THE INFLUENCE OF ANESTHETIC DRUGS AND TECHNIQUES ON INTRACRANIAL TENSION *

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Success or failure during intracranial operations often may be decided by the degree of care and skill that is exercised by the anesthetist. With the judicious use of the cautery, bone wax, gelfoam, and more recently induced hypotension, the neurosurgeon can to some extent control bleeding. Detailed surgical dissection will limit the incidence of local traumatic edema. But when swelling of the entire brain occurs, when cerebral congestion pushes the brain tissues beyond their normal boundaries, few effective means are known to combat this perhaps fatal complication.

Within recent years light has been shed on some of the factors that may influence the size of the brain at operation. Most of these factors have their roots in the respiratory system and involve deviations from the normal status quo. As a rule cerebral congestion is present only when the respiratory physiological balance is upset.

The term, physiological imbalance, is rather vague and connotes little that can be carried into practice. Reduced to its simplest terms with reference to respiration, it means the development of hypoxia or carbon dioxide retention during the course of operation. Thus, Brennan (1) stressed that the etiological factors associated with cerebral congestion included anoxia, respiratory obstruction and inadequate ventilation of the patient owing to faulty positioning. This emphasis upon physiological alterations was reinforced by pertinent animal experiments carried out by White *et al.* (2). This group found that pentobarbital sodium and ethyl ether produced relatively insignificant changes in total brain volume. However, anoxia and carbon dioxide retention resulted in pronounced swelling and congestion of the brain. More recently Hunter (3) has emphasized the necessity of smooth induction and maintenance of anesthesia, with avoidance of breath holding, spasms of coughing or "bucking" on the endotracheal tube, if increased intracranial tension is to be avoided.

That either anoxia or hypercarbia does produce dilatation of the cerebral vessels sufficient to increase the brain volume has been demon-

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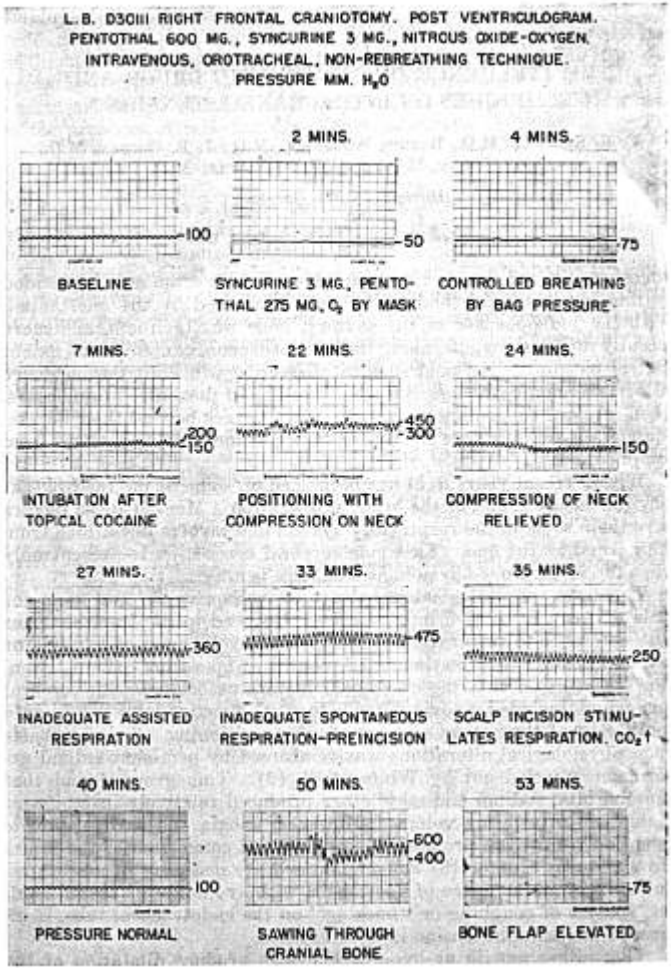


FIG. 1. Smooth induction and intubation. Note minimal increase in pressure with controlled respirations. Observe increase in pressure with inadequate assisted and spontaneous respirations. Note increase in pressure with compression of neck and direct pressure on brain (fifty minutes).

strated by Kety and Schmidt (4). It is probable that anoxia also increases the fluid component of the cells within the brain, and therefore their size, and thus may also act to increase swelling (2).

