

Letters

Sleeping Threshold Change Causing Failure of Artificial Cardiac Pacing

To the Editor.—Along with environmental, metabolic, and pharmacologic influences on artificial cardiac pacing, important threshold changes have been reported to occur incident to changes in physical activity.¹ Clinically evident interruption of pacing as the result of these influences has largely been reported as a result of drug administration.^{2,3} We have recently seen a patient in whom a pacemaker malfunction occurred only during sleep.

Report of a Case.—A 74-year-old white man underwent initial implantation of an artificial pacing system in November of 1962 for a stable arteriosclerotic third-degree heart block. Since that time the patient has undergone 12 different procedures incident to the management of various complications causing pacemaker failure. These include, over the years, skin erosion with extrusion of the pulse generator, wire fracture, infection of the battery pocket, electrode dislocation, battery fatigue, and finally threshold change. He was seen in the office on Dec 23, 1969, four weeks after replacement of a generator with a like power unit. He came in advance of his scheduled visit with a history that he had been awakened from a night's sleep and the preceding two evenings by a sensation of irregularity in his pulse. He confirmed this by palpation at the radial level but noted that as soon as he fully awakened and sat up, a satisfactory regular pulse rate was restored. The same sequence of events appeared on the morning of his office visit when he was dozing on the couch. At no time had he noted any irregularity during a waking period. In the office, satisfactory regular pacing was evident at the previously established rate of 72 beats per minute and no postural adjustments or respiratory alterations resulted in variation of that rate. He was promptly hospitalized for confirmation of this history by continuous monitoring of his electrocardiogram (ECG). On the evening of admission, when the patient slept, the previously noted waking, consistent one to one ventricular response to pacing artifact disappeared. Awakening him promptly reestablished capture. Percutaneous insertion of a Keith needle into the output control of the pulse generator

was done at the bedside in the waking state through a skin wheal of 1 cc of 1% xylocaine. A single counterclockwise turn of the needle resulted in cessation of the ventricular response to the pulse artifact confirming that the patient's threshold requirements were indeed almost exactly at the level of output power. Seven clockwise turns were then given to the percutaneous needle resulting in an increase in generator output. An additional period of 24 hours' constant monitoring of the patient's ECG while he was awake and asleep confirmed the reliability of the adjusted system. The patient was discharged the following day and to date (ten months later) has experienced no difficulty with this system.

Comment.—The careful history given by this patient suggested the cause of his pacing system malfunction. The threshold rise during the period of four weeks since implantation of this high-output unit is in agreement with the experience of others.¹ Although apparently infrequent in clinical expression, this mechanism of pacing interruption deserves recognition in the differential diagnosis of pacemaker malfunction.

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2. Morris JD, Judge RD, Leininger BJ, et al: Clinical experience and problems encountered with an implantable pacemaker. *J Thorac Cardiovasc Surg* 50:849, 1965.

3. Haywood J, Wyman MD: Effects of isoproterenol, ephedrine, and potassium on artificial pacemaker failure, abstracted. *Circulation* 32 (suppl 2):110, 1965.

Phenothiazine and Biperiden in LSD Reactions

To the Editor.—As drug abuse continues to distress us, we learn more about new phenomena connected with it and, in my opinion, we should report immediately any new development in this field.

Ordinarily phenothiazine is quite useful in combating the unpleasant symptoms of a lysergic acid diethylamide (LSD) "bad trip." But here is an account of an unusual situation. An 18-year-old boy who had had a "bum trip" on "acid" could not "come down" for two weeks. He experienced hallucinations continuously for 14 days after he drank wine with a group of friends and then was told by one of them that the wine contained a high dose of LSD. This was two weeks before his

visit in my office, still having hallucinations. He was persecuted by paranoid delusions, was in great distress, thinking that he would never recover.

I treated him with 150 mg of phenothiazine twice each day, with the provision that he might take 300 mg at night if necessary. I added 2 mg of biperiden to counteract possible parkinson's disease.

A week later, he reported that as long as he took the biperiden he could not calm down. Only after he stopped taking it, while continuing phenothiazine did his bad trip stop. He had never had symptoms of Parkinson's disease of any kind.

I am reporting this in order to alert my colleagues to the fact that it might be best to use phenothiazine without biperiden in combating LSD effects even if the dosage of phenothiazine is high.

I saw this patient again three weeks later and he informed me that at his prep school he gave biperiden to four boys. They had had "acid trips" before but not flash backs, yet everyone of them had a recurrence after taking one tablet of biperiden. He also reported that phenothiazine, 150 mg would stop his hallucinations but when he was concentrating on a chess game, the hallucinations would come back despite the phenothiazine. This occurred only after he reduced the dose of 300 mg a day to 150 mg at night, however.

Hallucinations occurred also when he was in a prone position in bed, but not when supine.

Many young people who experiment with drugs are at the same time cautious about medicine prescribed by physicians, and this young man wondered how long he would have to take the medicine prescribed.

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The Ankle Sprain

To the Editor.—In reply to Dr. Hilsinger's letter (214:1326, 1970), the severely sprained ankle is truly a problem. This is in no small measure an outcome of inadequate diagnosis and treatment.

The orthopedic literature is liberally sprinkled with papers emphasizing necessity for strain x-ray films of the ankle if there is a high index of suspicion of complete ligament tears.

The presence of a complete liga-