Cardioversion of Supraventricular Tachycardias

By CARLOS VASSAUX, M.D., AND BERNARD LOWN, M.D.

SUMMARY
Cardioversion was employed in 33 episodes of supraventricular tachycardia in 25 patients. Of these, 18 were episodes of paroxysmal atrial tachycardia with block and the remainder were atrial, junctional, and undefined supraventricular tachycardias. Cardioversion was successful in restoring sinus rhythm in 23 (70%). A major problem was to identify when digitalis intoxication was the cause of the supraventricular mechanism. By progressive titration of the shock energy, it was possible to determine the presence of digitalis intoxication. This was manifested by emergence of ventricular ectopic beats and acceleration in atrial rate. The use of lidocaine abolished the ventricular extrasystoles. Of the 10 patients failing to revert, seven had digitalis toxic arrhythmias. No complications were encountered.

Additional Indexing Words: Acetyl strophanthidin Atrial tachycardia Atrial flutter Nodal tachycardia Digitalis toxicity Lidocaine Paroxysmal atrial tachycardia with block

CARDIOVERSION has been primarily employed in patients having either atrial fibrillation or atrial flutter.1–11 These are the most common chronic disorders of atrial rhythm. Much experience has already been accumulated and many of the details of this procedure have been defined.12, 13

Frequently, the supraventricular tachycardias present difficult diagnostic and therapeutic problems. When they prove unyielding to vagal maneuvers, digitalis glycosides, and antiarrhythmic drugs, the patient is considered as a possible candidate for cardioversion. These refractory arrhythmias commonly occur in patients with significant heart disease who because of congestive failure have been receiving both digitalis and diuretic drugs. It is often uncertain whether or not the arrhythmia is a toxic reaction to the cardiac glycosides. In the presence of digitalis intoxication, electrical shock may provoke serious and even lethal disorders of the heart beat.14–16 How then is cardioversion to be used in patients with these rhythm disorders? The supraventricular tachycardias may seriously compromise cardiac function and must therefore be promptly reverted, and yet cardioversion presents a substantial hazard. The objective of the present report is to detail a significant experience with cardioversion of patients having supraventricular tachycardias and to outline an approach for managing these disorders safely.

Methods
The present study includes 33 episodes of supraventricular tachycardia occurring in 25 patients. These are drawn from approximately 900 cardioversions performed at the Peter Bent Brigham Hospital from December 1961 through July 1968. In all instances, the supraventricular tachycardia had proved refractory to antiarrhythmic drugs. The term "supraventricular tachycardia" includes paroxysmal atrial tachycardia with block (PAT with block), atrial tachycardia, A-V junctional tachycardia, and supraventricular tachycardia of undetermined mechanism. Table 1 presents the distribution of the different supraventricular mechanisms subjected to cardioversion. A number of patients developed more than one arrhythmia. Some of the clinical features

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Table 1

Incidence of Various Mechanisms among 25 Patients Exhibiting 33 Episodes of Supraventricular Tachycardia

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Episodes</th>
<th>Patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAT with block</td>
<td>18</td>
<td>13</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>A-V junctional tachycardia</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Undetermined tachycardia</td>
<td>7</td>
<td>6</td>
</tr>
</tbody>
</table>

* Total exceeds 25 since a number of patients developed more than one single type of arrhythmia.

are highlighted in table 2. On clinical grounds these arrhythmias have been divided into two categories: PAT with block, and other types of supraventricular tachycardias. An arrhythmia was diagnosed as PAT with block when the atrial rate was less than 240/min, an iso-electric base line was observed between successive P waves, and second degree A-V block was present. Atrial or A-V junctional tachycardias were designated when a specific ectopic pacemaker was demonstrable. When the ectopic pacemaker was not identified, the arrhythmia was classified as of undetermined mechanism.

