Retinal haemorrhage as a complication of gas encephalography and gas myelography
Prospective study using oxygen gas with a discussion of pathogenetic mechanisms

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SUMMARY Seventy patients, submitted to oxygen encephalography and oxygen myelography, were examined by retinal funduscopy to establish the incidence of intraocular haemorrhages, a complication reported to be frequent by other investigators. We did not discover any retinal haemorrhages. Pathogenetic factors influencing intracranial or intraocular venous pressure or both are discussed, particularly the effect due to different technical procedures during the examination, and the type of anaesthesia used.

The early studies of the cerebral ventricles were morphological, depending upon visual observation. Leonardo da Vinci, by using wax, made casts of the ventricles (Robertson, 1967). The injection of air into the subarachnoid spaces and ventricular system as a diagnostic aid was proposed by Dandy (1918). Later, Jüngling (1922) introduced oxygen. Lack of homogeneous material due to different patient groups and techniques has made it difficult to compare the many reports of general complications (Masson, 1933; Davidoff and Dyke, 1946; Whittier, 1951; Falk, 1953; Taveras and Wood, 1964; Ruggiero et al., 1969; Bergeron and Rumbaugh, 1971).

Complications in the visual system arising from pneumoencephalography are not well recognised. Masson (1933) reported a case of temporary blindness, and Hoyt and Beeston (1966) one case of retinal haemorrhage. Simon et al. (1973) observed intraocular haemorrhage resulting from pneumo-myelography. Moseley and Pilling (1976) reported retinal haemorrhage after pneumoencephalography in 20% of their patients. These reports stimulated us to examine the retinal fundus in patients submitted to oxygen encephalography (OEG) and oxygen myelography (OMG).

Patients and methods

In the second half of 1976, 70 patients from the Department of Neurology underwent OEG and OMG examinations. The investigations were carried out under local, general, or neuroleptic anaesthesia. Fundus examination using a mydriatic was performed before and within a couple of hours after the procedure.

Filling of the temporal horns of the lateral ventricles was obtained by placing the patient's head in a slightly extended lateral decubitus for a couple of minutes while rocking it gently; one temporal horn filled at a time. In no case was the somersault manoeuvre employed.

The investigation included 21 females and 49 males aged between 12 and 69 years with a mean of 40 years. The referring diagnoses are listed in Tables 1 and 2. Four patients were examined by OMG and 66 patients were submitted to OEG. The clinical status and age of each patient determined the type of anaesthesia as follows: local anaesthesia 56 patients, ketamine anaesthesia six patients, and fentanyl (neuroleptic) anaesthesia eight patients. Atropine and droperidol were used as premedication, and pancuronium bromide for muscle relaxation. Ephedrine was given before OEG but was not used in OMG. The technique was that of fractional OEG, introducing oxygen in small fractions after lumbar puncture. At the same time cerebrospinal fluid (CSF) was removed. Oxygen myelography was undertaken with an additional needle placed suboccipitally to withdraw. In OEG the
Table 1  Diagnoses in 66 cases of oxygen encephalography

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>Number of cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy</td>
<td>21</td>
<td>32</td>
</tr>
<tr>
<td>Intracranial haemorrhage (sequelae)</td>
<td>3</td>
<td>4.5</td>
</tr>
<tr>
<td>Cerebral ischaemic disease</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Cerebral and cerebellar atrophy</td>
<td>18</td>
<td>27</td>
</tr>
<tr>
<td>(Traumatic; Wernicke; aetiology unknown)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurosis and psychosis</td>
<td>16</td>
<td>24</td>
</tr>
<tr>
<td>(Post-traumatic; unspecified)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cephalalgia</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Chronic alcoholic disease</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Intracranial tumours</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Paralysis agitans</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Hereditary ataxias/muscular dystrophy</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Spasmodic torticollis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Cerebellar ataxias/torticollis</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Hydrocephalus/syringomyelia, syringobulbia</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Diabetes insipidus/hypothyroidism</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>81</td>
<td></td>
</tr>
</tbody>
</table>

Table 2  Diagnoses in four cases of oxygen myelography

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syringomyelia</td>
<td>2</td>
</tr>
<tr>
<td>Cervical medullary tumour</td>
<td>1</td>
</tr>
<tr>
<td>Myelopathy (aetiology unknown)</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>4</td>
</tr>
</tbody>
</table>

The average amount of oxygen injected was (62±16) ml, while 37(±11)ml of CSF was removed. In OMG the figures were 58(±23)ml and 26(±15)ml.

Results

We observed no cases of retinal haemorrhage or exudate in our patients.

Concerning general complications of the procedures, most patients complained of headache lasting for 24 to 36 hours, but no major accident, such as syncope or death, occurred.

Discussion

Hoyt and Beeston (1966) and later Simon and colleagues (1973) appear to have been the first to record retinal haemorrhages provoked by pneumoencephalography and pneumomyelography. These haemorrhages were either intra-or preretinal, and were usually asymptomatic (Moseley and Pilling, 1976). However, an affection of the vitreous body with gross visual reduction was reported by Simon et al. (1973).

