Prior to the introduction of muscle relaxants into anaesthetic practice, apnoea was considered a useful adjunct to abdominal and thoracic surgery in order to provide a steady state in the operating field. Ether apnoea met the requirements of respiration before the demand, by increasing the dosage of ether and lowering the volumes per cent of the carbon dioxide in the blood. It was readily reversed in 15 to 30 seconds (Guedel and Treweck, 1934). Waters (1936) developed the concept of controlled respiration in which he suggested that by lowering the Pco₂ below the threshold sensitivity, the respiratory centre would fail to respond with efferent stimuli; he warned against the dangers of hyperventilation. The possible dangers of hyperventilation when performed under anaesthesia were foreseen and investigated (Seevers et al., 1939). Patients were hyperventilated for periods of 11 to 21 minutes under anaesthesia, lowering the Pco₂ 31 mm below the mean normal and raising the pH 0.25 above the mean normal. No changes were noted clinically in these cases, but in the discussion which followed the authors stated that they were not advocating hyperventilation anaesthesia.

When muscle relaxants were first introduced it was customary to allow spontaneous respiratory effort (Griffiths and Johnson, 1942; Cullen, 1944), and it only was considered necessary to resort to artificial ventilation if overdosage had occurred (Gray and Halton, 1946). The use of apnoic doses of d-tubocurarine then followed and complete control of respiration was advocated by Harroun, Beckert and Fisher (1947). Neff, Mayer and Perales (1947) in their description of this method used the phrase “supplemental ventilation”, thus distinguishing it from Waters' original definition of controlled respiration. They observed no effects from overventilation, but suggested that adverse effects might occur. Gradual recognition that any degree of abdominal relaxation would impair respiratory effort resulted in the conception that in all cases receiving d-tubocurarine, respiration should be aided or assisted (Gray, 1948). The review of Watrous, Davis and Anderson (1950) stressed the hazards of excess carbon dioxide accumulation under anaesthesia, and advocated the use of control or assistance to respiration with all forms of inhalation anaesthesia. They felt that the apnoic technique utilizing d-tubocurarine was safe though, to some extent, it implied the use of hyperventilation, and suggested that apacnia might make some contribution to the degree of relaxation and the depth of anaesthesia. This suggestion has been confirmed by Dundee (1952), and also by Gray and Rees (1952) who showed that assisted respiration necessitated probably more both of relaxant and narcotic drugs. They advocated that the anaesthetist should control the respiration routinely for these reasons.

Clinical reports would seem to indicate that hyperventilation during anaesthesia has no adverse effect either during or after the operation (Stephen et al., 1958). Walley (1959) records that of patients treated for prolonged periods with intermittent positive pressure respiration, with Pco₂ levels within the range 20 to 30 mm Hg, only one developed tetany. It has also been shown that intermittent positive pressure respiration, if properly performed, has no effect on the cardiac function.
CENTRAL NERVOUS SYSTEM EFFECTS OF HYPERVENTILATION

The development of a technique employing deliberate hyperventilation as a means of reducing the requirement of anaesthetic or muscle relaxant was a natural sequela. Its basis rests upon the clinical impression that it is harmless, and upon the electroencephalogram tracings recorded in the alkalotic state. The wave patterns resemble those produced by halothane or ether anaesthesia, and it is suggested that they are due to lack of carbon dioxide (Geddes and Gray, 1959). However, the e.e.g. is open to widely differing interpretations (Watrous, Davis and Anderson, 1950). Hyperventilation with the development of slow waves parallels unconsciousness (Engel, Ferris and Logan, 1947). Cerebral hypoxia results in the appearance of slow waves in the e.e.g. Hyperventilation with 100 per cent oxygen, however, causes a 50 per cent decrease in the incidence of slow waves (Holmberg, 1953). Others have related the slow waves to cerebral anoxia and lack of glucose consequent upon cerebral vasoconstriction produced by the hyperventilation (Davis and Wallace, 1942). There is no doubt that the electroencephalogram tracings show similar patterns with anoxia and hyperventilation (Lennox and Behnke, 1936).

