INTRACRANIAL PRESSURE DURING RECOVERY FROM NITROUS OXIDE AND HALOTHANE ANAESTHESIA IN NEUROSURGICAL PATIENTS

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SUMMARY
Seven patients with intracranial disorders were studied during recovery from anaesthesia with nitrous oxide and halothane. Arterial, intracranial, and central venous pressure, and arterial carbon dioxide tension were measured and compared with the patient's clinical state. No patient had evidence of increased brain volume when the dura was closed. All had been hyperventilated during the surgical procedure. Cessation of hyperventilation and the continued administration of anaesthetics was followed by a moderate increase in intracranial pressure and a reduction in cerebral perfusion pressure, but critically low values were not seen. Spontaneous respiration returned when the PaCO_2 was in the range 33–51 mm Hg. When spontaneous respiration was judged to be normal, anaesthesia was interrupted and the endotracheal tube was removed. In the following minutes, until the patients were awake, the intracranial pressure decreased to normal or near normal values, with minimal change in PaCO_2. In these seven patients in whom there were no signs of brain swelling, the skull was closed, the patients were allowed to resume spontaneous respiration, and anaesthesia was terminated without major changes in intracranial pressure or cerebral perfusion pressure. However, hyperventilation is advocated after operation in patients with marked brain swelling.

All inhalation anaesthetic agents increase intracranial pressure (McDowall, Barker and Jennett, 1966; Fitch et al., 1969; Jennett et al., 1969). This increase is the result of cerebral vasodilatation and the consequent increase of cerebral blood volume. The intracranial hypertension is most pronounced in patients with intracranial space-occupying lesions, as these patients have less reserve space. Minor changes in blood volume may, therefore, cause major changes in intracranial pressure.

Another factor of major importance in anaesthesia for intracranial procedures is arterial carbon dioxide tension. Hypercapnia adds to the vasodilating effect of anaesthetic agents, while hypocapnia effectively counteracts it.

There are many published reports of intracranial pressure changes during induction of anaesthesia, but there has been little previous interest in the period following surgery. At this time, when artificial hyperventilation is interrupted and spontaneous breathing begins, arterial carbon dioxide tension must increase, probably leading to some degree of hypercapnia.

If this occurs, vasodilatation caused by anaesthetic agents will be potentiated and intracranial hypertension will result after operation.

Therefore we found it worthwhile to study this period by correlating the clinical state with arterial carbon dioxide tension and intracranial pressure.

MATERIAL AND METHODS
Seven patients were studied during recovery from general anaesthesia. The diagnosis, the intracranial pressure during 24 hr after anaesthesia, and the treatment directed against intracranial hypertension are presented in table I.

Intracranial pressure, arterial pressure and central venous pressure were recorded continuously via catheters in a lateral ventricle, in the radial artery of the non-dominant hand, and in a subclavian vein (Jørgensen and Henriksen, 1973). All catheters were placed after induction of anaesthesia. Pressures were referred to forehead level. Cerebral perfusion pressure (CPP) was calculated as the difference between mean arterial pressure (MAP) and mean intracranial pressure (ICP). Blood samples were drawn through the arterial catheter for determination of Po_2, Pco_2 and pH by standard Radiometer equipment.

Five patients underwent craniotomy for removal of
TABLE I. Details of the patients studied.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Intracranial pressure (mm Hg)</th>
<th>Plateau waves</th>
<th>Clinical state</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>38</td>
<td>hydrocephalus postencephalitis</td>
<td>20-40</td>
<td>—</td>
<td>alert/papilloedema</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>21</td>
<td>cerebellar angioma</td>
<td>10-35</td>
<td>—</td>
<td>alert/papilloedema</td>
<td>dexamethasone</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>57</td>
<td>cerebral metastasis</td>
<td>15-25</td>
<td>—</td>
<td>alert/papilloedema</td>
<td>dexamethasone</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>57</td>
<td>acoustic neuroma</td>
<td>5-10</td>
<td>—</td>
<td>alert</td>
<td>dexamethasone</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>40</td>
<td>acoustic neuroma</td>
<td>5-10</td>
<td>—</td>
<td>alert</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>55</td>
<td>cerebral atrophy</td>
<td>5-10</td>
<td>—</td>
<td>alert</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>20</td>
<td>chiasmatic haematoma</td>
<td>0-20</td>
<td>—</td>
<td>somnolent</td>
<td>dexamethasone</td>
</tr>
</tbody>
</table>

tumours or haematomas, and two patients were studied after ventriculography. Anaesthesia had been induced with barbiturate-relaxant, and, after endotracheal intubation, was maintained with 50% nitrous oxide in oxygen plus 0.5–1.0% halothane. Controlled hyperventilation by means of an Engström ventilator was used throughout the operative procedures to counteract the vasodilator effects of anaesthetic agents and to minimize brain swelling.

