthesia using this drug is still contra-indicated when there is raised intracranial pressure which existed pre-operatively unless facilities exist for pressure monitoring throughout the anaesthetic and for the relief of the pressure. This adds some weight to the

previous literature on the subject, which suggests a degree of caution.

Halothane

As mentioned above, it has repeatedly been shown to be a cerebro-vaso-dilator with an increase in cerebral blood flow of 14% and a decrease of 9% in the cerebral metabolic rate of oxygen (Wollman 1964). Smith (1973) showed a good correlation of $CMRO_2$ and cerebral arteriovenous oxygen difference, with the depth of halothane anaesthesia. The effect of halothane may be minimised or even abolished by prior induction of hypocapnia for 10 minutes, but not in all cases (Adams et al 1972).

It is now generally accepted that all volatile anaesthetic agents like halothane, i.e. trichlorethylene and methoxyflurane are capable of increasing C.S.F. pressure due to cerebro vaso-dilation and an increase in cerebral blood flow (Macdowell, Barker and Jennett 1966, Fitch, Barker and Macdowell 1969, Jennett, Barker and Fitch 1969).

Methoxyflurane

This resembles halothane in its cerebral metabolic effects which are not completely counteracted by moderate hyperventilation (Fitch et al 1969).

Nitrous Oxide

When administered in a concentration of 70% it causes a decrease in $CMRO_2$ of 25% without significantly affecting the cerebral blood flow during normocapnia (Wollman et al 1965). Besides the unaffected cerebral blood flow (Smith et al 1970) demonstrated that cerebral auto-regulation is well preserved during nitrous oxide anaesthesia in man, i.e. with an unchanged cerebral blood flow and a reduced $CMRO_2$, there is a relative increase in flow and Henriksen and Jorgensen (1973) found I.C.P.

increases associated with 66% nitrous oxide administration in patients with intracranial disorders, and concluded that it was a significant cerebro vaso-dilator.

Barbiturates

Pierce et al (1962) showed thiopentone to be a pronounced and dose-dependent cerebral vaso-constrictor in man. In anaesthesia it causes a reduction of about 50% of $CMRO_2$ and cerebral blood flow. Thiopentone affords some cerebral protection in hypoxia because of the diminished energy requirements associated with the reduced cerebral function.

Narcotic Analgesics and Neuroleptic Drugs

Morphine causes progressive and parallel decreases of $CMRO_2$ and cerebral blood flow of about 15% in dogs with normocapnia. Morphine per se is a cerebral vaso-constrictor and its effects are completely abolished by hypercapnic vaso-dilatation. Pethidine is similar. Fentanyl has been shown in normal man not to influence either C.B.F. or $CMRO_2$ significantly with normocapnia (Sari, Okuda and Takeshita 1972).

Broperidol is a more potent and long acting cerebral vaso-constrictor which does not influence $CMRO_2$. Often Fentanyl and Broperidol are administered for the purpose of neuroleptanalgesia and a significant decrease in intracranial pressure occurs in patients with normal C.S.F. pathways with neuroleptanaesthesia (Fitch et al 1969). Diazepam has recently been shown to cause a parallel depression of both C.B.F. and $CMRO_2$ in comatose patients with diffuse brain damage (Cotev and Shalit 1975).

Curare

It was previously claimed that Curare had no effect on the brain.
However Tarkkanen, Laitinen and Johansen (1974) found a significant
increase in intracranial pressure associated with Curare during
maintained normocapnia. They suggested that the pressure increase
was due to increased blood flow associated with histamine release. If
so, pancuronium might be preferable as a relaxant drug.

Comments Relating to Neuro-Anaesthesia

Cerebral vaso-dilators are therefore generally contraindicated as in
hypercapnia. The arousal effect associated with pain and anxiety during
an incomplete anaesthetic, might induce cerebral vaso-dilatation and
hyperaemia, and because of the loss of auto-regulation an increased
arterial pressure might be deleterious.

In the damaged brain, some degree of protection might be obtained by
deep barbiturate anaesthesia, so anaesthetic agents chosen should
reduce cerebral metabolism and preferably have an associated vaso-
constrictor effect. The anaesthetic technique should include passive
hyperventilation and avoidance of hypotension.

Thiopentone drip should be considered as a supplement to nitrous oxide
and oxygen. This was used in 166 patients who were curarised and
artificially ventilated and intracranial tension during the operations
was low (Hunter 1972).

With regard to endotracheal intubation, Misfeldt, Jorgensen and Rishog
(1974) studied the effects of endotracheal tubing and found a mean
increase in pressure of 16.6 mm Hg. They felt that critically small
C.P.P. values were prevented by an increase in arterial pressure and
concluded that the effect was harmless.

CHAPTER 10

OBSERVATIONS ON THE EFFECT OF SLEEP ON INTRACRANIAL PRESSURE IN CHILDREN

CHAPTER 10

OBSERVATIONS ON THE EFFECT OF SLEEP ON I.C.P. IN CHILDREN

INTRODUCTION

It has been known for some time that the symptoms of R.I.C.P. are
worse on waking and although most authors on the subject of V.P.M.
now recognise the value of a sleep record, there have been relatively
few reports in the medical literature concerning the changes in I.C.P.
during sleep (Cooper & Hulim 1966, Hulim & Cooper 1968, Di Rocco
et al 1975, 1976, and Rossi et al 1975).

SLEEP STAGES

A number of descriptions of the different phases of sleep have been
described, (Loomis et al 1937, Gibbs & Gibbs 1950, Dement & Kleitman
1957). The latter described four stages according to E.E.G. changes.

Stage 1   absolute lack of spindle activity, low voltage fast
pattern on the E.E.G.

Stage 2   presence of 'spindles', low voltage background, some
slower activity 3-6 Hz, spontaneous 'K' complexes
(identified by their wave form and widely spaced
occurrences).

Stage 3   high voltage slow waves with some spindles

Stage 4   waves greater than 100 μV in the delta range.

R.E.M. sleep shows mostly Stage 1 E.E.G. activity but rarely some of
Stage 2.

From a behavioural or consciousness point of view one can stage sleep
from the awake state to the deepest sleep, viz

     1   awake

2   Stage 1

3   Stage 2

4   Stage 3

5   Stage 4

6   R.E.M. or 'Paradoxical Sleep'

For practical purposes sleep has been simply classified in this
thesis according to the classification of Aserinsky & Kleitman 1955,
as:

1   R.E.M. or 'active' sleep

2   N.R.E.M. or 'quiet' sleep

The reason for this is that staging can be very difficult if not
impossible in young infants with rapid maturation of the E.E.G. e.g.
sleep spindles do not occur until 10 weeks of age.

R.E.M. OR PARADOXICAL SLEEP

or 'active' or 'primitive' sleep arises from the Pons (the arousal
centre of the reticular formation) and there is an increase in cortical
activity but a deepening of consciousness, i,e. during this stage one
is very difficult to arouse. There is a decrease in muscular tone,
dreams, erections and associated with the increased cortical activity,
bursts of rapid eye movements start at the same time. There may be
up to 50 conjugate eye movements from side to side in one burst.
In this phase of sleep the 'arousal centre' as it were goes to sleep
and therefore does not suppress but it facilitates an increased cortical
activity. The E.E.G. is then similar to the awake state, i.e. low
voltage fast activity. The awake state can be differentiated from
this stage of sleep by examining hippocampal leads. The increased
activity does not arise from the visual cortex and the pupils are
maximally meiotic.

R.E.M. sleep in the adult only follows the deeper stages of sleep. However, Jouvet 1964 found that in young kittens, paradoxical sleep can occur immediately on falling asleep and this is consistent with my observations in young children (vide infra).

If one is deprived of R.E.M. sleep it is made up the next nights. However, with severe R.E.M. deprivation it is still only possible when making up the loss to have a maximum of 60% of sleep as R.E.M. and it is still cyclical even in recovery.

