Intraocular haemorrhage as a complication of pneumoencephalography

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SYNOPSIS The ocular fundi of 20 patients were examined before and after pneumoencephalography. In four of these, fresh venous retinal haemorrhages were seen, and a further patient had developed an exudate. Possible reasons for a rise in retinal venous pressure include bodily inverting the patient, compression of the thorax, the use of positive pressure respiration, and the air injection itself. It may be advisable to take steps to limit the effects of such possible causative factors.

In 1973, Simon and colleagues published an account of several cases of intraocular haemorrhage resulting from air myelography carried out under general anaesthesia. They described three cases of symptomatic haemorrhage occurring in an uncontrolled series of 480 gas myelographies, while in a pilot series of 19 patients examined before and after myelography there were five patients with asymptomatic retinal or preretinal haemorrhages. At the time of the original presentation of this work, the authors were criticised for what appeared to be 'faulty' techniques and, indeed, they pointed out in a footnote that, after certain modifications to the procedure, no further haemorrhagic incidents occurred. It was also claimed that such complications were specific to gas myelography and did not occur with, for example, pneumoencephalography.

Retinal haemorrhage is not a recognised complication of pneumoencephalography (Mikkowski, 1969; Clark et al., 1970; Bergeron and Rumbaugh, 1971), although intracranial haemorrhage, usually subdural, is well documented (Bucy, 1942; Robinson, 1957; Khalifeh et al., 1964; Zotti, 1966; Calkins et al., 1967, Seshia, 1971; Mead, 1973); one case only of retinal haemorrhage appears to have been recorded (Hoyt and Beeston, 1966). However, many neuroradiologists or neuro-ophthalmologists seem to have encountered individual cases. It therefore seemed desirable to examine systematically a series of patients undergoing pneumoencephalography in order to detect subclinical retinal or preretinal haemorrhage, since, if this were occurring, the possibility of a more serious vitreous haemorrhage would always be present (Simon et al., 1973).

METHODS

Twenty patients undergoing pneumoencephalography under general anaesthesia at the National Hospital for Nervous Diseases, Queen Square, were examined funduscopically before and after the procedure. In the majority of cases, these examinations were within a few hours of the encephalogram, and a short acting mydriatic was employed. Fluorescein angiography was carried out in one case.

RESULTS

The patients have been divided into three groups:

1. Those in whom fundal examination was normal before and after the encephalogram (10 patients: Table 1).

2. Those in whom pre-encephalogram funduscopy revealed some abnormality which was unaffected by the radiological procedure (five patients: Table 2).

3. Those in whom some intraocular lesion was

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TABLE 1
PATIENTS WITH NORMAL FUNDI BEFORE AND AFTER PNEUMOGRAPHY

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>CH</td>
<td>58</td>
<td>Cerebral atrophy</td>
</tr>
<tr>
<td>PS</td>
<td>58</td>
<td>Atrophy + infarct</td>
</tr>
<tr>
<td>AM</td>
<td>33</td>
<td>Cerebellar atrophy</td>
</tr>
<tr>
<td>HP</td>
<td>60</td>
<td>Pituitary adenoma</td>
</tr>
<tr>
<td>MP</td>
<td>16</td>
<td>Retrobulbar neuritis</td>
</tr>
<tr>
<td>AT</td>
<td>39</td>
<td>Chiari malformation + ? syringomyelia</td>
</tr>
<tr>
<td>JB</td>
<td>14</td>
<td>? Vestibular neuritis</td>
</tr>
<tr>
<td>AB</td>
<td>38</td>
<td>?</td>
</tr>
<tr>
<td>TD</td>
<td>39</td>
<td>?</td>
</tr>
<tr>
<td>BM</td>
<td>51</td>
<td>Normal</td>
</tr>
</tbody>
</table>