Since the pressure of the subarachnoid cerebrospinal fluid reflects to some extent the tension to which the brain substance is being subjected, it was thought that minute to minute measurement of variations in this pressure during induction and maintenance of anesthesia in human beings might prove of value. By correlating the episodes during anes-

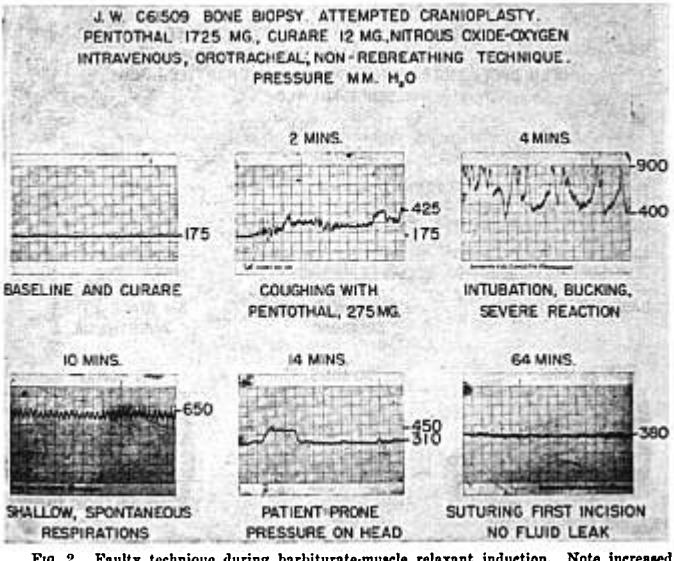


FIG. 2. Faulty technique during barbiturate-muscle relaxant induction. Note increased pressure is maintained. Record at ten minutes probably reflects inadequate ventilation. Observe pressure on dura at fourteen minutes causing increase in pressure.

thesia which led to increases in spinal fluid pressure, some objective evidence might accrue concerning the factors likely to prove most dangerous for the patient. Various combinations of drugs were employed during this study, and several techniques of producing anesthesia were utilized.

METHOD

Twenty-two patients who required operation for different neurosurgical conditions were investigated in the following manner. Before induction of anesthesia, the patient was turned on his side and a plastic

vinyldene catheter introduced about 2 inches cephalad into the lumbar subarachnoid space. This catheter was of such a size that it would pass through a 17 gauge spinal needle and would admit a 23 gauge intravenous needle. After its introduction through the spinal needle, the catheter was taped securely to the skin and the patient turned on his back. The procedure was similar to that performed for continuous spinal analgesia. The free end of the catheter was connected by means of small bore malleable copper tubing to a continuous recording Sanborn electromanometer. This was calibrated in millimeters of water

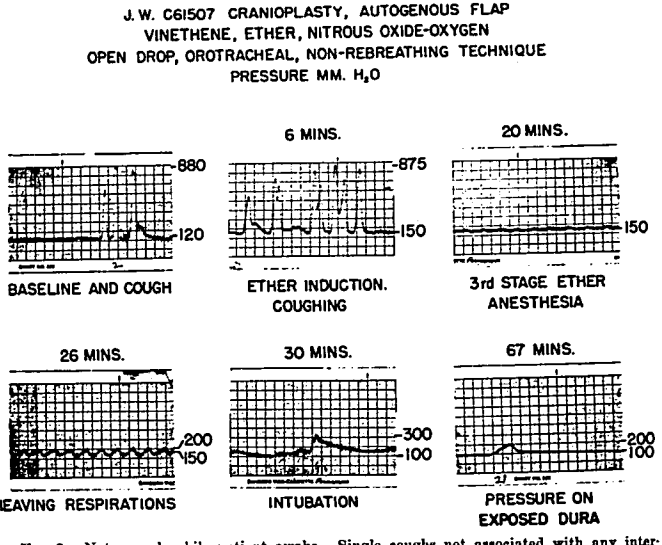


FIG. 3. Note cough while patient awake. Single coughs not associated with any interference with respiration at 6 minutes. Smooth induction and intubation. Effect of deep full respirations seen at 26 minutes. Note pressure on exposed dura at sixty-seven minutes.

to record the variations in cerebrospinal fluid pressure expected. The technique was sensitive enough to record the pulsations of the subarachnoid fluid which occur with each heart beat, and those which are associated with deep respirations (see below). The observations which were made may be discussed under several headings.