The haemorrhages seemed to be of venous origin (Hoyt and Beeston, 1966). Several factors may contribute to such bleedings, especially those causing raised intracranial or intraocular venous pressure or both, possibly as a result of special examination procedures. The technique of having the patient's legs higher than the head results in a stasis of cephalic venous vessels (Simon et al., 1973, Moseley and Pilling, 1976). This Trendelenburg position is particularly unfavourable if long-lasting. The somersault manoeuvre to fill the temporal horns with gas is another way of causing increased venous pressure (Moseley and Pilling, 1976) due to compression of the thorax and abdomen by the necessary support (Pearce, 1957). This mechanism is further illustrated by the haemorrhagic retinopathy caused by the Valsalva manoeuvre (Duane, 1973) and by the retinal angiopathy after heavy compression trauma to the chest, in Purtscher's disease (1910). In the present study none of these manoeuvres were undertaken, and this may be why there were no ocular complications.

Transient rise in intracranial pressure may be caused by the injection of air into the ventricular system (Cronquist et al., 1963). This rise may also be observed when using carbon dioxide, nitrous oxide, or ethylene. The use of these gases, however, leads to poorer radiographs, although the residual symptoms are considerably reduced compared with injection of air. Oxygen occupies an intermediate position between these gases and air.

In the present study the method was that of 'fractional pneumoencephalography'—that is, repeated injections of small quantities of oxygen and subtraction of cerebrospinal fluid. In OEG the volume difference between injected oxygen gas and withdrawn CSF was on average 26 (range 14–38) ml. Larger volumes of oxygen were thus used than in comparable investigations by Ruggiero et al. (1969) using air. The amount of oxygen injected into the 54 patients under local anaesthesia was 24 (range 15–33) ml and in the 12 patients under general anaesthesia 33 (range 15–31) ml. More oxygen was injected into the patients under general anaesthesia, probably because, unlike the conscious patients, they did not present adverse reactions such as headache, profuse sweating, nausea, and general discomfort.

Our investigation included only four patients submitted to OMG, and this is too small a number for analysis. The amount of air injected and CSF withdrawn does not influence the incidence of retinal haemorrhages (Simon et al., 1973) or general complications (Ruggiero et al., 1969).

Local anaesthesia does not increase intracranial pressure. Using ophthalmodynamometry, the intraocular pressure has been shown to be affected by...
lumbar spinal puncture in some patients (Finke and Jaenicke, 1970). All inhalation anaesthetics increase the intracranial pressure (McDowall et al., 1966), and mechanical obstacles constitute one of the main causes for this. The effect of compression of the thorax and abdomen has already been discussed. A report by Jeppson and Järpe (1960) revealed an increase in intracranial pressure caused by intubation which necessitated the use of muscle relaxation. The vasodilatation arising from general anaesthesia leads to a rise in cerebral blood volume and a consequent increase in intracranial pressure, for example with fluothane as used by Simon et al. (1973). However, in these respects the anaesthetic agents behave differently. Nitrous dioxide does not change the cerebral blood volume when hypoxia is avoided and there is no free air trapped inside the skull. Saidmann and Eger (1965), however, reported a substantial increase in CSF pressure using nitrous dioxide during pneumoencephalography. This gas, more soluble than nitrogen, is carried to the air-containing ventricles in a greater quantity than the amount of nitrogen removed from the ventricles. Nitrous dioxide is best avoided, unless used both as contrast gas for gas encephalography and as inhalation drug for general anaesthesia in the same patient (Ruggiero, 1974; Elwyn et al., 1976). Moseley and Pilling (1976) hyperventilated their patients to minimise rise in intracranial pressure. Hypocapnia counteracts the effect of vasodilatation while hypercapnia adds to it. In their discussion, they do not mention nitrous dioxide as a cause of intraocular haemorrhage among their patients.

The use of ketamine for general anaesthesia, as suggested by Corrsen (1967), offers two advantages—quick recovery from anaesthesia without respiratory depression, and preservation of pharyngeal reflexes. In our study patients given ketamine were young, since this drug is preferably not given to adults. However, it causes a moderate increase in intracranial pressure by raising the arterial blood pressure and cerebral blood volume (Gardener et al., 1971). Diazepam seems to counteract this effect when given in combination with ketamine (Gran, 1976, personal communication), and this tranquilizer was added in our study.

Analgesic and neuroleptic drugs were combined for general anaesthesia in our third group. The CSF pressure decreases, and the patients are awake and cooperative. Neuroleptic anaesthesia, which is easy to administer even by the radiologist, is now in common use.

Ruggiero et al. (1969) summed up the relevant factors influencing the complication rate for gas encephalography. They were more frequent if (a) examination was carried out under general anaesthesia, (b) patients were in poor general condition, and (c) diagnosis was that of cerebral tumour.

We did not observe retinal haemorrhages or general complications among our patients. Bearing in mind the results from Ruggiero’s study, the fact that most of our patients were submitted to local anaesthesia may be significant; tumours were diagnosed in only 10%. Epilepsy, cerebral and cerebellar atrophy, and mental disturbances were the most frequent diagnoses. The use of ketamine/diazepam or neuroleptic anaesthesia avoids any great rise in intracranial pressure. Oxygen minimises residual symptoms and can be used in larger volumes than air due to its rapid absorption. Retinal haemorrhages are infrequent complications of gas encephalography and gas myelography, and, if they occur, are, as a rule, hardly noticed by the patients (Moseley and Pilling, 1976). However, they are a potential threat to vision (Simon et al., 1973), and can be kept to a minimum by considering the factors known to increase the intracranial or intraocular venous pressure or both.

References


Finke, V., and Jaenicke, H. (1970). Kreislaufänderungen während lumbarpunktion und pneumoencephalographic (am hand ophthalmodynamographi-
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