TABLE 2
PATIENTS WITH ABNORMAL FUNDI, UNCHANGED AFTER PNEUMOGRAPHY

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>Funduscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP</td>
<td>34</td>
<td>Ischaemic papillitis</td>
<td>Papilloedema both eyes</td>
</tr>
<tr>
<td>BR</td>
<td>65</td>
<td>Chordoma</td>
<td>Left optic atrophy</td>
</tr>
<tr>
<td>MR</td>
<td>16</td>
<td>Hydrocephalus</td>
<td>Blurred disc margins</td>
</tr>
<tr>
<td>NS</td>
<td>43</td>
<td>Thalamic tumour</td>
<td>Discs pale</td>
</tr>
<tr>
<td>SR</td>
<td>31</td>
<td>Suprasellar tumour</td>
<td>Discs pale</td>
</tr>
</tbody>
</table>

TABLE 3
PATIENTS WITH INTRAOCULAR CHANGES AFTER PNEUMOGRAPHY (PEG)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>Pre-PEG funduscopy</th>
<th>Post-PEG funduscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>GC</td>
<td>41</td>
<td>Optic neuritis</td>
<td>Questionably swollen discs; venous pulsation normal</td>
<td>Scattered perimacular, blot haemorrhages, left more than right haemorrhages</td>
</tr>
<tr>
<td>TS</td>
<td>54</td>
<td>Cerebral atrophy</td>
<td>? Small haemorrhages left disc margin</td>
<td>Scattered blot haemorrhages both eyes plus flame on left</td>
</tr>
<tr>
<td>KF</td>
<td>11</td>
<td>Communicating hydrocephalus</td>
<td>Bilateral papilloedema</td>
<td>Florid, pre-retinal perivenous, haemorrhages both eyes</td>
</tr>
<tr>
<td>WH</td>
<td>49</td>
<td>Recurrent meningioma</td>
<td>Chronic papilloedema; small haemorrhage on right</td>
<td>Small, fresh, pre-retinal perivenous haemorrhages both eyes</td>
</tr>
<tr>
<td>LB</td>
<td>62</td>
<td>? Cholesteatoma</td>
<td>Normal</td>
<td>Small linear exudate on right</td>
</tr>
</tbody>
</table>

FIG. 1   Fluorescein angiography, carried out on patient GC, 48 hours after pneumoencephalography (a) left eye, 18 seconds, (b) right eye, five minutes. No significant angiographic abnormality is seen, but small haemorrhages (dark spots) are seen scattered throughout both fundi.
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FIG. 2 A patient in the Mimer III chair, in the 'hanging head' position. This may be sustained for several minutes while tomography is carried out. The cuirasse-type harness is shown.

FIG. 3 The patient is inverted during the somersault manoeuvre. The neck veins are engorged, and the facial skin is darker-hued than that of the hands or legs. Much of the weight is taken on the chest.

produced or exacerbated by the encephalogram (five patients: Table 3).

In every case, the encephalogram was carried out under general anaesthesia, with atropine premedication, induced with methohexitone and suxamethonium and maintained with a nitrous oxide/oxygen/trichlorethylene mixture and with pancuronium. Intermittent positive pressure respiration was always employed. The patients were placed in the Mimer III apparatus and, in every case, the somersault manoeuvre was used to fill the temporal horns.

There was no significant difference between the duration of anaesthesia in groups (1), (2), and (3), the mean for the first two groups being 130 minutes, and for the last 135 minutes. In all three the mean volume of air injected was 35 ml.

Of the five patients who showed fresh funduscopic abnormalities after the encephalogram, three denied any visual disturbance as a result of the lesions, while one (WH) still showed a hemianopia which had been present before the procedure. The last (GC) thought that some improvement in his visual acuity which he had
noticed shortly before the encephalogram had been maintained. This last patient was subjected to fluorescein angiography, which showed no abnormality (Fig. 1).

**DISCUSSION**

Since the haemorrhages seem to be of venous origin, factors causing raised intracranial and/or intraocular venous pressure must be sought. In this situation, several may be contributory.

**HEAD-DOWN POSITION** During much of the procedure, the patient’s legs are above the head and in certain positions—for example, when the anterior end of the third ventricle is being examined (Fig. 2) and during the somersault manoeuvre (Fig. 3)—this may be exaggerated. Although no correlation between the duration of anaesthesia and the incidence of haemorrhage was found, this factor cannot, we think, be disregarded. In the case of retinal haemorrhage after an encephalogram described by Hoyt and Beeston (1966), there was a suggestion that an excessive amount of time had elapsed with the patient inverted.