ANESTHETIC DRUGS

Several combinations of drugs were used during induction and maintenance of anesthesia. When handled expertly a rapid induction em-

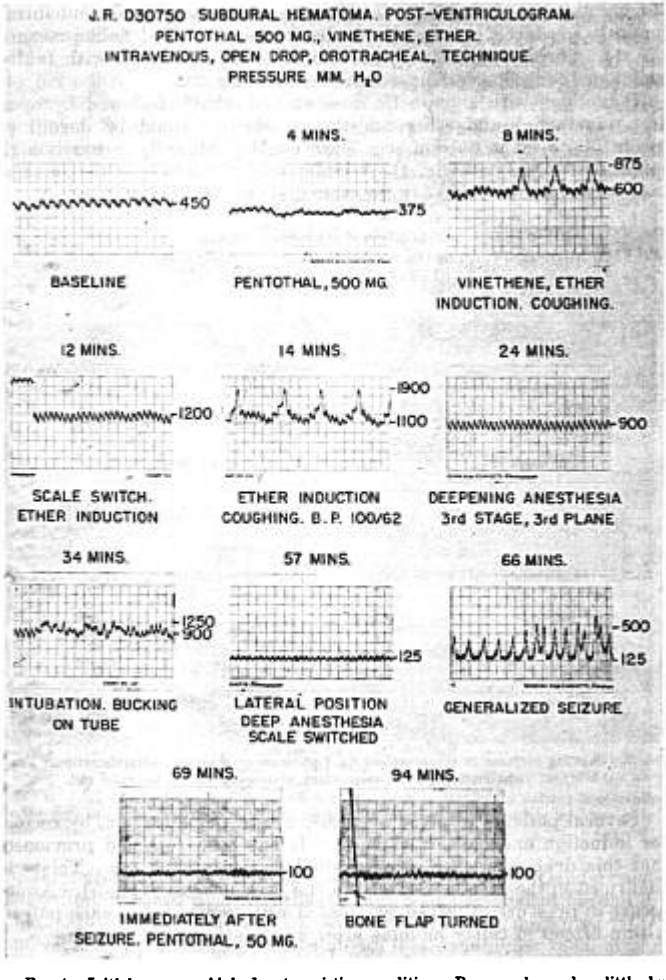


FIG. 4. Initial pressure high due to existing condition. Pressure lowered a little by pentothal. Stormy induction associated with coughing, breath holding, respiratory obstruction and laryngospasm. Intubation with "bucking" increased pressure further. Note pressure response with generalized seizure at sixty-six minutes.

ploying an ultra short-acting barbiturate and a muscle relaxant intravenously produced no significant rise in cerebrospinal fluid pressure (fig. 1). On the other hand, similar drugs administered with faulty techniques caused severe upsets in pressure (fig. 2).

Inductions with a hypnotic dose of pentothal®, followed by open drop vinethene® and ether anesthesia, likewise could be devoid of spectacular rises in tension (fig. 3), or could produce dangerously high spinal fluid pressures (fig. 4). Similar variations in records could be seen when nitrous oxide-oxygen-ether inductions were performed.

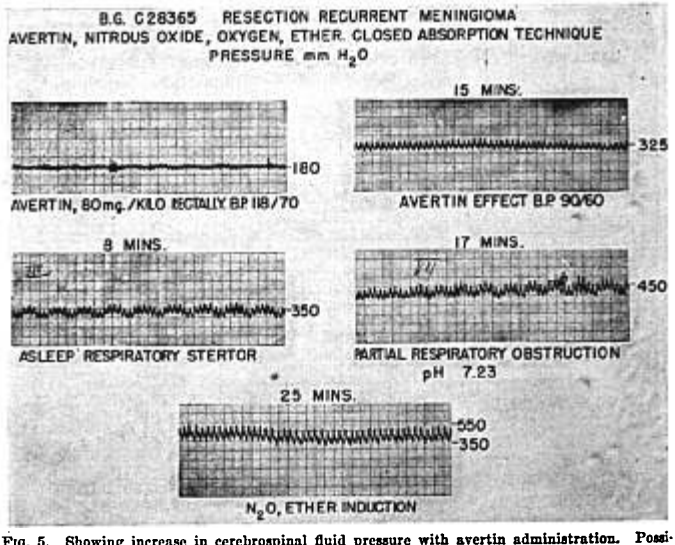


FIG. 5. Showing increase in cerebrospinal fluid pressure with avertin administration. Possibility of complicating partial respiratory obstructon cannot be ruled out.

Several patients received avertin®, 80 mg. per kilogram, by rectum for induction and basal anesthesia. It has been reported previously that this drug increases cerebrospinal fluid pressure (5). This was confirmed in the results obtained in the present study (fig. 5). An increase in pressure of 200 to 300 mm. of water occurred in each patient within fifteen to thirty minutes after administration.

