TABLE 2. Responses to Treatment with Domperidone Compared with Responses to Placebo

	No Further Symptom*	Recurrent Nausea	Recurrent Vomiting	Second Dose Given	Treatment Failure
Domperidone (n = 23)	7	16	15	8	10
Placebo (n = 23)	4	19	16	9	8

* No further nausea or vomiting after treatment. There was no significant difference between responses to domperidone and placebo.

we previously reported a protective effect against further nausea and vomiting when domperidone, 10 mg, was administered iv to a similar patient population, we felt it important to report and try to explain these findings.

Possible explanations include: 1) the necessity of an initial high blood level for effectiveness; 2) poor absorption from intramuscular sites; 3) insufficient dose; 4) a reduced potency of domperidone in the particular drug lot that we were using.

Either an initial high blood level, obtained with intravenous administration, is necessary for a therapeutic effect, or a larger intramuscular dose is necessary to maintain blood levels in the therapeutic range. Blood levels obtained when domperidone, 20 mg, iv, was given to treat apomorphine-induced vomiting in man were much higher, exceeding for an hour even the peak levels obtained after dosing with 10 mg, im (50 times higher at 15 min, ten times higher at 30 min, 2.4 times higher at 60 min, and two times higher at 120 min).§ These are the only clinical data available for comparison of blood levels achieved by intramuscular and intravenous routes.

Pharmacokinetic studies in human volunteers have shown peak plasma levels are reached 15–30 min after intramuscular administration.¶

Janssen R & D, Inc., analyzed the drug from the lot used and found normal activity.

Since side effects are absent at an intravenously administered dose of 20 mg, a higher intramuscular dose should be studied. However, domperidone's insolubility would require too large a solvent volume to be practical for larger doses.

We conclude that patients are not protected from postoperative nausea or vomiting when domperidone, 10 mg, is administered intramuscularly in single or repeated injections.

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¶ Unpublished research reports supplied by Janssen R & D, Inc., New Brunswick, New Jersey.

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Transient Left-bundle-branch Block during Anesthesia

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Cardiac conduction blocks during anesthesia are uncommon. We present the case of a patient in whom transient left-bundle-branch block developed in association with an episode of hypertension during anesthesia for cholecystectomy.

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REPORT OF A CASE

A 79-year-old woman admitted with the diagnosis of cholecystitis was scheduled for elective cholecystectomy. The patient had had a diaphragmatic hernia repair 15 years previously. She was not diabetic, was not taking digitalis or psychotropic drugs, and had no history of hypertension.

The patient weighed 50 kg. Blood pressure was 120/80 torr and

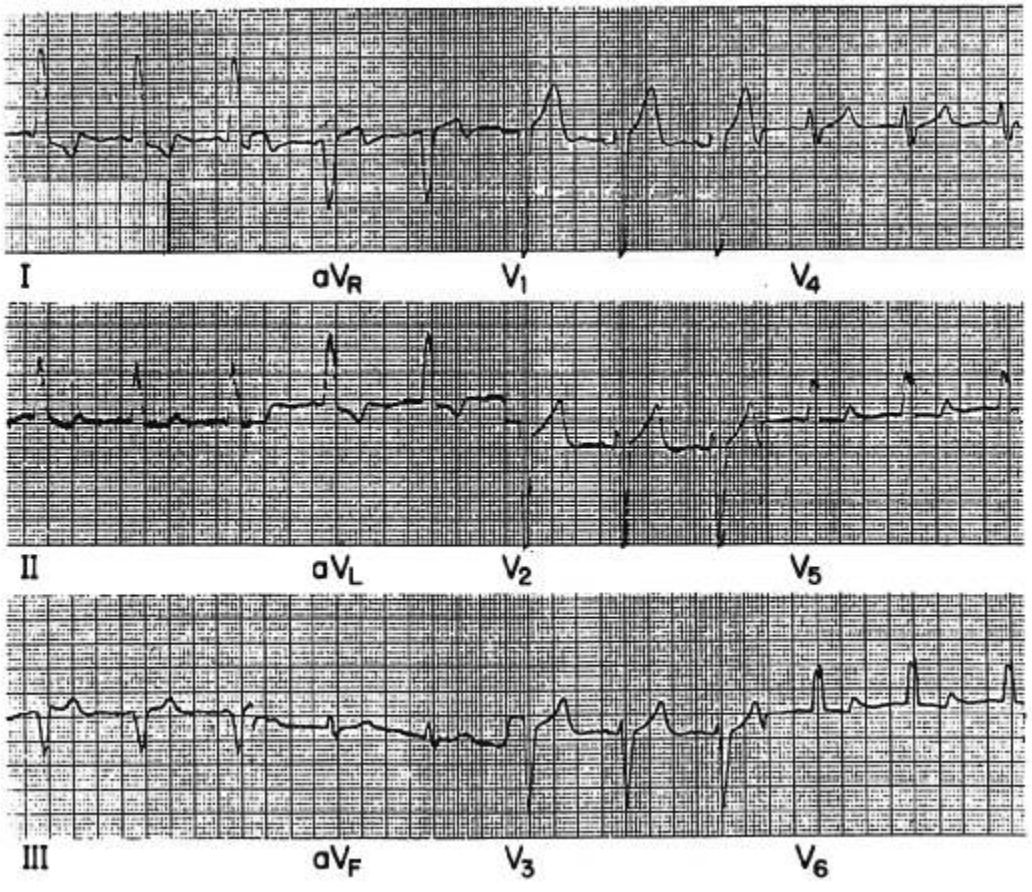

FIG. 1. Electrocardiographic tracing showing left-bundle-branch block.

pulse rate, 90/min and regular. She was anicteric, and all physical findings were normal. The chest x-ray showed aortic-arch calcification and the electrocardiogram, slight left venticular hypertrophy. Hepatic enzymes were slightly elevated: serum glutamic pyruvic transaminase (SGPT) 80 μ/ml, serum glutamic oxaloacetic transaminase (SGOT) 72 μ/ml, creatine phosphokinase (CPK) 200 mIU/ml and alkaline phosphatase 96 μ/ml. Serum bilirubin was 1.1 mg/dl.

