cells, and cultures and fungal staining reactions were negative. The specimens for biopsy revealed portions of normal pulmonary vein, bronchial mucosa, and normal alveoli, with a few chronic inflammatory cells.

Unfortunately, the etiology of the lesion remains unknown, as the patient declined further investigations. Six months later, the patient remains asymptomatic, and the mass has remained unchanged in size.

**DISCUSSION**

Cerebral embolization following the endobronchial instillation of particulate contrast material has not been previously reported. One would expect this complication to be exceedingly rare, but the incidence might be expected to increase if simultaneous fiberoptic transbronchial biopsy and selective bronchographic studies are performed.

Propylidone (Dionosil), one of the most common bronchographic agents, is extremely viscous, whether suspensions in oil or aqueous suspensions are used. If injected via the bronchoscope through a tube with a small bore, considerable pressures are generated. Brisk bleeding is not a rare complication following transbronchial biopsy, and inadvertent biopsy of a pulmonary vessel is usually the cause. If the contrast material is injected near the site of the bleeding and if there is a free communication between the pulmonary vessel and the bronchus, the high pressures required to inject the dye could conceivably lead to an "angiographic" injection. Presumably, this is what occurred in our patient, resulting in the microembolization of particulate contrast material to the cerebral cortex and the immediate grand mal seizure and subsequent transient neurologic defects. The direct visualization of systemic embolization, the time course and the prolonged period of recovery make it unlikely that the patient had an idiosyncratic reaction to either the contrast material or the local anesthesia with lidocaine; however, the possibility of a simultaneously occurring air embolus, caused by clearing of the dead space of the polyurethane catheter prior to injection of the contrast material, cannot be ruled out. As a result of this complication, we have abandoned the use of selective fiberoptic bronchoscopic bronchographic studies where bleeding has occurred following bronchial biopsy.

**REFERENCES**


Propranolol-Induced Dysfunction of the Sinus Node in Wolff-Parkinson-White Syndrome

Elliott M. Berry, M.B., and Yonathan Hasin, M.D.

We report the findings in a patient with Wolff-Parkinson-White syndrome (type A) who initially had recurrent fainting episodes. It appeared that they were caused by prolonged posttachycardiac depression of the sinus node, which was induced by treatment with propranolol. The possibility of covert dysfunction of the sinus node in patients with Wolff-Parkinson-White syndrome should be considered before commencing therapy with β-adrenergic blocking agents.

Syncopal attacks in patients with Wolff-Parkinson-White syndrome are rare, but when such attacks occur, they may be attributed either to paroxysmal tachycardia or to extreme bradycardia. We describe a patient who initially had syncopal episodes that only commenced after he had been receiving therapy with propranolol.

**CASE REPORT**

A 21-year-old man was admitted because of repeated episodes of fainting and palpitations. Six months before this admission, his condition had been diagnosed as Wolff-Parkinson-White syndrome, and treatment with propranolol (10 mg three times daily) had been started. Thereafter, the palpitations became less frequent, but they were followed by episodes of fainting. The findings from physical and laboratory investigations, including an electroencephalogram, were normal. A resting electrocardiogram was typical of Wolff-Parkinson-White syndrome, type A.

In an attempt to provoke a reentrant tachycardia, direct atrial pacing was performed. Figure 1 shows the patient’s 12-lead ECG. Alongside the limb leads, V1 and V2, are seen the widened QRS complexes induced by atrial pacing at 120 impulses per minute. Reentrant tachycardia could not be produced by programmed stimulation of the right atrium, bundle of His; however, after one minute of atrial stimulation, asystole occurred consistently and reached 5.0 seconds (Fig 2, top). Treatment with propranolol was then stopped, and the examination was repeated ten days later.

Figure 2, bottom (A), shows a reentrant tachycardia of 150 beats per minute, with retrograde conduction (presumably via the bypass tract) induced by stimulation of the His bundle. After the spontaneous cessation of the reentrant tachycardia (Fig 2, bottom [B]), there was no significant sinus arrest. Pacing of the atrium and His bundle at different rates disclosed a slightly prolonged recovery time for the sinus node.

It was thus assumed that the posttachycardiac depression of the sinus node was apparently not produced by the mecha-
nism activated by the reentrant tachycardia alone, but rather by an increased sensitivity to therapy with propranolol. All treatment was discontinued, and during 1½ years of follow-up, the patient has not fainted and has experienced only two episodes of palpitations.

**DISCUSSION**

Patients with Wolff-Parkinson-White syndrome may experience recurrent palpitations. Syncope rarely occurs and is considered to be secondary to paroxysmal tachycardia or atrial or ventricular fibrillation; however, abnormalities in the recovery time of the sinus node have been previously described in these patients, but the effect of therapy with β-adrenergic blocking agents was not investigated.