TECHNIQUES OF ADMINISTRATION

Induction: The smoothness of the induction period usually held the key to the degree of increased pressure observed. A rough, stormy

prelude, complicated by coughing, breath holding, straining, laryngospasm or upper respiratory obstruction, invariably produced considerable increases in pressure (figs. 2 and 4). Simple coughing, followed by normal respirations, produced no sustained rise (fig. 3). However, when this was complicated by upper respiratory obstruction and laryngospasm, the rise in pressure continued and as a rule was sustained. Accompanying these difficulties was a variable degree of cyanosis in the patient. The pressure returned to normal only when a free airway had been re-established with adequate ventilation.

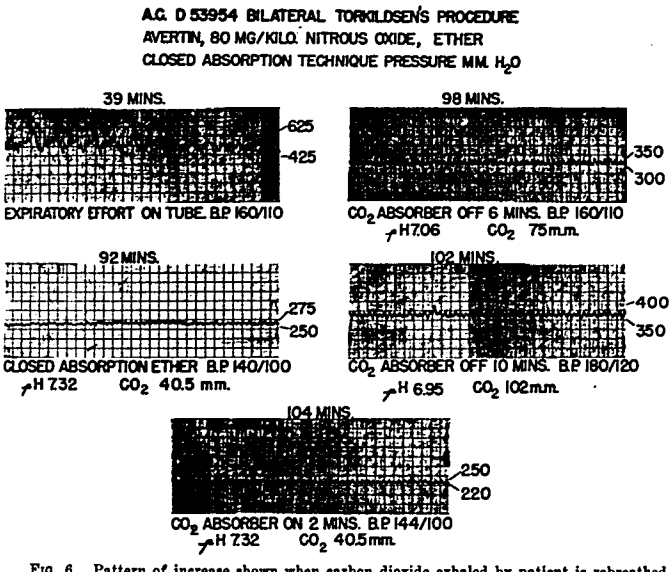


FIG. 6. Pattern of increase shown when carbon dioxide exhaled by patient is rebreathed. Note return to normal blood chemical findings and cerebrospinal fluid pressure level two minutes after absorber turned back into circuit.

Intubation: The importance and, indeed, necessity of endotracheal intubation in neurosurgical patients have been established beyond dispute. However, the technical process of intubation may give rise to undesirable increases in pressure unless it is done under ideal circumstances (figs. 1 and 2). If "bucking" on the tube, bronchospasm or breath holding occurs after intubation, normal respiratory exchange is interrupted and pressure rises occur.

Ventilation: When ultra short-acting barbiturates and muscle re-

laxant drugs are employed, it must be recognized that inadequate ventilation or respiratory exchange results owing to their depressant effect on the respiratory center and the motor muscles of respiration. This will lead to anoxia and retention of carbon dioxide (fig. 1) with increased spinal fluid pressure, unless the respirations of the patient are assisted adequately.

Carbon Dioxide Absorption: After surgical planes of anesthesia had been established, several patients were placed on a closed carbon dioxide absorption system, employing ether and oxygen for anesthesia. For a period of ten to fifteen minutes the carbon dioxide absorber was turned off and the patient was allowed to rebreathe his exhaled carbon dioxide. Cerebrospinal fluid pressure, arterial hydrogen ion concentration and arterial carbon dioxide tensions were measured during this period and correlated, together with the changes which occurred immediately after the absorber was again turned into the circuit (charts 1 and 2). Respirations became deep and full during this period, so one can be sure that the patient was adequately oxygenated. As might be expected from the work of Kety and Schmidt, there was a close relationship between increase in cerebrospinal fluid pressure and the carbon dioxide retention. As the carbon dioxide tension increased in the arterial blood, there was a corresponding increase in the cerebrospinal fluid pressure. The rapid return to pre-existing pressure levels after the elimination of carbon dioxide indicates the ready reversibility of this process (figs. 6 and 7).

Venous Pressure: Increasing the venous pressure within the cranial vault produced at times sharp rises in spinal fluid pressure. In figure 3, for example, simple coughing, both before and after unconsciousness was established, increased the pressure by several hundred millimeters of water. The extent to which an elevated venous pressure contributed to the total effect during episodes of struggling, respiratory obstruction and laryngospasm, is difficult to estimate. Compression of the neck to create a local mechanical increase in venous pressure was reflected by an increased fluid tension (fig. 1).