In adults about 20% of all sleep normally is R.E.M. whereas 50-60% of sleep in pre-term infants and full term newborns is R.E.M. (Goldie 1965).

During R.E.M. breathing and heart rate are irregular and accelerated and there is increased protein synthesis within the brain as evidenced by an increased C.B.F. (Kanzow et al 1962) and an increase in brain temperature. This has led to the suggestion that R.E.M. sleep is a time of brain growth and repair. If indeed it is a time of repair then the exaggerated pressure responses which are occurring (vide infra) and which are a reflection of increased C.B.F. are an attempt to compensate or repair hypoxic-ischaemic damage resulting from R.I.C.P. Furthermore, less R.E.M. is evident in older mentally retarded patients.

Also during R.E.M. sleep there is an increased urine osmolality, probably a result of enhanced A.D.H. production and excretion of adrenalin metabolites (Mandell & Mandell 1969).

In animal studies, the locus coeruleus with nor-epinephrine as a transmitter induces R.E.M. sleep.

Hypnotics lower the percentage of paradoxical sleep in a night and it may therefore be that by decreasing the time intervals when the

I.C.P. is raised that hypnotics help to control R.I.C.P. After cessation of hypnosis however there is still a need to make up lost R.E.M. sleep.

NON-R.E.M. SLEEP

or 'quiet' or 'slow' or 'forebrain' sleep is characterised by cortical spindles in adults which are 15-18 Hz and on which are superimposed high voltage slower (theta) waves (trace alternant E.E.G.). In this stage the 'arousal' centre is less depressed and so inhibits cortical activity and muscle tone increases. There is an increase in muscle tone, regular respirations and heart rate but no body or eye movements. Since non-R.E.M. is proportionately less in newborns and pre-term infants, it suggests that 'slow' sleep has to be learned. In the last few weeks before term, babies can sustain slow sleep for 10-15 minute periods. This pattern is lost if there is brain damage.

During non-R.E.M. there is an increased Human Growth Hormone Production (Honda et al 1969, Eastman & Lazarus 1973) and the latter found that this did not occur with hypopituitary dwarfism. Often with mentally retarded brain damaged children their odd dysplastic fascies and growth pattern, although evident, is not characteristic of any identifiable syndrome and it may be that interferences with normal growth hormone secretion because of disturbed non-R.E.M. sleep is at least partially responsible although other mechanisms associated with delayed maturation e.g. lag in myelination etc. may also play a part.

In animal studies activation of serotonin at the raphe nuclei level induces non-R.E.M. sleep.

ORIGINAL OBSERVATIONS

TYPICAL PRESSURE VARIATIONS THAT OCCUR DURING SLEEP

From a resting pressure level 'A' in the diagram over the child becomes drowsy and then lightly asleep. During this there is an increase in the mean level of I.C.P. with increased superimposed pressure fluctuations. This rise in pressure reaches a new level 'B' and then decreases in a symmetrical fashion.

During the pressure elevation there is an accompanying variable number of easily recognisable clinical features.

1 Rapid eye movements, continuous nystagmus, eye blinking or eyes only half closed.

2 Snoring and grunting

3 Grinning and smiling

4 Chewing or sucking

5 Facial flushing

6 Sighing

7 Apnoea

8 Moaning and becoming unsettled and eventually waking if huge pressure excursions occur

9 Hypnagogic myoclonic movements such as facial twitching, finger twitching which sometimes coincides with the peaks of the pressure wave, twitching of eyelids, tongue movements, lip smacking, scratching the nose and face, stirring and general body movements.

10 Deep noisy respiration

After the original elevation the pressure falls to a new level 'C' which is often less than the mean awake resting level. This non-R.E.M. phase is not accompanied by any identifiable clinical variations.

TYPICAL SLEEP CHANGES
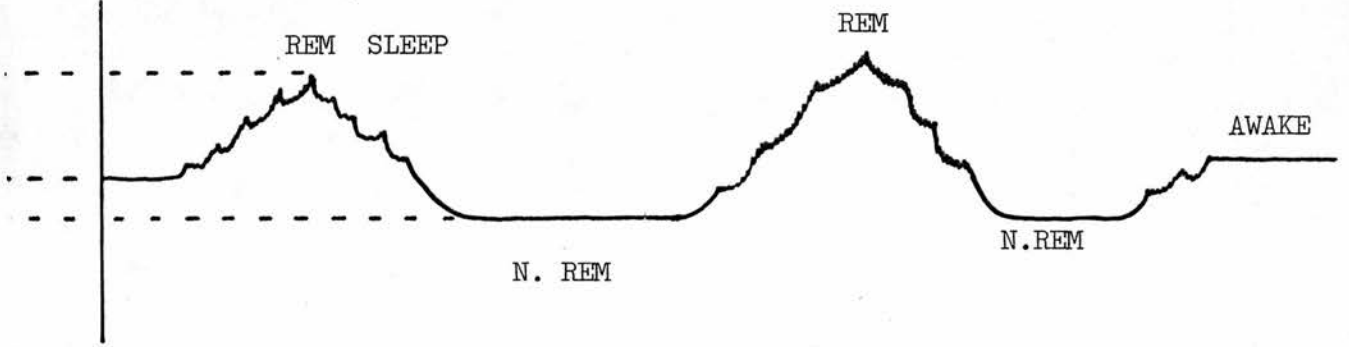
GRAPHIC ILLUSTRATION OF FREQUENCY AND
AMPLITUDE OF 'LIGHT SLEEP' PRESSURE CHANGES
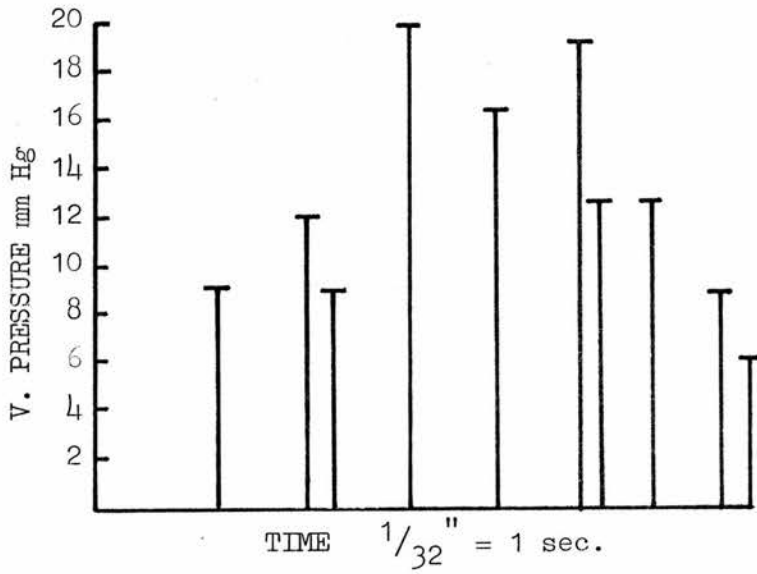
Later in sleep, pressure elevations occur similar to the first elevation. They may not necessarily be of the same amplitude or duration but are again accompanied by the clinical features of 'active' sleep listed above.

This pattern may be repeated a number of times throughout the night until just prior to waking when the pressure rises from the deep level 'C' with characteristic fluctuations to become stable again in the awake state at level 'A'. These changes are also detectable by pressure monitoring at the brain surface (Case Number 99).

When the Mean I.C.P. is in the Normal Range the pressure variations that occur with sleep (i.e. rhythmic increase in mean level and amplitude of pulse waves) do not extend outwith the normal range of I.C.P. e.g. Case Number 4, 65.

When there is a Raised Mean Pressure Level the amplitude of the pressure deflections with sleep is almost always less than a maximal stress response, except in Case Number 81 when the sleep pressure equalled the stress response (14 mm Hg).