It has been suggested that the deliberate use of hyperventilation under anaesthesia is by no means innocuous (Hewer, 1959). Lee (1960) also expresses these doubts but states that although the method may be doing some harm, clinically it shows no ill effects.

Cerebral blood flow is reduced by hypocapnia (Wolff and Lennox, 1930) due to cerebral vasoconstriction (Lambertsen et al., 1953) and, although the cerebral oxygen consumption per 100 g of brain is not reduced, the brain is working at a low mean oxygen tension (Kety and Schmidt, 1946). The normal compensatory cerebral blood vessel mechanisms are altered under anaesthesia (Schieve and Wilson, 1953), and the tone of the cerebral vessels is reduced by thiopentone (Schmidt, 1950). It would seem then that the results of cerebral circulation studies should not necessarily be interpreted as applying to patients under anaesthesia.

The other factor to be studied is the Bohr effect (Bohr, Hasselbalch and Keogh, 1904) as a cause of tissue anoxia. In the presence of hypocapnia the resulting alkalosis has been shown by Barcroft (1928) to increase the uptake of oxygen by the blood at any given oxygen tension. This feature of hyperventilation under anaesthesia was first discussed by Carryer (1947) who stressed the theoretical possibility that hyperventilation would reduce the tissue uptake of oxygen by the brain. Barach and his colleagues (1947), in their review of the physiology of pressure breathing concluded that the Bohr effect was disadvantageous at saturations in excess of 93 per cent and that tests of cerebral function indicated that all degrees of hyperventilation in normal atmospheres were detrimental to cerebral function. Wayne (1958) found that clinical differentiation between the effects of hyperventilation and anoxia upon cerebral function was not possible. In his demonstration of the analgesic effects of hyperventilation Clutton-Brock (1957) attributes the mechanism of its production to cerebral anoxia, and the reversal of the effect by amyl nitrite or 100 per cent oxygen inhalation would appear to confirm the hypothesis. It is suggested, however, that this explanation is not necessarily correct, for Robinson and Gray (1961) were unable to obtain reversal of analgesia with 100 per cent oxygen. Further, the initial decrease of analgesia obtained by the inhalation of amyl nitrite can also be achieved with ammonia. They ascribe this temporary reversal to an arousal response caused by the irritant vapour. The problem of the effect of alkalosis upon tissue uptake of oxygen was studied by Papadopoulos and Keats (1959). In this study they found that with hyperventilation under anaesthesia there was a rise in the fixed acids of the blood but considered this effect to be due to tissue compensatory mechanisms, rather than to tissue anoxia.

Animal work by Sugioka and Davis (1960) utilizing the oxygen electrode shows that dogs who are hyperventilated for 5 to 10 minutes demonstrate a fall in cortical tissue Po2, which parallels the fall in alveolar carbon dioxide, and which is reversed by the addition of carbon dioxide to the ventilating gases. A similar study by Adams (personal communication, 1960) tended to confirm this work in human subjects subjected
to hyperventilation under anaesthesia but in this instance the response was not so constant. Malette and Eiseman (1958) also concluded that cerebral anoxia resulted from hyperventilation after they had demonstrated a rise in lactic acid content of the dog cerebral cortex following hyperventilation with 100 per cent oxygen, the pH rising to 7.8.