Patients having PAT with block, when compared to the group with other supraventricular mechanisms, were more likely to be afflicted with rheumatic valvular disease, had a higher incidence of heart failure, were older, and were more frequently in chronic atrial fibrillation prior to onset of the supraventricular arrhythmia. Digitalis intoxication was considered as a possible cause for the supraventricular arrhythmia when the arrhythmia developed in patients with chronic atrial fibrillation after increasing doses of glycoside or diuretic drugs. Digitalis was thus implicated in 13 of the 18 episodes of PAT with block and in three of the 15 episodes of other supraventricular mechanisms.

Since PAT with block is frequently confused with atrial flutter, an additional group of 31 episodes of the latter arrhythmia were studied. These episodes were selected from 151 episodes of atrial flutter treated with cardioversion on the basis of an atrial rate of 240 or less. The single electrocardiographic feature that distinguished the patients in this group from patients having PAT with block was the presence of an undulating base line between atrial complexes in one or more leads (fig. 1). The 31 episodes of flutter occurred in 24 patients of whom eight had rheumatic heart disease, eight had coronary artery disease, six had no apparent organic cardiac derangement, one had congenital cardiac abnormalities, and one had pulmonary embolism.

Table 2

Some Clinical Features of Patients with Supraventricular Tachycardias

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>PAT with block</th>
<th>Other supraventricular tachycardias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>13 (18*)</td>
<td>12 (15*)</td>
</tr>
<tr>
<td>Sex:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Female</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Age (yr):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>15–83</td>
<td>7–82</td>
</tr>
<tr>
<td>Median</td>
<td>59</td>
<td>50</td>
</tr>
<tr>
<td>Heart disease:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RHD</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Valve operations</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>CAD</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>HCVD</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>WPW</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Unknown</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Chronic atrial fibrillation</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Digitalis toxicity†</td>
<td>13 episodes</td>
<td>3 episodes</td>
</tr>
</tbody>
</table>

* Number of episodes.
† See criteria in text.

Abbreviation: RHD = rheumatic heart disease; CAD = Coronary artery disease; HCVD = hypertensive cardiovascular disease; WPW = Wolff-Parkinson-White syndrome.

Although many of these patients were receiving digitalis, in the majority, treatment with the drug had been started after the onset of the arrhythmia. In none was there evidence of digitalis intoxication.

The technic for cardioversion has been described previously. A few features are of significance and require recapitulation. Patients were pretreated with pentobarbital sodium one or more hours before the procedure. The majority were anesthetized briefly with 25 to 75 mg of methohexital given intravenously. In the past 2 years diazepam was used almost exclusively. An initial dose of 5 mg was given intravenously and followed every 2 minutes with increments of 2.5 mg until sleep was induced. Blood pressure was checked between successive increments. An anteroposterior paddle placement was employed. In 11 of the 33 episodes of supraventricular tachycardia, the initial energy setting of the cardioversion discharge was 50 to 100 watt-seconds (ws), while in the remaining 22 episodes treated over the past 2 years it was 25 ws or less (table 3). If reversion did not occur, the energy was increased progressively from 5 to
CARDIOVERSION

Atrial Rate
200

Figure 1

With an atrial rate of 200 and varying degrees of atrioventricular block, PAT with block is suggested; however, an undulating base line between successive P waves (lead V1) indicates that the arrhythmia is atrial flutter.

Table 3

<table>
<thead>
<tr>
<th>Energy (ws)</th>
<th>Mechanism</th>
<th>PAT with block (no.)</th>
<th>Other supraventricular tachycardias (no.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Effective*</td>
<td>Initial</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>25</td>
<td>5</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>&gt;25</td>
<td>5</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

* Excludes final energy in those failing to revert.

Table 4

Failure of Cardioversion in Patients with Supraventricular Tachycardia

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Episode (no.)</th>
<th>Failure (no.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAT with block</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>A-V junctional tachycardia</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Undetermined tachycardia</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>10</td>
</tr>
</tbody>
</table>

Results

Cardioversion restored a sinus mechanism in 23 of the 33 episodes of supraventricular tachycardia (70%). Aside from the small group with atrial tachycardia, the highest incidence of failure was observed in patients exhibiting PAT with block (table 4). In only two of the 10 failures was a discharge of 400 ws reached. In eight, the procedure was discontinued before the maximal energy was employed until cardioversion was achieved or a maximum of 400 ws was administered.

