Total autonomic blockage and primary sinus node dysfunction in a patient about to undergo thyroidectomy.

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Key words: Thyroidectomy, propanolol, atropine, primary node, and dysfunction.

This is a report of a rare presentation of primary node dysfunction in a patient about to undergo thyroidectomy. A 40-year old female patient had been prepared with propanolol for thyroidectomy. In the operating room, on receiving intravenous atropine, she immediately went into asystole sinus arrest and arrhythmia with severe bradycardia. She responded to basic resuscitative measures and the operation was postponed. Following cardiologial and neurological evaluation, she was deemed fit to undergo thyroidectomy for a goitre compressing the trachea. A week later, she underwent thyroidectomy. Postoperative follow-up was uneventful.

The mechanism of complete autonomic blockage and the resulting sinus node dysfunction is discussed. Propanolol and atropine block the sympathetic and parasympathetic systems respectively. In a normal heart such blockade does not usually create problems, as the heart will beat at its own fast intrinsic rate of about 100 beats/minute. In contrast, if there is underlying primary node dysfunction or sinoatrial disease, there may be a severe bradycardia and possible complete arrest. The authors advise caution and an awareness of the possibility of such autonomic blockade occurring in a patient about to undergo thyroidectomy.

IIntroduction

We report a rare presentation of primary nodal dysfunction following autonomic blockade in a patient about to undergo thyroidectomy under general anaesthesia.

Case Report

A 40-year old healthy looking woman with no history of previous cardiac or liver disease or ay other major illness was booked for thyroidectomy. She had a slightly raised T4 value of 131.78nMol/L with early exophthalmos that was only detected on ophthalmological examination; clinically it was not obvious and she had no toxic symptoms or signs. She had not been on any anti-thyroid treatment prior to her attendance at the tumour clinic. She was prepared for thyroidectomy with propranolol four days before the scheduled operation. Before induction with general anaesthesia, intravenous atropine sulphate o.6 mg was administered on the table. The patient immediately went into momentary asystole, followed by bradycardia of 36 beats per minute, developed ectopics, ST segment depression and prolongation of atrioventricular conduction time. These changes were observed by the attending staffon a cardiac monitor which had been connected to the patient's chest, At the same time as the monitor changes were observed, she had a momentary clouding of consciousness with the blood pressure dropping to 90 mm Hg systolic and 60 mm Hg diastolic. She was immediately given oxygen by mask, transfused rapidly with normal saline and the foot end of the table elevated. As she began to improve she responded to questions and complained of severe occipital and frontal headaches. She was subsequently taken to the Intensive Care Unit for observation. Over a period of a few hours, using oygenation and intravenous fluids, the heart rate and rhythm stabilised to the preoperative value of 64 beats/minute and a normal rhythm. No drugs were used for resuscitation. She was transferred to a high obseration ward on the same afternoon. Clinically, the patient was well the next day with a steady pulse rate of 72 beats/minute, but she still complained of the headache, which lasted for two days. She was neurologically intact.

Following this unexpected turn of events, the patient was sent to the hospital cardiologist and neurologist for evaluation. An Intensive Care Unit electrocardiogram done on the day of the event was normal. The cardiologist suggested the possibility of

autonomic dysfunction. An electroencephalogram was reported as showing an epileptogenic focus, but neurologically she was considered to show no abnormalities clinically. The patient denied any history of fit or fainting attacks but confirmed attacks of headaches for some years. Review of her past medical history confirmed the episodic headaches but there was no record of a similar incident. A Caesarean section done in 1996 had been uneventful. She apparently had a pre-eclampsia at that time. A skull x-ray was normal but showed a prominent system of diplopic veins and marked indentations on the skull table from arachnoid lacunae; the radiologist reported these findings as being normal. Chest x-ray was normal including he the heard outline. Ophthalmological examination revealed mild axial bilateral exophthalmos, which was not obvious at clinical examination. Fundoscopy was normal.

The incidence was discussed in full with the patient, who was a nurse. It was explained that with appropriate management, the heart condition would not be a contraindication to an early thyroidectomy, which was still advisable as there was evidence of tracheal compression.

One week following this episode, the patient was subjected to a subtotal thyroidectomy under general anaesthesia. Tracheal shift, compression and retrosternal extension were confirmed at surgery. She did not receive preoperative medication and the operation was uneventful. The atropine had been diluted five to one and the total dose given slowly in aliquots before induction of anaesthesia.