Thus the effects of hyperventilation during clinical anaesthesia, with the concomitant hypocapnia, would appear to need further investigation. Many of the measurements have been made on conscious subjects, or upon animals, and none solves the problem of whether transient or permanent damage to the central nervous system has resulted. The method used by Nilsson (1953) in his investigation of cerebral damage following anaesthesia using controlled hypotension, suggested to the authors a possible means of determining the effects of hyperventilation under anaesthesia. This method of using the critical flicker fusion value with a tolerance test of hexobarbitone was developed by Berg (1949) to demonstrate latent neurological damage. The effects of anoxia on the electroencephalogram parallel the changes in critical flicker fusion value, and those resistant to anoxia show no change in either investigation (Gellhorn and Hailman, 1943). It is contended by Berg, Nilsson and Vinnars (1957) that the critical flicker fusion value is a more sensitive index of cerebral function than the electroencephalogram. The variations in the critical flicker fusion value with anoxia have been shown by Seitz (1940) to be a cortical rather than a retinal effect. The use of a tolerance test with critical flicker fusion assessment is well recognized, having been used to detect latent cardiovascular disease (Krasno and Ivy, 1950; Enzer, Simonson and Blankstein, 1942) and the anoxic effects of respiratory disease (Enzer, Simonson and Evans, 1945). Thus, the method of assessing whether cerebral damage due to anoxia has occurred would seem to be particularly well suited for the investigation of the cerebral effects of hyperventilation under anaesthesia.

METHOD

The instrument used for the determination of the critical flicker fusion value was a commercial Strobotac,* modified for clinical use. The results obtained have been compared with those obtained by the rotating disc method and found to be a highly reliable measure, though their ordering was not the same (McNemar, 1951). Berg (1949), who used a variation of light intensity for his determination of flicker fusion values, found that the results obtained by his method were comparable with those using a rotating disc.

To decrease the concomitant noise of the flash of the Strobotron* bulb it was enclosed in a double-walled asbestos box, being connected to the lamp socket by a wand. A further lead was then taken from the Strobolux* socket to a small frosted neon bulb which acted as the test light source, and which provided a light of constant intensity. The light carrier was a commercial adjustable light bracket, attached to which was a 12-inch (30.5 cm) cone. The neon bulb was screened to provide a test light source with a diameter of 0.42 inch (1.07 cm); this resulted in a constant visual angle of 1 degree being presented to the patient. Thus cone vision was maintained throughout the test and obviated the necessity of providing adaptation at each test (Granit, 1936) and the use of an artificial pupil (McNemar, 1951). In this manner, the critical flicker fusion value could then be obtained at the patient's bedside. The daily conditions of test were kept comparable commensurate with the difficulties of studying a postoperative patient. The use, however, of the hexobarbitone tolerance test on the day of each measurement obviated the necessity of providing adaptation at each test (Granit, 1936) and the use of an artificial pupil (McNemar, 1951). In this manner, the critical flicker fusion value could then be obtained at the patient's bedside. The daily conditions of test were kept comparable commensurate with the difficulties of studying a postoperative patient. The use, however, of the hexobarbitone tolerance test on the day of each measurement obviated the necessity of achieving laboratory-type conditions as each patient provided his own control on each separate day of the investigation. The recommendations of Simonson and Brozek (1952) were followed in the determination of critical flicker fusion value. Exposure was discontinuous, variation in rate of flicker was in steps, and at regular intervals, whilst the duration of exposure was kept constant. The duration of exposure, however, was doubled to 2 seconds in order that the subjects in the older age group could appreciate the occurrence of a change. In a test of the younger age group prior to the start of the investigation no significant variation could be found in the results if a longer

* General Radio Company, West Concord, Massachusetts, U.S.A.
or shorter period of exposure was allowed. Two readings were taken from a low to high rate of flicker on each occasion.

INVESTIGATION

One-tenth of a narcotic dose of hexobarbitone was used as the tolerance test, the dose being assessed on the basis of the weight, age, sex and physical state of the patient in accordance with the table published in *Intravenous Anaesthesia* (Adams, 1944). The c.f.f. value was assessed pre-operatively and the value was taken at 2-minute intervals following the injection of hexobarbitone, being repeated until it was normal or for a period of at least 8 minutes. The test was repeated post-operatively on successive days until no abnormality in the test was detected. Any depression of the c.f.f. value in excess of twice the standard deviation and of a length greater than 6 minutes was considered significant. The standard deviation for the critical flicker fusion value following the injection of hexobarbitone was determined for each patient, the range being 0.75 to 1.36 flashes per second.

