Effect of Potassium on Conduction and Ectopic Rhythms in Atrial Fibrillation Treated with Digitalis

By Charles Fisch, M.D., J. Paul Shields, M.D., S. Anthony Ridolfo, M.D., and Harvey Feigenbaum

Intravenous administration of potassium to patients with atrial fibrillation treated with digitalis resulted in depression of conduction through the A-V node, appearance of complete A-V dissociation and in some abolition of ventricular ectopic beats. The presence or absence of ectopic ventricular beats as well as the degree of depression of A-V node could be controlled at will by varying the rate of infusion. Termination of infusion was paralleled by prompt reappearance of all arrhythmias present before infusion started.

Potassium is used in the treatment of cardiac arrhythmias due to digitalis as well as some arrhythmias not associated with digitalis therapy. A number of the latter no doubt represent cases of arrhythmias secondary to potassium depletion such as are encountered in diarrheal states, vomiting, and severe heart failure. There are instances of cardiac arrhythmia, however, in which potassium depletion cannot be demonstrated, nor is there reason to suspect that a state of depletion exists; yet some of these respond to potassium. It is very likely that the response of many of these arrhythmias is due to the well-known, nonspecific, depressing effect of potassium and may have nothing whatever to do with replacement of serum or total body potassium or “antagonism” to digitalis. The direct relation of the degree of depression of myocardium to the rate of infusion and the concentration of potassium delivered to the heart itself was stressed in 1930 by Wiggers. Elevation of serum potassium has been noted to decrease conduction through the A-V node. Such a depression of conduction is manifest by prolongation of P-R intervals and occasionally by A-V dissociation due to interference. In patients with atrial fibrillation the depression of conduction is manifest by slowing of ventricular rate. Higher grades of block such as second degree or complete A-V dissociation have not been observed in humans.

Because of these earlier reports it was thought that this problem warranted further study with emphasis on (1) the relation of changes of ventricular rate and rhythm to rate of infusion and (2) the speed with which such changes may take place. Patients with atrial fibrillation seemed best suited for such an investigation because (1) it is easier to determine the “degree” of digitalization in patients with atrial fibrillation than in individuals with sinus rhythm; (2) if potassium and digitalis have an opposite effect, then administration of potassium to individuals with atrial fibrillation and A-V nodal escape beats or A-V nodal escape rhythm due to digitalis should result in increased A-V conduction with speeding of ventricular rate and disappearance of A-V nodal escape beats and A-V nodal rhythms; and (3) if disappearance of ectopic ventricular beats were due to opposite effects of potassium and digitalis, it should be accompanied by simultaneous increase in A-V conduction and speeding of ventricular rate.

Method

Seventeen patients with atrial fibrillation with varying forms of heart disease were selected from the Robert Moore Heart Clinic and wards of Indianapolis General Hospital. All but one had su-
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### Table 1.—Effect of Potassium on Heart Rate and Rhythm