I suspect that ketamine exaggerates the sleep responses in cases of R.I.C.P. e.g. in Case Number 72 the early sleep peaks occurred without ketamine to a level of 32 mm Hg. After ketamine was administered the maximum level now was 57 mm Hg and the time interval to reach this peak was shortened from 11 to 6 minutes.

EXAGGERATED PRESSURE RESPONSES TO SLEEP IN SOME CASES OF R.I.C.P.

Although there is always an increase in pressure to a moderate degree when there is R.I.C.P., on occasions these pressure responses may be grossly exaggerated. These are not typical 'plateau waves' e.g. Case Number 29, 9. In the case number 29 huge excursions of

ventricular pressure occur associated with facial flushing and rapid nystagmus.

A further case, number 32, shows similar ventricular pressure increases to levels in excess of 60 mm Hg and at the point where the pressure wave begins to decline, a period of apnoea ensues. This occurred constantly. For comparison rhythmical 'active' sleep changes in ventricular pressure are seen to occur in the same child at a much lower amplitude after temporary relief of his pressure by tapping the C.S.F. On this tracing is seen the 'sighing' effects on the respiratory wave, on the downward slope of the pressure variation. It may be in this child that apnoea is an exaggerated sighing or self-induced valsalva response.

Case Number 45 illustrates a drop in the level of pressure when accompanied by an absence of the respiratory component of the ventricular pressure wave (indicated by the arrows), thereafter the pressure level is minimally below the level which preceded it. The absence of this respiratory segment was not detected clinically. At this same time, in other patients, E.C.G. irregularities are seen to occur (Case Number 62). The E.C.G. irregularity is associated with a prolonged drop or 'step-down' in the ventricular pressure level and corresponds to a 'switch' to a different (non-R.E.M.) sleep phase. A similar pattern occurs in this child a few seconds later.

Ordinarily the timing of the peak of the pressure fluctuations consistently follows the T wave of the E.C.G. tracing, i.e. in diastole (Bering 1955) and this is illustrated in Case Number 62, thus maximising venous outflow from the head. In this same case there is a reduction in heart rate during sustained pressure increases

during sleep (prior to waking) on a 24 hour recording. It can also
be seen that the width of the band of pulses is greater, the higher
is the mean level of ventricular pressure, i.e. 20-35 mm Hg C.R.A.
at the highest ventricular pressure level.

SLEEP IN THE PRE-TERM

A child born at 36 weeks gestation at a chronological age of 2 weeks
showed persistent 'active' sleep pressure changes and some difficulty
phasing into 'quiet' sleep (Case Number 26). One might expect this
because of the relative excess of R.E.M. sleep at this age. However
this case was complicated in that the monitoring was performed after
a C.S.F. infection, which, as will be discussed below, also prolongs
'active' sleep.

SLEEP IN THE FULL TERM NEONATE

In 1891-2 Czerny observed irregular pulsations of the fontanelle
in sleeping infants, these pulsations occurred in a cyclical
fashion alternating with phases of completely regular fontanelle
pulsations. From pressure monitorings done in the neonatal period,
it is my impression that although rhythmical oscillations occur in
'active' sleep the ventricular pressure level does not usually
become more than marginally elevated, e.g. Case Number 36 and 80.

Case Numbers 36 and 37 were sleep monitorings performed on the same
child at age one and six months respectively, and the differences
in the pressure responses to sleep are obvious. In the latter case,
arrows on the figures depict sections of the ventricular pressure
recording when the pressure is more or less horizontal and this
phenomenon is not uncommon and are occurring at a similar time to
the 'sighs' in Case Number 32. This could point to a time, as
mentioned before, when central sleep mechanisms become reset for

either continuing pressure increases in the same R.E.M. phase of sleep or the start of declining pressures into 'quiet' sleep.

THE NEONATE WITH GROSS HYDROCEPHALUS

The already damped ventricular pressure changes in sleep compared to the older child are observed to be even more diminished in the presence of severe ventricular dilatation, possibly due to damping of impulses through a large volume of water (Case Number 60).

IDENTIFICATION OF STAGES OF SLEEP ON VENTRICULAR PRESSURE RECORDINGS

Case Number 62 in early sleep demonstrated typical clinical associations. The arrows indicated sleep spindles which can occur in R.E.M. or Stage 2 sleep. The sleep spindles did not appear constant in their relation to the peak of the waves. Likewise a polygraphic record (Case Number 86) confirmed eye movements, sleep spindles and the typical pressure oscillations in 'active' sleep.

A summated E.E.G. (cerebral function monitor) in Case Number 85 recorded a change in the base line at the time of these pressure changes in sleep. Again sleep spindles lasting approximately 2 sec, accompany the pressure fluctuations in Case Number 91 and occur at any point on the ventricular pressure wave, whereas in 'quiet' sleep when the amplitude of the E.E.G. increased there were no clinically detectable movements, sleep spindles, and the level of ventricular pressure was at its lowest recorded value.

PERIODICITY OF R.E.M./NON-R.E.M. CYCLES

In Case Number 55, the first elevation of pressure lasts $7\frac{2}{3}$ minutes, a sigh heralds quieter sleep which also lasts $7\frac{2}{3}$ minutes followed by a further section of light sleep lasting $7\frac{2}{3}$ minutes. This very regular pattern is not invariable, see also Case Number 72.

A further Case Number 84 with 'active' sleep peaks to 37.5 mm Hg
(mean of 24 mm Hg) had a pressure in excess of 20 mm Hg for 6
minutes. The first 'active' phase lasted 14 minutes, followed by a
'quiet' sleep phase of $26\frac{2}{3}$ minutes, then 'active' sleep for $16\frac{2}{3}$
minutes and 'quiet' sleep again for $25\frac{2}{3}$ minutes. Approximately
one third of sleep in this child is 'active' and two thirds 'quiet'
sleep. The awakening phase lasts 19 minutes with the usual pressure
sequences (indicated by the arrows) when there is no respiratory
component. It is tempting to suggest that the increase in pressure
is from an increase in C.B.F. at this time, needed to initiate
wakefulness and active thought; similar to the effects of CO_2 used
transiently at the conclusion of muscle relaxant anaesthesia to
initiate breathing.

Quiet sleep commenced 10 minutes after the onset of sleep in Case
Number 45.

FREQUENCY OF PRESSURE FLUCTUATIONS IN SLEEP

Manually measuring the intervals between the pressure fluctuations
in sleep was not always possible (because of the different recorder
speeds, apparatus etc.). However the mean interval between the
peaks during 'active' sleep in 5 cases was plotted against a number
of parameters.

1. The highest peak value in sleep

2. The resting awake level of ventricular pressure

3. Maximum stress ventricular pressure

4. Age

5. Degree of ventricular dilatation.

No consistent pattern or correlation emerged, however with a computer
analyser or micro-processor attached to the existing apparatus for

monitoring I.C.P., a more detailed analysis would be possible and one could detect more than random frequencies by Poisson and other formulae.

The mean intervals between 'active' sleep pressure peaks for these 5 children were:

1. 1 per 13 sec i.e. 4.62/min.
2. 1 per 21 sec i.e. 2.86/min.
3. 1 per 29 sec i.e. 2.07/min.
4. 1 per 41 sec i.e. 1.46/min.
5. 1 per 45 sec i.e. 1.33/min.

The frequency of the pressure deflections varied therefore in 'active' sleep. Case Number 10 the pressure waves occurred at 12 sec. intervals i.e. 5/min (close to the Traube-Hering-Meyer waves of 6/min). It is interesting that snoring began at the first high frequency value in this patient.