According to Berg (1958), the time during which the value is depressed below the normal range, together with the length of the depression and the number of days during which this depression is noted, is an indication of the severity of damage to the central nervous system.

ANAESTHESIA

No constant method of anaesthesia was selected for the investigation. All drugs given were noted, together with any other features relevant to the anaesthetic management, such as the sex and the age of the patient and the presence of concomitant disease. The blood pressure and pulse rate were recorded at 5-minute intervals. Hypotensive or hypoxic incidents during the course of anaesthesia resulted in the case being eliminated from the series. Hypotension was defined as a reduction of the systolic blood pressure by more than 25 per cent of its pre-operative value, and lasting longer than 10 minutes. A hypoxic incident was said to have occurred if obvious cyanosis was noted during the recovery period, or if intubation was difficult and prolonged.

Anaesthesia was given either by the authors or under their direct supervision. The type of respiration during anaesthesia was classified as spontaneous, assisted, controlled or artificial ventilation. The last was performed either manually or by using a ventilator, and in this group an attempt was made to carry out deliberate hyperventilation. The Radford nomogram was used to determine normal ventilation requirements, and the use of the volume control of the respirator or a ventilation meter measured the increase required to produce hyperventilation. Rate of ventilation was kept constant and the increase was made in the tidal volume. When respiration was spontaneous care was taken to avoid rebreathing. The utilization of subapnoic doses of d-tubocurarine with subsequent identification of respiratory effort, made it possible to achieve controlled respiration utilizing d-tubocurarine.

The duration and depth of anaesthesia in the series is indicated in tables I and II.

<table>
<thead>
<tr>
<th>Type of respiration</th>
<th>Less than 120 min</th>
<th>More than 120 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cases</td>
<td>Range (min)</td>
<td>No. of cases</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>11</td>
<td>60–105</td>
</tr>
<tr>
<td>Assisted</td>
<td>5</td>
<td>45–115</td>
</tr>
<tr>
<td>Controlled</td>
<td>11</td>
<td>45–85</td>
</tr>
<tr>
<td>Hyper-ventilated</td>
<td>19</td>
<td>60–105</td>
</tr>
</tbody>
</table>

CARBON DIOXIDE AND OXYGEN STUDIES

The oxygen content of the inspired gases was monitored by a Beckman portable oxygen analyzer. The efficiency of ventilation and particularly hyperventilation was assessed by one of the following methods or by a combination of them: (i) rebreathed gas samples, (ii) volumes per cent of arterial carbon dioxide, (iii) arterial blood pH.

The rebreathed gas sample gives a reliable measurement of Pco₃ of mixed venous blood, even in the presence of cardiovascular or a respiratory disease, there being a constant difference from arterial blood of 6 mm Hg carbon dioxide tension (Hackney, Sears, and Collier, 1958). The method of collection was that described by Campbell and Howell (1960), who also confirmed the accuracy of the method with a variation of plus or minus 3 mm Hg Pco₃. The validity of the method when
used with artificial ventilation has been confirmed, the standard deviation for individuals in this case being 3.8 mm Hg carbon dioxide tension (Cooper and Smith, 1961). The collected sample was analyzed on a Hayes gas analyzer using saturated sodium hydroxide to avoid absorption of nitrous oxide (Prime, 1950). On each change of the absorbing solution its accuracy was checked against gas samples of known carbon dioxide concentration. Arterial blood carbon dioxide content was determined by the Van Slyke manometric blood gas analyzer. The arterial blood pH was determined by a Cambridge pH meter, the sample being collected from the brachial artery.

The average pre-operative arterial Pco₂ was 39.6 mm Hg as assessed by the rebreathed gas method, all patients had been premedicated with hyoscine alone. The average arterial Pco₂ in cases with a positive result was 22.9 mm Hg, the average carbon dioxide content was 31.5 volumes per cent and the mean pH was 7.7.

