CARDIAC SLOWING DURING STRABISMUS SURGERY*†

BY

HOWARD REED AND THOMAS McCAUGHEY

Children's Hospital, Winnipeg,
and
Faculty of Medicine, University of Manitoba, Canada

CARDIAC arrest is a frightening complication which may confront a surgeon at any operation. It is fortunate that it is relatively uncommon, but there is a danger of false security. Eye surgeons face this complication less frequently than general or thoracic surgeons, but few of those who have practised for many years have not met cases of actual or threatened cardiac arrest. Kirsch, Samet, Kugel, and Axelrod (1957) have estimated that cardiac arrest occurs in about 1 in 1,500 cases in general surgery and in about one in 3,500 cases in eye operations. Snyder, Snyder, and Chaffin (1953) considered that cardiac arrest occurs in about 1 in 2,500 cases. Milstein (1956) quoted estimates varying from 1 in 858 to 1 in 4,950 operations. Gartner and Billet (1958) estimated that about 45 deaths occurred during eye surgery in the United States of America every year. This problem is, therefore, a very real one.

Review of the Literature

The oculo-cardiac reflex was described simultaneously by Aschner (1908) and Dagnini (1908). Aschner's report is particularly comprehensive. He investigated this reflex in animals and was the first to record that pressure upon the eyeballs caused bradycardia. He experimented by cutting the oculomotor, trochlear, abducens, facial, and auditory nerves, and found that an intact trigeminal nerve was essential for the production of the reflex. He also found that stimulation of any of the branches of the trigeminal nerve produced the reflex, and that pressure upon the tissues remaining at the apex of the orbit after enucleation would cause slowing of the heart.

He made the interesting comment that pressure upon both eyes of a rabbit might slow or even arrest the heart beat temporarily but it was not possible to kill the rabbit in this way. He observed that, whereas in dogs and human beings the reflex acted mainly upon the heart, in rabbits the respiration was more seriously affected. In the case of one rabbit respirations were slowed for 3 minutes, but they gradually returned to normal despite continued pressure upon the eyes.

Aschner further discovered that the reflex was more readily evoked under anaesthesia than in the waking state. Since these early observations, many reports have appeared showing that serious bradycardia, vomiting, and shock may

be caused by retrobulbar haemorrhage resulting from injury, and by enucleation of an eye. These reports have been concerned with vagal inhibition due to a variety of causes unrelated to squint surgery.

It was not until Sorenson and Gilmore (1956) reported the occurrence of cardiac arrest during a squint operation that the importance of this reflex in squint surgery was realized. In this case traction upon the medial rectus caused the heart to stop, but when the muscle was released the heart rate returned to normal. They studied seventeen further cases and found that traction upon the recti produced bradycardia in sixteen, and that seven developed extra systoles and one ventricular fibrillation. They concluded that pre-operative hypodermic atropine did not completely inhibit the reflex.

Kirsch and others (1957) reported the results of a study of more than fifty cases of eye surgery. Kirsch's interest in the subject was stimulated by the fact that he had had one fatal case of cardiac arrest which occurred during an operation for retinal detachment. He found that the only stimuli which produced this reflex were:

(*a*) Pressure on the globe;

(*b*) Manipulation of the extra-ocular muscles;

(*c*) Pressure on the tissue remaining at the apex of the orbit after enucleation of the globe.

He concluded that the reflex was just as easily elicited under local as under general anaesthesia and that the depth of the anaesthesia and the nature of the anaesthetic had no effect upon the sensitivity of the reflex. However, in twelve cases in which retrobulbar procaine with epinephrine and hyaluronidase was injected behind the globe, the reflex was abolished within 90 seconds. He therefore recommended that retrobulbar procaine should be given in every operation for squint, retinal detachment, or enucleation.

The results obtained by Bosomworth, Ziegler, and Jacoby (1958) differed considerably from those of Kirsch. These authors made a similar study of the effect of retrobulbar Xylocaine in seventeen patients, and found that twelve still exhibited cardiac slowing after the injection.

Present Investigations

The present study was undertaken, therefore, to determine the factors tending to cause cardiac arrhythmias, and to attempt to show the most efficient means of making squint surgery as safe as possible.

Since the oculo-cardiac reflex is but one of the many vagal reflexes many of the findings may well apply in other fields of surgery.