Frequencies of the wave forms were also analysed in Case Number 69, 70 and 81. The frequencies appear to be very consistent in the same patient but varied from patient to patient. Case Number 79, an 18 year old girl, showed consistent waves every 18 sec, i.e. 3.3/min over long periods of active sleep. These are neither the classical 'B' or 'C' waves nor do they correspond to any component of the heart beat or respiration.

A child with elevated I.C.P. due to cerebral tumour also showed amazingly regular recurring pressure waves in sleep every 60 sec and if one was omitted then the amplitude of the succeeding pressure elevation at the 2 min interval was larger. In a further patient, Case Number 84, the waves of 'active' sleep occurred at 40 and 60 second intervals.

Generally therefore the frequency of the pressure fluctuations in 'active' R.E.M. sleep in this series varied from 1-5/min and was quite consistent in each individual patient.

A graphic illustration is seen on page 139 (Case Number 70) where the amplitude and frequency of the pressure fluctuations of 'active' sleep are seen extending outwith the normal range of intracranial pressure.

CHANGES IN C.P.P. WITH 'ACTIVE' SLEEP

No continuous systemic arterial pressure recordings were possible throughout this series, however with a mercury sphygmomanometer, B.P's were recorded and in Case Number 40 the C.P.P. at rest was 66 mm Hg but in early sleep dropped to 60 mm Hg transiently. A similar drop in C.P.P. from 80 mm Hg (awake) to 70 mm Hg ('active' sleep) was seen in Case Number 89.

Since the ventricular pressure changes are due to an increased C.B.F. then the lower C.P.P. values must reflect a drop in S.A.P. during sleep. It is noteworthy that Cooper & Huline 1966 found no close correlation between S.A.P. and I.C.P. during 'plateau' waves in sleep.

EFFECT OF SEDATION ON SLEEP INDUCED VENTRICULAR PRESSURE CHANGES

There has been little opportunity to observe such changes as generally no sedation has been offered prior to V.P.M. In Case Number 25 light sleep was observed in a child of $7\frac{1}{2}$ months, $7\frac{2}{3}$ minutes after administering chloral and the pressure elevation lasted $8\frac{1}{3}$ minutes.

A further child (Case Number 69) given chloral moved into 'ative' sleep quickly and remained in this phase for 5-8 minutes. After the next 15 minutes in quiet sleep he enters another active phase

which lasts 25 minutes.

15 mgm Phenobarbitone was given to a restless child during the course of V.P.M. and 'quiet' sleep changes were recognisable on the tracing 30 minutes later but no earlier 'active' sleep. R.E.M. sleep appeared $2\frac{3}{4}$ hours after giving the Phenobarbitone with the characteristic clinical and ventricular pressure changes observed. As mentioned previously, hypnotics lower the percentage of paradoxical sleep in a night and it appears in these children that chloral and Phenobarbitone therapeutically shortened or abolished the invariable early sleep pressure elevations. Since one needs to make up lost R.E.M. sleep it is necessary to proceed cautiously when small amounts of sedative are used, because later in the night longer periods of paradoxical sleep might appear (Case Number 69 - R.E.M. phase lasting 25 minutes) which compromise intracranial dynamics.

SLEEP CHANGES IN POST-INFECTIVE STATES

Characteristically children with post-meningitic hydrocephalus or hydrocephalus complicated by recent ventriculitis, show a very disordered 'saw tooth pattern' on their ventricular pressure recordings during 'active' sleep. This 'active' or R.E.M. phase appears to be quite protracted and any environmental noise tends to wake them easily, something which should not happen in paradoxical sleep when normally one is more deeply unconscious. These children tend to be 'light sleepers' according to their parents, Case Number 57.

PLATEAU WAVES IN SLEEP

In this series plateau waves were seen to begin during the course of sleep (Case Number 41) or in the awake state (Case Number 41) or in a semi-conscious state (Case Number 89).

It is interesting that the plateau waves may be very silent and

unaccompanied by any change in vital signs, so much so that nursing observers have written on the recording at the time such comments as 'settled' etc. Sometimes however in sleep, these plateaus are associated with the child moaning restlessly.

MONOAMINES DURING PRESSURE CHANGES IN SLEEP

This has been dealt with in another section of this book (Chapter 8) but briefly no significant changes occurred in the main monoamine metabolites during the pressure changes associated with 'active' or 'quiet' sleep or immediately on waking.

HYDRANENCEPHALY

Case Number 68 was a child who had a recorded I.C.P. within the normal limits but during 'active' sleep no demonstrable pressure fluctuations occurred although the appropriate cardio-respiratory pulses were seen. Presumably this child has a choroid plexus which is the origin of these C.R.A.'s and experience indicates that often these children have a choroid plexus lying exposed on the surface of a remnant of mal-developed brain. At advanced chart speeds tall T waves and short periods of asystole are noted which may correspond to changes in sleep phases. It is not surprising that without an intact cerebro-vascular tree for CO_2 mediated changes to act upon that the usually observed pressure fluctuations in sleep are absent.

CO_2 estimations were slightly lower than the normal range on two C.S.F. samplings, however this again may be a dilutional effect. Left sided E.E.G. changes occurred during sleep in this child so some brain tissue must be producing these potentials, and since the child's pressure was normal, I.C.P. could not have been influencing the E.E.G. record. It is known that children with space occupying lesions often have their E.E.G. findings complicated by R.I.C.P. e.g. posterior

rhythmic delta activity seen with infratentorial tumours is said to be due to R.I.C.P., from distension of the third ventricle and distortion of the adjacent thalamic nuclei (Martinus et al 1968).

The E.E.G. changes in children with R.I.C.P. usually are not specific or recognisable as such (Townsend 1978).

DISCUSSION

It is known that in young children during R.E.M. sleep the respiratory pattern is irregular and some bursts of apnoea occur and that often a deep breath tends to initiate this phase of sleep (Simpson 1979). It has previously been reported that large inter-mittent increases in pressure occur in R.E.M. and Stage 2 sleep (Cooper & Huline 1966) and this is confirmed in this series. However it is mostly not preceded by a non-R.E.M. phase in children and the rhythmical pressure fluctuations do not extend outwith the normal range when the mean I.C.P. is normal. When there is existing R.I.C.P. the R.E.M. sleep elevations in pressure occur as varying wave frequencies from 1 to 5/minute.

During the change to non-R.E.M. sleep sighing (or a spontaneous valsalva), E.C.G. and respiratory abnormalities are common. During non-R.E.M. sleep the respiration becomes very regular (there are however still bursts of apnoea in recent respiratory studies above Simpson 1979) and the E.E.G. is of much higher amplitude.

Since CO_2 enters the brain quite rapidly and the C.S.F. is slightly more acidic than arterial blood (Kraaus et al 1972) and since it is the most important known effector of changes in C.B.F. responsible for initiating rises in pressure, CO_2 would seem to be responsible for the sleep-pressure changes by increasing the I.C.V. However, CO_2 is remarkably constant in health and when the I.C.P. is normal,

the pressure variations with sleep, although rhythmical and different from the awake state, are not elevated beyond the normal range. Therefore these variations are most likely due to alterations in the vasomotor tone of the cerebral vasculature in the normal child.

It seems unlikely that in health the ventricular pressure changes are due to altered CO_2 responsiveness in the child, as this is known only in pathological states such as cyanotic congenital heart disease, living at high altitudes, hypothalamic conditions etc. resulting in the respiratory centre being reset.

It is my opinion that ventricular pressure changes in early sleep in the normal child are controlled by inate biological rhythms by means of an integrated switch system for initiating light, deep sleep or wakefulness. The most likely site for this switch is the formatio-reticularis with its known control of consciousness and sleep-waking rhythms. Any interference with the intricate inter-connections from here to the cortex, thalamus and down to the lower brain stem following injury or disease may produce changes in consciousness, motor, sensory or automatic imbalance. Hydrocephalic children or children who have in the past experienced R.I.C.P. are susceptible to pressure effects in this area of the reticular formation and by altered CO_2 responsiveness or some other mechanism cause an alteration in the cerebral vascular tone producing an exaggerated response to sleep. The apnoea and bradycardia which follows these large pressure changes in sleep (e.g. Case Number 29) may be a result of increased I.C.P. directly influencing the lower brain stem or secondary to interference with the medullary afferents to the reticular formation.