**RESULTS**

Of 61 cases investigated the results in 10 were rejected as being unsuitable. Of the remaining 51 patients (table III) artificial ventilation was carried out in 24 and an attempt was made to achieve hyperventilation. This was actually achieved in

### Table II

<table>
<thead>
<tr>
<th>Induction</th>
<th>Control</th>
<th>Artificial ventilation</th>
<th>Positive post-op. c.f.f. test</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cases</td>
<td>Range of dose (mg)</td>
<td>No. of cases</td>
<td>Range of dose (mg)</td>
</tr>
<tr>
<td>Thiamylal</td>
<td>9 (-)</td>
<td>150-500</td>
<td>14 (9)</td>
</tr>
<tr>
<td>Gaseous</td>
<td>18 (3)</td>
<td>-</td>
<td>10 (9)</td>
</tr>
<tr>
<td>Relaxant</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d-Tubocurarine</td>
<td>14 (2)</td>
<td>5-40</td>
<td>19 (13)</td>
</tr>
<tr>
<td>Suxamethonium</td>
<td>6 (-)</td>
<td>40-200</td>
<td>5 (5)</td>
</tr>
<tr>
<td>No relaxant</td>
<td>7 (1)</td>
<td>-</td>
<td>Nil</td>
</tr>
<tr>
<td>Supplemental agents</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhalation</td>
<td>20 (2)</td>
<td>-</td>
<td>15 (10)</td>
</tr>
<tr>
<td>(ether, cyclopropane, halothane)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pethidine</td>
<td>4 (-)</td>
<td>30-75</td>
<td>9 (5)</td>
</tr>
<tr>
<td>None</td>
<td>4 (1)</td>
<td>-</td>
<td>7 (7)</td>
</tr>
</tbody>
</table>

*Note: Figures in brackets denote number of cases in which hyperventilation was confirmed.*
18 patients. Twenty-seven patients were included in the control group. Respiration was classified as “spontaneous” in 4, “assisted” in 8, and “controlled” in 15.

Of the 24 patients artificially ventilated, 18 were proved to have been subjected to hyperventilation for periods varying from 60 to 265 minutes and 14 of these showed evidence of cerebral damage as judged by the response to the c.f.f. test.

In the non-hyperventilated group 3 patients showed a positive response to the c.f.f. test and in each case hyperventilation had in fact been performed. One of these patients was from the group which had been classified as “assisted” and two were from the “controlled” respiration group.

The average duration of depression in these 17 patients was 1.5 days. In 10 cases the response was positive for 1 day only; in 6 cases positive for 2 days; and in 1 case it was positive for 3 days. The average maximum depth of depression from normal was 3.9 flashes per second, the range being 1.7 to 9.1 flashes per second. The average length of depression was 10 minutes, the range being 6 to 18 minutes. Figure 1 illustrates the results in a positive case. It can be seen that critical flicker fusion value did not fall after the injection of hexobarbitone on the day before operation. The length of time taken for recovery after the injection of hexobarbitone was greatest on the first postoperative day, reducing gradually until no fall was recorded on the third postoperative day.

It illustrates the wide scatter of flicker fusion values obtained on different days, and thus the value of the hexobarbitone injection as a control measure. The rise in the value obtained after the injection of hexobarbitone on the third postoperative day is within the limits set by con-

![Graph](https://academic.oup.com/jb/article-abstract/34/5/296/263435)
Considering only those changes in excess of twice the standard deviation as significant; the carbon dioxide estimations confirm that hyperventilation had been performed throughout the period of anaesthesia.

Thus the degree and duration of the depression of the hexobarbitone critical flicker fusion test may be considered as slight, for the majority of cases returned to normal within 1 day. Nevertheless, these results show that some slight degree of cerebral damage can occur with hyperventilation.

Thus of 21 patients in whom hyperventilation had actually been carried out the c.f.f. value was depressed in 17. Six of the 24 patients in whom artificial ventilation had been carried out had not been hyperventilated as shown on gas analysis.