Methods of Study

77 children undergoing squint surgery, whose ages ranged from 1 to 14 years, were used as subjects of study, and continuous electro-encephalographic and electrocardiographic recordings were made throughout the operation. The former gave an objective estimate of the depth of the anaesthesia and the latter supplied an exact record of the heart's action.

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Anaesthesia

The routine anaesthetic procedure was as follows:

(1) Atropine was injected subcutaneously about 45 min. before the operation in doses of 0·25 to 0·4 mg., varying with the age of the child.

(2) Rectal or intravenous pentothal was used for induction.

(3) An injection of succinyl-choline chloride was given to assist endotracheal intubation.

(4) Anaesthesia was maintained with a mixture of oxygen, nitrous oxide, and ether or cyclopropane by a non-rebreathing technique.

This procedure was modified in cases in which atropine was withheld or gallamine or retrobulbar anaesthetics were given. If any respiratory depression due to gallamine was evident at the end of the operation, edrophonium was given to reverse its effects.

Operative Procedure

In each case the usual squint operation was performed. When the extra-ocular muscle was mobilized with the squint hook, traction was applied to the muscle. The ECG recording gave an accurate and permanent record of any degree of cardiac slowing (Fig. 1, opposite). The heart rate was determined from the record before, during, and after the stimulus. This was found to be a more reliable method of estimating bradycardia than precordial auscultation with a stethoscope strapped to the chest. Slight degrees of cardiac slowing of less than 10 to 15 per cent. were almost impossible to detect clinically, but they were readily measured on the ECG tracing.

Results

Four measures to prevent vagal inhibition of the heart were tested:

(1) Retrobulbar local anaesthetics such as procaine or xylocaine.

(2) Gallamine triethiodide (Flaxedil).

(3) Subcutaneous atropine.

(4) Intravenous atropine.

(1A) *Procaine.*—This drug was used in twelve cases. When cardiac slowing had been elicited by traction on the extra-ocular muscle, a retrobulbar injection of 1·5 ml. 2 per cent. procaine was given (Fig. 2, opposite). After an interval of 5 minutes traction was again applied to the muscle. In eleven cases bradycardia still occurred, the reflex being abolished in one case only.

It might be argued that, if the injection had been given accurately in the region of the ciliary ganglion, the reflex would have been completely abolished. In every case, however, the pupil dilated, indicating that the posterior ciliary nerves were affected by the local anaesthetic. It would thus appear that a

1

2

3

PULL   RELEASE

DEMEROL   RECTAL PENTOTHAL   CYCLOPROPANE

1

2

3

PULL   RELEASE
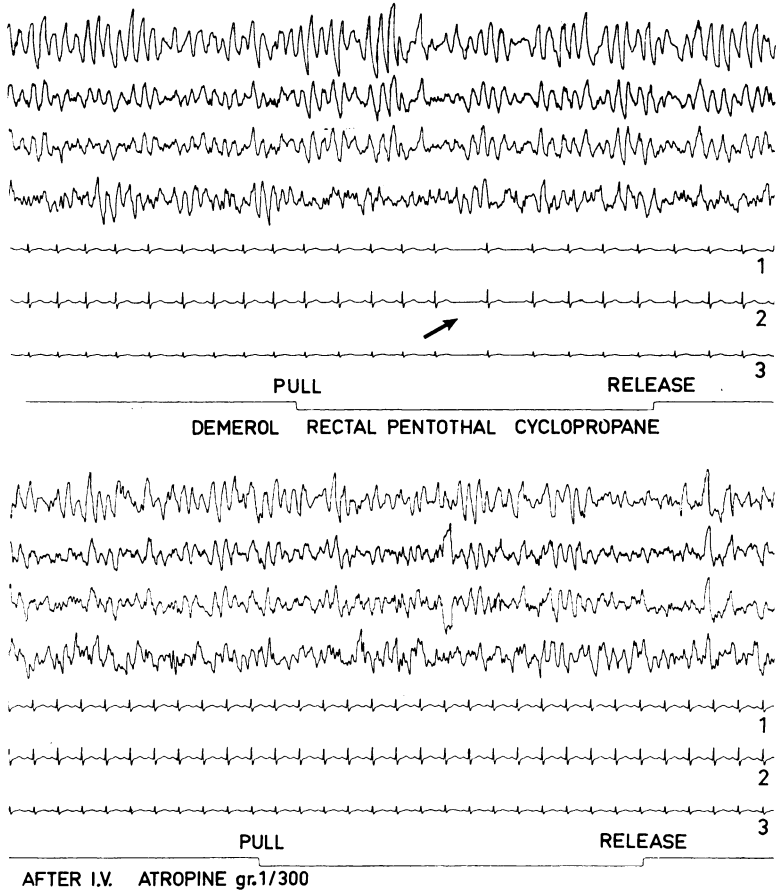
AFTER I.V.   ATROPINE gr.1/300

