patients with recurrent tachycardia due to WPW syndrome.\(^2\)\(^-\)\(^4\) Together with the five patients briefly noted in the article by Gallagher and associates,\(^4\) there are now approximately 12 such patients successfully treated with pacemaker therapy that we are aware of, without any untoward effects. The lack of enthusiasm for this particular treatment in WPW syndrome since our initial report has been a concern to us, particularly when compared to the enthusiasm attending the generally unsuccessful surgical approach in this disease. The article by Gallagher and associates\(^5\) further emphasizes the difficulties in surgical correction of the WPW syndrome. The elegant electrocardiographic studies carried out by Dr. Gallagher and associates\(^5\) seem to have been generated by a fascination with the electrophysiologic mechanism, rather than the clinical effectiveness of surgery. In that series of 30 attempts at surgical correction, there were 17 successful procedures, six which were unquestionably successful, five failures and two deaths. Without even considering the operative morbidity one can hardly develop much enthusiasm for this procedure. If one looks at the total world experience, it is characterized by the frequent development of complete atrioventricular block necessitating pacemaker implantation and generally unsuccessful interruption of the accelerated conduction pathway. With this in mind, one must question the advisability of further surgical attempts in this disease.

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References


The author replies:
To the Editor:

We appreciate Dr. Goldstein’s remarks concerning the place of permanent pacemaking in the therapy of reciprocating supraventricular tachycardia (SVT) due to the Wolff-Parkinson-White syndrome. Overlooked in his review of the literature were the contributions of Iwa et al.,\(^1\)\(^-\)\(^4\) who as early as 1970 were using implantable radiofrequency pacemakers for this problem. The lack of enthusiasm for pacing merely points out the inherent limitations of this method of treatment. There is, of course, a place for this particular type of therapy as evidenced by our use of it in five cases reported and in two subsequent cases. We, in fact, seek to assess the effect of pacing during reciprocating SVT at multiple cycle lengths from multiple cardiac chambers in every patient we evaluate, and the decision to surgically interrupt the accessory pathway in the reported cases was in part due to the inadequacy of pacing to effectively terminate SVT. In cases where pacing is effective, the pacing site must be close to the site of the accessory pathway. This would necessitate thoracotomy for implantation of atrial electrodes in many instances, though the coronary sinus might be useful in cases of left sided pathways. Transvenous right ventricular pacing would suffice for right sided pathways, but a subxiphoid exposure would be necessary to implant ventricular electrodes on the left ventricle in the case of a left sided pathway. The endpoint of pacing therapy in any event is not necessarily the ability to terminate an episode of SVT at will. Consideration must also be given to the frequency of attacks, since the result could hardly be considered therapeutically optimal if the arrhythmias continually recurred, or activity states had to be modified in order to avoid provoking arrhythmias.

To view the problem of arrhythmias associated with the Wolff-Parkinson-White syndrome merely as one of controlling reciprocating tachycardia is an oversimplification. While it is true that one mode for the initiation of atrial fibrillation is that of deterioration of reciprocating SVT, this relationship is not invariable. Other mechanisms exist that would not be prevented by pacing, such as paroxysmal atrial flutter-fibrillation as a primary disorder or secondary to coexisting anomalies. In addition, we have observed atrial fibrillation resulting from ventricular premature beats which conduct retrogradely over the accessory pathway to the atrium, arriving there in the atrial vulnerable period. Atrial fibrillation is not infrequently provoked during pacing attempts to terminate the supraventricular tachycardia. Such an occurrence in patients with short antegrade refractory periods of the accessory pathway would be fatal. We recognized that the series reported is a referral one, as evidenced by the high incidence of patients presenting with ventricular fibrillation. We can merely state that every candidate submitted to surgery had a lifethreatening or disabling arrhythmia that had not proved amenable to attempts at control by pacing or drug therapy. As for the results reported, apparent undue emphasis has been ascribed by Dr. Goldstein to the presence of the delta wave. Of the six patients he considered to be "questionably successful" none have had recurrence of arrhythmias despite having presented with intractable arrhythmias. Three of these patients have subsequently undergone electrophysiological study and in two definite functional changes have been demonstrated in the accessory pathway. Of the five failures Dr. Goldstein refers to, three are free of arrhythmia, albeit at the expense of His bundle ablation in two.

One could thus summarize that in a group of 30 patients with intractable arrhythmia unresponsive to pacing or drug therapy, there are 28 survivors, 26 of whom are asymptomatic. Surgical therapy of the Wolff-Parkinson-White syndrome thus appears entirely feasible as a treatment modality for selected patients with the syndrome.

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The Action of Propranolol in Man and Animals

To the Editor:

We should like to take exception to the statements by Nies and Shand that the action of dl propranolol against digitalis-induced cardiotoxicity is primarily related to its quinidine-like effects, with the implication of a predominant direct myocardial action.

This position ignores the work of Gillis demonstrating that glycoside-induced toxicity correlated with increased sympathetic neuronal activity and the occurrence of ventricular tachycardia.\(^3\) When propranolol converted the electrical activity to normal, the sympathetic nerve activity was either normalized or abolished.

We have shown that propranolol alters the electrical mechanism of death in the glycoside intoxicated heart from fibrillation to asystole.\(^8\) Asystole is observed in neurally deprived animals and in vitro hearts while fibrillation is noted in intact animals. We further noted that in the isolated heart, propranolol prolongs the time to the induction of cardiac rhythm disorders by inhibiting the myocardial
Letter: The action of propranolol in man and animals.
J Somberg, H Cagin, J Kleid, R Gillis and B Levitt

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