The patient was discharged the day after surgery. Follow-up was to continue until her cardiac status was determined with certainty and appropriate long-term management recommended.

Discussion

In the senior author's Unit (EK), all patients for thyroidectomy are prepared with propranolol[1]. By so doing, the sympathetic drive is controlled and the response is monitored by changes in the pulse rate. Our practice has been to slow the rate to just over 60 beats/minute preoperatively. In her case, she had received 80 mg 12 hourly orally for four days. On the day before surgery the dose had been increased to 8 hourly because her pulse rate had remained on the high side. In accordance with current recommendations, her last dose was given on the morning of the operation[1,2]. Inadvertent propranolol over dosage due to liver saturation was unlikely as our patient was not a chronic user, she had received it for 4 days only, furthermore, oral propanolol is subject to 80% liver first-pass metabolism therefore most of the drug is inactivated following ingestion and absorption[3]. Her pulse rate preoperatively had been 64 beats per minute and other ward nursing observations had been within normal limits – Temp. 36.5^{0}C, Respiratory Rate 18 per minute and Blood Pressure 120/80 mm of mercury. Atropine had been given on the operating table in the usual manner before induction at a dose of 0.6 mg intravenously. Her untoward and unexpected reaction had been immediate with initial asystole, bizarre electrical complexes, arrhythmias and severe bradycardia.

Autonomic blockade in the investigative setting can be achieved with 0.2mg/Kg propanolol intravenously followed after 10 minutes by 0.04mg/Kg of atropine sulphate intravenously[4]. Such autonomic blockade can separate patients with asymptomatic sinus bradycardia into a group with primary sinus node dysfunction (slow intrinsic heart rate) and a group with autonomic imbalance (normal intrinsic heart rate)[4]. The normal intrinsic heart rate at rest is usually about 100 beats per minute[3].

The operating room cardiac monitor showed severe depression of cardiac conduction with slowing of the intrinsic heart rate to 36 beats/minute. The pattern is in keeping with a diagnosis of primary sinus node dysfunction, the machine did not have a memory store for subsequent study, and moreover we were unable to confirm this diagnosis, as the hospital had no facilities for invasive assessment of nodal function. Repeat provocative testing was too risky without a reliable cardiac laboratory. Sinus node dysfunction was the most likely diagnosis clinically as other causes of sinus bradycardia such as myocardial infarction, hypothermia and hypothyroidism had been excluded[5,6].

It is of interest that Davidson's textbook of medicine 17th edition [6] recommends the use of atropine to correct haemodynamic instability resulting from sinus bradycardi. It seems that such a recommendation may need to be qualified when there is partial beta blockade, because the administration of atropine can precipitate complex autonomic blockade in the susceptible patient. The use of drugs such as propanolol and atropine is common in patients undergoing thyroidectomy. Drug induced autonomic dysfunction which results in overt manifestation of a covert conduction defect prior to thyroidectomy must be a rare event in general surgical practice. None of the authors had encountered such an event in a patient about to undergo thyroidectomy.

We consider it important that surgeons and anaesthetists be aware of such a possibility in patients being prepared for thyroidectomy. In certain circumstances sinus node dysfunction can become manifest in the presence certain cardio-active drugs[4]; we suggest that atropine should be added to the list of drugs, because

it has the potential albeit rare to cause sinus node dysfunction when administered to patients who are on propanolol for thyroidectomy.

References

1. Mieny C J. General Surgery academia, Pretoria 1992; Chapter 1, p.22.
2. King M, Bewes P C, Cains J, Thomtom J. Primary Surgery, Volume One Non-Trauma. Ist edn. Oxford University Press. Oxford 1993; Chapter 21.7 Pages 349.
3. Laurence D R, Bennett P N.Clinical Pharmacology 7th edn ELBS Churchill Livingstone Edinburgh 1994; Page 417.
4. Braunwald E, Isselbacher K J, Petersdorf R G, Wilson J D, Martin J B, Fauci A S. In: Harrison's Principles of Internal Medicine 11th edn McGraw-Hill Book Company, New York Page 918.
5. Forrest A P M, Carter D C, Macleod I B. In: Principles and Practice of Surgery. 3rd edn. Churchill Livingstone Edinburgh 1995; Chapter 11 Page119.
6. Edwards C R W, Bouchier I A D, Haslett C, Chilvers E R. In: Davidsons's Principles and Practice of Medicine. 17th edn ELBS Churchill Livingston Edinburgh 1995; Chapter 5 PP 227 and 263.