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Preoperative ICP Monitoring (x).

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SUMMARY :

Preoperttive intracranial pressure (ICP) measurement gives very valuable information on some neurorsurgical problemss, like post - traumatic intracranial massive hematomas. post - traumatic hydrocephaly, and predicting the prognosis etc.

It is an early alarming system for the Neurosurgeons, that pathological process is under the development, especially on the cases whom unconsciouss and to follow up is clinically impossible. On the other hand, accordingly with the ICP measurement method, it is also possible to check the brain - volüme index and pressure volume response in the craniospinal axis. Another important point of ICP measurement is that it can be used to release cerebrospinal fluid to lower the high ICP accordingly with the measurement method.

INTRODUCTION

First description of intracranial pressure (ICP) measurement was experimentally made by LEYDEN in 1866 and its technique was *introduced* into clinical practice in 1960 by LUNDBERG[3]. Since then, the indications for ICP monitoring have increased to include head injury, subarachnoid and intracranial hemorrhage, brain tumors, hydrocephalus, hypoxic brain damage and encephalitis[6].

The widespread use of intracranial pressure *(ICP)* monitoring in clinical neurosurgery has facilitated the management of elevated ICP. Although there is general agreement with regard to the diagnostic and theurapeutic value of ICP monitoring, from the several publications, there appear to be still some doubt its accuracy[28]. Therefore, it is necessary to have the entire monitoring system free of microleaks to prevent spurious ICP readings and all connections must be watertight[28].

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ICP monitoring *methods* vary; but only three methods have been widely used, of which has its advantages and drawbacks[23],

a) *Extradural monitoring :* In this method, either the sensor is placed in direct apposition to the dura or the signal is transmitted to an extracranial sensor using a fluid filled extradural screw. The method affords the advantages of relatively easy placement and a low incidence of serious infection, as the dura remains intact. Major disadvantages are signal damping, problems of sensor plate - dura interrelationships and a tendency to over-read when compared to ventricular pressure. In addition, the method does not allow assessment of intracranial compliance[2, 7, 23, 34].

b) *Subarachnoid/Subdural monitoring :* The ICP is transmitted via a fluid column to a sensor using a subarachnoid catheter or extradural screw placed in the subdural or subarachnoid spaces. The main advantages are ease of placement, avoidance of brain puncture and independence from brain shift or small ventricular size. The drawbacks are that blockage of the screw may occur at high ICP levels, surface bleeding may be caused when the dura and arachnoid are opened; cerebrospinal fluid (CSF) leak especially with the catheter, may occur and there is potential risk of meningitis owing to the dural breech. In addition Mendelow, et al, reported that both the LEEDS and RICHMOND screws tend to under-read in the subdural space at ICP values above 20 mm Hg. Also, recently it was reported that the ICP recorded in the subarachnoid space may vary with the site of measurement in patients with unilateral pathology.[6, 22, 23, 44, 45].

c) Intraventricular monitoring : The ventricular fluid pressure method (VFP) involves the insertion of a catheter or a reservoir into the frontal horn of the lateral ventricle with connection by fluid line to on external sensor. The method has the advantage of high quality and most accurate recordings and allows CSF drainage and pressure/volume *analysis*. (Fig: 1-2-3). In acute pressure *situations* the combination of immediate diagnosis and immediate ttaetment may be lifesaving and in many patients continous drainage of ventricular fluid under pressure control has shown to be a valuable aid for prolonged treatment of intracranial hypertension. (Fig: 4-5-6-7-8). In addition, the indwelling catheter or reservoir facilitate ventriculography, sampling of CSF and intraventricular administration of drugs as necessary. Recordings of the VFP and volume/pressure *response* (VPR) may be more or less *useful* in different *kinds* of patients and in different situations (Fig 3).

The disadvantages of this method are that the brain must be punctured and when the ventricles are small and shifted they may be diffucult to locate.

Generally, no attempt to be made more than three to puncture the ventricle and if unsuccessful, it would be wise to turn subarachnoid/subdural monitoring method[1, 11, 12, 15, 16, 19, 20, 26, 34, 35, 36, 41, 21, 23, 28, 37, 39, 42].

WHAT IS NORMAL ICP?

A catheter inserted into the lateral ventricle records a pulsatile pressure of 0-10 mm Hg (0-136 mm H20) relative to the foramen of Monro when the patient lying flat. In normal circumstances, however, ICP may transiently increase to 100 mm Hg during coughing or straining. Although it has been traditional to measure lumbar CSF pressure in milimetres of water. It is more valuable to measure ICP in milimetres of mercury as it may need to be critically compared with arterial pressure. (Fig: 1). When the ICP is displayed on an oscilloscope or chart recorder two frequencies of pulsation are seen: a rapid *oscillation* which corresponds to arterial pulsation and a slower waveform synchronous with respiration. *They* are produced by changes in intrathoracic pressure. (Fig: 1)[23].

WHAT IS ELEVATED ICP?

A sustained increase in mean ICP above 15 mm Hg *abnormal* but ICP may in some circumstances rise to the level of the arterial pressure. ICP levels between 20-40 mm Hg are considered to be moderately elevated (Fig: 5, 6, 7, 8) and those over 40 mm Hg severly elevated (Fig: 4). Elevations in ICP may take the form of a steady increase or more often appear as periodic waves. The considerable variation in ICP that occurs with time is the *reason* why it is important to monitor it continously. (Fig: 5, 6, 8). *Three* forms of pressure wave were distinguished by LUNDBERG: PLATEAU waves rising abruptly to 50 mm Hg or more, for 5-20 minutes and then falling spontaneusly (Fig: 6); SHARPLY PEAKED waves of variable height occuring at a frequency of 0,5-2/minutes and often corresponding to changes in respiration (Fig: 5, 7, 8) and WAVES related to TRAUBE-HERING plots of blood pressure arising 5-8 times/minute[23].

WHAT ARE THE CLINICAL CORRELATES OF RAISED ICP?

With increasing experience of ICP monitoring it has become evident that the only way reliable to gauge its level and its variations over time is to meassure continously and include at least one overnight record[20, 23].

