The Significance of Sinus Bradycardia in Acute Myocardial Infarction*

CAPTAIN ROBERT F. HADEN (MC), USA,** PER H. LANGSJOEN, M.D.,
CAPTAIN MORTON I. RAPOPORT (MC), USA
COLONEL JULES J. MCNERNEY (MC), USA

El Paso, Texas

The cause of bradycardia incident to acute myocardial infarction has not been clearly established. As a natural result, its prognostic significance, the importance of its treatment, and the preferred treatment is not well defined. We hope through a review of the literature and our own clinical experience to help clarify these problems.

Sinus bradycardia occurs in a significant number of cases of acute myocardial infarction. Binder and co-workers noted bradycardia of less than 60 per minute in only 2 per cent of 300 acute myocardial infarctions. Imperial et al., however, found sinus bradycardia in 14.9 per cent of 153 cases. On reviewing 50 cases of acute myocardial infarction between 1959 and 1960, we found an incidence of 14 per cent after eliminating all cases in which the sinus bradycardia might have been due to other specific causes, such as digitalis or quinidine.

Lengyel et al. found severe sinus bradycardia to be the first electrocardiographic abnormality in non-anesthetized dogs with experimental ligation of the anterior descending branch of the left coronary artery. Bradycardia incident to myocardial infarction has frequently been attributed to vagal stimulation, but no one has explained exactly how this is mediated. Birchfield, Menee, and Bryant demonstrated that atropine was of no benefit in patients with sinus bradycardia in acute myocardial infarction and concluded that increased vagal tone was not responsible. Corday and Gold noted bradycardia in dogs with acute infarction even after completely denervating the heart. It would seem reasonable to assume that increased vagal tonus is not the cause of all cases of sinus bradycardia incident to acute myocardial infarction.

Sinus bradycardia is known to occur in conditions of myocardial anoxia from varying causes. The slowing of the fetal heart rate during fetal distress or anoxia has been well documented. Varmon and Melville noted significant bradycardia in 65.4 per cent cholesterol fed rabbits following 40 minutes of anoxia induced by inhalation of 10 per cent oxygen. McKain et al., working with dogs, noted sinus bradycardia as a constant result of myocardial ischemia caused by cross clamping the aorta. Bradycardia may occur as a precursor to induced cardiac standstill in human beings whether produced by cardioplegics, hypothermia, or cross clamping of the aorta. We have found in cardiac monitoring during cardiac and other forms of surgery that bradycardia was a common manifestation of myocardial anoxia whether due to blood loss, airway obstruction, shock, or compromise of the coronary circulation.

Birchfield, et al. and James, in separate studies, demonstrated faulty blood supply to the sinus node in their cases of sinus bradycardia complicating acute myocardial infarction. Right coronary artery thrombosis was found in one of our four necropsied cases with sinus bradycardia complicating acute myocardial infarction. Since the sinus node is usually supplied by

*From William Beaumont General Hospital. This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any acceptance of the opinions advanced or any recommendation of such products as may be named.

**Presently at Fort Sill, Oklahoma.
the right coronary artery, this case could be explained by ischemia of the sino-atrial node.

The prognostic significance of bradycardia is likewise not clear. Imperial et al. noted a lower mortality in his cases of acute myocardial infarction with sinus bradycardia and concluded that this might be a basis for a more optimistic outlook. Binder and Sbertoli and Billings and co-workers noted a significant increase in mortality under these circumstances. No very conclusive figures are available. We feel it may be an ominous prognostic sign in some cases.

The following illustrative cases are presented:

Case 1

This 47-year-old white man with a one-half year history of typical angina on effort, was admitted with crushing substernal chest pain of eight hours duration. His pain occurred suddenly while reading in bed, and radiated through the chest into the middle of the back. It was associated with numbness of the arms. He was mildly dyspneic and was admitted to the hospital the following morning.

Past history and review of symptoms were non-contributory. Physical examination: pulse 54 and irregular; blood pressure 110/70; respirations 18/min.; temperature 98°F. oral; examination revealed a slightly obese, middle-aged man in acute distress with crushing anterior chest pain. The physical examination was normal with the exception of sinus bradycardia with a non-phasic sinus arrhythmia.

Laboratory and x-ray data: the electrocardiogram was within normal limits except for sinus bradycardia and non-phasic sinus arrhythmia; the cardio-lipin micro flocculation test was negative; prothrombin activity was 100 per cent. The white blood count was 7,400 per cmm. with 75 per cent neutrophiles, 17 per cent lymphocytes, 2 monocytes, 1 eosinophil, and 2 basophils.

Hospital course: he was placed in an oxygen tent and given 16 mg. of morphone sulfate subcutaneously. He was kept at absolute bed rest and his vital signs were observed every 15 minutes. Approximately three and one-half hours after admission, he complained of dizziness. He was seen immediately by the intern who noted a slow irregular pulse. In a few seconds, he gasped

Figure 1: Electrocardiogram showing sinus bradycardia and a non-phasic sinus arrhythmia. The patient died three hours later and postmortem examination revealed occlusion of the right coronary artery 3 cm. from its origin caused by hemorrhage into a large atheromatous plaque.
and stopped breathing regularly and had only intermittent deep gasps. The peripheral pulse was no longer palpable, and blood pressure could no longer be obtained. Intermittent positive pressure breathing was immediately administered. Auscultation over the precordium revealed an irregular slow rhythm that was interrupted periodically by asystole the length of which gradually lengthened. Intracardiac epinephrine was of no benefit and he expired.

