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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.

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In Reply:—We appreciate Stemp's constructive comments regarding our clinical case report.[1] As pointed out by Stemp, 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 hypoxia[4] 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 competitive antagonist of adenosine, can antagonize adenosine-mediated AV block.[3,4,7] In addition, dipyridamole or erythro-9-(2-hydroxy-3-nanyl)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 Stemp, however, that aminophylline is a viable therapeutic option for bradyarrhythmias in the setting of acute IMI.