The Cushing triad of headache, vomiting, and papilloedema is the traditional clinical indicator of intracranial *hypertension*. Papilloedema is a reliable

sign that ICP is or has recently been *increased*, but it takes time *to* develop and headache and vomiting may be absent during severely increased ICP. Other signs such as drowsiness, bradycardia, arterial hypertension, pupillary dilatation, decerebrate rigidity and respiratory changes which are seen in some patients with high ICP are related *casually* more to brain *shift*, distortion and herniation than to specific pressure levels[9, 10, 14, 16, 27]. However, increasing ICP in such patients is often a forerunner of and an important clue to impending neurological deterioration[20, 23].

WHAT HARM DOES RAISED ICP DO?

Papilloedema, which may lead *onto* optic atrophy and blindness, is a direct consequence of raised ICP. The other harmful effects of intracranial hypertension are mediated either through reductions in cerebral blood flow (CBF) or through brain shift[16, 23, 27].

A dramatic correlate of brain death following *overwhelming hedd* injury or subarachnoid hemorhage is the appearence of "false block" of the carotid system in which angiographic *contrast* medium injected into the internal carotid artery fails to enter its intracranil portion. In this situation the ICP is or has recently been, at the level of the arterial pressure so that there is no perfussion pressure avaliable to drive the contrast medium accross the cerebrovascular bed. In the normal cerebrovascular bed, CBF can be maintained within the usual limits in the face of rising ICP until the difference between arterial pressure and ICP falls to 40 mm Hg, below this CBF *falls* precipitously. In the damaged brain, where this autoregulatory mechanism is impaired, CBF falls at lesser levels of intracranial hypertension[14, 23].

One wonders from the both clinic and experimental evidences, whether there is an arbitrary level of raised ICP at whichtreatment should be started. It is known that neurological function is deterioted by any increase *in* ICP which, produces cerebral ischemia or aggravates brain shift[9, 10, 18, 61, 28].

WHAT ARE THE INDICATIONS FOR ICP MONITORING AND HOW MAY IT HELP THE PATIENT?

ICP monitoring is most *frequently* used in severe head injured cases to see if any complication of trauma (acute hematoma, delayed hematoma, post traumatic hydrocephaly) developing, although there is against opinions to use the ICP monitoring[1, 5, 8, 13, 15, 21, 26, 29, 30, 36, 41]. It is also widely used in obtunded patients with subarachnoid hemorrhage, intracranial hemorrhage and brain tu-

mors who are awaiting surgery, in hydrocephalus, in benign intracranial hypertension, and in hypoxic brain damage[4, 23, 40, 43].

It is believed that *the* information of ICP is beneficial to the patient in several ways. First, it is important to delineate those patients who do not have elevated ICP, so that they can be spared needless and expensive therapy. It is also *important* to detect those patients who have raised ICP so that this may be treated ideally *by removing* the cause (removing hematoma, insertion of shunt etc)[1, 4, 5, 8, 13, 15, 21, 28, 29, 40, 43].

By monitoring the ICP, the effectiveness of treatment can be directly *measured* and an ineffective mode of therapy quickly changed to one that works[23]. On the other hand, the ICP monitor provides the sole guide the progress of intracranial events in the patients who are not accessible by neurological examination and clinical following, like the ones *artificially* ventilated *under* muscle relaxants. (Figures: 9, 10, 11).

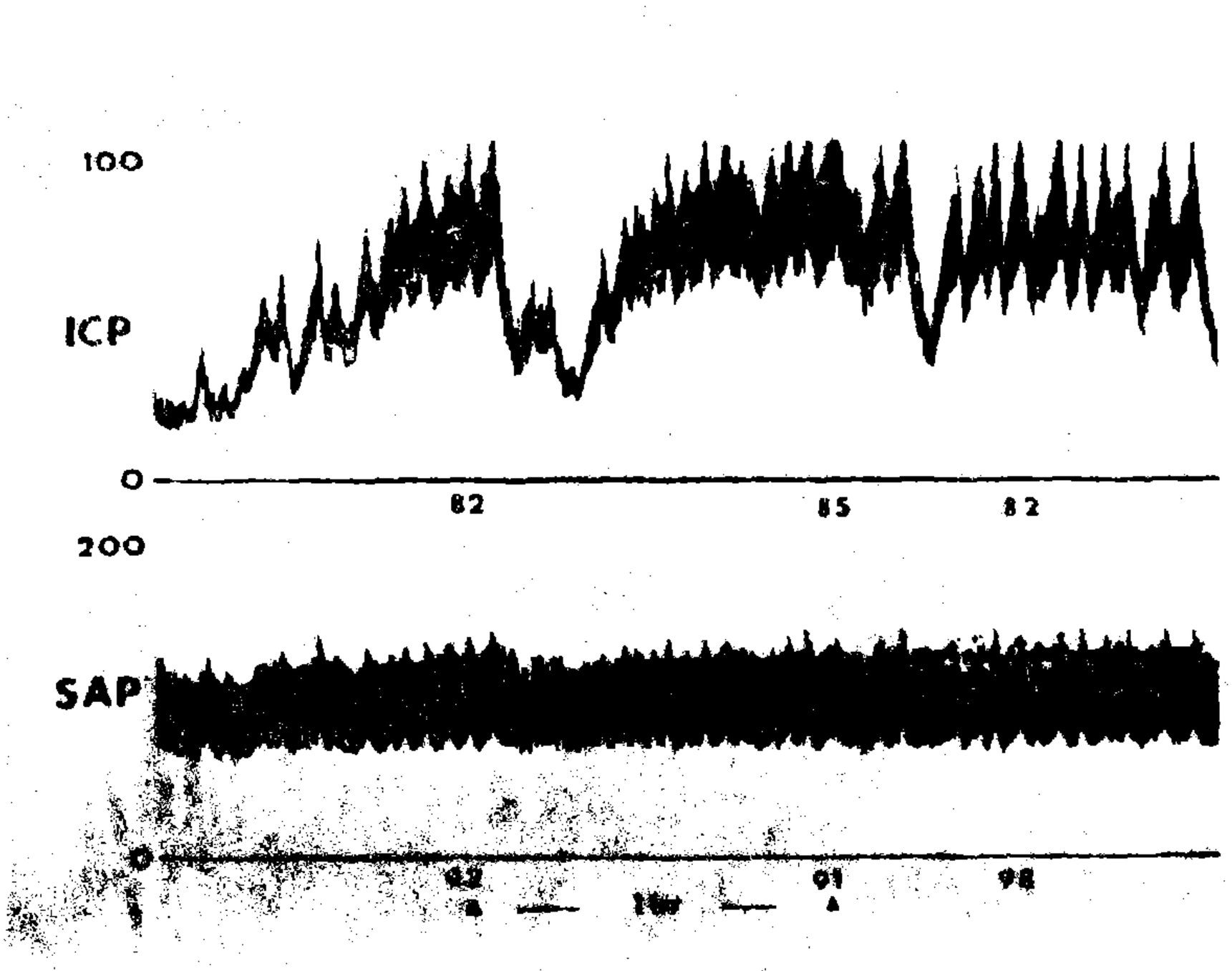

Figure 1 : Simultaneusly recorded ICP and systemic arteriyel pressure graphics.