CHAPTER 11

DISCUSSION

PATHOPHYSIOLOGY

    (a)   Cerebrospinal Fluid

    (b)   Blood

    (c)   Brain

COMPLICATIONS

FUTURE CONSIDERATIONS

SUMMARY

CHAPTER 11

DISCUSSION

PATHOPHYSIOLOGY

In a child with a fused cranio-vertebral axis, the modified Monro-Kellie Doctrine holds true, i.e. an increase in the volume of one part within the cranium (80% brain, 10% blood, 10% C.S.F.) causes a decrease in volume of another, tending to keep the I.C.P. normal, by means of the following buffers:-

(i) C.S.F. displacement to the spinal axis (accounts for 70% of the compensation, e.g. as occurs on coughing).

(ii) decreasing the C.B.V.

and in children particularly below the age of 18 months, there is a further compensatory mechanism:-

(iii) an increase in the OFC (remember sutures can splay up to about 12 years of age).

For these buffers to be effective the rise in I.C.P. must occur slowly. The increase in volume of one component may give rise to no rise in I.C.P. or only a slight rise (because of the above), when these compensatory mechanisms are exhausted however, R.I.C.P. occurs.

The amount of buffering available within the cranium is termed compliance $\frac{\triangle V}{\triangle P}$, (the inverse of which, $\frac{\triangle P}{\triangle V}$ is elastance) and follows a hyperbolic curve.
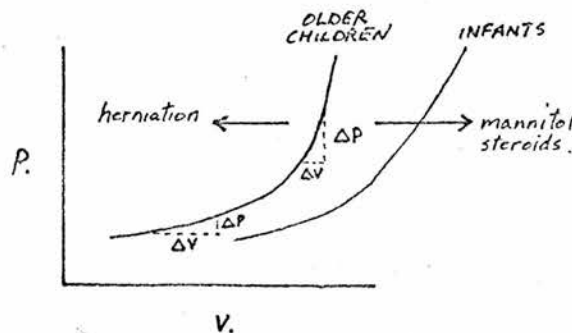
At a point low on the curve, a larger volume of C.S.F. is accommo-
dated with a proportionately smaller pressure increase than occurs
at a high point on the curve. Mannitol and steroids reduce the
I.C.P. and shift the curve to the right, herniation shifts it to the
left. Hyperventilation only decreases the I.C.P. and does not shift
the curve to the right, because when hyperventilation is stopped,
the pressure returns to its previous level.

This dynamic state exists in situations where there is a fused cranio-
vertebral axis, but in infants who have the ability to expand their
head, the curve should probably be permanently shifted to the right
and move slowly to the left with growth over the first 18 months of
life. This accounts for the less frequent observation of plateau
waves in infants, where slowly developing R.I.C.P. is accommodated
by increasing the OFC. With a rapid increase in I.C.P. in infants
e.g. acute toxic encephalopathy, head injury etc., the buffering
mechanisms do not come into operation early enough and dramatic
signs of increased pressure with gross opisthotonous, facial swelling
etc. are evident.

With a progressive but slow build up in the I.C.P. over a period of
time, the pressure increases are less well compensated for as they
are with sudden increase in I.C.P. and an estimate of the residual
compliance left within the cranium can be obtained by the addition
of saline or the subtraction of C.S.F. from the system by methods
described by Marmarou & Shulman (1976).

The importance of this clinically is that R.I.C.P. may occur
without alteration in the patient's state but persistent small
incremental increases in blood volume etc. are not then readily
compensated for and sudden large pressure waves (plateaus) occur.
Plateau waves are always pathological and occur as a result of

diminished or absent compliance. As mentioned before they are
infrequent in infantile hydrocephalus and the only children who have
displayed them in this series were those older children with an
absence of a spinal compartment as a buffer, e.g. a child with a
theco-peritoneal shunt which subsequently blocks, or his hydro-
cephalic state becomes non-communicating. With this type of shunt
one can imagine that the spinal subarachnoid space is not encouraged
throughout the early years to distend and accommodate or absorb C.S.F.,
thus subsequent non-communication or blockage leaves the child with
virtually no protective mechanisms and low compliance so that
brain swelling and plateaus occur.

Langfitt (1968) considered the direct cause of plateau waves to be
cerebral congestion resulting in an increase in C.B.V. I think that
a further explanation is more likely, namely a focal (or possible
global) increase in brain swelling leading to acute non-communication.
Brain swelling certainly accompanies a rapid increase in I.C.P., even
if the pathogenesis of the R.I.C.P. is hydrocephalus. Plateau waves
are sometimes self limiting, exactly how this comes about is not
clear.

(a) <u>Cerebrospinal Fluid</u>

C.S.F. production $\rightleftharpoons$ C.S.F. absorption

C.S.F. production at a rate of 0.3-0.5 ml/min arises 70% from the
choroid plexus and 30% extrachoroidal, i.e. brain lymph, and is
normally in equilibrium with C.S.F. absorption and this dynamic
relationship is proportional to the level of C.S.F. pressure
(Cutler 1968).

C.S.F. absorption is also possible two ways:

    (i) at the junction of the cortical subarachnoid space and
    saggital sinus by means of a one way valve which opens at

5 mm Hg C.S.F. pressure (i.e. pressure dependent absorption)

and   (ii) small amounts of C.S.F. are probably absorbed via the

spinal nerve roots.

C.S.F. absorption can be tested by the constant infusion manometric

test (saline injected at the lumbar level according to the suggestion

of Latyman & Hussey 1970 and how well the pressure is accommodated,

less than or greater than 22 mm Hg is recorded).

Hydrocephalus is due to an increased production (e.g. choroid

papilloma) blocked C.S.F. pathways (e.g. aqueduct stenosis) or

decreased absorption (e.g. post subarachnoid haemorrhage) and

whatever mechanism is producing the hydrocephalus the ventricles

dilate according to the formula:

$$P = \frac{2T}{r}$$

where T is tension in the expanding walls

P is pressure in the ventricles

r is radius

Initially therefore a high pressure is needed to dilate the

ventricles, but as dilatation commences then relatively less pressure

is needed to continue the expansion, active hydrocephalus.

Eventually the pressure may drop to normal levels and the ventricles

stop enlarging, the state of arrested hydrocephalus, when there is

a stable ventricular size, normal pressures and a developmental state

commensurate with normal growth.

Clinically therefore, in the ideal management of cases of infantile

hydrocephalus, one should allow ventricular dilatation to occur:

(a) hoping for spontaneous arrest and shunt avoidance,

(b) to promote alternative C.S.F. pathways,

(c) until any further degree of dilatation would result in unfavourable neurological or mental outcome. In our unit, this means not allowing the cortical mantle to be reduced below 15-20 mms.

For this plan to work properly, a daily non-invasive bedside assessment of ventricular size should be done with either a rapid C.T. scanner or two dimensional echoencephalography and the ventricles allowed to expand. If they stop expanding at a cortical mantle of say 25 mm, well and good, but if not when a pallium of 15 mms is reached, V.P.M. with a complete sleep-awake cycle, is undertaken. Earlier invasion to measure pressure may mean that the procedure needs repeating later (because the child has raised pressure with a good size cortical mantle or his development of hydrocephalus is proceeding in a step-wise fashion).