<table>
<thead>
<tr>
<th>Patient</th>
<th>Solution infused</th>
<th>Rate of infusion</th>
<th>Ventricular rate prior to infusion of potassium (mEq. per L.)</th>
<th>Slowest ventricular rate during infusion of potassium (mEq. per L.)</th>
<th>Change in rhythm</th>
<th>Ventricular rate 2 hours after infusion</th>
<th>Before infusion</th>
<th>During infusion</th>
<th>End of infusion</th>
<th>1 hour after infusion</th>
<th>2 hours after infusion</th>
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<tr>
<td>1</td>
<td>K phosphate</td>
<td>Varying</td>
<td>50</td>
<td>34</td>
<td>A-V dissociation</td>
<td>60</td>
<td>4.9</td>
<td>6.2</td>
<td>6.7</td>
<td>5.5</td>
<td></td>
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<tr>
<td>2</td>
<td>K phosphate</td>
<td>Varying</td>
<td>53</td>
<td>35</td>
<td>A-V dissociation</td>
<td>57</td>
<td>5.7</td>
<td>4.7</td>
<td>5.3</td>
<td>5.9</td>
<td>5.7</td>
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<tr>
<td>3</td>
<td>NaCl-KCl</td>
<td>Varying</td>
<td>60</td>
<td>41</td>
<td>A-V dissociation</td>
<td>69</td>
<td>5.4</td>
<td>4.6</td>
<td>4.8</td>
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<td></td>
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<tr>
<td>4</td>
<td>K phosphate</td>
<td>Varying</td>
<td>63</td>
<td>39</td>
<td>A-V dissociation and disappearance of ventricular bigeminy</td>
<td>65</td>
<td>5.5</td>
<td>4.0, 4.7</td>
<td>5.9</td>
<td>4.9</td>
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<td>5</td>
<td>K phosphate</td>
<td>Varying</td>
<td>57</td>
<td>40</td>
<td>Appearance of ventricular escape beats and idioventricular rhythm</td>
<td>76</td>
<td>5.3</td>
<td>4.0</td>
<td>4.7</td>
<td>6.3</td>
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<tr>
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<td>K phosphate</td>
<td>Varying</td>
<td>68</td>
<td>45</td>
<td>Disappearance of ventricular escape beats and idioventricular rhythm</td>
<td>65</td>
<td>5.0</td>
<td>5.4</td>
<td>7.0</td>
<td>6.6</td>
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<tr>
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<td>NaCl-KCl</td>
<td>Varying</td>
<td>60</td>
<td>60</td>
<td>A-V dissociation and disappearance of ventricular bigeminy</td>
<td>4.0</td>
<td>4.1</td>
<td>4.4</td>
<td>4.5</td>
<td>4.1</td>
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<td>Varying</td>
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<td>41</td>
<td>A-V dissociation</td>
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<td>4.6</td>
<td>4.9</td>
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<td>K phosphate</td>
<td>Varying</td>
<td>74</td>
<td>54</td>
<td>No change</td>
<td>89</td>
<td>5.7</td>
<td>4.5</td>
<td>5.1</td>
<td>4.9</td>
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<td>Varying</td>
<td>74</td>
<td>65</td>
<td>A-V nodal escape beats</td>
<td>87</td>
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<td>A-V nodal escape beats</td>
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<td>63</td>
<td>No change</td>
<td>79</td>
<td>4.6</td>
<td>4.7</td>
<td>5.5</td>
<td>5.6</td>
<td>5.0</td>
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<td>Uniform</td>
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<td>60</td>
<td>No change</td>
<td>85</td>
<td>5.0</td>
<td>4.6</td>
<td>5.3</td>
<td>4.5</td>
<td>4.6</td>
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<td>Uniform</td>
<td>62</td>
<td>71</td>
<td>Speeding of A-V nodal rhythm present prior to infusion</td>
<td>68</td>
<td>3.3</td>
<td>2.7</td>
<td>3.6</td>
<td>3.9</td>
<td>3.5</td>
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<tr>
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<td>89</td>
<td>79</td>
<td>No change</td>
<td>88</td>
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<td>4.1</td>
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<td>5.4</td>
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<tr>
<td>16</td>
<td>NaCl-KCl</td>
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<td>88</td>
<td>58</td>
<td>A-V nodal escape beats</td>
<td>83</td>
<td>4.6</td>
<td>4.4</td>
<td>5.4</td>
<td>5.7</td>
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<tr>
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<td>NaCl-KCl</td>
<td>Uniform</td>
<td>78</td>
<td>78</td>
<td>No change</td>
<td>92</td>
<td>3.6</td>
<td></td>
<td></td>
<td>4.5</td>
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</table>

praventricular conduction, the latter (patient no. 14) had atrial fibrillation with complete A-V dissociation. Each patient was carefully examined and those falling in functional group IV or showing signs of renal impairment were excluded from the study. All patients were “fully” digitalized with digitoxin. The majority exhibited A-V nodal escape beats at the end-point of digitalization. So as not to be misled by the vagal effect of digitalis each patient was exercised and given 1.25 mg.
of atropine subcutaneously on the day prior to administration of potassium. Those who did not show a rise in ventricular rate over 90 were selected for further study.

