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Treatment of Complete Heart Block in a Patient with Coronary Artery Disease

To the Editor:—In their article describing acute complete heart block (CHB) during anesthesia in a patient with coronary artery disease,¹ Park and Lowenstein report the CHB in their patient to have been resistant to atropine and isoproterenol and discuss etiologic mechanisms and management options for CHB.

Although they cited Shah *et al.*'s article on atropine-resistant CHB,² they omitted discussion of the role of adenosine in ischemic CHB and the implications for treatment thereof. As the electrophysiology of the atrioventricular node has been further elucidated over the last decade, evidence has accumulated that ischemia-induced sinus slowing and atrioventricular block are mediated by adenosine released from myocardial cells rendered hypoxic.³⁻⁸ Historically, these bradycardias were attributed to increased vagal tone because of the commonly associated nausea and diaphoresis. In fact, however, ischemia in dogs and humans is frequently resistant to atropine,^{7,9} and in the setting of inferior wall myocardial infarction, aminophylline, a known adenosine antagonist, has been reported to restore sinus rhythm in patients with atropine- and isoproterenol-resistant complete atrioventricular block.^{2,10}

As coronary artery bypass surgery is used more frequently in the management of patients in the immediate peri- and postinfarction period, it is likely that episodes such as that described by Park and Lowenstein, already familiar to cardiologists in the emergency and coronary care setting, will be managed by anesthesiologists in the cardiovascular operating room with increasing regularity. Because aminophylline may prove to be the only effective pharmacotherapy in such resistant bradyarrhythmias, anesthesiologists should be familiar with this therapeutic option.

LEO I. STEMPE, M.D.
Cardiothoracic Anesthesiology/G3-203
The Cleveland Clinic Foundation
One Clinic Center
9500 Euclid Avenue
Cleveland, Ohio 44195-5076

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In Reply:—We appreciate Stempel's constructive comments regarding our clinical case report.¹ As pointed out by Stempel, heart block is a common occurrence in the setting of an inferior wall myocardial infarction (IMI). In one series of 288 patients with acute IMI,² second- and third-degree heart block was diagnosed in 14% of the patients. These blocks could be grouped as those that developed within 6 h of the first signs of infarction ("early blocks") and those that developed later ("late blocks"). The early blocks were usually responsive to atropine, whereas the late blocks were not. These late blocks are thought to be mediated by release of adenosine by ischemic myocardium.³ Animal studies provide evidence that adenosine released in hypoxia⁴ prolongs the atrioventricular (AV) interval by increasing the atrial-to-His bundle interval. The mechanism for this prolongation is an action upon an extracellular A-1 receptor.^{5,6} Therefore, aminophylline, a

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competitive antagonist of adenosine, can antagonize adenosine-mediated AV block.^{3,4,7} In addition, dipyridamole or erythro-9-(2-hydroxy-3-nonyl)adenine (an inhibitor of adenosine deaminase), which impair adenosine metabolism, can potentiate the effect of adenosine.⁸

In the case we reported,¹ there was no IMI documented prior to the patient's coronary artery bypass grafting, although he had been "ruled in" for a non-Q wave myocardial infarction. If, as we suspected, he had ischemia of the AV node in the periinduction period, his complete heart block would have been an "early" block, making it less likely to be adenosine-mediated and aminophylline-responsive. The editors suggested that in the interest of brevity we omit a full discussion on the role of adenosine in ischemia-induced heart blocks. We agree with Stempel, however, that aminophylline is a viable therapeutic option for bradyarrhythmias in the setting of acute IMI.

KYUNG WON PARK, M.D.
EDWARD LOWENSTEIN, M.D.
*Department of Anesthesia and Critical Care
Beth Israel Hospital
330 Brookline Avenue
Boston, Massachusetts 02215*

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The Use of a Nasogastric Tube as an Aid in Blind Nasotracheal Intubation

To the Editor:—We propose the following method to improve the success rate of blind nasotracheal intubation. First, anesthesia of the nasal mucosa, nasal pharynx, and larynx are achieved *via* a combination of topical and translaryngeal administration of local anesthesia. Second, a nasogastric tube is placed in the tracheal tube. The tip of the nasogastric tube, emerging about 20 cm from the tip of the tracheal tube, is inserted into the nasal cavity to the level of the pharynx. The nasogastric tube acts as a guide as the endotracheal tube is introduced into the nares. When the tracheal tube reaches the pharynx, the nasogastric tube is removed. The tracheal tube is then advanced until its tip reaches the inlet of the esophagus, confirmed by the disappearance of breath sounds. The tracheal tube is then gradually pulled back as

far as the junction of the larynx and the esophagus, and maximum breath sounds return.* Third, the cuff of the tracheal tube is inflated with 15-20 ml air, causing the tip of the tracheal tube to rise from the posterior pharyngeal wall and directing the outlet of the tracheal tube toward the trachea.¹ This purpose is also accomplished by the Endo-

* This first step was described by Kondou *et al.*, who did not, however, refer to the use of the nasogastric tube into the larynx. Kondou K, Toyama T, Shibata Y, Nagao T, Taki K, Ito M, Takeuchi T: An aid of nasotracheal intubation. *Journal of Japanese Dental Anesthesiology* 16:59-62, 1988

FIG. 1. The nasogastric tube is inserted into the trachea through the tracheal tube.

