
Reprint requests: Dr. Kahn, 1128 Medical Drive, Tyler, Texas 75701.

FATAL SUBDURAL HEMATOMA

Clinical pacemaker failures are usually manifested by return of complete heart block, recurrence of Stokes-Adams attacks or sudden death. In the case presented, the patient sustained cerebral trauma during a Stokes-Adams attack, but symptoms of his subdural hematoma did not become evident until five days after re-establishment of pacing. The possibility of intracranial trauma should be considered following Stokes-Adams attacks especially if changes in cerebral status occur.

Implantable pacemaker systems are generally accepted as definitive treatment for patients with symptomatic complete heart block. However, the unpredictability of battery life remains a problem. Opinion regarding replacement has varied considerably. While Chardack1 recommends elective replacement at 30 months, Zoll2 recommends replacement only because of malfunction or evidence of battery exhaustion.

The following patient is of interest since he developed a previously unreported complication of pacemaker failure after 43 months of continuous pacing.

CASE REPORT

An 82-year-old white man was admitted to the Kansas City Veterans Administration Hospital in October, 1967 with syncopal episodes due to pacemaker failure. The patient had transient complete heart block in 1955. Following the onset of Stokes-Adams attacks in October, 1962, a pacemaker with myocardial electrodes was inserted at another hospital. Failure of this pacemaker occurred in February, 1964 and it was replaced at our hospital by an Electrodyne pacemaker with myocardial electrodes. The patient was seen at regular intervals with pacing at 72 per minute and asymptomatic. In October, 1969, syncopal episodes recurred and the patient was returned to the hospital the following day. The pulse rate was 28 per minute. Pacing was re-established using a temporary transvenous catheter electrode. Because of his advanced age (82), it was felt inadvisable to attempt another thoracotomy. A Medtronic implantable transvenous pacemaker was obtained for insertion. While discussing the proposed procedure with the patient he complained of pain in the hip which had been aggravated during insertion of the temporary catheter electrode. Therefore, following implantation of the permanent pacemaker system, x-ray films of the hips were obtained, but no evidence of bony injury was seen. For the first five days following the implantation of the new unit, the patient did well, pacing at 73 beats per minute without extrasystoles or arrhythmia. He was then noted to be increasingly lethargic, his fluid intake had become inadequate and his blood urea nitrogen had risen to 38 mg percent. However, in spite of correction of fluid and electrolyte problems, the patient's level of consciousness continued to deteriorate during the next 36 hours. No localizing signs were present. A spinal tap showed xanthochromic fluid and the possibility of subdural hematoma was then considered. Bilateral carotid arteriography was undertaken and demonstrated bilateral space-occupying lesions. Following neurosurgical consultation, bilateral frontoparietal trepanations with evacuation of subdural hematoma from both sides was accomplished. Postoperatively, the patient showed some improvement, but never regained consciousness. His condition gradually deteriorated, and in spite of intensive supportive management, he developed pneumonia and expired in January, 1968. Throughout the prolonged postoperative course, there was no difficulty with the cardiac pacemaker system.

Autopsy examination showed resolving bilateral subdural hematomas. There were old infarcts in both occipital lobes of the cerebrum and acute confluent bronchopneumonia of the lungs. The transvenous pacemaker electrode was in good position in the apex of the right ventricle and throughout most of its course had become covered with the smooth thin layer of tissue. At the point where the electrode traversed the tricuspid valve was an area of fibrous thickening.

---

*From the Department of Surgery, Veterans Administration Hospital, Kansas City, Missouri and the University of Kansas School of Medicine, Kansas City, Kansas.
**Chief, Surgical Service and Associate Professor of Surgery.

CHEST, VOL. 57, NO. 3, MARCH 1970
DISCUSSION

In reviewing the course of the patient reported, the diagnosis of subdural hematoma was not initially considered. Although the patient gave a history of striking his hip, he gave no history of head trauma and had done well initially. At the time he became lethargic, he was noted to be dehydrated and because of his advanced age, this was thought to explain his difficulty. Even when loss of consciousness occurred, he was thought to have sustained a cerebral vascular accident, particularly as these have been reported as the cause of death of patients having implanted pacemakers.10-12 It was only after finding xanthochromic discoloration of the spinal fluid that the correct diagnosis was suspected. The delay between the onset of cerebral trauma and his difficulty must be quite uncommon. One can only speculate as to the reason for this. Possibly the lack of trauma is related to the fact that loss of consciousness is not always sudden. Penton, Miller and Levine11 provide a detailed description of the symptomatology in Stokes-Adams attacks and note that patients frequently have a aura or premonition prior to the actual loss of consciousness. Additional evidence to support the possibility of premonitory symptoms which permit the patient to place himself in a more favorable position is also suggested in the report of Burchell.14

Certainly, intracranial trauma should be considered in the differential diagnosis of patients who show changes in consciousness or headache following Stokes-Adams attacks or pacemaker failure.

REFERENCES

14. Burchell, H.B., Connolly, D.C., and Ellis, F.H.
PSEUDOHYPERTENSION


Reprint requests: Dr. Heilbrunn, VA Hospital, 4801 Linwood Boulevard, Kansas City, Missouri 64136.

CHEST, VOL. 57, NO. 3, MARCH 1970

Pseudohypertension Secondary to Aortic Valve Prosthesis*

Anthony R. Geraci, M.D. and Herman L. Falsetti, M.D.

A 57-year-old woman was suspected of having systemic hypertension two years after replacement of her aortic valve with a Starr-Edward prosthesis. The opening and closing sounds of the prosthesis were audible in the antecubital fossa with the blood pressure cuff inflated to 300 mm Hg. Further physical examination and phonocardiography clarified the origin of the sounds leading to the correct diagnosis of pseudohypertension secondary to aortic valve prosthesis. This adds one more cause to the list of possible errors in measuring blood pressure by auscultation.

In the usual clinical situation, blood pressure is measured by auscultation. Although quite adequate in most instances, the measurement of blood pressure may lead to errors which either over or underestimate the actual pressures. The most frequent causes of error are well known: improper cuff sizes, an auscultatory gap, diseases associated with a wide pulse pressure, and the noncompressible brachial artery syndrome.

Recently, we have examined a patient in whom hypertension was suspected. When the auscultatory measurement of her blood pressure was compared with other methods of estimating the arterial pressure, it was apparent that the systolic blood pressure obtained by auscultation was incorrect. Further study revealed the auscultatory artefact to be due to transmission of the opening and closing sounds of a prosthetic aortic valve. This report thus adds one more cause to the list of errors in the auscultatory measurement of blood pressure. Although an uncommon occurrence in other patients examined to date, the number of such patients will probably increase in the future as more aortic valve surgery is performed.

CASE REPORT

A 57-year-old white woman was seen in June 1968, two years after replacement of her aortic valve. She had been asymptomatic until March of 1964, when she noted the onset of shortness of breath on exertion. Her disability became progressive, and she was hospitalized in April, 1966. Physical examination at that time revealed a blood pressure of 115/90 mm Hg in both arms; the pulse was 84 and regular, and the jugular venous pulse was normal. On auscultation a grade 4/6 harsh systolic ejection murmur

*From the Department of Medicine, State University of New York at Buffalo and the Buffalo General Hospital, Buffalo, New York.

Supported in part by research grants from the Heart Association of Western New York.