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Table 5
Clinical Features of 10 Cardioversion Failures

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Heart disease</th>
<th>Mechanism</th>
<th>Chronic atrial fibrillation</th>
<th>Cardioversion energy (Ws)</th>
<th>PVB*</th>
<th>Atrial rate increase</th>
<th>Digitalis toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.M.</td>
<td>35</td>
<td>M</td>
<td>RHD</td>
<td>PAT c B</td>
<td>+</td>
<td>100</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>D.A.</td>
<td>50</td>
<td>F</td>
<td>RHD</td>
<td>PAT c B</td>
<td>+</td>
<td>100</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>A.K.</td>
<td>37</td>
<td>F</td>
<td>RHD</td>
<td>PAT c B</td>
<td>0</td>
<td>200</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>I.Z.</td>
<td>50</td>
<td>M</td>
<td>RHD</td>
<td>PAT c B</td>
<td>+</td>
<td>25</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>J.W.</td>
<td>67</td>
<td>F</td>
<td>RHD</td>
<td>PAT c B</td>
<td>+</td>
<td>400</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>B.D.</td>
<td>69</td>
<td>F</td>
<td>HCVD</td>
<td>PAT c B</td>
<td>0</td>
<td>50</td>
<td>0</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>D.O.</td>
<td>7</td>
<td>F</td>
<td>Unknown</td>
<td>AT</td>
<td>0</td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>J.F.</td>
<td>35</td>
<td>M</td>
<td>RHD</td>
<td>AT</td>
<td>0</td>
<td>200</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>J.W.</td>
<td>67</td>
<td>F</td>
<td>RHD</td>
<td>UT</td>
<td>+</td>
<td>400</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>L.B.</td>
<td>82</td>
<td>F</td>
<td></td>
<td>Post-operative</td>
<td>JT</td>
<td>0</td>
<td>200</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Abbreviations: PVB = premature ventricular beats; RHD = rheumatic heart disease; HCVD = hypertensive cardio-vascular disease; PAT c B = paroxysmal atrial tachycardia with block; AT = atrial tachycardia; UT = undetermined tachycardia; JT = nodal tachycardia.

### RELATION OF ATRIAL RATE TO RESULT OF CARDIOVERSION IN 33 EPISODES OF SUPRAVENTRICULAR TACHYCARDIA

**Figure 2**

Low atrial rate does not preclude cardioversion, note apparent lack of correlation between rate and success of reversion.

delivered, in six of these because of the occurrence of increasing numbers of multiformal ventricular ectopic beats. In seven of the 10 failures evidence suggested that the arrhythmia was due to digitalis intoxication (table 5). Of the seven failing to revert and judged to be in digitalis toxicity, six developed ventricular ectopic beats during cardioversion; the one patient without ectopic beats had received a number of antiarrhythmic drugs just prior to the procedure. The extrasystoles increased in frequency with higher energies of discharge. In six of the seven with digitalis intoxication the atrial rate accelerated after electrical shock. All but two of the patients judged to be digitalis intoxicated had been in chronic atrial fibrillation prior to the development of the supraventricular mechanism.

It has been stated that when the rate of an arrhythmia is slow the disorder is difficult to restore to a sinus mechanism by means of cardioversion. This conclusion is not borne out by the present experience (fig. 2). Thus the mean atrial rate in the 10 failures was 183/min as compared to 171/min in the 23 successfully treated. The slowest rate at which cardioversion was effective was 130/min. The energy requirement for reversion was unrelated to the atrial rate.