FIG. 1.—Tracing showing bradycardia on traction upon the right medial rectus muscle during a strabismus operation before and after intravenous atropine.
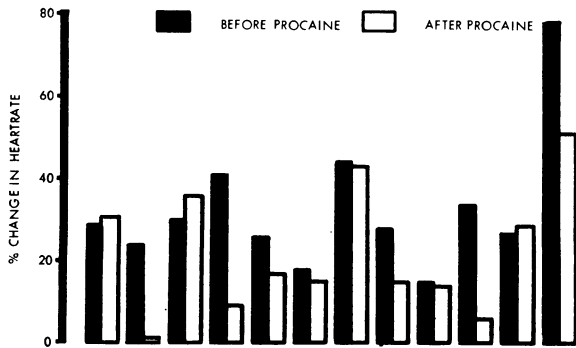
FIG. 2.—Effect of retrobulbar injections of procaine in inhibiting the oculo-cardiac reflex.

retrobulbar local anaesthetic is an unreliable method of abolishing the reflex.

These results differ from those of Kirsch and others (1957), who reported that a retrobulbar injection of a local anaesthetic gave almost complete protection from vagal inhibition during eye surgery. However, as Kirsch used hyaluronidase with the local anaesthetic, it was decided to repeat our experiments using Xylocaine 1 per cent. with hyaluronidase.

(1B) *Xylocaine with Hyaluronidase.*—When bradycardia had been produced by traction on the extra-ocular muscle, 1·5 ml. Xylocaine 1 per cent. with hyaluronidase was injected retrobulbarly in twenty cases (Fig. 3). After an interval of 5 minutes for the anaesthetic to take effect, traction was again applied to the muscle. In all cases cardiac slowing was demonstrable, although in many cases it was minimal. In five cases cardiac slowing was actually increased after the injection of Xylocaine and hyaluronidase. This increase may not be of significance because it is possible that the degree of slowing is dependent in part upon the degree of traction upon the muscle. We had no means of measuring the amount of pull on the muscle. These findings are therefore more qualitative than quantitative in nature. It will be noted that they differ from those of Kirsch but conform to those of Bosomworth and others (1958).
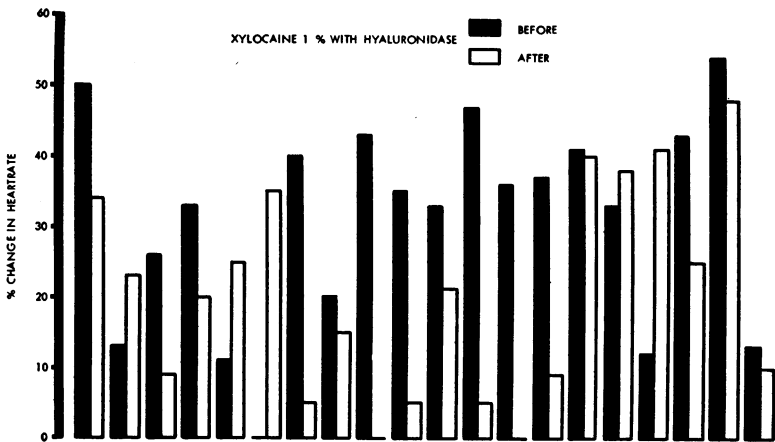

FIG. 3.—Effect of retrobulbar Xylocaine with hyaluronidase in inhibiting the oculo-cardiac reflex.