The patient was premedicated with morphine sulfate, 10 mg, and atropine sulfate, 0.4 mg, im, an hour prior to operation. She was monitored with an EKG capable of recording all leads. Blood pressure was 120/70 torr and pulse rate 80/min prior to induction of anesthesia. Innovar® (droperidol, 2.5 mg, and fentanyl, 0.05 mg/ml), 1 ml, iv, and thiopental, 0.2 g, iv, were given. Following administration of succinylcholine, 80 mg, iv, endotracheal intubation was performed. Anesthesia was maintained with nitrous oxide, 4 l/min, and oxygen, 2 l/min, in a semiclosed circuit. Fentanyl, 0.05 mg, iv, was given to a total dose of 0.15 mg. *d*-Tubocurarine, 24 mg, iv, was given with return of spontaneous respiration. Vital signs and the EKG remained unchanged.

When the surgeon began to explore the porta hepatis, blood pressure suddenly rose to 160/100 torr. Despite administration of fentanyl, 0.1 mg, iv, and Innovar®, 2 ml, iv, the blood pressure rose to 200/120 torr. At this time a left-bundle-branch block was evident on the cardiac monitor. This was confirmed by an EKG tracing (fig. 1). Halothane, 2 per cent, was added, and the blood pressure gradually fell to 110/60 torr, at which point the left-bundle-branch block disappeared.

The remainder of the surgical procedure was uneventful. The patient was monitored in the recovery room for 18 hours. The EKG returned to preoperative status and the blood pressure was 120–110/70–60 torr. Both the EKG and vital signs remained stable for the nine days the patient spent in the hospital postoperatively. Serially determined serum enzyme levels (CPK, CPK-MB, lactate dehydrogenase, SGPT, SGOT) were normal.

DISCUSSION

Left-bundle-branch block (LBBB) refers to an EKG pattern in which there is alteration of the normal sequence of impulse spread from the septum to the left ventricle,[1] or to an alteration in the manner of transmyocardial spread of excitation throughout the left ventricle.[2] A localized lesion in the left bundle is not necessary; in many cases no such lesion is evident.[3] A delay of impulse conduction through a functionally altered portion of the bundle is enough to produce the EKG pattern of LBBB.[2]

The upper portion of the interventricular septum in 90 per cent of individuals is supplied by the right coronary artery. The lower portion of the interventricular septum carries the major part of the two main bundle branches and the Purkinje arborization.

This is supplied mainly by penetrating branches of the left anterior descending coronary artery (LADCA). The septal branches of the LADCA fix it to the epicardium and limit its range of excursion during systolic filling.[5] Occlusion of the LADCA or one or more of its branches may produce LBBB.[6]

The essential electrocardiographic features of LBBB are a prolonged QRS interval (0.12 sec or more), slurring and notching of the QRS complex and prolongation in time between the onset of the QRS and the peak of R or R^1 to 0.05 sec (normal 0.035 sec maximum) in left precordial leads. In LBBB the initial septal activation is from right to left (normally left to right), and there is no Q wave in left precordial leads.

The majority of cases of bundle-branch block are due to coronary atherosclerosis with recent or old occlusion and associated hypertension. LBBB is commonly indicative of left ventricular hypertrophy, including hypertrophy of the left septal wall due to aortic valvular or subvalvular, coronary, or hypertensive heart disease or idiopathic myocardiopathy.[7] Transient LBBB during anesthesia has been described. In one instance the authors ascribed its development to preoperative lithium therapy.[8] In the second patient, the LBBB was heart rate-related, occurring with a heart rate of more than 115/min.[9] When the heart rate slowed, the LBBB reverted to normal. This was not a factor in our case.

Our patient experienced LBBB when hypertension occurred during anesthesia. We believe there are two possible mechanisms: 1) Zink and co-workers[10] have described dysrhythmias during epinephrine infusion during halothane anesthesia. These occurred only when a critical level of blood pressure was reached. Stretch of Purkinje fibers has been shown to slow conduction velocity and to increase the rate of diastolic depolarization, both of which encourage re-entry. It is likely that a similar mechanism could produce a bidirectional block and cause LBBB.[11] This is particularly true in a patient who had evidence of left ventricular hypertrophy, albeit slight, on the EKG preoperatively with no evidence of hypertension. 2) Light or ineffective anesthesia may lead to a sudden increase in blood pressure and stroke work, directly increasing myocardial oxygen consumption. Increases in heart rate may be masked by reflex action. Although coronary blood flow is autoregulated according to myocardial energy needs, in coronary heart disease there is a point at which perfusion distal to the obstructed area of the artery can no longer increase.[12] An increase in left ventricular end-diastolic pressure leads to an increase of intraventricular pressure, producing further compromise. It is possible that the sudden elevation in blood pressure in this patient prevented segmental increase in blood flow to the LBBB area, with temporary ischemia of the septum and LBBB, which disappeared with effective control of blood pressure.

While LBBB itself is a relatively benign dysrhythmia, it is important during anesthesia, since it may indicate the development of myocardial infarction, in which transient LBBB can occur. It is difficult to diagnose myocardial infarction on the EKG in the presence of a bundle-branch block.

We present a case of transient bundle-branch block occurring during an episode of hypertension in an anesthetized patient. Mechanisms of its production are reviewed.

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