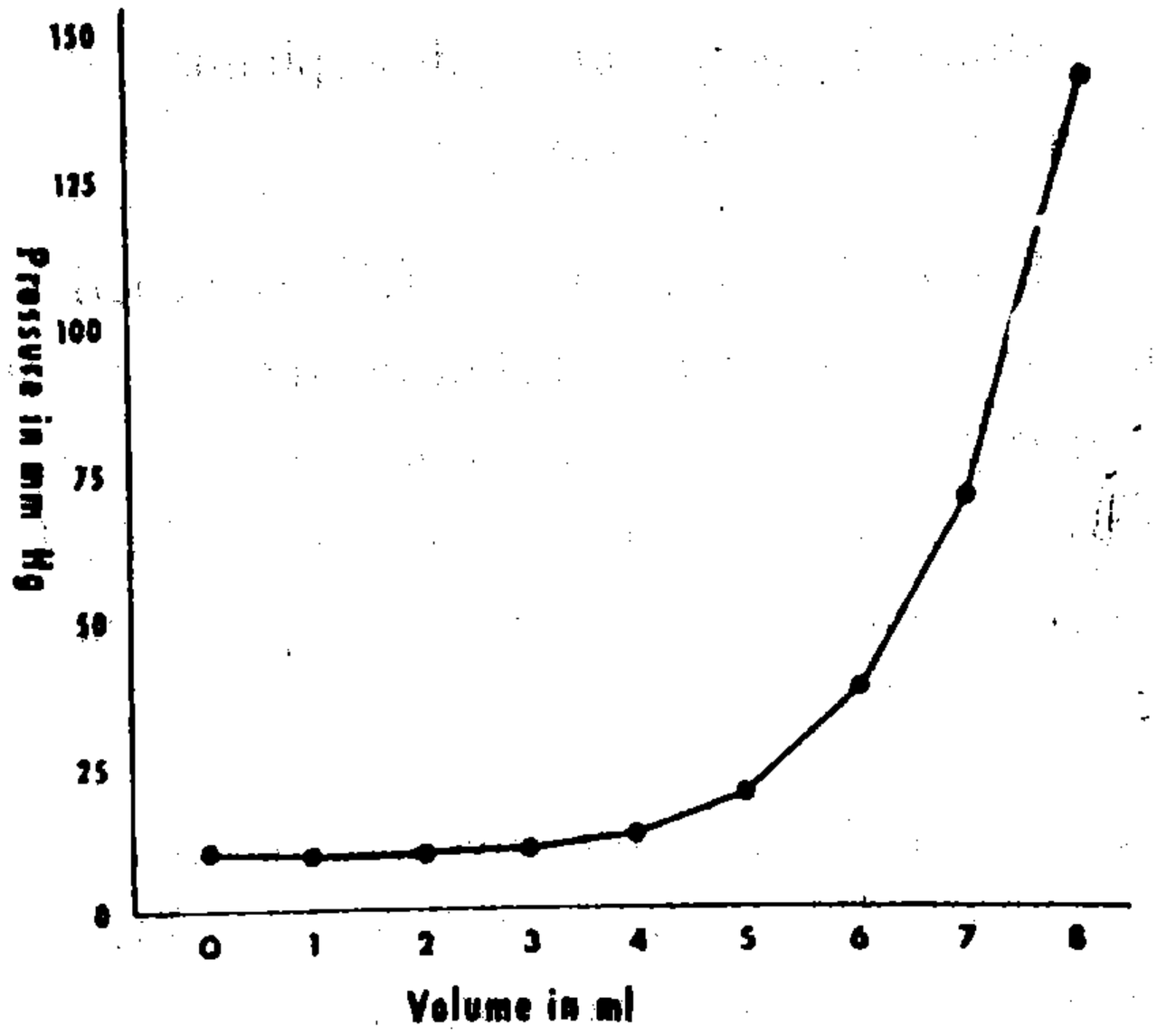

Figure 2 : This figure shows the relations between the intracranial pressure in mmHg and intracranial volume in ml. (V/P graphic)