**PAT with Block**

Digitalis intoxication has been regarded as a frequent cause of PAT with block. In the 13 patients having PAT with block in our series, the etiology was uncertain, and the arrhythmia did not respond to the usual antiarrhythmic agents employed to combat overdosage of digitalis. Cardioversion was not attempted immediately; indeed, it was the last resort. This is supported by the fact that the mean duration of arrhythmia prior to reversion was 8 days with a range of 1 to 30 days. Eight of
the 13 patients had chronic atrial fibrillation prior to development of PAT with block. The remaining five patients had frequent paroxysms of diverse atrial arrhythmias over many years. The development of PAT with block in patients with long-established atrial fibrillation has been regarded as strong evidence of digitalis intoxication. In 13 of the 18 episodes of PAT with block, digitalis was initially suspected as the cause of the arrhythmia. Yet seven of these 13 episodes, or over half, were readily restored by means of cardioversion to sinus rhythm, and the clinical impression of digitalis intoxication was not confirmed.

In the 12 episodes of PAT with block in which reversion was successful, it was achieved with low energies. In all but one, less than 100 ws was effective. In this one episode, the initial discharge setting was at 100 ws; however, when the patient presented with the same arrhythmia at a subsequent occasion, he responded to a single shock of 25 ws. In seven of the 12 episodes the reversion energy was 25 ws or less; in two it required 5 ws, and in one, 1 ws.

The special problems presented by this group are illustrated by the following clinical summaries:

**Case A.K.**

A 37-year-old woman with asymptomatic mitral stenosis had frequent paroxysms of supraventricular arrhythmia over a period of 2 years. P waves could not be identified; however, right intra-atrial leads indicated the mechanisms to be atrial tachycardia. Quinidine, procainamide, and diphenylhydantoin in substantial doses did not prevent recurrences. The atrial arrhythmia exhibited at atrial rates ranging from 170 to 200/min and responded to cardioversion discharge of 50 ws. The most effective prophylactic measure was digitalization, and she was therefore maintained on 0.25 mg of digoxin daily.

When she came to the hospital and was found to be in PAT with block, digitalis was not suspected as the causative agent since she denied symptoms or change in drug schedule. The first cardioversion discharge of 5 ws resulted in ventricular bigeminy and acceleration of atrial rate (fig. 3). Lidocaine and procainamide in doses of 150 mg and 250 mg, respectively, controlled the ectopic beats. Higher energies up to and including 200 ws were unsuccessful in restoring sinus rhythm. Careful questioning after the procedure indicated that she had suffered for several weeks from chromatopsia and anorexia. Examination of the drug that she had been taking indicated that digitoxin, 0.2 mg daily, rather than digoxin had been prescribed, and she had been taking this medication for 3 months. It required large doses of potassium and procainamide administration over a period of 3 days before sinus rhythm was restored.

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**Figure 3**

A 5-ws cardioversion discharge resulted in ventricular bigeminy and acceleration of atrial rate (204 to 330) in a patient (A.K.) having PAT with block due to digitalis overdose.
Comment. Three points deserve emphasis:

1. The atrial tachycardia responded to a cardioversion discharge of 50 ws; however, when digitalis was the provocative factor four times this amount of energy was without effect. (2) When the arrhythmia was digitalis-induced PAT with block, an initial discharge of 5 ws resulted in ventricular bigeminy and a transient increase in atrial rate to 330. Had higher energies been employed without antiarrhythmic drug protection, more serious or even fatal arrhythmias might have resulted. (3) After discontinuation of digitalis it required administration of potassium and procainamide for 3 days before the PAT with block gradually receded. In about 200 patients having PAT with block and treated to date, such a slow resolution of the arrhythmia had not been observed. It is our view that electric shock was the factor responsible for the unusual persistence of the PAT with block.

Case B.D.

A 69-year-old woman with hypertensive cardiovascular disease, was admitted to the Peter Bent Brigham Hospital for the first time because of increasing congestive heart failure over the preceding 5 days. On admission she received 1.25 mg of digoxin intramuscularly in divided doses and thereafter a single dose of 0.25 mg orally. During the initial 14 hours the rate of the sinus tachycardia slowed from 150 to 110/min; thereafter the heart rate jumped to 180/min. P waves could not be identified in standard leads. The presumption was that the arrhythmia was an atrial tachycardia due to digitalis. Neither diphenylhydantoin in a dose of 250 mg nor propranolol, 10 mg, both given intravenously, had any effect on the arrhythmia. A right intra-atrial lead established the mechanism as atrial tachycardia (fig. 4).

