LETTERS TO THE EDITOR

A REVIEW OF ANAESTHESIA IN OPHTHALMOLOGY

Dear Sir,

I write to you regarding the above article by E Y Yap, W K Chan and R F T Tan that was featured in the June 1993 issue of SMJ[1]. Although I enjoyed the brief review, I note that there was no contribution from an anaesthesiologist. The article was also probably not reviewed by an anaesthesiologist judging from the presence of several inaccuracies. There are also points which I disagree with. These are as follows:

1. The authors mentioned that lignocaine or bupivacaine with or without adrenaline 1:1,000 may be used for local or regional anaesthesia. The correct concentration usually used is 1:200,000. 1ml of solution with 1:1,000 adrenaline contains 1 mg of adrenaline – if injected this will cause severe hypertension and tachycardia. If the authors meant that a 1:1,000 solution of adrenaline may be added to the local anaesthetic solution, they should add that only 0.05 ml of the adrenaline solution (containing 50 mcg of adrenaline) should be added to 10ml of local anaesthetic solution, giving a final concentration of 1:200,000 (5 mcg of adrenaline per ml).
2. The authors mentioned that their patients undergoing regional anaesthesia are given 100% oxygen through nasal catheters. When we say that patients are given 100% oxygen, we mean that the inspired oxygen concentration is 100%, and not the concentration of oxygen flowing within the nasal catheter. It is impossible to give an inspired concentration of 100% oxygen via nasal catheters due to dilution with ambient air.
3. The authors mentioned that 'succinylcholine is ... not used in penetrating injuries as it increases intraocular pressure and therefore the risk of expulsion (of intraocular contents)'. The use of succinylcholine in penetrating eye injuries is controversial, and this dogmatic statement does not do justice to the issues involved. Succinylcholine has the major advantage of allowing the anaesthetist to intubate the trachea within 30 seconds of administration, as compared to at least 90 seconds with even large doses of non-depolarising agents. This is a significant advantage in the context of penetrating eye injuries where the patients present as emergencies with full stomachs, with the associated risk of pulmonary aspiration during induction of anaesthesia (which may be life-threatening). Intubating conditions are also ideal with succinylcholine, whereas with non-depolarising agents the patient may not be fully paralysed when the anaesthetist attempts to intubate even after 90 seconds. If the patient coughs or bucks due to inadequate paralysis, the effects on the intraocular pressure may be even more disastrous than the slight rise due to succinylcholine.

These factors should be considered when the anaesthetist makes the decision about whether to use suxamethonium in a patient with perforating eye injury. Obviously, it is essential to do nothing which will compromise the patient's life, while efforts are being made to salvage what remains of the patient's sight. This issue is further discussed in standard anaesthesia texts [2,3].

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AUTHOR'S REPLY

Dear Sir,

Here are our responses to the 3 points raised:

1. We apologise for not stating more precisely in the paper about the way we used adrenaline. We use 1:1000 solution of adrenaline drawn into a 2cc syringe. 1 – 2 drops of this was then mixed into a 20cc vial of lignocaine 2% via a 23G needle. This gave an extremely low concentration of adrenaline in the lignocaine, much less than the 1:200,000 concentration you had mentioned.
2. We totally agree with your comments on this point.
3. We take your point that the use of succinylcholine in perforating eye injuries is controversial. The decision as to which paralytic agent to use for intubation should be ultimately the anaesthesiologist's choice after consultation with the ophthalmologist as to the extent of the patient's ocular injuries and the potential risk of expulsion of ocular contents.

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Answer to Electrocardiographic Case

Diagnosis: Right bundle branch block with left anterior hemiblock and prolonged PR interval – trifascicular block.

DISCUSSION

The electrocardiogram in Fig 1 illustrates complete right bundle branch block (RBBB), left anterior hemiblock (LAHB) and a prolonged PR interval. The presence of the left anterior hemiblock is diagnosed on the ECG by the presence of counterclockwise rotation of the frontal plane QRS loop, resulting in initial ventricular activation proceeding inferiorly and posteriorly, whereas the superior and anterior portion of the left ventricle is activated late. This results in an initial r wave in II, III and aVF and q wave in I and aVL. The late activation of left, superior and anterior left ventricle results in left axis deviation (> 30 degrees) and rS waves in II, III and aVF. There is also a terminal r in aVR and aVL, with the terminal r in aVL occurring before the r wave in aVR.

The above ECG features of RBBB and LAHB with prolonged PR interval suggest that the block may be a trifascicular block or block within the atrioventricular (AV) node[1]. In many cases, PR prolongation represents delay in the AV node and not below the His bundle, so diagnosis of trifascicular block from the ECG alone is inferential, and an electrophysiological study is sometimes needed to be absolutely certain.

The RBBB and LAHB, as seen in this patient, is the most common form of bifascicular block. It is seen in approximately 1% of hospitalised patients. Bifascicular block is rarely found in the absence of heart disease with perhaps < 0.1% incidence in the clinically normal population[2]. It is most commonly associated with coronary artery disease, congestive cardiomyopathy and aortic valve disease, especially calcific aortic stenosis[3]. Rarer causes include Lenegre's disease and Lev's disease. Congenital diseases associated with RBBB+LAHB include endocardial cushion defects, ventricular septal defects and following surgical repair of Fallot's tetralogy. In one study by McAnulty, 47% had associated coronary heart disease and 23% primary conduction system disease[4].

The importance of the above findings is the progression to complete AV block. McAnulty[4] studied the 2-year course of 257 patients with various types of bifascicular block and found that complete heart block occurred in only 5%. Another group found that AV block developed at a rate of < 1% per year in the patients without underlying heart disease, during an average followup of 4 years, whereas AV block developed at a rate of approximately 2% per year in patients with chronic bifascicular block and coexisting heart disease[5]. The method of investigation of choice is prolonged electrocardiographic monitoring such as the Holter. Prolonged electrocardiographic monitoring in our patient documented intermittent complete heart block to be the cause of giddiness. In some patients, when the complete heart block cannot be documented, an electrophysiological study may be helpful to decide if permanent pacing would be useful. Patients with HV interval of 70-100 ms have a 4% annual incidence of complete heart block and probably should be paced, whereas patients with an HV interval >100 ms have an 8% annual incidence of complete heart block and should undergo implantation of a permanent pacemaker[6-9]. Pacing abolishes symptoms in approximately two-thirds of patients with chronic bifascicular block and syncope but does not seem to improve overall long term survival[8-11]. Our patient underwent a permanent pacemaker implantation.

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