OTHER OBSERVATIONS

Evidence that the recordings made did reflect the pressure within the cranial cavity can be seen in figure 1, where pressure on the brain substance associated with removing the bone flap increased the pressure recorded. This was demonstrated also when pressure was placed over a pre-existing skull defect in one patient (fig. 2).

In another patient (fig. 4) who had experienced spinal fluid pressures up to 1900 mm. of water during induction, a generalized seizure was recorded. The administration of pentothal intravenously assisted in arresting this and returning the pressure to normal levels.

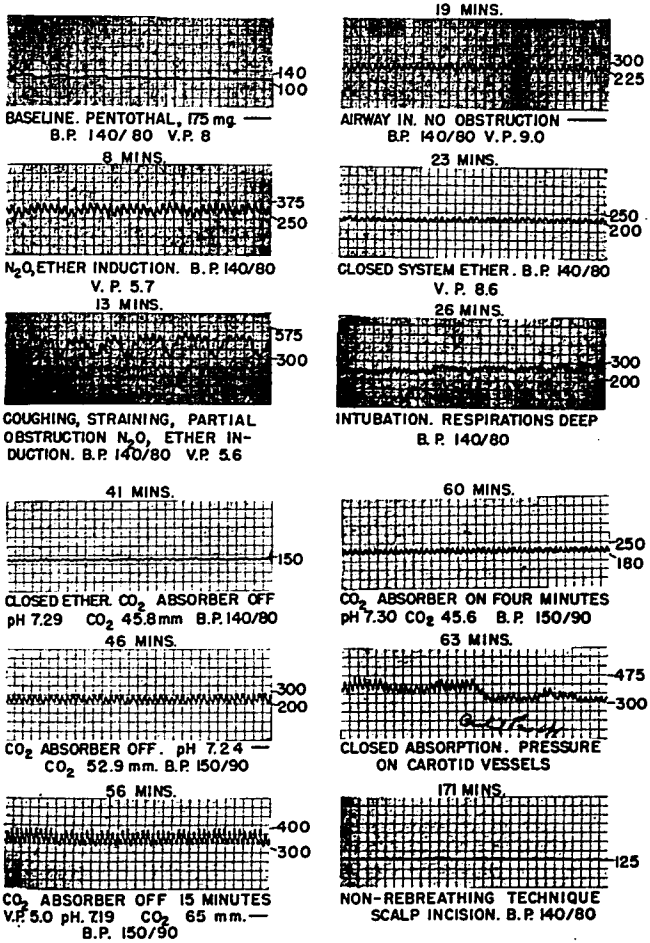


FIG. 7. Induction complicated by straining and partial respiratory obstruction. Intubation smooth. Note small variations of spinal fluid pressure coincident with deep respirations at twenty-six minutes. Typical response to rebreathing of carbon dioxide. Pressure becomes normal as scalp incision is made. V.P. = Venous Pressure.

Deep, full respirations were reflected by small rhythmic variations in the spinal fluid pressure (fig. 7). Normal respirations as a rule produced no noticeable pattern. When controlled respiration was carried out on the apneic patient, only minute increases in pressure were seen (fig. 1).

DISCUSSION

The only drug which appeared consistently to increase cerebrospinal fluid pressure was avertin®. It is possible that in one or 2 patients the increases observed were due in part to the diminished ventilation or partial upper respiratory obstruction coincident with the administration (fig. 5). Anesthesia *could* be induced without significant rises in pressure using the other drugs or combinations enumerated. When increases in spinal fluid pressure occurred, some error in technique was observed to account for them. It is doubtful whether anesthetic drugs *per se*, with the probable exception of avertin, are capable of increasing cerebrospinal fluid pressure.

Over a period of years clinical opinion has averred that ethyl ether will cause cerebral congestion and brain swelling. It is well known that a smooth induction with this drug is one of the most difficult things to master in anesthesia. Probably in the past much of the blame for brain swelling should have been directed at the laryngospasm, respiratory obstruction and anoxia which accompanied the inductions, rather than blamed on the drug itself.

In patients who have high initial spinal fluid pressures, as in figure 4, even the most expert anesthetist may experience a troublesome induction with ether unless extreme caution is taken. Such difficulties may be circumvented by using techniques which do not rely on the integrity of the respiratory center for successful completion, that is, a combination of an ultra short-acting barbiturate and a muscle relaxant. The responsibilities of the anesthetist are many in such instances because the respirations are purposefully depressed to apnea. However, with a versatile administrator the possibilities of increasing cerebrospinal fluid pressure in such a case are probably fewer with the latter method.