(2) *Gallamine Triethiodide (Flaxedil).*—This drug is used primarily as a muscle relaxant, but its vagolytic quality is a valuable side-action, especially in eye surgery. It was used in eleven cases. When it had been proved that traction on the muscle had caused a slowing of the heart, intravenous Flaxedil was given (Fig. 4, opposite). In eight patients some slowing of the heart still occurred though it was considerably reduced in degree. The reflex was completely abolished by Flaxedil in three cases. These findings suggest that Flaxedil is more effective than the retrobulbar anaesthetics in inhibiting the oculo-cardiac reflex.
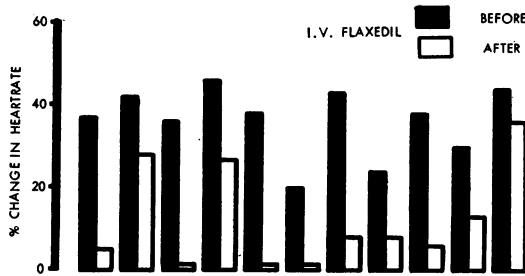
FIG. 4.—Effect of Flaxedil in inhibiting the oculo-cardiac reflex.

(3) *Subcutaneous Atropine.*—34 patients were given subcutaneous atropine as a pre-anaesthetic medication (Fig. 5). Of these 22 (64·5 per cent.) showed some bradycardia when traction was applied to the muscle. 24 patients received no premedication by atropine, and of these 21 (87·5 per cent.) showed bradycardia. It seems evident therefore that premedication with atropine does reduce the danger of vagal inhibition, but does not give complete protection.
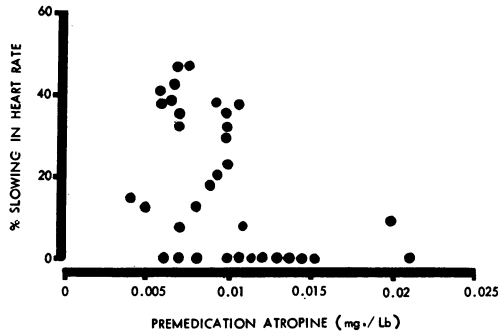

FIG. 5.—Effect of various quantities of premedication atropine in inhibiting the oculo-cardiac reflex.

No special provisions were made for an analysis of the weight/dose ratio and the dose/time relationship.

(4) *Intravenous Atropine.*—Twenty patients received intravenous atropine after it was shown that the reflex could be elicited. Only one of them showed any bradycardia when traction was re-applied to the muscle. Intravenous atropine thus seems to be the most effective means of abolishing the oculo-cardiac reflex.

Related Factors.—In an attempt to determine other factors which might be associated with a particularly sensitive oculo-cardiac reflex, scattergrams were made, plotting age, weight, and the heart rate before stimulation against the percentage slowing (Figs 6, 7, and 8, overleaf).
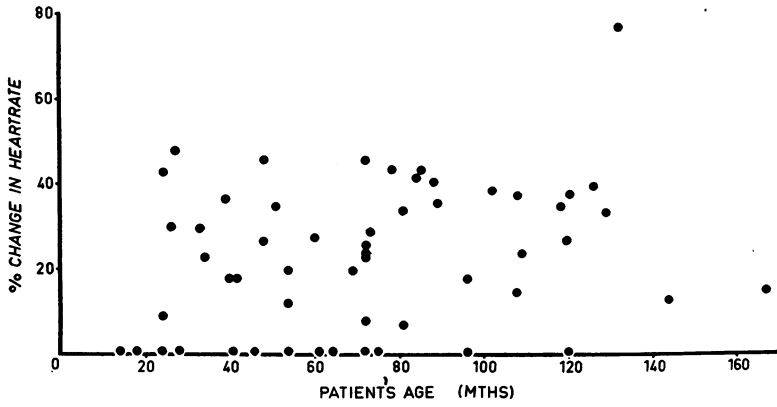
FIG. 6.—Percentage slowing of heart rate related to patient's age in months.
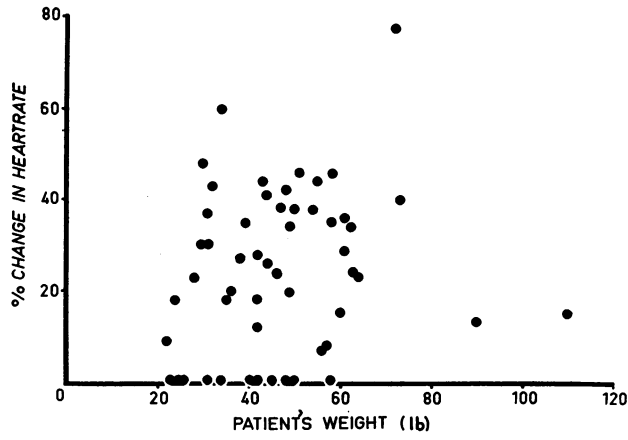

