7.1 percent. Lung volumes, as anticipated, were scarcely changed from preoperative values.

There are certain other features which render the present case unique in the world literature, so far as we are aware. We have been able to trace only one previous account of multiple unilateral bronchial ruptures. Burke reported the case of a 26-year-old man involved in an automobile accident, in whom it was found at thoracotomy that the bronchus to the right upper lobe had been severed at its origin from the mainstem bronchus, while the intermediate bronchus had been severed approximately one centimeter proximal to the middle lobe orifice. Such an injury is unusual, and it is generally agreed that most cases demonstrate disruption of the mainstem bronchus within 2.5 cm distance from the carina. Our patient supported this observation, save that his coincidental congenital anomaly resulted in rupture both of the mainstem bronchus and also of the independent bronchus leading to the right lower lobe, both structures being located in the same vertical plane. The anomaly itself appears to be extremely rare, and is not referred to in standard texts of bronchial anatomy.

It has been stated that, while traumatic rupture of the bronchus often occurs in the absence of bony injury in patients under the age of 30, over that age it is always associated with fractured ribs on the same side. In our patient repeated x-ray examinations failed to reveal any evidence of fractured ribs, but he suffered a severe fracture of the right scapula.

Finally the present case further substantiates previous reports emphasizing the importance of retaining a high index of suspicion of underlying bronchial injury in the early evaluation of patients who have sustained significant chest trauma.

ACKNOWLEDGMENTS: We are grateful to Drs. J. E. Bachynski and W. E. Beamish for their advice in interpreting the radiographs; to the staff of the pulmonary function laboratory, University of Alberta Hospital, for technical assistance; and to Mrs. Audrey Rothwell for her help in preparing the manuscript.

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Intermittent Heart Block Related to Treatment of Hypertension in a Patient with Acute Myocardial Infarction*

Kenneth Gershengorn, M.D., and Jacob I. Haft, M.D.

A patient with an acute diaphragmatic myocardial infarction and persistent hypertension developed varying degrees of A-V block including complete heart block as his blood pressure was lowered. Block could be abolished by allowing the blood pressure to rise. His bundle electrograms during heart block demonstrated the conduction defect to be proximal to the His bundle in the A-V node. The patient was managed with a stand-by demand pacemaker that allowed safe normalization of his blood pressure.

The treatment of moderately severe hypertension in the face of an acute myocardial infarction is controversial and warrants very careful manipulation. Maintenance of a high pressure is associated with a severe strain to an already damaged myocardium. Lowering of the pressure may reduce coronary blood flow and lead to further damage. Nevertheless, it is commonly felt that where chest pain continues with hypertension in a patient with an acute myocardial infarction, efforts should be made to lower the pressure. We are reporting on a patient with such a clinical state, in whom efforts to lower the pressure were rewarded by relief of the chest pain but were accompanied by progressive degrees of heart block including reversible complete heart block.

CASE REPORT

The patient, a 49-year-old postal employee was entirely well until 30 hours prior to admission when he experienced the sudden onset of sharp precordial chest pain radiating to the left arm, associated with shortness of breath. The pain persisted for five minutes and subsided. That evening he experienced two more episodes of chest pain, once after dinner and again while resting. Later that night, he was awakened from sleep by a more severe precordial pain, associated with diaphoresis, that lasted 30 minutes and abated spontaneously. On the day of admission, while walking in the street, he suffered another episode of severe chest pain, lasting 45 minutes, associated with diaphoresis. Shortly after, he presented to the emergency room at the Bronx Veterans Administration Hospital.

The patient denied any previous episodes of chest pain, shortness of breath, orthopnea, paroxysmal nocturnal dyspnea or edema. He denied any major illnesses or operations and had not been hospitalized before. He did not smoke but admitted to drinking one pint of gin per day on weekends only.

*From the Cardiology Section, Department of Medicine of the Veterans Administration Hospital, Bronx, New York and the Mount Sinai School of Medicine, of the City University of New York.

Reprint requests: Dr. Haft, VA Hospital, 130 West Kingsbridge Road, Bronx 10468

CHEST, VOL. 61, NO. 4, APRIL, 1972
His mother had died of complications of diabetes at the age of 67 and his father had succumbed at the age of 75 with a myocardial infarction. He has no siblings.

On admission to the coronary care unit, the patient appeared to be a mildly obese man in no distress. His blood pressure was 210/130, pulse was 100 per minute and temperature and respirations were normal. Examination of the fundi revealed grade 1 hypertensive retinopathy. Lungs were clear bilaterally. Examination of the heart revealed the PMI to be at the midclavicular line in the fifth intercostal space. The rhythm was regular at a rate of 100 per minute. S₁ > S₂ and A₂ > P₂. Both S₃ and S₄ gallop sounds were heard. No murmurs were audible. The rest of the physical examination was within normal limits.

Initial chest x-ray examination revealed cardiomegaly and minimal pulmonary vascular congestion. Initial ECG revealed ST elevation in leads II, III and aVF and a sinus tachycardia at a rate of 100 per minute (Fig 1).