Postmortem examination revealed complete occlusion of the right coronary artery 3 cm. from its origin caused by hemorrhage into a large atheromatous plaque. The lumen of the left coronary artery was also markedly narrowed, but not completely occluded.

In the presence of a normal electrocardiogram, the bradycardia might have been an ominous sign.

Case 2
This 44-year-old white man, who had had mild hypertension for several years, suddenly experienced acute crushing anterior chest pain on the evening of admission. He was brought immediately to the hospital.

Past history and review of the systems were non-contributory. Physical examination: blood pressure 170/100; pulse 56; respirations 20; temperature 98.6°F. He was a well developed and nourished man complaining of anterior chest pain radiating down the inner aspects of both arms. The physical examination was not remarkable except for sinus bradycardia noted on auscultation of the heart.

Laboratory and x-ray data: an electrocardiogram on admission revealed an acute anterior myocardial infarction (Fig. 2).

Hospital course: He was placed at bed rest in oxygen and given 16 mg. of morphine sulfate intravenously. His pain receded and he was comfortable. His vital signs were recorded every 15 minutes and remained stable with no evidence of falling blood pressure, rhythm change or respiratory change. He died suddenly three and one-half hours after admission.

Necropsy revealed thrombosis of the anterior descending branch of the left coronary artery.

Figure 2: Electrocardiogram showing acute anterior myocardial infarction. The patient died suddenly three hours later. Necropsy revealed thrombosis of the anterior descending branch of the left coronary artery.
CASE 3

This 40-year-old white man was in good health until the morning of admission when, while eating his breakfast, he suddenly became weak and nauseated. This was soon followed by left chest pain of a crushing nature which radiated to both shoulders and to the left jaw. He was taken to his local dispensary and found to be ashen gray, cold and clammy and immediately brought by ambulance to William Beaumont General Hospital. After the onset of the pain, he was not able to recall accurately anything until the following day when he discovered he was in the hospital.

Physical examination on admission revealed an ashen gray man in obvious distress with chest pain, disoriented, and unable to answer questions intelligently. The blood pressure was 70/50. The pulse was 40/min. and irregular with frequent premature ventricular contractions with runs of bigeminal rhythm. The lungs were clear to auscultation. The remainder of the physical examination was not remarkable.

Laboratory findings: Serial electrocardiograms revealed an acute posterior myocardial infarction (Fig. 3). Serum oxalic transaminase was 21 units on the day of admission. The following day, it was 41 units, and on the third hospital day it was 30 units. Sedimentation rate was 6 mm./hour on admission and on the second hospital day was 21 mm./hour. The white blood count on admission was 8,000 and on the following day, 10,700 WBC per cmm. with normal differentials. Urinalysis was normal. A cardio-lipin micro flocculation test for syphilis was negative.

Hospital course: He was immediately given oxygen. Intravenous 5 per cent dextrose and water with one ampule of levarterenol was started. The blood pressure was brought up to 105/70 promptly. However, he was still ashen gray and disoriented. It was noted that his heart rate was 40 per minute with frequent premature ventricular contractions with runs of bigeminal rhythm. This was considered a dangerous rhythm because of the possibility of ventricular fibrillation or standstill. Isoproterenol 0.2 mg. was given subcutaneously and over the next 30 minutes, his pulse rate increased to 68 per minute with no premature ventricular contractions. He was more alert and his vital signs were stable. After 12 hours, the levarterenol was discontinued. Isoproterenol 5 mg. every four to six hours sublingually was required for the first two days to maintain a pulse of 50 per minute. He convalesced from his myocardial infarction without complications. He is now two years post-infarction and is asymptomatic on no medication.

FIGURE 3: Serial records revealed an acute posterior myocardial infarction with sinus bradycardia of 30/min.
We feel this man demonstrated marked bradycardia with signs of irritability due to anoxia much as is seen in anoxia from other causes. The administration of isoproterenol resulted in an apparent excellent response.

**Comment**

Bradycardia and other arrhythmias associated with myocardial anoxia are also frequently associated with shock and commonly respond to pressor drugs. Corday and Gold
c have postulated that this effect is due to: (a) enhancing the coronary flow, thus improving the nutrition of the myocardium or removing irritating metabolites; (b) stimulating an inhibitory center in the myocardium; or (c) acting in some unknown manner upon the myocardium directly. Vasopressor agents such as isoproterenol are also known to increase the cardiac rate in the absence of hypotension in heart block with Adams-Stokes syndrome and in sinus bradycardia with Adams-Stokes syndrome. Atropine is also commonly recommended for sinus bradycardia. There appears to be no clear pattern regarding the need for treatment or the type of treatment to be given.