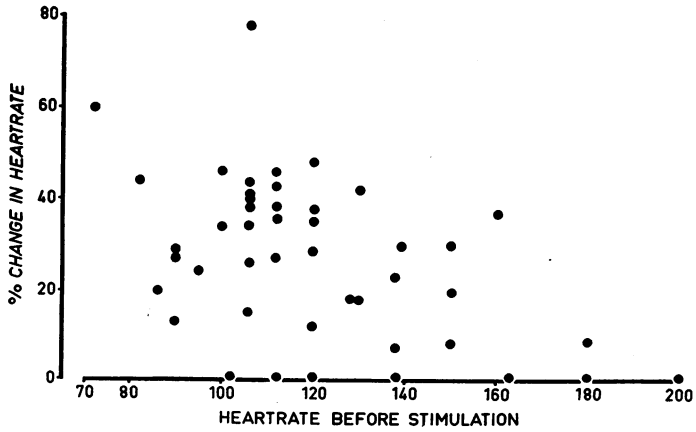FIG. 7.—Percentage slowing of heart rate related to patient's weight in pounds.

FIG. 8.—Percentage slowing of heart rate related to heart rate before stimulation.

These scattergrams demonstrated no apparent correlation between the age, weight, or cardiac rate before stimulation and the percentage slowing. On numerous occasions we attempted to forecast whether a patient was or was not particularly sensitive to traction upon the ocular muscle on the assumption that an anxious, frightened child would show a more marked reflex. But this we found it was impossible to do.

An interesting study of the oculo-cardiac reflex was made by Gastaut and Fischer-Williams (1957), using the ocular compression test. They showed that, in epileptics and healthy people, ocular compression had little effect upon the heart rate. In patients subject to syncopal attacks, however, a greater tendency to cardiac slowing was found. It might, therefore, be that a history of fainting spells is the most valuable guide to the tendency of an individual to have an unusually sensitive oculo-cardiac reflex.

Anaesthetic Considerations.—Light rather than deep anaesthesia appeared to favour the production of the reflex, which appeared most readily when the patient was under cyclopropane anaesthesia, with assisted or controlled respiration. A non-breathing technique was used with assisted or controlled respiration to offset the possibility of carbon dioxide accumulation from hypoventilation. At no time did the EEG pattern show evidence of hypoventilation. No attempt was made to measure oxygen or carbon dioxide levels during anaesthesia.

We found that it was possible to demonstrate the reflex almost without exception by dispensing with atropine and using the following induction sequence: rectal or intravenous pentothal, succinyl choline for intubation, and nitrous oxide, oxygen, and cyclopropane with assisted or controlled respiration. Ether made it more difficult to demonstrate the reflex and the deeper the ether anaesthesia the greater the difficulty.

Succinyl choline can itself cause bradycardia in infants and children. However, this drug was used only for the intubation and its vagal effects disappeared long before traction was applied to the eye muscles.

Discussion

All anaesthetists and ophthalmologists should be aware of the dangers of cardiac arrest during squint surgery. In particular the pulse rate should be monitored by precordial auscultation during traction on the extra-ocular muscles. Our experience leads us to believe that the danger is not great, especially if adequate doses of atropine and Flaxedil are given.