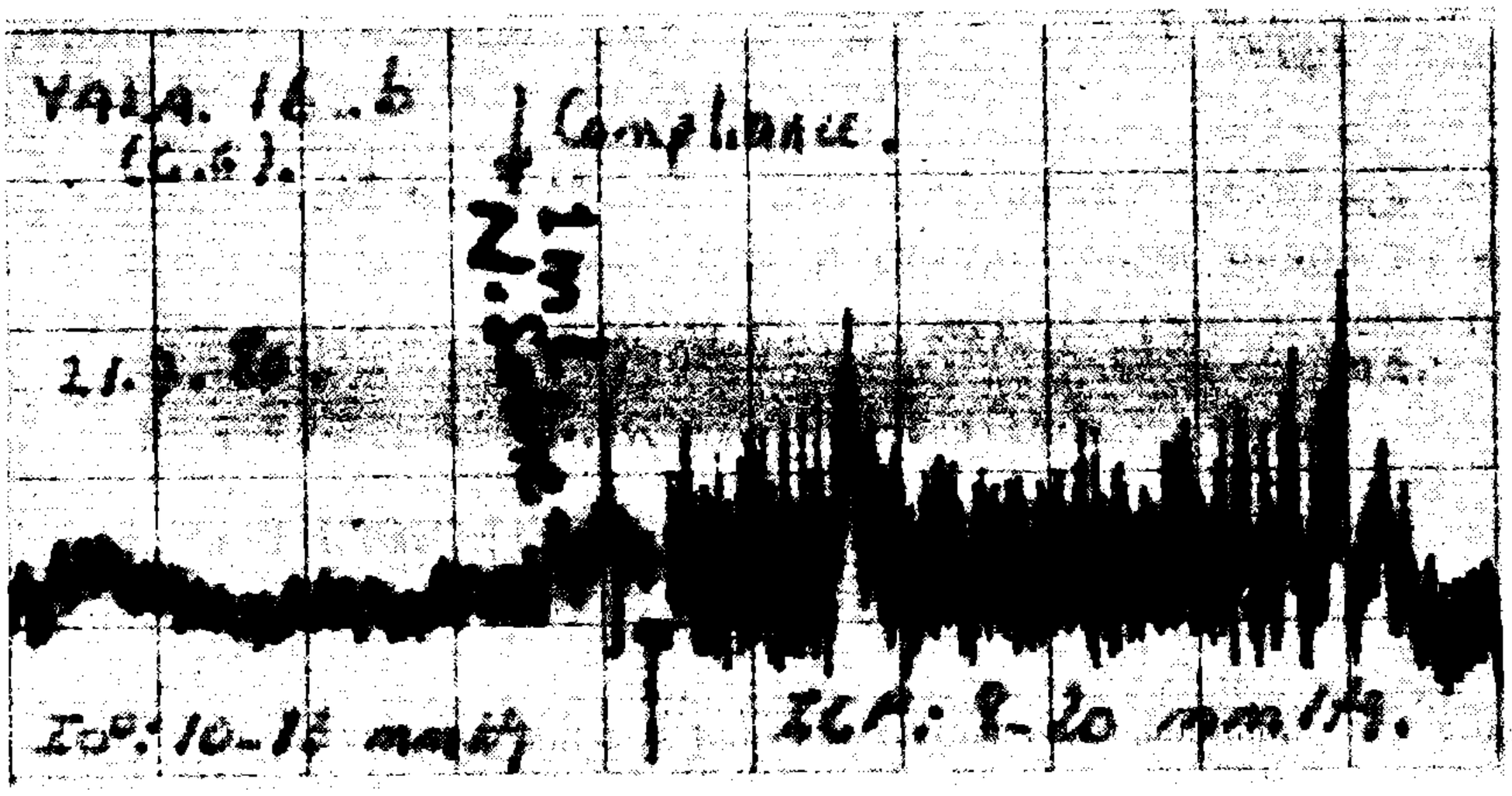

Figure 3 : ICP recording with V/P analysis (compliance).

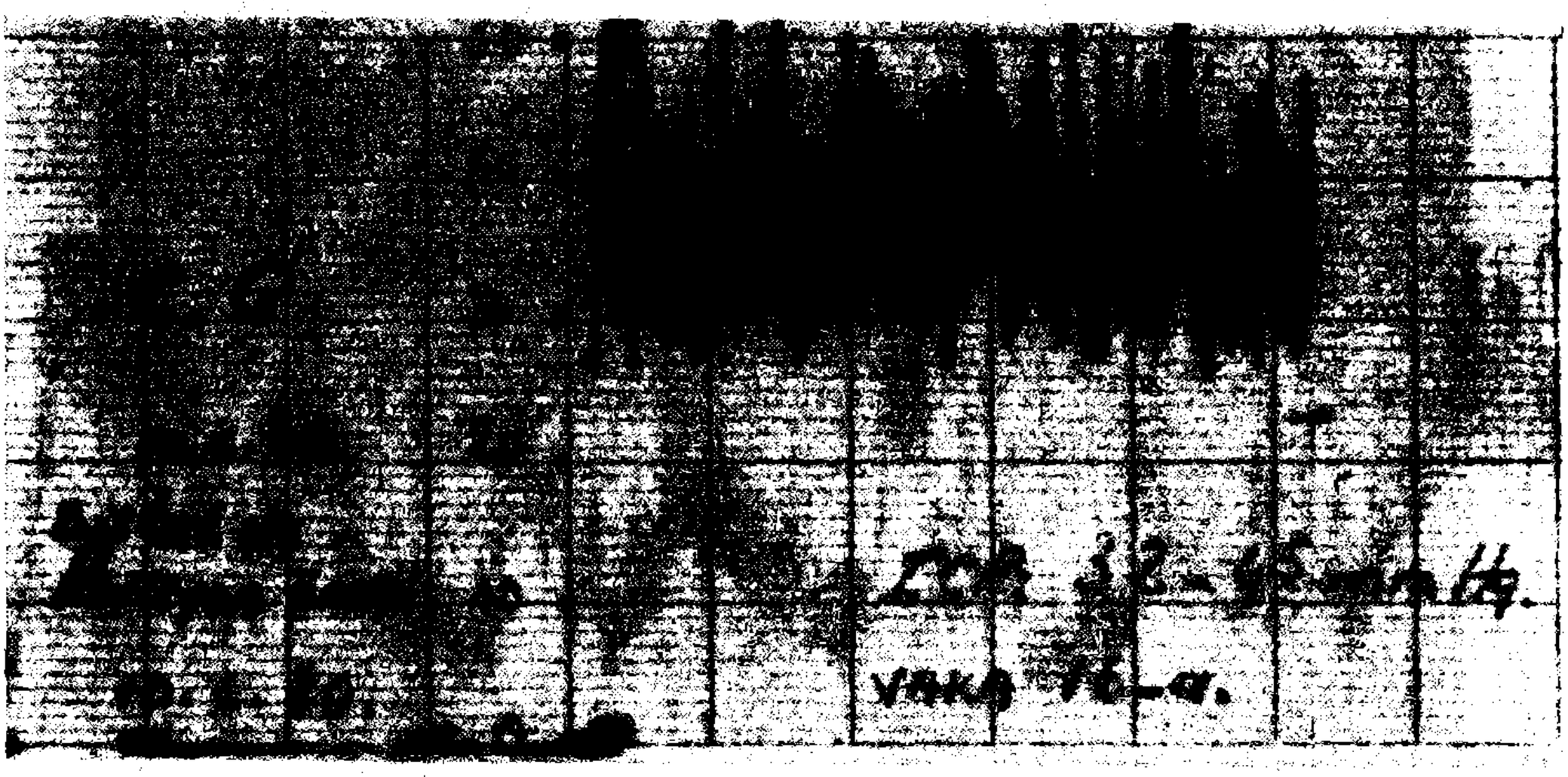

Figure 4 : High ICP recording indicate immediate action to lower the ICP.

