Clinical Reports

Intraoperative diagnosis of rate-dependent bundle branch block

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Rate-dependent left bundle branch block (LBBB) occasionally occurs during anaesthesia when the heart rate exceeds a critical value. While it is usually a benign disorder, it may mask the electrocardiographic manifestations of myocardial ischaemia and the ST-T wave pattern associated with LBBB may be mistaken for those of ischaemia. This case report presents two cases in which rate-dependent LBBB was clearly documented during the perioperative period. It demonstrates the use of pharmacologic agents (e.g., atropine and neostigmine) and physiologic manipulations (e.g., carotid sinus massage) to alter the heart rate and confirm the diagnosis of benign rate-dependent LBBB in the operating room. These interventions should be used with caution in patients who have hypertension, angina, cerebrovascular, or AV node disease or in the setting of myocardial ischaemia or severe bundle branch disease.

Key words

COMPLICATIONS: arrhythmia; HEART: arrhythmia; diagnosis, treatment.

Transient bundle branch block has been associated with hypertension,¹ increased heart rate under general² and local anaesthesia,³ preoperative lithium therapy⁴ and without obvious aetiologic factors⁵ during anaesthesia. However, the use of intentional measures to initiate or terminate the bundle branch block has not been described in the anaesthesia literature. This report presents two cases of rate-dependent LBBB which occurred during anaesthesia in association with spontaneous changes in heart rate and which were induced or treated by pharmacologic and mechanical interventions in order to confirm their diagnosis.

Case report #1

A 55-year-old man with a history of a cerebrovascular accident and transient ischaemic attacks was scheduled for carotid endarterectomy. The preoperative history included untreated mild hypertension, with a blood pressure range of 150–160/80–90 mmHg, mild exertional angina, chronic obstructive pulmonary disease, and previous alcohol abuse. The physical examination was unremarkable except for mild obesity and a left carotid bruit. Preoperative laboratory studies were within normal limits. The electrocardiogram (EKG) revealed normal sinus rhythm (NSR) with a rate of 71 beats/min and left ventricular hypertrophy by voltage criteria. An EKG on a previous admission showed NSR with a left bundle branch block pattern at a rate of 90 beats/min. Carotid arteriography demonstrated left internal carotid stenosis with an ulcerated plaque.

The patient was premedicated with morphine 10 mg and scopolamine 0.4 mg intramuscularly and arrived at the operating room sedated, with the EKG revealing NSR with normal conduction at 58 beats/min

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FIGURE 1 V_2 EKG tracing (25 mm/s) from patient #1. The first strip (1a) illustrates the change in conduction from normal to a left bundle-branch block (LBBB) pattern following 0.5 mg of intravenous atropine. Although P waves are not obvious, their presence was confirmed in lead II (not shown). Note that the transition in conduction occurred at a heart rate between 70–75 beats/min and that the change in R-R wave cycle length which triggered it is quite subtle. The second strip (1b) illustrates the reversion of conduction from LBBB pattern to normal in the V_2 lead, after neostigmine was given. Note that the abnormal conduction persisted until the heart rate was slower than that which triggered it (zone of linking).

and a blood pressure (BP) of 220/90 mmHg. After preoxygenation, anaesthesia was induced with 200 mg of thiopental, 300 μ g of fentanyl, and 50 per cent N_2O in oxygen. After intravenous injection of pancuronium ($0.08 \text{ mg} \cdot \text{kg}^{-1}$ in divided doses) for muscle relaxation and trimethaphan (4 mg over two minutes) for blood pressure control, the trachea was intubated. The heart rate increased to 85 beats/min with a BP range of 150–215/70–90 mmHg (mean of 80–115 mmHg), and a left bundle branch block pattern was noted on the EKG monitor. The pattern persisted despite control of his blood pressure with trimethaphan and enflurane. An arterial blood gas showed $PO_2 = 122 \text{ mmHg}$, $PCO_2 = 33 \text{ mmHg}$, $pH = 7.48$, $K^+ = 3.9 \text{ mEq/l}$, $Hb = 13.2 \text{ mg/dl}$, and ionized $Ca^{++} = 2.3 \text{ mEq/l}$. When the heart rate decreased to 70 beats/min, the EKG pattern reverted back to normal sinus rhythm. No ST segment or T wave changes were noted. Atropine (0.5 mg IV) was administered by slow intravenous titration to diagnose the LBBB as rate-dependent and the LBBB pattern returned when the heart rate rose to 80 beats/min (see Figure 1a). During the remainder of the case, he exhibited rate-dependent LBBB, which occurred consistently when his heart rate was greater than 75 beats/min. The change in conduction was not triggered by changes in blood pressure and it did not influence systemic blood pressure. No other EKG changes were noted. Reversal of the

pancuronium with neostigmine lowered his heart rate and the LBBB pattern reverted to normal conduction (see Figure 1b). Carotid massage, to lower the heart rate, was not performed because of the patient's carotid disease.