Of the 4 patients in whom hyperventilation was confirmed, and in whom the postoperative c.f.f. test showed a negative result, 2 had a positive pre-operative hexobarbitone critical flicker fusion result which was due to concussion. This normal result may possibly be considered to be due to the beneficial effects of hyperventilation in the reduction of cerebral oedema, a normally acceptable practice in neurosurgical operations (Lundberg, Kjallquist and Bien, 1959).

The remaining 2 patients were hyperventilated for periods of 60 and 90 minutes respectively and showed no abnormality in the postoperative flicker fusion results. The anaesthetic agent in each case was nitrous oxide only and each received d-tubocurarine 30 mg. The patient hyperventilated for 60 minutes was noted pre-operatively to have atheroma and emphysema; this might be expected to result in an abnormal pulmonary blood flow/ventilation ratio, with consequent inadequate removal of carbon dioxide. The other patient underwent a laparotomy for abdominal carcinomatosis (probably pancreatic in origin) and showed no such abnormality; this remains inexplicable. Some authors consider that the period required to produce alkalosis would be in excess of 90 minutes (Rollason and Parkes, 1957; Pask, 1958), and if the ratio between alveolar Pco₂ and pulmonary ventilation is not always predictable (Sunahara et al., 1957), then this may account for the normal result obtained in this latter case.

The efficacy of the method as a means of estimating cerebral damage was demonstrated in 2 of the 10 rejected cases.

**CASE 1.** This patient had an anoxic episode of 30 minutes duration following surgery. The hexobarbitone critical flicker fusion test was still positive on the fourth postoperative day but was not followed further due to the necessity of modifying the instrument at this time.

**CASE 5.** A positive result was still present on the tenth postoperative day. The anoxic episode having been due to haemorrhage which required 45 pints (25.6 l.) of blood for its replacement, subsequent operation terminated the investigation.

In both these cases the depression of the critical flicker fusion value following the injection of hexobarbitone was gross and prolonged, being much in excess of that seen in the cases who showed a positive result in the investigation of hyperventilation.

**DISCUSSION**

It would appear from these cases that hyperventilation under anaesthesia can cause demonstrable cerebral damage. The changes are minor in degree and duration but are similar to those which have been shown to be associated with cerebral damage and the consequences of cerebral hypoxia, both in this series and in other investigations (Berg, 1958). It is worth noting that in the control series studied by Berg, Nilsson and Vinnars (1957) during their investigation of cerebral damage associated with the use of controlled hypotension under anaesthesia, three cases were found postoperatively to have a positive hexobarbitone critical flicker fusion result lasting for two or three days. These patients were anaesthetised with nitrous oxide and oxygen, d-tubocurarine and pethidine; it is possible that they were inadvertently hyperventilated.

It is difficult to discount the numerous documented reports of cases of hyperventilation who appear to do well clinically, and it may be that the overall benefits of reduction in the amount of anaesthesia and relaxant outweigh the slight cerebral damage which may occur. However, it is not essential to hyperventilate these cases to obtain this benefit, as control of the respiration with a near normal Pco₂ will achieve this result (Gray and Rees, 1952). Intermittent positive pressure respiration with 5 to 15 l./min is not sufficient to produce alkalosis (Motley et al., 1948).
unless efforts are deliberately made to hyperventilate the patient, and thus even artificial ventilation without resort to any means of monitoring the Pco₂ will not result in the production of a lowered Pco₂ with consequent cerebral vasoconstriction.

In this series the age of the patient offered no protection from the apparent harmful effects of hyperventilation; cerebral atheroma did not seem to mask the effects of a lowered Pco₂, nor did the recovery period appear shorter in patients in the lower age groups, as might be assumed to be the case from cerebral circulation studies.

The fact that these results are similar to those obtained with patients in other series with permanent cerebral damage, does not imply that the effect of hyperventilation during anaesthesia is permanent, but the results also can be correlated with those obtained from cases of concussion, and although the positive critical flicker fusion result is transient, concussion is considered to produce permanent cerebral damage although of minor degree.