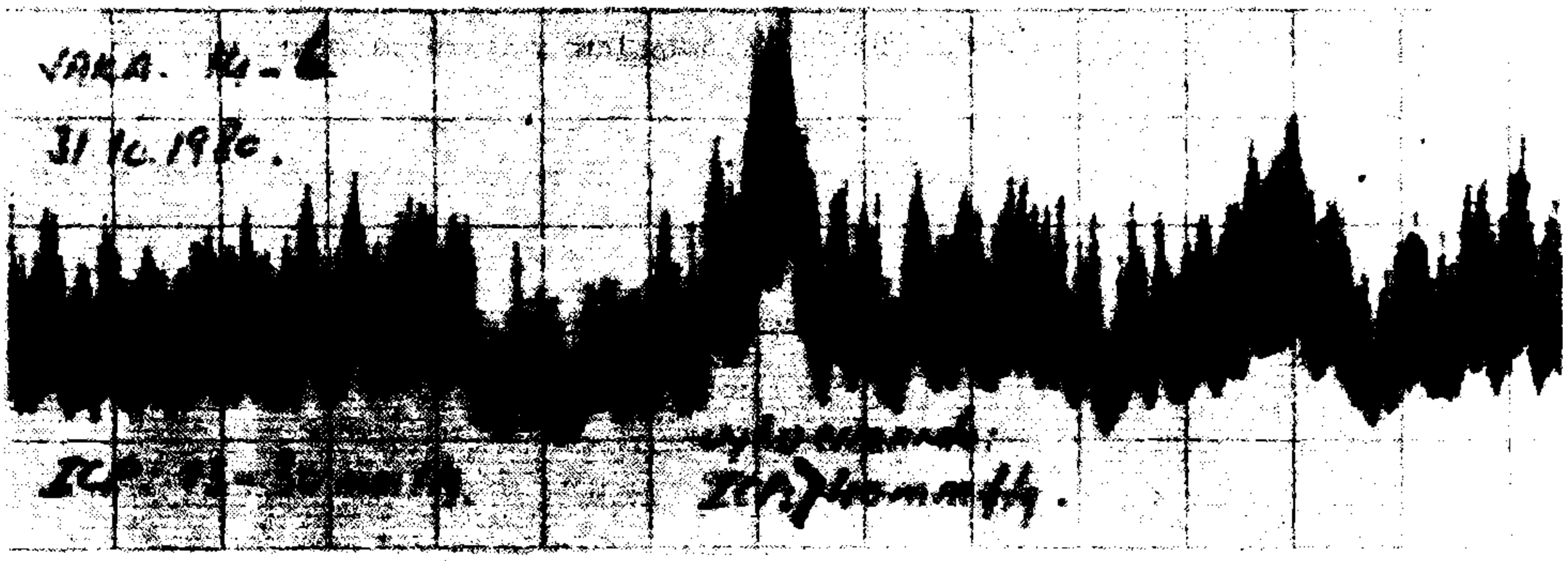

Figure 5 : ICP recording with a moderate high course and peaks during sleep.

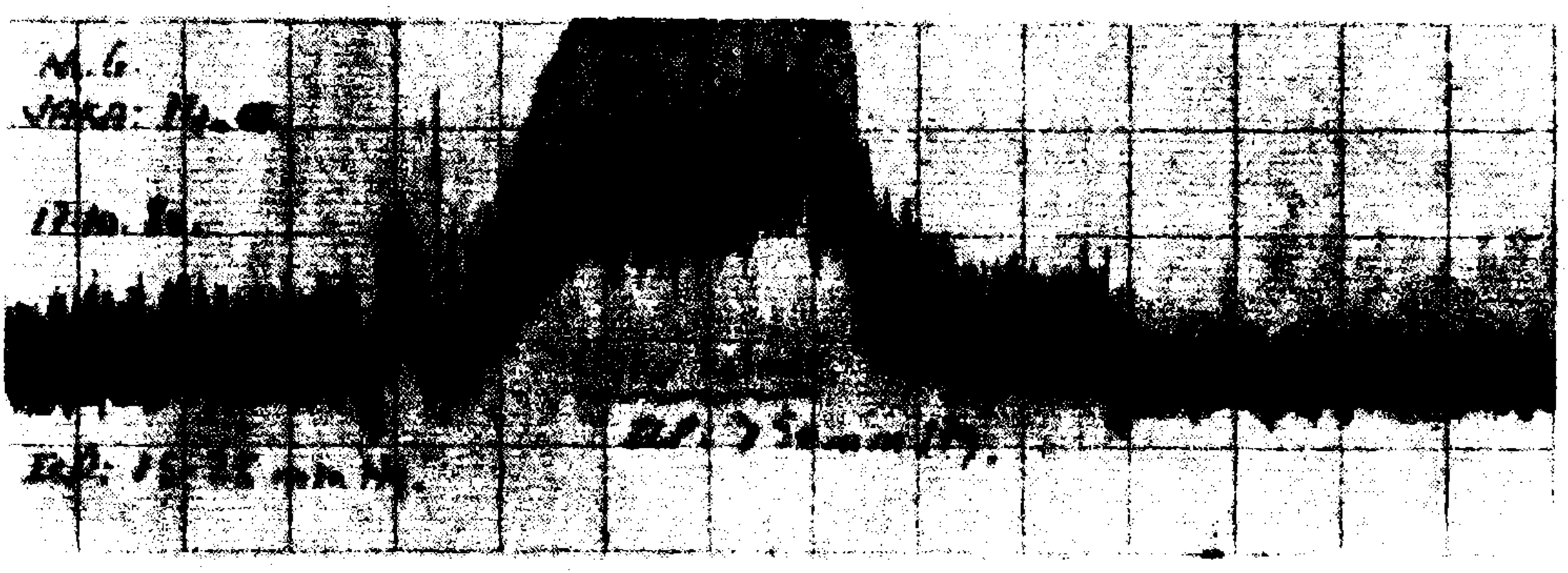

Figure 6 : With a moderate high course ICP recording and during sleep at times PLATEAU waves present.