The presence of normal serum electrolytes, the fact that she had received but a small dose of digoxin, and the failure to respond to diphenylhydantoin and propranolol suggested that the mechanism was not digitalis induced. Cardioversion was therefore attempted. An initial discharge of 5 ws was followed with shocks of 25

![Figure 4](http://circ.ahajournals.org/)

The diagnosis of atrial tachycardia with 1:1 conduction was confirmed by a right intra-atrial lead. The arrhythmia was suspected to be due to digitalis intoxication; however, neither diphenylhydantoin nor propranolol restored sinus rhythm. Propranolol induced transient 2° A-V block.
CARDIOVERSION

Figure 5

Cardioversion discharges of 25 and 50 ws resulted in progressive acceleration in atrial rate from
160 to 200/min suggesting that the arrhythmia was due to digitalis intoxication.

and 50 ws. The atrial rate which was 160 increased transiently to 200 after the last shock (fig. 5).
This acceleration suggested that the mechanism was due to digitalis overdosage. To resolve the
uncertainty an acetyl strophanthidin tolerance test was carried out.18 After an intravenous dose
of only 0.3 mg acetyl strophanthidin, the atrial rate increased from 160 to 190 with emergence of
2:1 A-V block, suggesting that the arrhythmia was due to digitalis overdosage (fig. 6). Within
the ensuing 24 hours, after discontinuing use of digitalis and administering potassium and
propranolol, a heart rate similar to the previous was maintained, with emergen-ce
of atrial rate
200

Comment. Electric shock resulted in acceleration of atrial rate similar to that occurring
after additional doses of digitalis. When digitalis is not responsible for supraventricular
tachycardia, transthoracic shock, even at a high energy level, does not significantly
accelerate the heart rate. The absence of ectopic beats after electrical discharge may
have been due to the rapid ventricular rate as well as to pretreatment with antiarrhythmic
drugs. Although diphenylhydantoin19–21 and propanolol22 have been regarded as highly
specific for digitalis-induced arrhythmias, in this patient they were without effect and
thereby led to an incorrect interpretation of the cause for the arrhythmia.

Case R.S.

A 60-year-old woman with terminal rheumatic heart disease with two previous mitral valvular
operations was admitted to the Peter Bent Brigham Hospital with far-advanced right and
left sided congestive heart failure. She had been on maintenance digoxin and required large doses
of ethacrynic acid and furosemide daily. She had been in chronic atrial fibrillation over many years.
On admission she was found to be in PAT with block with an atrial rate of 145 and variable
degree of A-V block with periodic 1:1 atrioventricular response. It was presumed that she
was in digitalis intoxication. However, antiar-
rhythmic drugs and potassium supplementation
did not alter the arrhythmia; cardioversion was
therefore employed. The initial shock of 1 ws
resulted in transient A-V junctional mechanism
promptly followed by sinus rhythm (fig. 7).

Comment. When an arrhythmia develops in
a digitalized patient there is usually no certainty
as to the role of digitalis. The electrocardio-
graphic pattern is never decisive in determin-
ing whether the disorder has been induced by
the cardiac glycosides. Cardioversion utilizing
Acetyl strophanthidin (AS) in a dose of 0.3 mg given intravenously resulted in acceleration of atrial rate and emergence of 2° A-V block suggesting digitalis intoxication as the cause of the tachycardia.

In patient having PAT with block a single 1-ws cardioversion discharge resulted in nodal rhythm which in 30 sec shifted to a sinus pacemaker.

small energies may provide information not otherwise obtainable. If the arrhythmia is readily reverted, it is unlikely that digitalis drugs are implicated. If, however, the electrical discharge accelerates the atrial or junctional rate and provokes ventricular ectopic beats, the abnormal rhythm is digitalis-induced. In such instances cardioversion, even with high energies, usually is ineffective in restoring sinus rhythm. The use of high energy shocks is hazardous.

