INTRAOCULAR PRESSURE DURING RETROBULBAR INJECTION

The recent publication of Lampard and Morgan (Aust. vet. J. 53: 490-491), its conclusions, and experimental method require close scrutiny.

Although retrobulbar injection is commonly used prior to intraocular procedures in human ophthalmic surgery in some countries, such injections are aimed at producing anesthesia, mydriasis, akenesis and hypotony in the absence of general anesthesia (Havener 1974 a). Havener warns that hyaluronidase must be added to the local anesthetic solution to prevent undesirable tensions of the orbit and that propitiousness of the orbit caused by “injection of large volumes without the aid of hyaluronidase is quite undesirable and dangerous in intraocular surgery” (Havener 1974 b). Hyaluronidase was not used in this study. Retrobulbar injection of air or saline is not commonly used “to produce exophthalmus” in human or veterinary ophthalmic surgery. Because of the increased incidence of vitreous loss during intraocular surgery, the technique is not used in dogs, nor is retrobulbar injection of saline used to produce exophthalmos in routine veterinary ophthalmic surgery.

Although raised intraocular pressure (IOP) associated with retrobulbar injections is widely recognised by veterinary ophthalmologists, experimental methods and results presented in this study do not justify the conclusions reached.

The site of retrobulbar injections (depth, inside or outside the extraocular muscle cone, etc) mentioned in the Summary is not stated.

In the Summary, the post injection fall in IOP is said to be associated with decrease in anterior chamber volume. Anterior chamber volume or anterior chamber depth were not measured. Clinical estimates and measurements of anterior chamber depth can be made with routine biomicroscopy. Changes in anterior chamber volume should be measured objectively to yield valid results. In the absence of objective measurements of biomicroscopic estimates of changes in such parameters, simple ‘observation’ alone is unlikely to yield quantitatively reliable estimates. No estimates of anterior chamber depth are presented to support ‘decrease in anterior chamber volume’.

The effect of post injection massage, usually recommended in human ophthalmic surgery (Havener 1974b), was not investigated.

With few exceptions most general anesthetics result in a fall in IOP thought to be caused by an increase in aqueous outflow facility (Havener 1974c). In the protocol used, three anesthetic or tranquilizing agents were used in each of the experimental animals — sodium thiopentone, 1% chloralose and urethane 10% — during the period of decay in IOP although the doses are not stated. No controls for these agents, either singularly or in combination, were used. Studies in dogs have shown that different anesthetics and levels of anesthesia cause differences in facility of aqueous outflow (Pellfer et al 1975). Based on other studies (Kornblueth 1959) these agents must have contributed to the decay in IOP but the extent to which these results have been influenced cannot be determined. Neither chloralose nor urethane is routinely used for anesthetic maintenance of dogs or cats in veterinary practice (Lumb and Jones 1973; British Veterinary Codex 1965). The rationale for their use in this experiment is unclear.

With the exception of two dogs whose weights are given in the legends to Figures 2 and 3 the number of experimental animals or their body weights are not stated in the paper. The results for cats used in the experiment are not given. Unfortunately, conclusions based on body weight are presented in the Summary.

Arterial blood pressure, rectal temperature and end-expiratory pCO₂ were measured. Results are not stated. End-expiratory pCO₂ is related to arterial pCO₂ the values for which may affect aqueous production and IOP. Mean arterial blood pressure levels may also affect these parameters (Kaskel et al 1974). Without stated results, significance of these values and rectal temperature measurements is unclear.

The relationship if any between the ‘unit of fluid’ (chosen such that 10 retrobulbar increments elevated IOP to about 100 mm Hg), and the body weight is not shown.

No control group was used to determine the effect of anterior chamber cannulation on the results of the experimental procedures. Although IOP measurements in the cannulated eye may simulate those measured by indirect means, the effects of experimental procedures, especially those involving physical manipulation, cannot be directly transposed from the cannulated to the uncanalculated eye without confirmatory evidence.

In ‘some experiments heparinised isotonic saline was injected into the anterior chamber’, exactly which animals or groups, the dose of heparin and/or saline used and the IOP reached prior to the study of decay of IOP were not stated.

Lignocaine hydrochloride 2%, epinephrine 1 : 80,000, and combinations of the two were given by retrobulbar injection added to physiological saline. The dose, and the number and species of animals subjected to this treatment were unspecified. The graph and the text figures 2A and 2B are referred to. Presumably this refers to 2 and 3 respectively. The symbols defined in the legends do not agree with those in the graphs. The graphical symbols used, relating ‘total volume of retrobulbar injection’ to ‘intraocular pressure’ (sic) in what appears to be 4 separate lines are undefined. The interpretation of the experimental results is thus impossible.

Assuming that the omissions referred to above were due to editorial or printing errors, it is unfortunate that the results of the experiments were not subjected to statistical analysis. Mean and standard deviations of plotted points were not shown. The statement ‘no significant difference was seen when epinephrine alone was used and the presence of epinephrine did not significantly modify the effect of lignocaine’ must be questioned, solely on the lack of statistical evidence. With no quantitative data available this conclusion is questionable.