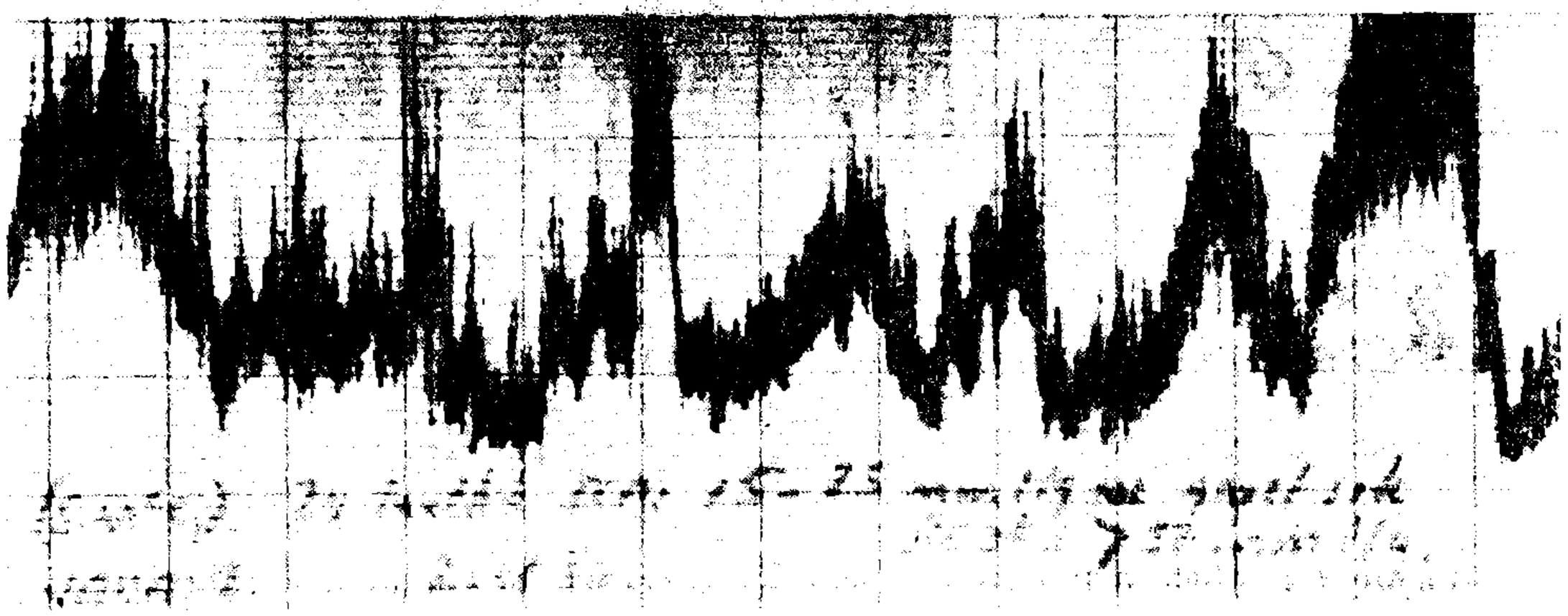

Figure 7 : With a moderate ICP recording but very often peaks appear.

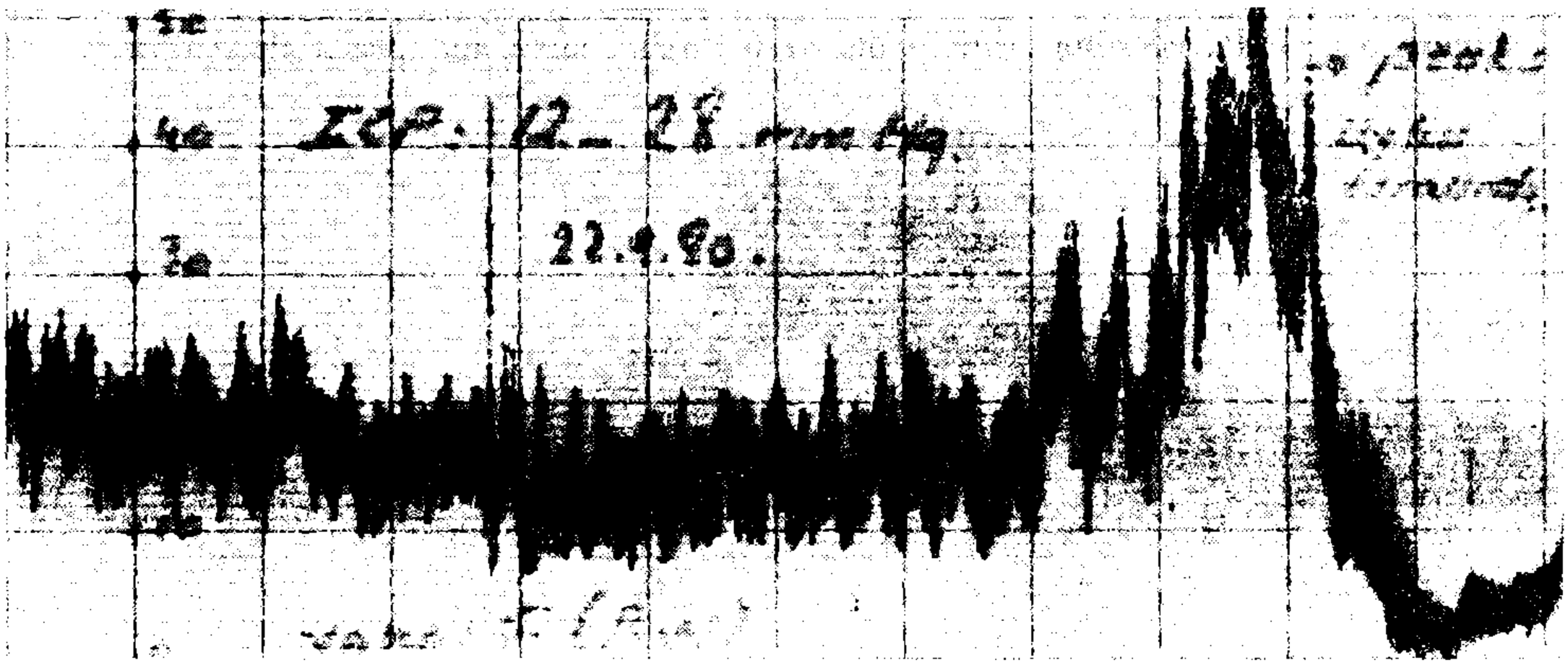

Figure 8 : ICP recording with a moderate high course and P5AKS during sleep.