One of the proposed causes of the sick sinus syndrome is imbalance in parasympathetic and sympathetic innervation. Thirty percent of the patients with a clinical history suggestive of the sick sinus syndrome do not have a prolonged asystole after pacing; however, it has recently been shown that after treatment with propranolol, some of these patients develop a prolonged asystole after pacing. It was suggested by Strauss and co-workers that this finding supports the concept of sympathetic innervation activated by the reentrant tachycardia alone, but rather by an increased sensitivity to therapy with propranolol. All treatment was discontinued, and during 1½ years of follow-up, the patient has not fainted and has experienced only two episodes of palpitations.

**FIGURE 1.** Twelve-lead ECG showing sinus rhythm and conduction characteristic of Wolff-Parkinson-White syndrome (type A). Leads L1' through V2' show aberrant conduction during atrial pacing at 120 impulses per minute.

**FIGURE 2.** Top, Asystole of five seconds' duration after cessation of atrial pacing at 120 impulses per minute. Bottom, A (left), Initiation of reentrant tachycardia by stimulation of His bundle. B (right), Spontaneous interruption of tachycardia. Only mild depression of sinus node is evident.
dysfunction as one factor in the development of the sick sinus syndrome.

The patient described herein represents a typical case of the Wolff-Parkinson-White syndrome. Both the progressive widening of the QRS complex during atrial pacing, with the initiation of the ventriculogram before the appearance of the His potential, and also the occurrence of paroxysmal tachycardia following appropriately timed stimulation are characteristic phenomena. The additional malfunction of the sinus node was clearly demonstrated after therapy with β-adrenergic blocking agents; however, the recovery time of the sinus node was prolonged slightly, even after discontinuation of therapy, suggesting an underlying abnormality of the sinus node. We, therefore, assume that the reason for the syncopal attacks in our patient was repeated paroxysmal tachycardia followed by prolonged sinus arrest accentuated by sensitivity to therapy with propranolol. Thus, while patients with Wolff-Parkinson-White syndrome may benefit from treatment with β-adrenergic blocking agents to reduce the episodes of paroxysmal tachycardia, it should be noted that in some cases, there may be additional dysfunction of the sinoatrial node, which would contraindicate such therapy. In these patients, alternative antiarrhythmic therapy should be sought. The use of a challenge dose of propranolol during atrial pacing may be useful to uncover those patients who have such a tendency to malfunction of the sinus node.

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The degree of pleural involvement varies from series to series, but such involvement has been present in at least one-third of the cases related to administration of procainamide or hydralazine. Detailed analysis of the pleural effusions occurring in drug-induced lupus erythematosus have not been included in the major reviews of drug-induced lupus erythematosus. To date, there has been no report of in vivo lupus erythematosus cells in the pleural fluid in cases of drug-induced lupus erythematosus. Recently, we obtained in vivo lupus erythematosus cells from the pleural fluid of an elderly man who had been receiving therapy with procainamide (Pronestyl) hydrochloride for nine months.

CASE REPORT

An 81-year-old man who had been receiving procainamide (Pronestyl) hydrochloride (2 gm/day orally for nine months because of ventricular irritability) initially had acute pleuritic pain in the chest and a left pleural effusion. The electrocardiogram showed atrial fibrillation, with a suggestion of pericarditis. The findings from urinalysis, the blood urea nitrogen level, and the creatinine level were within normal limits. Levels of arterial blood gases with the pa-

Drug-Induced Lupus Erythematosus

with in Vivo Lupus Erythematosus Cells in Pleural Fluid*

Alfred I. Kaplan, M.D., F.C.C.P.; Fouad Zakher, M.D.; and Stanley Sabin, M.D., F.C.C.P.

Pleural involvement in drug-induced lupus erythematosus is not uncommon. Lupus erythematosus cells were found in vivo in the pleural fluid of an elderly patient who had received procainamide (Pronestyl) hydrochloride (2 gm daily) for nine months. Patients who initially have pleural effusions while receiving drugs capable of inducing lupus erythematosus should have the fluid analyzed for lupus erythematosus cells to help clarify the cause of the effusion.

Procainamide (Pronestyl) hydrochloride and other drugs have caused a syndrome which closely resembles systemic lupus erythematosus. Since the first description of the procainamide-induced lupus erythematosus, multiple case records have confirmed the frequency of this complication. Procainamide is the most common drug implicated in this illness. The drug-induced syndromes frequently have initial findings of polyarthralgias, myalgias, fevers, and pleuritic involvement. When compared with systemic lupus erythematosus, renal disease in the drug-induced syndrome is very infrequent. The results of serologic tests mimic those seen in systemic lupus erythematosus, with the exception that the serum level of complement is not decreased and antidouble-stranded DNA also tends to be absent.

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