INTRA-OCULAR PRESSURE DURING RETROBULBAR INJECTION

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SUMMARY: It was found that the use of retrobulbar injections can cause quite large transient rises in intra-ocular pressure which can be significantly reduced by using local anaesthetic in the injected saline, and by injecting in small steps, typically 0.2 to 0.3 mls/kg body weight at 1 minute intervals for a total of not more than ten steps. After a moderate retrobulbar injection, the intra-ocular pressure was found to return to near normal in a few minutes, but with some decrease in anterior chamber volume.

Introduction

In ophthalmic surgery, it is common practice to use an injection of air or saline into the retrobulbar space in order to produce exophthalmus, making the eye more accessible for surgery. The obvious main disadvantage of using such a retrobulbar injection, is the resulting increase in intraocular pressure (IOP), which increases the likelihood of vitreous being lost during surgery (Blogg 1975). This study was aimed at gaining information about the extent of the rise of IOP, the mechanisms causing it, the time course of the IOP after retrobulbar injection, and the mechanisms involved in this time course.

Methods

Anaesthesia was induced in dogs and cats with thiopentone sodium intravenous injection, the larynx and trachea sprayed with lignocaine hydrochloride and the trachea intubated. A polythene cannula was tied into a cephalic vein and maintenance anaesthesia achieved with chloralose 1% urethane 10% in water with 1% sodium metaborate, given intravenously. The animals breathed air spontaneously throughout, via the endotracheal tube. Arterial blood pressure was monitored via a strain gauge transducer connected to a P.T.F.E. (“teflon”) femoral arterial cannula, whilst temperature and end-expiratory pCO₂ were monitored with a rectal thermistor probe and an infra red carbon dioxide analyser respectively.

For each animal used, a unit of fluid was determined from experience such that about 10 retrobulbar injections, each of one unit, would raise the IOP to about 100 mm Hg. As dogs and cats of various sizes were used, the unit varied from 1 ml to 5 ml, meaning that conclusions cannot be drawn from comparisons between animals, but only between the two eyes of the same animal.

A retrobulbar needle was introduced through the temporal canthus of each eye and tubes attached. A pressure measuring needle was introduced into the anterior chamber at the limbus and connected by P.T.F.E. (“teflon”) tubing to a low volume displacement strain gauge pressure transducer (S.E. Laboratories type 4-81). The whole preparation was then left for half an hour or more to stabilize, and care was taken to avoid any movement of the needles for the rest of the experiment. In some experiments, heparinized isotonic saline was then injected into the anterior chamber via the pressure measuring needle, and the decay curve recorded for comparison with the decay curve after retrobulbar injection.

Retrobulbar injection was made in steps of the previously determined unit at 40 second intervals, ten being the usual number of steps (see figure 1). After the last injection, the intraocular pressure was recorded for several minutes. For one eye of each animal, physiological saline only was used, and for the other eye, the following substances were added — either: Lignocaine hydrochloride 2%, or: Epinephrine 1/80,000, or: Combination of the above.

Results

The typical response to a retrobulbar injection was a sudden rise in IOP, followed by a decay towards normal, with time constants of the order of a minute. Plots of peak pressure, and pressure 30 seconds after the time of peak pressure (figure 2a) showed quite conclusively and consistently, that the presence of lignocaine reduced...
the rise in IOP. As would be expected, no significant difference was seen when epinephrine alone was used (see figure 2b), and the presence of epinephrine did not significantly modify the effect of lignocaine on the time scale of these experiments.

Comparisons of pressure decay curves after retrobulbar and intraocular injections showed similar time courses. However it was obvious that neither was a simple, nor even well repeatable, curve. Observation of anterior chamber depth after each series of retrobulbar injections showed that the anterior chamber volume had decreased. These results suggest that the normal mechanisms for controlling intraocular pressure by adjustment of aqueous production and outflow play a significant part in re-establishment of physiological pressures. In all cases, substantial exophthalmus was produced and persisted long after the IOP had returned to near normal.

Discussion

A doubling of the IOP can result from quite small retrobulbar injections if given quickly, e.g. 1.5 mls in a 3 kg cat, 5 mls in a 19 kg dog. Pressures several times normal could result clinically from quite moderate injections. The effectiveness of small steps and the use of lignocaine in minimising these increases suggests that the tone of the extra-ocular muscles (presumably principally M. retractor bulbi) is at least partly responsible for the increase by resisting the forward motion of the globe.

The decay is apparently caused primarily by a rebalancing of aqueous production and outflow, to reduce anterior chamber volume, although other factors also contribute. For example (a) the presence of lignocaine is known to actually reduce the IOP (Syndacker et al, 1954), (b) ocular rigidity is not constant under raised pressure (Duke-Elder 1961) and (c) the globe probably moves further forward during the period of decaying pressure.

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References


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