In the recovery room, his EKG showed NSR at 71 beats/min, unchanged from his preop EKG. Subsequent postoperative EKGs revealed a LBBB pattern with rates of 96–127 beats/min. Cardiac enzymes remained normal. Twenty-four hour Holter monitor confirmed the rate-dependent left bundle branch block.

Case report #2

A 67-year-old woman was scheduled for elective left total knee replacement. Past history was unremarkable except for degenerative joint disease of the knee joints. Physical examination revealed a systolic ejection murmur, grade II/VI, heard maximally at the lower left sternal border. The electrocardiogram revealed normal sinus rhythm at the rate of 73 beats/min with normal conduction. Other preoperative lab studies were normal.

The patient was brought to the operating room without premedication. She received fentanyl 50 μ g and diazepam 2.5 mg IV and was placed in the left lateral decubitus position for insertion of an epidural catheter in the L3-4 interspace. After test doses of Marcaine 0.75 per cent to detect subarachnoid or intravascular injection, a total of 20 ml of Marcaine 0.75 per cent was injected through the epidural catheter, resulting in a T8 sensory level of anaesthesia. The blood pressure decreased from 150/90 mmHg to 140/80 mmHg and the heart rate increased from a sinus rhythm at 90 beats/min to 98 beats/min, with a change in conduction from normal to a left bundle branch pattern. Conduction became normal transiently with carotid massage. During the remainder of the case, the conduction varied between the left bundle branch pattern and the normal pattern with the transition occurring consistently at a rate of 95–97 beats/min. The patient did not have any chest pain during the procedure. In the recovery room, the left bundle pattern appeared after atropine (0.25 mg IV) was given to treat a low systemic blood pressure with an increase in the heart rate to 98 beats/min (Figure 2a). After the application of carotid sinus massage, the heart rate decreased and the conduction returned to normal (Figure 2b). The patient recovered uneventfully.

FIGURE 2 Lead I EKG tracing (25 mm/s) from patient #2. The first strip (2a) illustrates the change in conduction from normal to a LBBB pattern following 0.25 mg of intravenous atropine. Carotid sinus massage lowered the heart rate and conduction returned to normal in the second strip (2b). The transition in conduction occurred at a rate of 95–97 beats/min.

Discussion

Rate-dependent left bundle branch block (LBBB) occurs when the heart rate exceeds a certain critical value.^{6,7} Many of the previous reports in the anaesthesia literature have emphasized the association of rate-related BBB with bundle branch disease and myocardial ischaemia and infarction.^{1–3,5} Postoperatively, patients presented in these case reports had serial determinations of serum CPK isoenzymes,^{1–3,5} postoperative intensive care admission,² and even cancellation of the surgical procedure.^{2,5} This case report emphasizes that rate-dependent BBB is often benign. It is not usually associated with ischaemia^{6,7} and does not usually affect ventricular performance.^{8,9} As with fixed LBBB, transient rate-dependent left bundle branch block is usually found in patients with hypertension or coronary artery disease (as in the first patient), although up to ten per cent may have no evidence of organic heart disease (as in the second patient).^{10–13} The rate-dependent block tends to become fixed over a period of time.⁷

Although rate-dependent LBBB is usually a benign disorder, diagnosing and treating it are clinically important for several reasons. First, it may mask the electrocardiographic manifestations of other less benign disturbances, such as myocardial ischaemia and infarction.¹⁴ Second, the ST-T wave changes associated with LBBB may be mistaken for ST-T changes due to ischaemia. Rate-related LBBB may also be mistaken for slow ventricular tachycardia and may be inappropriately treated. These two case histories illustrate the use of pharmacologic and

mechanical manoeuvres helpful in diagnosing benign rate-dependent LBBB during anaesthesia.