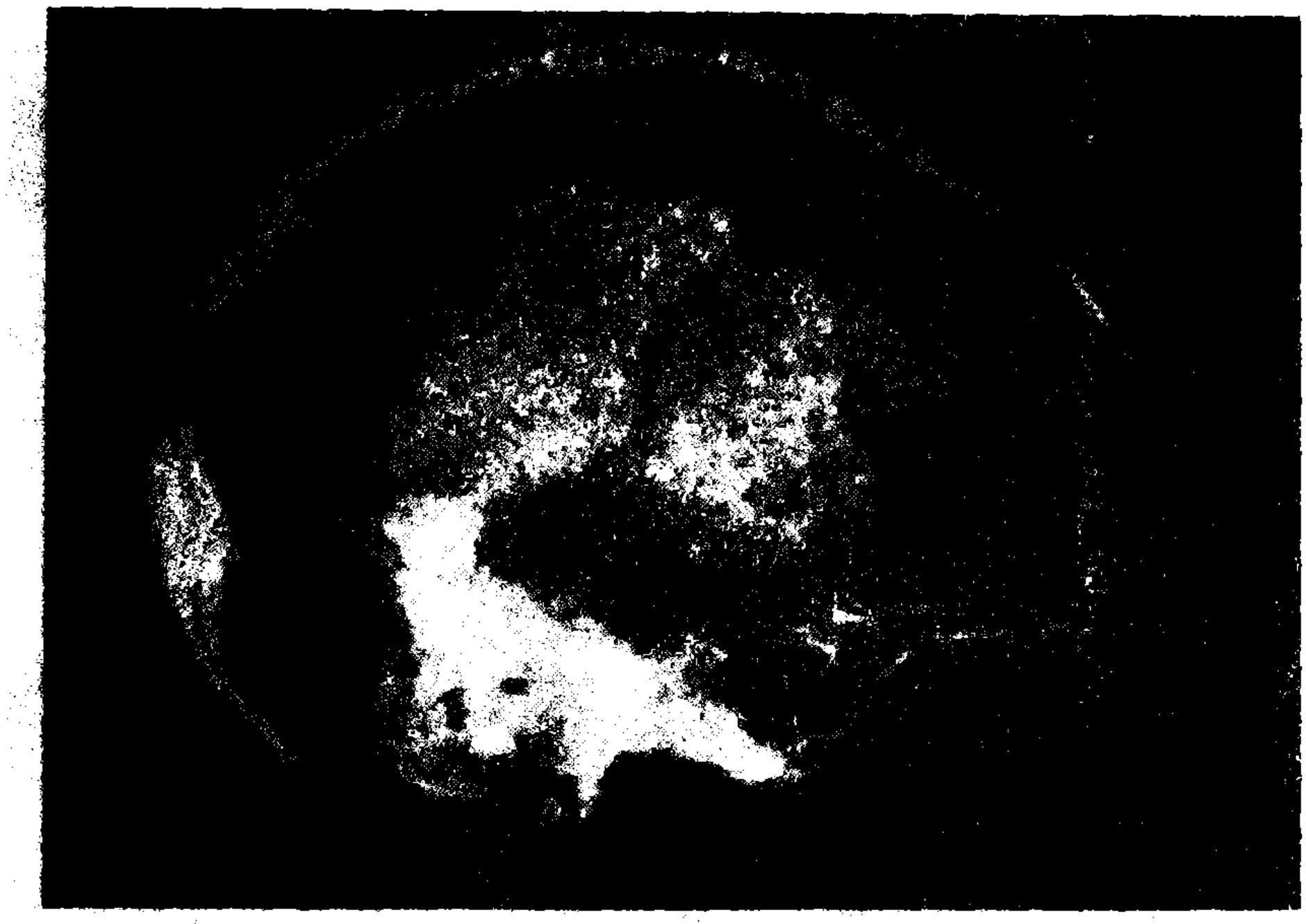

Figure 9 : A Case, involved in a road traffic accident, being deeply unconscious and his skull XR shows multipl linear fractures which render him to develop an extra-dural hematom, but his initial CAT scanning showed no hematoma (see figure — 10).