**Differentiation of PAT with Block from Slow Atrial Flutter**

PAT with block is frequently confused with flutter. About 20% of patients with atrial flutter have atrial rates within the range ascribed to...
PAT with block. Differentiation between the two disorders is important since approximately 70% of episodes of PAT with block and usually none of flutter are due to digitalis intoxication. In the present series, patients with PAT with block were selected from a larger group on the basis of failure to respond to antiarrhythmic drugs, and therefore the PAT with block was presumed not to be due to digitalis. However, in one third of the episodes, the arrhythmia probably resulted from digitalis intoxication on the basis of their response to cardioversion and their subsequent clinical course. By contrast with this group, in none of the 24 patients with atrial flutter, having slow atrial rates, was the arrhythmia due to overdigitalization. An additional important feature differentiating these two mechanisms is the atrial rate (fig. 8). Only one patient with flutter had an atrial rate less than 200, while only one patient having PAT with block had a rate in excess of 220/min. Cardioversion was successful in all the patients with atrial flutter.

Supraventricular Tachycardias Other than PAT with Block

Of the 15 episodes in this group, 11 were restored to sinus rhythm. One patient with long-standing atrial fibrillation developed supraventricular tachycardia as a result of excessive digitalis. Despite high energy shocks and the use of antiarrhythmic drugs, the arrhythmia could not be terminated. There were three other failures. The mean energy requirement for termination of tachycardia was 42 ws. Five patients were restored to sinus rhythm with 5 ws or less. The lowest effective energy was 1 ws in a patient with supraventricular tachycardia of undetermined mechanism. Included in this group were two patients with Wolff-Parkinson-White syndrome with atrial rates of 190 and 184 and 1:1 A-V conduction in whom cardioversion was achieved with 25 ws and 75 ws, respectively. No complications were encountered.

Discussion

The role of cardioversion for terminating supraventricular tachycardias, exclusive of atrial fibrillation and atrial flutter, hitherto has not been defined. To date there are four reports in the English language literature dealing with eight cases of supraventricular tachycardia. An additional nine cases may be gathered from various general reports on the subject of cardioversion. The paucity of experience probably relates to the reluctance of using this technic in patients having arrhythmias which may be due to digitalis. Indeed, a number of deaths following cardioversion occurred in patients with digitalis-induced supraventricular mechanisms. Yet the greatest urgency for reversion to a normal rhythm frequently exists in patients with these particular disorders. Usually the tachycardia has been present for several days. Since these arrhythmias generally occur in patients with substantial organic heart disease and advanced degrees of decompensation, the rapid ventricular rate further encroaches upon an already marginal cardiac reserve. Furthermore, since digitalis is commonly suspected as the provocative agent, the patient is denied the benefit of digitalis as well as diuretic drugs. When antiarrhythmic agents fail to restore sinus rhythm, the physician is then left with few if any therapeutic alternatives.

The present experience provides a clinical approach to these arrhythmias as well as guidelines for the safe use of cardioversion. When confronted with a supraventricular tachycardia, the first objective is to identify
precisely the underlying mechanism. It is important to distinguish between PAT with block and other supraventricular mechanisms. In the former, unlike the latter group of disorders, the incidence of digitalis intoxication is high. In the absence of identifiable P waves, the differentiation can be readily made in a majority of cases on the basis of ventricular rate. In PAT with block, the ventricular rate is usually irregular due to variable degrees of atrioventricular block. If the mechanism is regular and the ventricular rate exceeds 120, PAT with block is unlikely unless the arrhythmia is in the phase of 1:1 atrioventricular conduction. If this is the case, carotid sinus stimulation is diagnostic if it slows the ventricular rate and divulses the hitherto concealed P waves. In the absence of such a response, it is mandatory to identify the atrial mechanism. This can now be readily accomplished by passing a pacemaker electrode wire into the right atrium. The thin wire is inserted percutaneously through a polyethylene catheter placed in the basilic or jugular vein.