The change to aberrant conduction commonly occurs at "physiologic" heart rates, less than 80/min, with a change of heart rate by only 1–2 beats.^{10–13,15,16} Studies using atrial pacing show that small decreases (10 msec) in R-R wave cycle length may trigger bundle branch block.^{12,15} These changes are often too small to recognize grossly on the surface EKG. Hence, multiple cycles before and after the change in conduction are needed to recognize the critical cycle length. The onset of tachycardia-dependent bundle branch block is sudden in most patients¹⁶ and once initiated, it persists until the heart rate is slower than that which triggered it.^{12,16,17} The R-R cycle length at which the bundle branch block appears is 80–170 msec shorter than that where conduction returns to normal (zone of linking).^{15–17} The critical rate is also dependent upon the rate of change in cycle length. If the heart accelerates rapidly, the bundle branch block appears at slower rates. If it decelerates rapidly, normal conduction returns at more rapid rates. The cases presented in this report exhibited the above characteristics of tachycardia-dependent LBBB, with transitions between 73–75 beats/min in the first patient and 95–97 beats/min in the second patient.

Electrophysiologic studies have shown that patients with rate-dependent BBB have prolonged bundle branch refractory periods, which do not decrease normally with faster heart rates.¹⁶ The prolonged refractory time of the involved bundle branch is thought to be due to partial depolarization during the relative refractory period (phase 3 block).^{7,18,19} Since the rate of rise of phase 0 of an action potential is determined primarily by the resting membrane potential,¹⁸ if a cell is hypopolarized, the rate of rise of the action potential is slow and conduction is therefore delayed.

Pharmacologic and physiologic manipulations which alter heart rate can be used to change conduction in patients with rate-related bundle branch block. Normal conduction is changed to left bundle branch block in these patients by increasing heart rate with exercise, valsalva, arterial cuff release, amyl nitrate, or atropine.^{20,21} Atropine decreases the functional and effective refractory period of the AV node; it does not have a direct effect on His-Purkinje conduction time or refractory

periods.²² Manipulations which slow heart rate (e.g., carotid massage, deep inspiration, and pharmacological agents like neostigmine, edrophonium or propranolol) change the aberrant conduction back to normal with the slowing of heart rate.^{20,21} This case report demonstrates the use of selected pharmacologic agents (e.g., atropine and neostigmine) and physical manipulations (e.g., carotid sinus massage) to alter the heart rate and confirm the diagnosis of benign rate-dependent left bundle branch block in the perioperative period. These provocative manoeuvres should be used with caution in patients with hypertension, angina, cerebrovascular or AV node disease. Prevention of tachycardia and in some cases deliberately slowing the heart rate, can be of diagnostic and therapeutic value, as demonstrated in use of carotid massage to aid in the diagnosis of acute myocardial infarction in the presence of rate-dependent LBBB¹⁴ and to reduce heart rate in the presence of myocardial ischaemia. However, carotid massage is not advisable in patients with cerebrovascular disease. Propranolol and edrophonium may cause AV block in patients with AV nodal disease. In addition, deliberately increasing heart rate with atropine to induce a rate-dependent BBB may carry the risk of causing an arrhythmia or precipitating myocardial ischaemia. Diagnosis of a rate-dependent BBB should first be attempted by observation of changes in conduction with spontaneously occurring changes in heart rate. If the diagnosis is not obvious and the BBB persists, then judiciously reducing the heart rate may prove helpful. Increasing the heart rate to induce a BBB should be attempted with extreme caution by careful titration of atropine only after myocardial ischaemia has been ruled out. Drug doses should be appropriately chosen to avoid inducing tachyarrhythmias or precipitating myocardial ischaemia. However, in the proper circumstances, the manipulations can be helpful in establishing the diagnosis of benign rate-dependent LBBB in the operating room.

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Résumé

Le bloc de branche gauche (LBBB) dépendant de la fréquence survient occasionnellement durant l'anesthésie lorsque la fréquence dépasse un certain seuil critique. Cette anomalie est habituellement bénigne mais elle peut masquer les manifestations électrocardiographiques d'ischémie myocardique et la forme du tracé ST-T associée à ce bloc de branche peut être interprétée à tort comme indiquant un épisode ischémique. Cette observation présente deux cas où ce bloc de branche lié à la fréquence est clairement démontré durant la période péri-opératoire. On y démontre l'utilité de certains agents pharmacologiques (atropine et néostigmine) et de certaines manœuvres physiologiques (massage du sinus carotidien) pour modifier la fréquence cardiaque et confirmer le diagnostic de cette anomalie. Ces diverses manipulations doivent s'employer avec prudence chez les patients qui présentent de l'hypertension, de l'angine, de l'insuffisance cérébrovasculaire de même que chez les malades présentant des anomalies du nœud auriculo-ventriculaire, de l'ischémie myocardique ou des troubles de conduction marqués dans le faisceau droit et gauche.