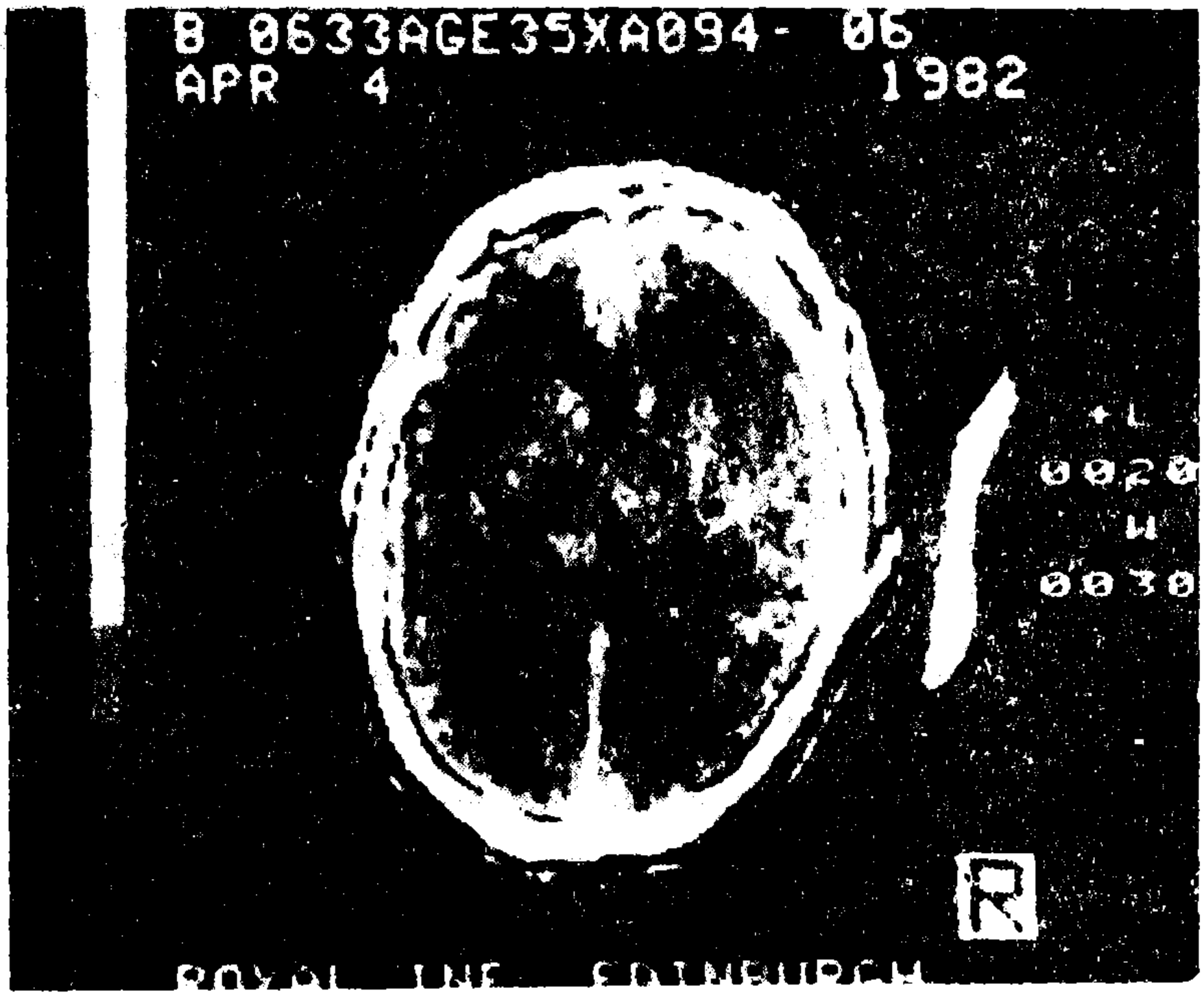

Figure 10 : CAT scanning of the case with multipl linear skull fracture and Loss of consciousness, this initial CAT did not revealed any intracranial hematoma.

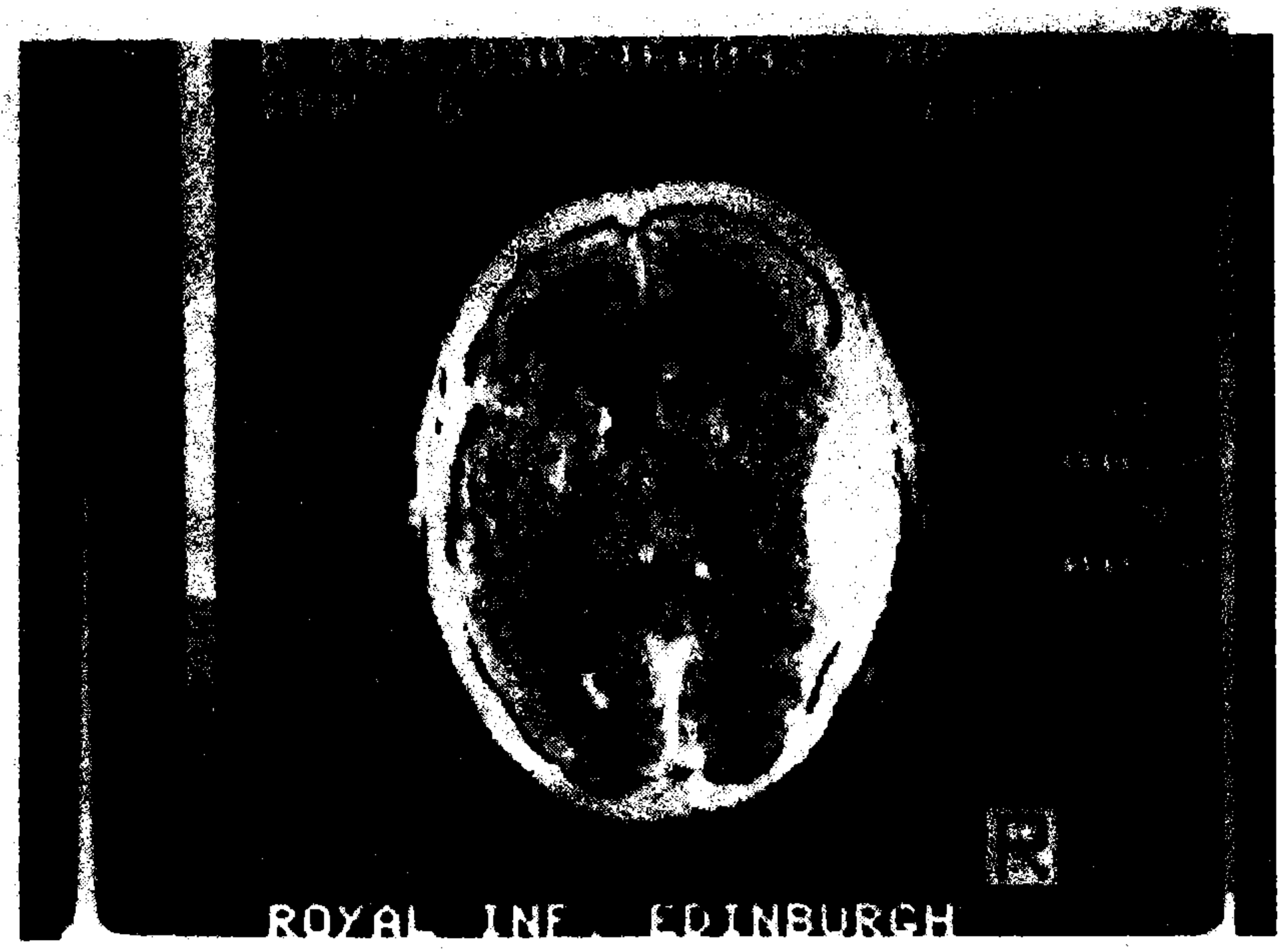

Figure 11 : Being ventilated under muscle relaxants ICP monitored, in other words, not being able to be accessible with neurological examination, this case's ICP level 2 days later of his ventilation ICP raised and immediate invtstigation with repeat CAT scanning showed under he linear fractur a huge EXTRADURAL HEMATOMA present. Otherwise this case, if ICP had not been monitored, would have died as his huge extradural hematoma would have been missed.

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