**COMPRESSION OF ABDOMEN AND THORAX** In the chair of the Mimer III, the anaesthetized patient is supported, in part, by a flexible cuirasse, and much of the weight is taken on this in the ‘brow down’ position. We have noticed that the neck veins may be engorged and the face rather dusky (Fig. 4) in the erect position, when the pressure on the thorax should be least. Compression injury of the chest is almost certainly the causative factor in Purtsher’s disease—traumatic retinal angiopathy—in which retinal exudates and haemorrhages were originally (Purtsher, 1910) thought to be due to an associated head injury. Stockhusen (1938) described traumatic retinal angiopathy in a pilot who crashed while wearing a halter-type safety harness. This was a relatively severe injury, but bilateral retinal haemorrhage occurring after a relatively mild seat-belt injury in a hypertensive man of 66 years was noted by Hoare (1970). It is of interest to note that our patient AB had developed retinal haemorrhage at the age of 20 years (18 years before the encephalogram) after aerobatics but was unaffected by the radiological procedure. Abnormal fluorescein angiographic findings in traumatic asphyxia—a severe form of crush injury in which retinal lesions are found along with severe systemic disturbance—have been reviewed by Ravin and Meyer (1973). The normal findings in the present case are not at variance with this since, clearly, the degree of compression during encephalography is much milder, although, in a discussion of what he termed ‘Valsalva haemorrhagic retinopathy’, Duane (1973) suggested that frank haemorrhages occurred only with a severe rise in venous pressure, and in a previously diseased retina.

**POSITIVE PRESSURE RESPIRATION** All the patients in the present study were ventilated with the Manley apparatus, without a significant negative phase. Although the consequent elevation of intrathoracic pressure would be intermittent, it might act in concert with any others to increase
the intracranial venous pressure. In the small additional series of Simon et al. (1973) carried out after the discovery of the venous haemorrhages (about 10 cases), the respiration did include a negative phase (−5 to −10 cm water); no further haemorrhages were seen. However, other modifications to the techniques had also been introduced. The French authors made no comments on the degree of assistance of ventilation; we routinely hyperventilate the patients, so that the paCO₂ is around 30 cm Hg. This may cause dilatation of the intracranial veins associated with shrinkage of the brain (Moseley and du Boulay, unpublished observations), but we have no information as to the effects on the intraocular venous pressure.

EFFECTS OF AIR INJECTION  It is well known that injection of air at encephalography causes an increase in intracranial pressure, although this may be transient (Cronquist et al., 1963). Finke and Jaenicke (1970), using the technique of ophthalmodynamometry, studied 60 patients undergoing pneumoencephalography carried out in a similar fashion to our studies, but under local anaesthesia. Twenty-four showed an increase of more than 5% in intraocular pressure as a result of lumbar puncture alone, with a further increase after air was injected. A further six patients showed an elevation of pressure only after air injection. These changes were thought to be due to alterations in ophthalmic arterial pressure, however.

The lack of any apparent correlation between the volume of air injected and the incidence of retinal haemorrhage, in either our series or that of Simon et al. (1973), would suggest that this is not a very important factor, at least quantitatively. A major difference between the two series is that in gas myelography a considerable quantity of cerebrospinal fluid is withdrawn, which is thought to cause intraspinial and possibly intracranial venous dilatation (Masserman, 1935), whereas, in our practice, only a small specimen is taken for analysis.

Of the factors discussed the combination of the first two—compression of the thorax, aggravated by the positions adopted during encephalography—would seem not only to be the most likely to be responsible, but also to be amenable to improvement. Discussions with the manufacturers of the equipment as regards the cuirasse have been initiated and work is projected on assessing the effects of encephalography without the somersault.

Lastly, it must be pointed out that two of the five patients who developed funduscopic abnormalities during encephalography had evidence of raised intracranial pressure. The possibility of intraocular haemorrhage must be added to the known risks of carrying out pneumoencephalography in these patients.

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REFERENCES