Hospital Course

The patient was monitored in the coronary care unit and treated with sedation and hydrochlorothiazide. We were reluctant to treat his hypertension aggressively at this time because we felt he already had had an infarction, and that his blood pressure would fall spontaneously. He remained stable with no chest pain and with no significant change in blood pressure until 36 hours after admission, when he awakened with severe substernal chest pain associated with nausea and striking ST elevation in leads II, III and aVF and the appearance of significant Q waves in these leads. At this time, his blood pressure was 180/130. Meperidine was administered and 30 minutes later, his blood pressure fell to 120/80. At this time, his ECG was noted to change to a Wenkebach rhythm followed rapidly by complete heart block with a narrow QRS complex at 60 per minute. The chest pain disappeared following the meperidine. Over the next four hours, his blood pressure rose steadily to 200/130 and his ECG reverted to a Wenkebach rhythm with 3:2 block. It was noted that as his blood pressure rose, he began to experience pressure across the chest and therefore, at this time, trimethaphan 500 mg in 500 ml DSW was administered intravenously with the rate at 30 to 50 drops per minute as needed to lower the blood pressure.

Over the next several hours, when his blood pressure fell with the administration trimethaphan, all traces of pain would disappear. At the same time, the rhythm would revert from a sinus tachycardia with first degree heart block through increasing Wenkebach phases to complete heart block. When the trimethaphan drip was decreased, his blood pressure would gradually rise, complete heart block would disappear and Wenkebach periods would ensue with the block changing from 3:2 to 7:6 as his blood pressure climbed. If the blood pressure rose to a diastolic value of 120 or higher, only sinus tachycardia with first degree heart block was noted (Fig 2). A temporary transvenous pacemaker was withheld at this time, because we felt, in agreement with previous work, that complete heart block with a narrow complex and a diaphragmatic myocardial infarction need not be treated with a pacemaker, contrary to the experience with anterior myocardial infarction.¹⁻¹⁰

Ninety-six hours after admission, however, because of persistent hypertension and development of block whenever the blood pressure was lowered, a transvenous pacemaker was inserted percutaneously via the femoral vein to enable the use of antihypertensive medications safely. Prior to positioning the catheter tip in the right ventricle for pacing, a His bundle electrogram was recorded.

Over the next three days, his blood pressure was lowered to a diastolic of 100-110 with a combination of methyldopa and hydrochlorothiazide and he was noted to have prolonged periods of complete heart block, always with a narrow complex and a ventricular rate above 65 per minute. Enzymes revealed progressive elevation of SGOT and LDH, diagnostic of an acute myocardial infarction. Seven days after admission, the patient was in normal sinus rhythm with a blood pressure of 160/110 and was free of chest pain. The temporary pacing catheter was removed and the remainder of his hospital course was uneventful.

Discussion

The occurrence of hypotension is common following myocardial infarction, both in normotensive and previously hypertensive individuals.¹ Hypertension is less commonly seen. Increased blood pressure following infarction may be transient or persistent. The former is generally of mild degree and causes little concern. Persistent moderately severe hypertension is infrequent, but difficult to manage during the course of acute infarction. The high pressure increases the work load placed on an ischemic left ventricle and may produce severe chest pain.²⁻⁴ Rapid lowering of the pressure will reduce

**Figure 1.** ECG on admission demonstrates sinus rhythm with a P-R interval of .18 seconds. There is ST segment elevation in leads II, III, and aVF with depression in aVR. Q waves are present in leads II, III, and aVF.

coronary blood flow and theoretically could potentiate or aggravate the infarction. In the patient described above, we were faced with this difficult management problem. We elected originally to treat his high blood pressure conservatively to prevent a rapid fall and the possibility of severe hypotension. It is impossible to ascertain, with present diagnostic methods, which patients with acute myocardial infarction will drop their blood pressure spontaneously. In these patients, antihypertensive medications may result in a persistently low pressure.

After 36 hours our patient’s blood pressure remained elevated. At this time he developed chest pain and more marked ST elevations. As the blood pressure was lowered, first inadvertently with meperidine and later purposely with trimethaphan, progressive heart block ensued. Fortunately, this was completely reversible. It is suggested that as the blood pressure fell to the normal range, the blood flow to the region of the A-V node decreased and the resulting progressive degrees of heart block appeared. Diaphragmatic myocardial infarction is frequently associated with occlusive lesions in the right coronary artery. The A-V node artery, in 90 percent of cases, is a branch of the right coronary and compromise to its flow may lead to nodal ischemia and resulting block at the nodal region. The His bundle electrogram recorded during such an episode of complete heart block revealed the site of block to be proximal to the His bundle and confirmed that block was occurring in the A-V node. Every QRS was preceded by a His bundle spike, whereas only conducted P waves were followed by such an electrical event (Fig 3). This finding is in agreement with the work of Rosen and associates11 who found that complete heart block associated with diaphragmatic myocardial infarction usually occurs proximal to the His bundle, in the node, as contrasted with complete heart block associated with anterior myocardial infarction, when block usually occurs distal to the His, presumably in both bundle branches.

The treatment of persistent hypertension in the face of an acute myocardial infarction must be individualized. In patients with diaphragmatic myocardial infarction, the possibility of heart block appearing with lowering of the blood pressure must be considered. The insertion of a temporary transvenous pacemaker may be effective in stabilizing such a situation and providing a margin of safety for antihypertensive therapy.

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