BLINDNESS ASSOCIATED WITH RETROBULBAR HEMORRHAGE

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The exact sequence of events leading to blindness in patients with retrobulbar hemorrhage remains unclear. However, it is believed that an abnormally high pressure developed within the bony orbit can play an important role in the pathogenesis of this complication. This concept is supported by the clinical finding of blindness coinciding with the onset of intraocular hypertension, lid edema, chemosis and proptosis, clinical features considered characteristic of intraorbital hypertension caused by an expansile mass in the retrobulbar compartment. This entity has attracted much attention in recent years because such complications occurred in individuals who had the cosmetic procedure of blepharoplasty (Hartley et al., 1973). While some have advocated decompression of the globe by perforating the anterior chamber of the eye as the treatment for this complication (Hartley et al., 1973; Hueston & Heinze, 1974), our clinical experience indicated that a more conservative approach in management is preferred (Huang et al., 1977). This includes an immediate control of factors such as systemic hypertension or coagulation abnormality. To further support this method of management, an experimental study was designed to investigate the effects of an abnormally high pressure within the bony orbit upon the ocular structures. The information accumulated from this study forms the basis of this report.

MATERIAL AND METHODS

In 24 rabbits, autogenous blood obtained by cardiac puncture was injected into the retrobulbar space in one eye and as a control, 0.9 percent saline solution was injected into the opposite eye. No anesthesia was used. The injection created a situation closely resembling the clinical entity of retrobulbar hemorrhage (Figure 1). The intraocular pressure was then measured with the Schioetz tonometer in both eyes immediately, and then 10, 15, 30, 45 and 60 minutes, two, three, 24 and 48 hours after injection. Simultaneously, the visual status was qualitatively measured by observing the size of the pupil and by noting the eyelid movements in reaction to bright light stimulation.

The orbital contents were also removed at the same time intervals for histological studies to ascertain whether any changes had taken place in the optic nerve, retina, retinal and periocular vessels.
Fig. 1. The onset of chemosis, lid edema and proptosis were immediate after injections. The appearance resembles the clinical entity of retrobulbar hemorrhage.

RESULTS

The intraocular pressure measured in the unanesthetised raptts, prior to retrobulbar injection of whole blood and of 0.9 percent saline solution ranged from 6.5 mmHg to 12.2 mmHg with a mean pressure reading of 9.2 mmHg. An intraocular pressure of 40 to 40 mmHg was achieved by injecting either the whole blood or 0.9 percent saline solution into the retrobulbar space. However, the pressure had never exceeded 55 mmHg. A loss of pupillary reactivity to bright light stimulation was usually accompanied by absent light-eyelid reflexes (Table 1). In most instances, the pressure began to decrease within minutes following the injection and came down to normal limits within two to three hours (Figure 2). The light perception remained absent for at least another three to four hours and frequently remained somewhat sluggish for the first 24 to 36 hours. A full recovery of these reflexes and a resolution of proptosis were observed in all animals by 48

Table 1.

| Light-pupillary reflex | ± | ± | + | +++ | +++ |
| Light-eyelid reflex | ± | + | ++ | ++++ | ++++ |

<table>
<thead>
<tr>
<th>Time elapsed after injection</th>
<th>0 hr</th>
<th>1 hr</th>
<th>2 hrs</th>
<th>3 hrs</th>
<th>24 hrs</th>
<th>48 hrs</th>
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</thead>
<tbody>
<tr>
<td>¼ hr</td>
<td>½ hr</td>
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(−) to (+++): Denote the magnitude of reaction to light stimulation.