When V.P.M. is undertaken and the pressure is found to be raised in the awake state, then shunting is necessary, where normal pressures are found in the awake and sleep recordings, no shunt is inserted but close follow-up is still warranted. In situations with equivocal pressure results, i.e. normal awake but elevated sleep pressures, I think a conservative approach is justified, but only if very close follow-up with serial scans, neurological and developmental assessments are insisted upon and a further period of V.P.M. done at the slightest indication. Lorber (1978) has reported children and young adults with extremely thin cortic es who were of normal intelligence, however these represent a very small isolated group of individuals that are certainly not generally representative of patients with very thin cortical mantles. Intelligence falls off in an exponential fashion as the cortex decreases below 15 mms.

The terminology of shunt dependent and shunt independent hydrocephalus used in the literature is confusing and best avoided. The former merely refers to treated active hydrocephalus and the latter arrested hydrocephalus.

The concept of normotensive hydrocephalus (Hakim & De Davila 1964, Adams et al 1965) is different: the original description of this, in geriatric patients with ventricular dilatation and dementia and normal pressures is understandable pathologically. Arteriosclerotic vessels result in global or periventricular cerebral atrophy as a result of diminished C.B.F. causing a passive expansion of the ventricles and hence normal pressures. In my experience, normotensive hydrocephalus is not an entity in childhood and reports suggesting that it is (Hammock et al 1976, Milhorat & Hammock 1972, Di Rocca et al 1975) have not demonstrated normal pressures during sleep. Hammock et al (1976) monitored 3 patients correctly and in 2 of these, there was substantial elevation of the pressure during sleep to 550 mm saline and 450 mm saline. Clearly then, this is not normotensive and we should be reserving the term for cases with normal or low pressure at all times. Most cases thought to have normotensive hydrocephalus with slow ventricular dilatation, are, in fact, intermittent active hydrocephalus, i.e. the elevated nocturnal pressures are slowly dilating the ventricles a little each night. That this situation is not uncommon is seen by the proportion of hydrocephalic children in this series with equivocal pressure results (21.8%).

The above situation should not be confused with the common ventricular dilatation which follows cortical atrophy or an expanded cortical subarachnoid space seen in a variety of paediatric conditions, e.g. following subdural collections. This also has been inappropriately

termed ex vacuo hydrocephalus.

(b) Blood

Little is known about the pathophysiology of C.B.F. in normal
children and even less in brain damaged children. An increase in
one compartment within the cranium results initially in a decrease
in C.B.V. (this is the buffering mechanism which comes about by
collapsing cerebral veins, so increasing venous resistance,
decreasing the C.P.P. and then a fall in arterial resistance occurs
to maintain a normal C.B.F.). When this buffer is exhausted, further
rises in I.C.P. result in further arteriolar dilatation and an
increase in intracranial volume with R.I.C.P. which causes further
arteriolar dilatation etc. until I.C.P. is equal to S.A.P. This
is the 'carotid stop' or 'false block' (Langfitt & Kassell 1966,
Balslev & Jorgensen 1972) when angiographic material injected into
the internal carotid artery fails to enter its intracranial portion,
i.e. there is no perfusion pressure to drive it across the vascular
bed. Pressure in the cerebral veins just prior to entry into the
saggital sinus must be just above the level of the I.C.P. or else
venous collapse occurs (Johnson & Rowan 1974).

Focal or global brain swelling (oedema) often accompanies R.I.C.P.
in children after the compensatory mechanisms are exhausted.

Cerebral blood flow in the brain arises from two systems:

    1. Extraparenchymal

    2. Intraparenchymal

Extraparenchymal

Control of this is via the cerebral arterioles in the subarachnoid
space, where flow is controlled at certain levels of I.C.P. (between
50 and 150 mm Hg) via an active cerebral autoregulation system.
Both cortical vessels and the large vessels at the base of the brain

are richly innervated by adrenergic and cholinergic nerve fibres
and when there is a drop in the C.P.P. or a rise in the I.C.P., there
is a reflex sympathetic blockade of carotid vessels in an attempt to
increase the C.B.F.

(i) At I.C.P. levels of 0-50 mm Hg the C.B.F. is fairly constant
and depends passively on the C.P.P.

(ii) At I.C.P. levels of 50-99 mm Hg there is an increase in C.B.F.
from arteriolar dilatation (and a mostly systolic increase
in S.A.P.)

(iii)At I.C.P. levels of 95-153 mm Hg the C.B.F. gradually ceases
either focally or globally limiting substrate to the
metabolising brain and at this stage is again dependent
passively on the C.P.P.

The C.B.F. falls precipitiously below a C.P.P. of 40 mm Hg (Johnson
et al 1972) and in brain damage where there is loss of autoregulation,
the C.B.F. falls at lesser levels of I.C.P., i.e. with higher C.P.P.
values. For practical purposes the C.P.P. is the arterial entrance
pressure (i.e. S.A.P.) minus the venous exit pressure at the
superior saggital sinus (i.e. almost the I.C.P.), therefore
C.P.P. = S.A.P. - I.C.P. In brain oedema, the C.P.P. is a less
reliable indicator of C.B.F.

Intraparenchymal

These vessels are poorly innervated with nerve fibres and control
of their flow is related to local metabolism, i.e. pH or H^+.

$$\left. \begin{array}{l} H^+ \text{ (increased metabolism)} \\ pCO_2 + P_aO_2 \end{array} \right\} \longrightarrow \uparrow r \text{ C.B.F.}$$
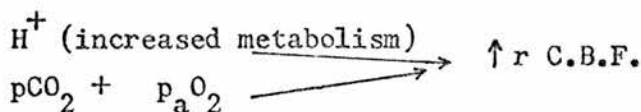
without a change in the C.P.P. and this is called metabolic
autoregulation. So although there may be a low C.P.P. and a drop
in C.B.F. there will be no residual damage if the metabolic auto-

regulation is intact. One can even tolerate absent C.B.F. with extreme lowering of metabolism for a short time with hypothermia.

Generally, however, with an intact metabolic autoregulation C.P.P. levels of less than 45 mm Hg will cause no change in the E.E.G., oxygen consumption or residual damage. If, however, the C.P.P. is of the order of 20-25 mm Hg despite intact metabolic autoregulation flattening of the E.E.G. and residual damage occurs.

Ideally, therefore, all unconscious neurological patients should have C.B.F. and CMR_o estimates done to keep therapy balanced. If the CMR_o is lost, one cannot rely on C.P.P. levels as 'luxury perfusion' or 'relative hyperaemia' may occur with focal oedema. There are as yet far too many technical limitations restricting a minute by minute print-out of these two parameters and at the present time C.P.P., arterial p_aCO_2 and a single estimate (Xenon) of C.B.F. are the most one can hope for. If a non-invasive continuous and reliable measure of C.B.F. was available for the infant, this would add a further parameter and stricter criteria for insertion of a shunting device.

(c) <u>Brain</u>

An increase in this intracranial component takes the form of cerebral oedema, cerebral congestion, cerebral tumour with surrounding oedema and megelencephaly.

The brain is virtually incompressible but in combination with its blood vessels, it assumes visco-elastic properties, i.e. stress relaxation. With continued application of pressure the tissue deforms and displaces with subsequent pressure decay.

The main displacements or herniations are:

    (i) <u>Cingulate herniation.</u> This compresses the anterior

        cerebral artery and gives rise to cortical ischaemia in the

leg area with clinical diplegia.

(ii) Transtentorial herniation, which may be anterior, posterior or
central with varying combinations of clinical signs, the most
readily recognised being the triad of diminished conscious state,
ipsilateral pupillary dilatation and hemiparesis.

(iii) Cerebellar herniation either upwards compressing the superior
cerebellar arteries or downwards (tonsilar) with occlusion
of the posterior inferior cerebellar artery, neck stiffness,
lower cranial nerve palsies and sudden cardio-respiratory
failure.