Experimental results for comparisons between ‘pressure decay curves after retrobulbar and intraocular injections’ were not given. The statement ‘however, it was obvious that’ in regard to these results is thus unjustified. Further, the statement ‘these results suggest that the normal mechanisms for controlling IOP by adjustment of aqueous production and outflow play a signifiant part in re-establishment of physiological pressures’ is not based on any evidence or experimental results presented in the paper. Although the techniques are available in the literature (Duke-Elder 1968a) no mention of measurements of aqueous production or the coefficient of aqueous outflow facility (C) is made. In the absence of quantitative data on aqueous production, outflow, or of the pressure decay curves themselves, the similarity of these unreported pressure curves is insufficient evidence on which to base the above statements.

On the basis of data presented, ‘the effectiveness of small steps and the use of lignocaine in minimising these increases’ cannot be ascertained.

The presumption that M. retractor bulbi is principally responsible for the increase in IOP is unestablished on the basis of the experimental results. The rectus muscles have similar origins and insertions to M. retractor bulbi in the dog and cat (Prince 1962) and may have been similarly affected by retrobulbar lignocaine injections. The exact position of the injection in relation to the extraocular muscles was not stated. The effects of epinephrine on the ocular blood supply and IOP, and of lignocaine on the autonomic innervation of both the ciliary body and aqueous outflow mechanisms have not been discussed.
The reference cited (Duke-Elder 1961) (sic) for the statement ‘ocular rigidity is not constant under raised pressure’ does not support this statement. In the only reference to ocular rigidity on the page cited Duke-Elder states ‘the rate at which pressure falls (after manometric infusion) is governed largely by the facility of aqueous outflow and the ocular rigidity; the latter must be assumed or measured at some point in the experiment’. The interpretation placed on Duke-Elder’s statement by the authors differs from his actual wording. In his next statement Duke-Elder states ‘although this technique (of manometric infusion to raise IOP as used by the authors) is quick and convenient, it has a number of serious objections, particularly the absence of equilibrium in the IOP or the rate of aqueous outflow’ (Duke-Elder 1968b).

The fluid infused may affect the facility of outflow by causing alterations in the endothelial cells of the trabecular meshwork (Barany 1964; Rohen 1963, cited by Duke-Elder). In this study the authors infused the eye, raised the IOP and compared pressure decay with decay in IOP after retrobulbar injection. Whether their conclusions can be related to an uncontrolled eye is uninvestigated and unproven.

The effective diffusion of the injected liquid within the orbital tissues and the subsequent possible effect on the decay of IOP was not investigated or discussed.

In spite of considerable species variation in the parameters discussed, for example, aqueous outflow facility (C) (Peiffer et al 1975; Duke-Elder 1968c; Melton and De Ville 1960; Melton and Hayes 1959) — the results, discussion and conclusion have been presented without regard to species.

Uncritical acceptance of the authors’ interpretations may lead to possible undesirable consequences to patients. Interpretation of the conclusions from these experiments should await confirmation by a fully controlled, statistically evaluated study using currently acceptable techniques of ophthalmic and scientific investigation.

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INTRAOCULAR PRESSURE DURING RETROBULAR INJECTION

Dr Slatter, in his letter to the Editor, raises a number of points, many of which are valid, and would be worth pursuing. He may well be right, in that ‘raised intra-ocular pressure associated with retrobulbar injections is widely recognised by veterinary ophthalmologists’. However conversations with a number of veterinary practitioners in general practice (and after all how many veterinary ophthalmologists are there in Australia?) convinces me, that a quantitative appreciation of this phenomenon is not common.

Our simple, unsophisticated and rather direct experiments on a small number of dogs (4) and cats (6) were designed to have, at least, a first look at this question. With this small number of animals a statistical presentation of results could be misleading. In some of these experiments tonometry, before and after needle puncture of the anterior chamber, was carried out with an applantation tonometer of McKay-Marg type and these experiments showed that a good puncture followed by a period of re-equilibration produced no change in the resting intra-ocular pressure. Similarly in some experiments tonometry was carried out before the induction of general anaesthesia (see below) following the application of one drop of topical anaesthetic to the cornea. Again our anaesthetic regime did not significantly alter intra-ocular pressure.

Our paper is not and does not purport to be, an in depth analysis of intra-ocular pressure changes due to fluid in the retrobulbar space. Such an in depth analysis would embrace many of the points raised by Dr Slatter and several others which he does not mention, for example, the effect of tamponade of the vessels in the retrobulbar space, the pressure changes in the posterior chamber (after all it is from here that vitreous loss occurs) and the pressure in the retrobulbar space itself. I shall comment on only a few of the points which Dr Slatter raises.

Previous experiments in our laboratory have shown that IOP is, over a wide range of conditions, related to arterial and venous pressures. Thus

\[ IOP = CVP + K(MAP - CVP) \]

where \( CVP \) is central venous pressure
\( MAP \) is mean arterial pressure
\( K \) is constant of order of 0.2

The rationale for the anaesthetic regime which was used was a quick humane induction with a relatively evanescent drug, namely thiopentone sodium, and a very long period (several hours) of anaesthesia with very good cardiovascular stability following the administration of a single dose of the cardiovascular-urethane mixture. This is one of the routine methods of anaesthesia in experimental physiology and pharmacology. Admittedly its direct effects on the mechanism of eye (K in the above equation) would need to be investigated but

References