Only on one occasion did the heart appear to stop (Fig. 9, overleaf). It was arrested for about four beats whilst traction was applied to the medial rectus. When the muscle was released, the heart recommenced at once. We believe that if the traction had been maintained vagal release would have occurred and the heart would have started despite the continuing traction.
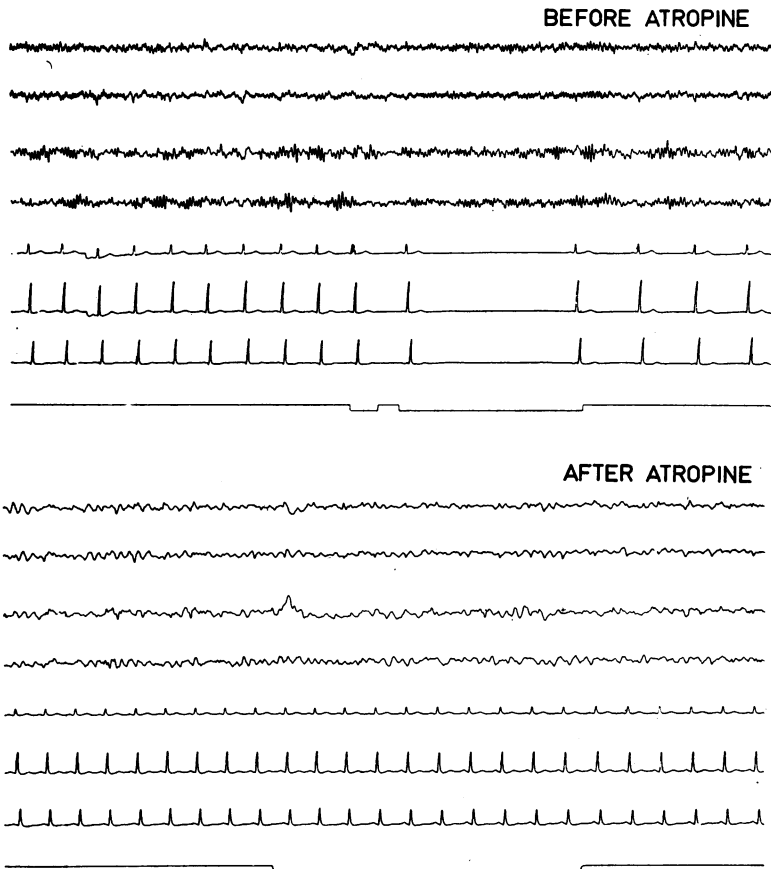
BEFORE ATROPINE

AFTER ATROPINE

FIG. 9.—Cardiac arrest for about four beats during application of traction to the medial rectus.

Sevéral authors have reported that vagal stimulation may cause cardiac irregularities in addition to simple slowing. In all the cases under investigation a simple bradycardia occurred. It is well known, however, that vagal inhibition in the presence of anoxia or an abnormal myocardium may produce cardiac arrhythmias.

Young, Sealy, Harris, and Botwin (1951) studied the effects of hypercapnia and hypoxia on the response of the heart to vagal stimulation. They concluded that anoxia and increased carbon dioxide accumulation enhanced the risk of cardiac arrest. Milstein (1956) held the same view. Radnai and Mosonyi (1957) showed that bulbar compression caused greater bradycardia in patients with anaemia or with angina pectoris than in normal patients. As a result of an electrocardiographic study on the effects of eyeball compression in 148 Africans, Schramroth (1958) concluded that myocardial abnormality or the use of digitalis increased the risk of ectopic rhythms. Rhode, Grom, Bajares, Anselmi, Capriles, and Rivas (1958) studied the

electrocardiographic alterations which occurred during operations upon the extra-ocular muscles and studied the action of the reflex in dogs.   They concluded that cardiac arrest occurred only if the reflex acted during the state of anoxia.   This is the view of Snyder, Snyder, and Chaffin (1953), who studied 61 cases of cardiac arrest in infants and children.   They quoted numerous authorities and concluded that anoxia and increased carbon dioxide accumulation rendered the myocardium irritable.   If at such a time the oculo-cardiac reflex acts, serious degrees of arrhythmia and arrest are likely to occur.   Reid, Stephenson, and Hinton (1952) arrived at the same conclusions.

During all our operations great care was taken to ensure that the patient was adequately oxygenated.   This is the probable explanation for the absence of cardiac arrhythmias apart from simple slowing.   We believe that a good airway and good oxygenation are probably the most important factors in the prevention of cardiac arrest in squint surgery on children.

Summary and Conclusions

An attempt was made to assess the value of various measures used to inhibit the oculo-cardiac reflex and thus to prevent cardiac slowing during squint surgery.   This was done by applying traction to the extra-ocular muscles during strabismus operations, and recording the heart rate with an electrocardiogram.   The change in heart rate during stimulation was measured and the percentage of slowing was calculated.

The results obtained pointed to the following conclusions:

(1) The usual doses of subcutaneous atropine given as premedication before an anaesthetic may give some protection against the oculo-cardiac reflex but the protection is not complete.

(2) Intravenous atropine gives almost complete protection against vagal inhibition.

(3) Retrobulbar local anaesthetics appear to have little effect in preventing cardiac slowing.

(4) Gallamine triethiodide gives considerable protection against cardiac inhibition.

(5) During an eye operation, the pulse should be monitored carefully whenever the extra-ocular muscles are being manipulated.   If bradycardia should occur, the oculo-cardiac reflex may be inhibited by an intravenous injection of atropine.

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