These findings appear to confirm those which have been obtained in animal experiments and to confirm the theoretical objections to hyperventilation as an adjunct to anaesthesia. The most satisfactory method of obtaining adequate respiratory exchange in the presence of relaxant drugs would seem, then, to be to return to the true concept of controlled respiration as originally defined by Waters (1936).

SUMMARY
The effect of hyperventilation during anaesthesia upon the cerebral cortex, was assessed by the critical flicker fusion test. Latent cerebral damage was detected by the use of hexobarbitone as a tolerance test.

Fifty-one cases were satisfactorily completed; there were 27 patients in the "control" group and in 24 patients an attempt was made to hyperventilate throughout the duration of anaesthesia.

It was found that in 21 patients hyperventilation had been satisfactorily performed and that in 17 of these a positive postoperative hexobarbitone c.f.f. test was obtained.

Comparison of results obtained in the hyperventilated series with those found in patients who had definite hypoxic incidents show that the degree of cerebral damage is slight following hyperventilation.

REFERENCES
SOMMAIRE
L’effet sur le cortex cérébral de l’hyperventilation pendant l’anesthésie fut recherché par le “flicker fusion test”. Une altération cérébrale latente fut détectée par l’emploi de l’hexobarbital comme test de tolérance.

51 cas furent complétés d’une manière satisfaisante; 27 malades étaient dans la groupe “de contrôle” et chez 24 malades on a essayé d’hyperventiler pendant toute la durée de l’anesthésie.

On a constaté que chez 21 malades l’hyperventilation avait été effectuée d’une manière satisfaisante et que chez 17 de ceux-ci, le c.f.f. test post-opératoire à l’hexobarbital fut positif.

La comparaison des résultats obtenus dans la série d’hyperventilation avec ceux trouvés chez les malades qui eurent des incidents manifestes d’hypoxie, montre que le degré d’atteinte cérébrale est minime à la suite de l’hyperventilation.

ZUSAMMENFASSUNG
Der Hyperventilationseffekt auf die Großhirnrinde während der Narkose wurde mit dem kritischen Flackerfusionstest geprüft. Durch Anwendung von Hexobarbital als Toleranztest liess sich ein latenter Hirnschaden feststellen.

51 Fälle wurden in zufriedenstellender Weise abgeschlossen; 27 Patienten gehörten der “Kontrollgruppe” an, und bei 24 Patienten wurde eine Hyperventilation während der ganzen Dauer der Narkose angestrebt.

Es fand sich, dass bei 21 Patienten die Hyperventilation richtig durchgeführt worden war und dass in 17 von diesen ein positiver postoperativer Hexobarbital-kFF-Test erzielt wurde.

Ein Vergleich der Ergebnisse aus der Versuchsreihe mit Hyperventilation mit denen von Patienten, die deutliche hypoxische Zustände hatten, ergab, dass der Grad der Hirnschädigung nach Hyperventilation leicht ist.

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UNIVERSITY OF CAMBRIDGE POSTGRADUATE MEDICAL SCHOOL
SYMPOSIUM FOR ANAESTHETISTS

to be held in the Postgraduate Lecture Room,
at Addenbrooke’s Hospital, Cambridge, on SATURDAY, JUNE 23, 1962

SOME EMERGENCIES

Morning session
10.30 EMERGENCIES IN INFANCY AND CHILDHOOD

11.30 COFFEE

11.45 THE PHYSIOLOGY AND MANAGEMENT OF DROWNING

Afternoon session
2.30 ACUTE RESPIRATORY FAILURE

3.30 THE USE AND ABUSE OF DRUGS IN EMERGENCY SITUATIONS

4.30 TEA

These meetings are open to anaesthetists of all grades and any members of other Departments who are interested. Course fee £1 1s.; charge for buffet lunch at the Hospital 5s.

Those intending to attend are asked to notify the Secretary of the Medical School, Tennis Court Road, Cambridge, before June 16, 1962, if possible, stating whether lunch is required and enclosing the appropriate fee.