Occasionally herniation of the brain can occur after successful
C.S.F. shunting. This only happens when there was advanced
ventricular dilatation pre-operatively and the post-operative course
is complicated by premature suturral fusion with consequent restriction
of the intracranial volume and with growth tonislar herniation becomes
apparent (Hoffman & Tucker 1976).

A complete discussion of cerebral oedema is beyond the scope of this
chapter, suffice it to say that the commonest brain oedema, 'vasogenic
oedema', which results from head injury, tumours and infections, arises
from a disruption of the blood-brain barrier (morphologically a site for
protein blockage at the tight junctions formed by membraneous fusion of
adjacent endothelial cells lining the cerebral vasculature). This
oedema is mostly confined to the white matter and spreads widely
giving rise to herniation or blockage of C.S.F. with acute reduction
in compliance.

Cerebral oedema is the basic pathology in a number of conditions
with R.I.C.P. and depending on the aetiology each requires specific
management considerations. For example, severe head injury with coma,
decerebrate responses to pain or combined head and chest injury. In

these situations the cerebral oedema may occur alone or in association with haematoma formation and the general outline of principles of management below is but one suggested line of action (Miller 1978).

(i) At presentation supportive and specific treatment and investigations and I.C. Mannitol.

(ii) Ventilate to pCO_2 25-30 mm Hg. If an associated chest injury then muscle paralysis also, to give positive pressure ventilation.

(iii) Monitor I.C.P.

(iv) If the I.C.P. rises

    (a) check the position of the patient, e.g. for jugular compression.

    (b) check airway, tube length and secretions.

    (c) arterial blood gases.

    (d) check B.P.

    (e) recalibrate transducer.

(v) If the I.C.P. rises in excess of 30 mm Hg re C.T. scan to determine if brain swelling present or a haematoma has developed or is increasing in size. If a haematoma, give pre-operative bolus of Mannitol.

(vi) If brain swelling only, hyperventilate to pCO_2 20 mm Hg or less.

(vii) If R.I.C.P. persists and there is a ventricular cannula, closed external drainage against a pressure of 15-25 mm Hg.

(viii) If there is still R.I.C.P. Mannitol I.V. with or without a repeat dose.

(ix) If resistant R.I.C.P. then deep barbiturate coma with hypothermia.

One should arbitrarily commence treatment of R.I.C.P. when

(i) an increase in I.C.P. is associated with neurological

deterioration which may mean progression to ischaemia or brain shifts.

(ii) Any I.C.P. in excess of 30 mm Hg, based on the principle of maintaining C.P.P. in excess of 60 mm Hg, depending on the S.A.P. (Becker et al 1977, Miller et al 1977).

Other specific conditions with brain swelling include the encephalo-pathies e.g. Reyes syndrome where ideally an epidural pressure monitor is inserted and R.I.C.P. controlled by means of hyper-ventilation, Mannitol, steroids, appropriate fluid restriction, and attention to other physiological parameters, e.g. osmolality, temperature, seizures etc.

Burns and scalds present a number of problems in the management of their brain swelling. With severe burns there is hypovolaemia, and Mannitol in this situation may induce severe metabolic acidosis and renal failure. Steroids will increase the already increased risk of infection, e.g. pseudomonas, and often with burns about the head and neck, there will be respiratory restriction and 'flash' burning of the upper airways limiting the use of naso-tracheal or endo-tracheal tubes. The indications for I.C. pressure monitoring here is coma and clinical evidence of unremittant brain swelling. An epidural transducer, which can be placed at any point on the head, is the most appropriate method for monitoring I.C.P. The encephalopathy which occasionally accompanies minor scalds, is managed with variable results by the use of Mannitol and steroids.

For severe brain swelling which accompanies diabetic ketoacidosis the response to Mannitol and steroids is often poor however lesser degree are not infrequently seen and preventing the condition happening is by far the most important aspect of management. A precipitous drop in blood sugar, which decreases osmolality, sets up

an osmotic gradient between blood and brain shifting fluid into the brain.

Megalencephaly is any oversized and overweight brain irrespective of any neurological or mental dysfunction. This entity lacks an adequate classification in the literature although attempts have been made (Forrest 1900, De Myer 1972). It is probably best considered as:

(i)   the familial 'large-heads' in the population who usually have no signs or symptoms of R.I.C.P., mostly are of normal intelligence and apart from some initial concern about their rate of head growth in early infancy, do not present themselves as a problem. No series of histological features has been forthcoming for this group.

(ii) the anatomic megalencephaly made up of cases, e.g. neuro-fibromatosis, tuberose sclerosis etc. where neurological or mental derangements may occur. Pathologically they are recognised as showing an increase in cell size without biochemical accumulates within the cell. De Myer (1972) reviewed 18 such cases in whom he found no clinical evidence of R.I.C.P. However Portnoy et al (1978) found 7 cases with R.I.C.P. in this group, due to an increase in saggital sinus pressure and they postulated that this resulted in a secondary increase in parenchymal fluid volume and constituted one end of a spectrum of disorders which was closely allied to Benign Intracranial Hypertension resulting from diminished C.S.F. absorption into the saggital sinus, to frank communic-ating hydrocephalus with absent C.S.F. absorption.

It is my opinion the careful observation will often reveal signs of R.I.C.P. e.g. distended scalp veins, 'sunsetting' etc.

transiently, and only at an early age in these children.
However, with a normal ventricular size on scanning and
transient signs, measurement of the C.S.F. pressure has not
been done and seems hardly justifiable.

(iii) the remaining cases are of so called metabolic megalencephaly
where intracellular biochemical accumulation results in
histologically distended cells, e.g. gangliosidoses, Hurler's
syndrome. These often have signs of R.I.C.P. e.g. papill-
oedema, neck retraction etc.

Numerous other causes for R.I.C.P. in children can be found in the
literature and these are outlined in Table 9 and factors which
aggravate existing R.I.C.P. are listed in Table 10.

COMPLICATIONS OF V.P.M.

There is less than a 1% chance of inducing an intracranial haemorrhage
with ventricular puncture, however if repeated ventricular tapping
is carried out I.V.H. will be the inevitable result. For this reason
where control of C.S.F. pressure is likely to be protracted a
ventriculostomy reservoir is best electively inserted. This also
lessens the likelihood of puncture porencephaly and discomfort to the
patient. It is a very worthwhile practice to insert a reservoir in all
cases requiring a C.S.F. shunt, they rarely block, in fact only 1 case
in this series was suspected to have a transiently blocked reservoir.
Apart from the reasons mentioned above, it eases management of
shunt blockage, allows pressure monitoring, ventricular contrast
studies, instillation of antibiotics etc.

Opening or penetrating the dura has an intracranial infection rate
(meningitis or ventriculitis) variously reported, from less than 1%
to greater than 5% (Sundbarg 1972, Rosner & Becker 1976) depending

on the number of times the dura is interrupted. Langfitt (1973) suggested that infection complicated pressure monitoring in less than 1% of cases when the procedure was carried out by experienced personnel. Infection is practically a negligible complication when pressure monitoring lasts for less than 3 days, however the prolonged use of steroids will obviously increase its incidence.

At the Royal Hospital for Sick Children in Edinburgh, we do not routinely use antibiotics for children undergoing V.P.M. but C.S.F. is collected early in the procedure and at its conclusion for microscopy and culture. In the 100 cases here reported one child possibly developed a C.S.F. infection as a result of V.P.M. but responded well to appropriate antibiotic treatment.

Leakage of C.S.F. from a tense fontanelle for a variable time after monitoring is not infrequent, particularly where the ventricular pressure was found to be elevated. Asepsis with sterile cotton wool, antibiotic sprays, colloidion etc. are all that is required for this complication.