At the end of the procedure the patients were placed in bed in a supine position. Hyperventilation with 50% nitrous oxide in oxygen and 0.5–1.0% halothane was continued for at least 10 min in order to maintain PaCO₂ at an unchanged value. Then a set of control values of MAP, ICP and CVP were measured and an arterial blood sample was drawn for gas analysis (first measurement).

Ventilation was then reduced stepwise until the beginning of spontaneous respiratory movements. The next values were obtained at this time (second measurement).

The ventilator was then disconnected, and respiration was assisted manually with an anaesthesia bag until spontaneous ventilation was judged clinically to be adequate, whereupon the third measurement was made.

The endotracheal tube was now removed and halothane and nitrous oxide administration was discontinued. Oxygen was given by mask until the patient responded to verbal stimulation, and the last set of pressure and gas values was obtained (fourth measurement). This sequence was used to avoid coughing and high intracranial pressure peaks during removal of the tube.

The data are shown in figures 1–3.

RESULTS

Control measurements during hyperventilation (first measurement), showed PaCO₂ values between 20 and 32 mm Hg (mean: 24.1 mm Hg). Perfusion pressures were between 48 and 86 mm Hg (mean: 64.9 mm Hg). Only one patient (no. 1), following a ventriculogram, showed an elevated ICP (15 mm Hg) during hypocapnia.

After hyperventilation was discontinued, PaCO₂ increased until spontaneous respiration started, at Pco₂ values in the range 33–51 mm Hg (mean: 40.3 mm Hg) (second measurement). At this time ICP had increased to 2–39 mm Hg (mean: 17.9 mm Hg); thus CPP was reduced to 44–82 mm Hg (mean: 58.2 mm Hg).

Four to 28 min (mean: 19 min) passed before spontaneous respiratory movements were seen, and a further period of 3–8 min (mean: 6 min) until respiration was judged clinically to be sufficient. The appearance of adequate spontaneous respiration was associated with only minimal changes in PaCO₂, 35–55 mm Hg (mean: 42.0 mm Hg) and ICP, 11–39 mm Hg (mean: 21.1 mm Hg) (third measurement). CPP decreased slightly in two patients (nos. 2 and 4), but increased in the other five patients because of an increase in arterial pressure.

When the patients were awake (fourth measurement) PaCO₂ was 29–53 mm Hg (mean: 39.0 mm Hg). In
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**MEAN VALUES**

<table>
<thead>
<tr>
<th>ICP</th>
<th>MAP</th>
<th>CPP</th>
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</thead>
<tbody>
<tr>
<td>69.0</td>
<td>76.1</td>
<td>86.3</td>
</tr>
<tr>
<td>17.9</td>
<td>21.1</td>
<td>21.1</td>
</tr>
<tr>
<td>91.1</td>
<td>58.3</td>
<td>65.0</td>
</tr>
<tr>
<td>64.9</td>
<td>40.3</td>
<td>39.0</td>
</tr>
<tr>
<td>25.0</td>
<td>-1.3</td>
<td>-1.3</td>
</tr>
</tbody>
</table>

**Fig. 2.** Cerebral perfusion pressure (CPP) in seven patients during recovery from anaesthesia. (1) 50% nitrous oxide in oxygen and 0.5% halothane and controlled hyperventilation. (2) 50% nitrous oxide in oxygen and 0.5% halothane at onset of spontaneous respiration. (3) 50% nitrous oxide in oxygen and 0.5% halothane, when spontaneous respiration is judged to be adequate. (4) 100% oxygen after removal of the endotracheal tube, when the patients are responding to verbal stimulation.

The autoregulatory mechanisms of cerebral arterioles will keep blood flow constant at perfusion pressures of as little as 40 mm Hg in normal brain (Häggendal et al., 1970; Zwetnow, 1970), whether this reduction is a result of an increase in intracranial pressure or of decrease in arterial pressure. However, in patients with intracranial disease, autoregulation may be impaired locally or globally. Thus blood flow may decrease in these patients when a decrease in perfusion pressure is only moderate.

Several factors may contribute to the reduction in cerebral perfusion pressure during general anaesthesia:

1. An increase in intracranial pressure resulting from an increase in blood volume caused by hypercapnia and anaesthetic agents.
2. A reduction in arterial pressure because of the cardiovascular effects of hypoxia and anaesthetics.
3. An increase in venous pressure as a result of inappropriate positioning of the head with compression of jugular veins.
The influence of these factors upon cerebral vascular dynamics has been examined thoroughly during induction of anaesthesia and before the skull is opened (McDowall, Barker and Jennett, 1966; Fitch et al., 1969; Henriksen and Jørgensen, 1973; Jørgensen and Henriksen, 1973).