If the atrial rate exceeds 220/min and there is an advanced degree of A-V block, the mechanism almost invariably is due to atrial flutter. This is confirmed by the presence of an undulating base line between successive P waves even in a single lead. Once the rhythm is identified as atrial flutter, no further temporizing is necessary. Cardioversion is the treatment of first choice. The success rate is nearly 100%. Reversion can be accomplished at low energies and without any complications.

If the mechanism is found to be atrial or junctional tachycardia and the arrhythmia has proved refractory to vagal maneuvers and such antiarrhythmic drugs as diphenylhydantoin and propranolol, cardioversion is then the indicated treatment. Under these circumstances the likelihood that the arrhythmia is due to digitalis intoxication is less than 10%. Again low discharge energies are effective, and complications are few and minor.

If the mechanism is identified as PAT with block, the initial treatment should be with antiarrhythmic drugs. If, however, the patient had not been receiving digitalis drugs, there should not be any hesitation in employing cardioversion. The response is the same as in atrial flutter. When the possibility of digitalis intoxication is entertained, treatment should be started with such drugs as diphenylhydantoin, propranolol, lidocaine, and procainamide. The arrhythmia presents no therapeutic emergency since the ventricular rate usually is not rapid. If these drugs prove ineffective, cardioversion is the next appropriate measure. However, the fact that drugs considered to be antidotes for the arrhythmias of digitalis intoxication have failed does not completely rule out the possibility that the arrhythmia results from digitalis overdosage. Indeed, in the present study one third of the patients having PAT with block and subjected to cardioversion responded as though they were overdigitalized. This was judged by development of accelerated atrial rates or emergence of ventricular ectopic beats or both following low-energy shocks of 1 to 10 ws.

In the present series, complications were prevented by titration of energy during cardioversion to either an effective or minor toxic end point and by the intravenous use of lidocaine. When the shock does not accelerate the atrial rate but causes ventricular extrasystoles, it is safe to proceed with higher energies provided that lidocaine completely suppresses these ectopic beats. When the shock accelerates the atrial rate and provokes ventricular premature beats, it is usually unlikely that higher discharge energies will restore a normal mechanism. When lidocaine proves ineffective or merely abolishes the ectopic beats transiently, cardioversion should be abandoned.

It has been demonstrated that low-energy shocks may expose the state of overdigitalization. In the experimental animal the early period of diastole is the most sensitive part of the cardiac cycle to electric shock-evoked digitalis arrhythmias. Stimuli with very low-energy content, of as little as 1 microjoule, are frequently sufficient to induce a repetitive ventricular response. In cardioversion the electric impulse is delivered during inscription
of the QRS complex and corresponds to the absolute refractory period. This part of the cardiac cycle is generally refractory to shock-evoked digitalis arrhythmias. When such arrhythmias develop during cardioversion, the presence of an advanced degree of digitalis poisoning may be suspected. The employment of low-energy discharges in cardioversion may not only provide a definitive therapeutic result, but at times may afford otherwise unobtainable evidence of the presence of digitalis intoxication.

References
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Anatomy and the Curriculum

Why Not?*

In contrast to the "monolithic" concept held by many—that anatomy is a study of dried bones and cadavers—the present research frontiers of anatomy are most exciting. The anatomist may still dissect the body as part of his teaching responsibilities, but he also utilizes a variety of research technics which are heavily dependent on the methods and instrumentation of chemistry and physics. He dissects tissues into their component structures under the binocular microscope and cells into their subcellular organelles by means of the ultracentrifuge. Although the modern anatomist is still studying bones, he is concerned with the dynamic aspects of bone, i.e., with the fourth dimension of structure—time. The new vistas of anatomy are developing as rapidly as are those in other fields of biomedical research.—From J. ARTHUR MYERS: Masters of Medicine: An Historical Sketch of the College of Medical Sciences, University of Minnesota 1888-1966. St. Louis, Warren H. Green, Inc. 1968, p. 263.

*Some men see things as they are, and say "Why?"
I dream of things that never were and say "Why not?"—G. B. Shaw
Cardioversion of Supraventricular Tachycardias
CARLOS VASSAUX and BERNARD LOWN

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