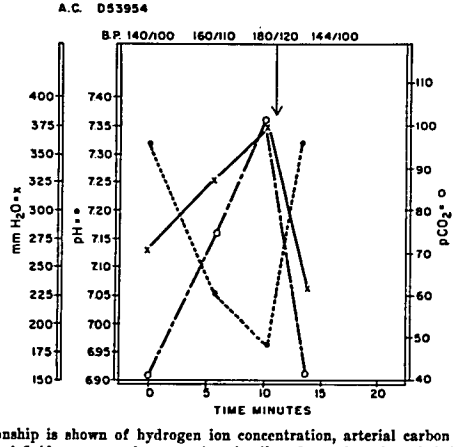
Most of the aberrations in spinal fluid pressure were seen in the induction phase of anesthesia, including endotracheal intubation. As noted above, interference with normal respiratory exchange was the basic fundamental disturbance when the pressure rose. The etiological factors causing this can be overcome only with experience and meticulous attention to detail on the part of the anesthetist. A smooth induction repeated time after time is not entirely a science; it is an art which is acquired only after long diligence and application. Much assistance can be given to the development of this art if a thorough knowledge of respiratory physiology is gained. The maintenance of adequate oxygenation and elimination of carbon dioxide constitute the

keystone to success. The accomplishment of this in anesthesia is much easier said than done.

Pitfalls during intubation may be avoided when adequate technical skill is developed and if the patient is sufficiently anesthetized and relaxed so that reflexes are not initiated by the presence of the foreign body. A rapid, atraumatic intubation does not cause any increase in cerebrospinal fluid pressure. The continued presence of the tube ensures a greater probability of free exchange of gases to and from the blood stream than by any other method.

The maintenance of adequate pulmonary ventilation during operation is of great importance if cerebral congestion is to be avoided.

CHART 1

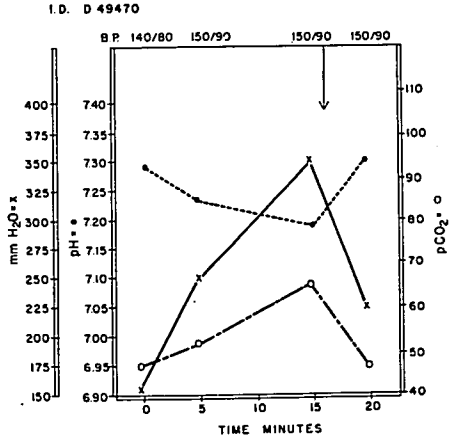


The relationship is shown of hydrogen ion concentration, arterial carbon dioxide tension and cerebrospinal fluid pressure when a patient is allowed to rebreathe exhaled carbon dioxide in a closed circuit system.

Diminished tidal volume, with its associated hypercarbia, will very quickly increase cerebrospinal fluid pressure (fig. 1). Clinically, this situation is likely to develop slowly in a benign manner, particularly if the anesthetist is employing drugs which interfere with normal tidal volume. With any drugs except nitrous oxide, ether and trichloroethylene, it is probably necessary to assist the patients' respirations in order to maintain adequate exchange.

The efficacy of closed carbon dioxide absorption systems is open to question during long operating procedures. The present absorbing mediums for carbon dioxide do not remain 100 per cent efficient over

CHART 2



Carbon dioxide rebreathing in a closed nonabsorption system causes increased subarachnoid fluid pressure while arterial carbon dioxide tension rises.

long periods of time. As a result there may be a gradual accumulation of carbon dioxide in the blood stream, and this may produce sufficient vasodilatation to increase the brain volume. Again the process may be benign and accompanied by few objective signs. Changing the soda lime frequently may prevent the situation from developing. Perhaps the best solution is to employ a nonrebreathing technique in which the anesthetist can be assured that all carbon dioxide is eliminated, providing respiratory ventilation is adequate (6). Certainly it is apparent how rapidly a decrease in carbon dioxide tension is reflected in the fall of cerebrospinal fluid pressure (charts 1 and 2).

SUMMARY

A method of recording variations in cerebrospinal fluid pressure instantaneously and continually is described.

The influence which anesthetic drugs have on cerebrospinal fluid pressure in a group of 22 neurosurgical patients is noted.

The effects of smooth and of complicated inductions of anesthesia on spinal fluid pressure are determined.

The etiological factors leading to anoxia and carbon dioxide retention, and their influence on cerebral congestion and swelling, are discussed.

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