No damage to S.C.S. shunts or reservoirs has resulted from needling them, there have been no C.S.F. fistulas, gross cortical damage or any other complication attributable to pressure monitoring and when one considers the acute complications of R.I.C.P. mentioned throughout this thesis, and the more chronic effects of R.I.C.P. (see published paper in Appendix by Minns et al 1977) the advantages for the individual patient for whom pressure monitoring is indicated, far outweighs any possible disadvantages.

FUTURE CONSIDERATIONS

A number of areas requiring further research have become evident during the preparation of this thesis and some of these are: an

indepth follow-up should be undertaken of the children who have
not had shunts inserted or who have had them removed on the basis
of pressure results, especially those with equivocal pressures;
the relationship of C.B.F. to particular levels of I.C.P. in the
patient of less than 18 months of age; techonological advances
which would make possible a non-invasive estimate of C.B.F. in normal
and sick children; refinements of methods for regional cerebral
metabolism in children in the clinical setting, and a reliable
telemetric device for long term out-patient assessment of I.C.P.
on those children with persistently abnormal C.S.F. dynamics.

SUMMARY

One hundred cases of children who underwent V.P.M. have been
discussed in detail and examples of their pressure tracings
illustrated. The technique employed has been justified from both
a theoretical and practical standpoint and other methods of measure-
ment and monitoring of I.C.P. reviewed, with their relative advantages
and disadvantages and their most useful clinical setting.
From this and other studies it is evident that I.C.P. monitoring
is a useful clinical tool in paediatrics and it has afforded me
the opportunity also to report and discuss the original results of
the effect of I.C.P. on monoamine metabolites in children's C.S.F.,
the not previously reported effect of various types of seizures
have on the I.C.P, and the fascinating effect of sleep on I.C.P.
I have put forward a hypothesis for this sleep effect based on
observations of pressure, E.E.G., E.C.G. and respiration.

The Chapter on Ketamine and anaesthesia has added some weight to
other studies regarding the effect of Ketamine on the C.S.F.
pressure, but raised doubts about its convulsant action. I have

shown the previously overlooked effects of intubation on I.C.P.

and described a momentary pressure reflex associated with

spreading the vocal cords, which together with the sleep effects of

pressure, may well be related to the sudden infant death syndrome

(S.I.D.S.).

From this study many ideas for the future have sprung that

hopefully

....are begot in the ventricle of memory, nourished

in the womb of pia mater and delivered upon the

mellowing of occasion.

Shakespeare.

TABLE 9

CAUSES OF R.I.C.P. IN CHILDHOOD

1. PRIMARY HYDROCEPHALUS

   sporadic or familial aqueduct stenosis (intraluminal veils or subependymal vascular anomalies)

   Dandy Walker syndrome

   Chiari malformation with or without myelomeningocoele

   idiopathic

   other malformations: hydranencephaly, porencephaly, holoprosencephaly.

2. INTRACRANIAL HAEMORRHAGE

   (a) PRE TERM

   intraventricular, periventricular or subependymal haemorrhage. Cerebellar or intracerebral haemorrhage especially in L.B.W. infants with R.D.S.

   (b) NEWBORN

   subarachnoid or subpial haemorrhage

   subdural haemorrhage (mechanical injury)

   extradural haemorrhage rarely

   (c) INFANTS AND OLDER CHILDREN

   head injury: subdural (BBS), epidural, subarachnoid.

   A.V. malformations e.g. cryptic angiomas, hereditary telangiectasia etc. multiple haemangimatosis.

   ruptured aneurysms, great vein of Gaten, anterior cerebral (Berry), mycotic.

   Haematologic: haemophilia, leukaemia, petechial or massive thrombocytopenia in aplastic anaemia, disseminated intravascular coagulation etc.

   cortical vein or dural sinus thrombosis due to sepsis, cyanotic C.H.D., hypertonic dehydration, suppurative mastoiditis and septic cavernous sinus thrombosis.

   jugular vein or superior vena caval occlusion.

3. CEREBRAL TUMOURS

papilloma of choroid plexus, arachnoid cysts, neuroblastomas, retinoblastomas, medulloblastomas, craniopharyngiomas, ependymonas, sarcomas, melanomas, spinal cord tumours and abscesses. The above can also cause spontaneous haemorrhage exaggerating R.I.C.P.

4. PSEUDOTUMOUR CEREBRI

from nalidixic acid, tetracyclines, steroid withdrawal adrenal hyperplasia, menarche, obesity, hypocalcaemia, hypoparathyroidism, vitamin A intoxication, lateral sinus thrombosis from middle ear infection or mastoiditis, head injury, histiocytosis-X.

5. THROMBO-EMBOLIC CEREBRAL DISEASE

cyanotic C.H.D. < 2 years; cortical vein or cerebral art thrombosis, > 2 years cerebral abscess.

cerebral emboli; post rheumatic, bacterial endocarditis, intracardiac mural thrombi from cardiomyopathies or endocardial fibroelastosis, left atrial myxoma.

sickle cell disease

collagen diseases e.g. S.K.E.

Kohlmeier-Degos syndrome

Moyamoya

neurofibromatosis

radiation induced arterial injury

carotid artery thrombosis following: structural lesions of the arterial wall, congenital dissecting aneurysms, following angiography, injury to the tonsilar bed, tonsillectomy, blunt trauma to the neck.

6. INFECTIONS

acute bacterial meningitis especially tuberculous, cryptococcal and coccidioidal with possible secondary subdural effusions, empyemas and abscesses.

viral encephalitis

exogenous acute toxic encephalopathies: pertussis, smallpox vaccination, lead, plant ingestion, salt poisoning, water intoxication, alcohol ingestion, hexachlorophene, aflatoxin ingestion, Jamaican vomiting sickness, phenylketonuria, drug abuses.

endogenous encephalopathies: aminoacidurias, e.g. maple syrup
urine disease, homocystinuria, organic acidurias, porphyria,
thyroid crisis, uraemia, hepatic failure, hyperbilirubinaemia,
hypercalcaemia, Reyes Syndrome.

Roseola infantum.

7. MISCELLANEOUS

Vitamin A abnormalities especially hypovitaminosis A due to
cystic fibrosis or biliary atresia.

Burns from diffuse cerebral insult or scalds and stings
encephalopathy.

Diabetic ketoacidosis during acute treatment.

Hypertensive encephalopathy

Pulmonary disease: emphysema, cardiac failure, obesity, kypho-
scoliosis, polio, muscular dystrophy.

Crouzons disease, galactosaemia.

Iron deficiency anaemia , rapid somatic growth after malnutrition.

Adverse reactions to penicillin, subacute sclerosing panenceph-
alitis.

Guillian Barré Syndrome, Wiskott Aldrich syndrome, osteopetrosis.

Degenerative brain diseases: Tay Sachs, Canavans, Alexanders maple
syrup urine disease, generalised gangliosidosis, mucopoly-
saccharidoses, meta chromatic leukodystrophy, gauchers disease.

neurocutaneous syndromes: basal cell carcinoma, incontinenti
pigmenti.

anatomic megalencephaly associated with gigantism, dwarfism,
tuberous sclerosis and familial types.

TABLE 10

FACTORS AGGRAVATING R.I.C.P.

1. RESPIRATORY INFECTIONS, airway blockage, neck obstruction etc.

2. SYSTEMIC HYPERTENSION

3. R.E.M. SLEEP

4. PYREXIA

5. VOLATILE ANAESTHETIC AGENTS

6. KETAMINE

7. FITS

8. SUPER ADDED BRAIN SWELLING e.g. about tumours

9. C.N.S. INFECTIONS

10. LARYNGOSCOPY AND INTUBATION

11. PATIENT POSITION

12. STOOLING

13. PERSISTENT CRYING

14. PLATEAU WAVES

15. ANY OTHER CAUSE OF HYPERCAPNIA, HYPOXIA or RAISED C.V.P.

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