It is generally accepted that patients are treated most safely with a barbiturate-relaxant induction (Pierce et al., 1962) followed by endotracheal intubation and the rapid establishing of controlled hyperventilation to reduce the period of hypocapnia and hypoxia. Inhalation anaesthetic agents should not be administered before hypocapnia can counteract their vasodilator effects (Adams et al., 1972; Misfeldt, Jørgensen and Rishøj, 1975).

Until now little attention has been paid to intracranial pressure changes in the period immediately after operation. This may be a result of the general belief that, at this time following the removal of space-occupying lesions, the risks of intracranial hypertension and mass shift are much reduced. However, a tumour may not have been removed radically, and local or diffuse brain swelling may develop or be present because of an increase in blood volume and oedema. Further, in patients who have undergone neuroradiological examinations, the risks of intracranial hypertension and mass shift are still present, since a possible tumour or haematoma has not been removed. Leech, Barker and Fitch (1974) report briefly on the increase in intracranial and systemic pressure in patients brought from the hypocapnic to the normocapnic state, despite the appearance of slackness of the dura when the skull is being closed. However, no information about $P_{\text{aco}_2}$ changes were presented.

What factors influence the intracranial pressure changes during recovery from anaesthesia?

1. Concentrations of anaesthetic agents decrease rapidly, and if there are remaining residual effects from these upon cerebral vascularity, ICP must decrease while anaesthetics are exhaled.
2. After hyperventilation during the operative procedures one must allow $P_{\text{aco}_2}$ to increase before the patient will be able to breathe spontaneously. This will increase ICP.
3. While the concentration of anaesthetic agents decreases and $P_{\text{aco}}$ increases, arterial pressure increases, and thus improves cerebral perfusion pressure.
4. Any oedema or haematoma initiated during operation or during hypoxia by inadequate perfusion pressure will increase ICP and bring about a risk of mass shift and herniation.

In the present study of patients, none of whom had evidence of severe brain swelling at closure of the dura, all of whom had received nitrous oxide and halothane during hypocapnia, and had the endotracheal tube removed after a period of hypoventilation and were then awakened, we found the following:

1. The intracranial pressure increased when hyperventilation was discontinued and $P_{\text{aco}_2}$ increased, but from the onset of spontaneous respiration little further increase was observed.
2. The cerebral perfusion pressure did not decrease to less than the critical 40 mm Hg value, because the arterial pressure increased simultaneously, both during hypoventilation and during lightening of anaesthesia.
3. The intracranial pressure decreased in all patients when the administration of anaesthetics was interrupted, despite nearly unchanged $P_{\text{aco}_2}$ values.
4. In most patients spontaneous respiration returned at normocapnia and before severe hypercapnia.
occurred. This indicates that the respiratory depression from nitrous oxide and halothane is without significance during this period.

It is concluded that patients without evidence of marked brain swelling may be allowed safely to resume spontaneous respiration, and have the endotracheal tube removed and be awakened from nitrous oxide and halothane anaesthesia. However, even in these patients some degree of intracranial hypertension may occur and a risk of hypercapnia exists. Therefore, if evidence of severe brain swelling is present at the time of closure of a craniotomy, it is likely that hyperventilation is indicated at least until nitrous oxide and halothane have been eliminated and probably for at least the next 24 hr, in order to minimize further brain swelling and to counteract development of secondary hypoxic and ischaemic brain damage.

REFERENCES


PRESION INTRACRANEAL DURANTE LA RECUPERACION DE ANESTESIA CON HALOTANE Y OXIDO NITROSO EN PACIENTES NEUROQUIRURGICOS

SUMARIO

Se estudiaron siete pacientes con perturbaciones intracraneales durante la recuperación de anestesia con halotane y óxido nitroso. Se midieron la presión arterial, intracraneal y el sistema de venas central, así como la tensión arterial provocada por el dióxido de carbono y se compararon con la condición clínica del paciente. Ninguno de los pacientes demostró aumento de volumen del cráneo cuando estaba cerrada la duramáter. Todos habían tenido una gran ventilación durante la operación quirúrgica. Después de la hiperventilación y la administración continua de anestésicos, se produjo un aumento moderado de presión intracraneal y una reducción en la presión cerebral por perfusión, pero no se observaron valores bajos críticos. La respiración espontánea reapareció cuando el Paco₂ se hallaba dentro de los límites de 33-51 mm Hg. Cuando se consideró que la respiración espontánea era normal, se interrumpió la anestesia y se extrajo el tubo endotraqueal. Durante los siguientes minutos, hasta que se despertaron los pacientes, disminuyó la presión intracraneal hasta alcanzar valores normales o próximos, con un cambio mínimo de Paco₂. En estos siete pacientes cuyos cerebros no presentaron señales de hinchazón, el cráneo estaba cerrado, se dejó a los pacientes que reanudasen la respiración espontánea y se interrumpió la anestesia sin cambios importantes en la presión intracraneal o en la presión cerebral por perfusión. Sin embargo se recomienda la hiperventilación después de la operación en pacientes con una hinchazón notable del cerebro.