In reviewing 50 cases of acute myocardial infarction occurring between 1959 and 1960, we found an incidence of sinus bradycardia of 14 per cent. Bradycardia was considered to be present when the heart rate was less than 60 per minute. Of this group of seven, four died suddenly. This was the first myocardial infarction for each of these seven patients. This is a mortality of 57 per cent which is considerably higher than our overall mortality. On reviewing all of our myocardial infarctions from 1955 through 1961, we found 508 cases with 92 deaths for a mortality rate of 18.1 per cent. Although our series is limited, this strongly suggests that sinus bradycardia is a poor prognostic sign.

On our service, bradycardia associated with acute myocardial infarction has been a significantly frequent occurrence. Furthermore, our data suggested that it may sometimes be a precursor to cardiac standstill or ventricular fibrillation. From our observations, mainly in cardiac surgery, we feel it reasonable to presume that the slowing of the heart rate might be a direct manifestation of myocardial anoxia. This condition during surgery has frequently responded well to positive inotropic agents such as adrenalin and isoproterenol. We, therefore, have used the milder of these agents (isoproterenol) in the treatment of sinus bradycardia in acute myocardial infarction. We have treated six patients, one mentioned in this paper, with good results and no undesirable reactions in a limited series.

**Summary**

Through review of the literature we have attempted to show that sinus bradycardia may occur in various anoxic states. We have also found that bradycardia is a common occurrence in experimental myocardial infarction in animals accomplished by ligating various branches of the coronary arteries.

Sinus bradycardia occurs in from 2 to 15 per cent of acute myocardial infarctions in humans. We feel there is evidence to suggest that it may possibly be a manifestation of myocardial anoxia and as such may in some cases be a prelude to cardiac standstill or ventricular fibrillation. It therefore deserves close attention and possibly treatment. In our limited experience, isoproterenol has proved effective.

**Acknowledgment:** The authors would like to express their thanks to Mrs. Edna Waddell, Mrs. Geneva Flynn, and Major William R. Thomas, for technical assistance in writing this manuscript.

**Resumen**

Por medio de una revisión de la literatura hemos intentado demostrar que la bradicardia sinusal acontece en varios estados anóxicos. También hemos encontrado que la bradicardia es una ocurrencia común en el infarto miocárdico experimental en los animales, realizado por la ligadura de varias ramas de las coronarias.

La bradicardia sinusal ocurre en el 2 al 15 por ciento de los infartos agudos del miocardio humano. Creemos que hay evidencia que sugiere que posiblemente sea una manifestación de anoxia que miocardio y como tal, puede, en algunos casos, ser un preludio del paro cardiaco o de la fibrilación ventricular. Por esto merece atención estrecha y tratamiento posiblemente. Nuestra experi-
ENCIA LIMITADA MUESTRA QUE EL ISOPROTERENOLO HA SIDO EFECTIVO.

RESUMÉ

GRACE A UNA REVUE DE LA LITTÉRATURE NOUS AVONS ESSAYÉ DE MONTER QUE LA BRADYCARDIE SINUSALE PEUT SURVENIR DANS DES ÉTAT ANOXIQUES DIVERS. NOUS AVONS ÉGALEMENT TRouvé QUE LA BRADYCARDIE SE RENCONTRE COMMUNÉMENT DANS L'INFARCTUS MYOCARDIQUE EXPÉRIMENTAL DE L'ANIMAL, RÉALISÉ PAR UNE LIGATURE DE DIVERSES BRANCHES DES ARTÈRES CORONAIRES.

La bradycardie sinusale survient dans 2 à 15 p. cent de l'infarctus myocardique aigu de l'homme. Nous pouvons suggérer qu'elle peut être une manifestation de l'anoxie myocardique, et de ce fait peut dans quelques cas être un prélude à l'arrêt cardiaque ou à la fibrillation ventriculaire. C'est pourquoi nous mérité une attention suivie et un traitement. Dans notre expérience limitée, l'isooproterenol s'est montré efficace.

ZUSAMMENFASSUNG


REFERENCES


For reprints, please write Capt. Haden at U. S. Army Hospital, Fort Sill, Oklahoma.

INTRACARDIAC ELECTROCARDIOGRAPHIC CHANGES AT THE TRICUSPID VALVE

Intracardiac electrocardiographic changes occurring at the tricuspid valve were studied by Dr. K. K. Datey, Bombay, India, in 51 cases. In the majority the transition from R. V. to R. A. was manifested by the appearance of large biphasic P waves and by sudden fall in the voltage of QRS with change in its pattern. When the catheter tip was at a low atrial level the change in the P waves was minimum, but these became large and biphasic at the mid-atrial level. However, the voltage and pattern of QRS changed in all cases except three. During the withdrawal of the catheter from R. V. to R. A., transient ventricular and atrial premature beats appeared. However, in presence of atrial pressure pulse only atrial premature beats appeared except in cases of Etoab's anomaly, where ventricular premature beats appeared. Significance of these findings was discussed.

Presented at the 7th International Congress on Diseases of the Chest, New Delhi, India, February 20-24, 1963.