Each patient rested for 30 minutes before administration of potassium was begun. Electrocardiograms were taken every 5 minutes during the control period and during infusion of potassium. Frequent tracings were taken thereafter for a period of 2 hours. Six patients (nos. 3, 6, 14-17) were given 200 ml. of isotonic saline over a 30-minute period before potassium was started. Eleven patients were given buffered potassium phosphate dissolved in distilled water to a concentration of 150 mEq. per liter and 6 other patients (nos. 3, 7, 14-17) received the potassium in form of potassium chloride. The rate of infusion was uniform in 6 patients and in 11 it was started at a rapid rate but this speed could not be maintained for longer than 3 to 5 minutes because of intense local pain. The average amount of potassium administered throughout the experiment varied from .5 to 1.0 mEq. per minute, depending on the degree of pain. The infusion was not discontinued until at least .5 mEq. per Kg. of body weight was administered. Six patients were given 1.25 mg. of atropine subcutaneously 2 hours after the administration of potassium was discontinued, and the ventricular rate was recorded for an hour. Serum potassium levels were determined during the control period, during the infusion, at the end of infusion, and 2 hours after the experiment was discontinued. The serum levels were determined by means of the Beckman flame photometer with a lithium interval standard.

The electrocardiograms were analyzed with regard to (1) ventricular rate, (2) presence or absence of ectopic ventricular complexes, (3) evidence of A-V nodal escape beats and A-V nodal rhythm, (4) change of T waves.

RESULTS

Effect on A-V Conduction and Ventricular Rate. Thirteen of the 17 patients exhibited a decided slowing of the ventricular rate during the infusion of potassium (table 1). In 6 individuals the ventricular slowing was secondary to a complete A-V dissociation (nos. 1-4, 7, 8). In 4 patients (nos. 5, 10, 11, 16) there were accompanying A-V nodal escape beats or A-V nodal rhythm and in 1 (no. 6) there was a disappearance of idioventricular rhythm. The patient (no. 14) with atrial fibrillation and A-V dissociation during the control period maintained the A-V dissociation throughout the experiment, but the ventricular rate did speed under the influence of the infused potassium.

In 2 of the patients (nos. 1 and 2) with complete A-V dissociation the rate of the
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Fig. 2. Atrial fibrillation with ventricular bigeminy showing appearance of A-V dissociation and disappearance of ventricular ectopic beats. These changes took place rapidly and were dependent entirely on rate of infusion of potassium.

pacemaker located in the A-V node (or bundle of His) varied with the speed of infusion of potassium. These changes are clearly demonstrated in figure 1 (patient 2). The control strip in this figure shows atrial fibrillation. Tracing taken 5 minutes after beginning of infusion of potassium and after 60 seconds of "wide-open" flow of the solution, shows complete A-V dissociation with R-R intervals of 1.80 seconds. At this point the patient experienced severe pain in the arm and the infusion was stopped. A tracing 5 minutes later still shows complete A-V dissociation but with a faster ventricular rate (R-R interval of 1.52 seconds). At this point a slower and more uniform rate of infusion of potassium was resumed. Fifteen minutes after start of the experiment supraventricular control of ventricles returned, interrupted by an occasional A-V nodal escape beat after an R-R interval of 1.50 seconds. The last 2 strips recorded 10 minutes and 75 minutes after the infusion of potassium was discontinued reveal atrial fibrillation with a ventricular rate similar to that noted prior to the experiment. A total of 30 mEq. of potassium was infused over a period of 55 minutes. The serum potassium levels were 5.7 mEq. before infusion, 4.7 mEq. midway in the infusion, 5.3 mEq. at the end of infusion, 5.9 mEq. 1 hour after infusion, and 5.7 mEq. 2 hours after infusion, respectively.

In 6 patients (nos. 1, 5, 9, 10, 12, 13) the ventricular rate 2 hours after the end of the administration of potassium exceeded the control rate by 10 beats per minute, the average rise being 16 beats per minute. In the remainder there was no rise or the increase was less than 10 beats per minute.

The 6 patients who received atropine 2
hours after infusion was discontinued exhibited ventricular speeding but the increase was not greater than that observed when atropine was administered on the day before administration of potassium.

**Effect on Ventricular Ectopic Beats.** During the administration of potassium the idioventricular rhythm (patient 6) and ventricular bigeminy (patients 4, 5, 7) disappeared either at the time of maximum depression of conduction through the A-V node (patients 5, 6) or with appearance of complete A-V dissociation (patients 4, 7). In all 4 patients in this group the ventricular ectopic beats reappeared: in 2 (patients 4, 5) within 5 minutes after the infusion was halted and in the other 2 (patients 6, 7) within 30 minutes after the infusion was discontinued. Elimination of the ventricular ectopic beats with simultaneous decrease or cessation of conduction through the A-V node and the effect of rate of infusion on such changes are shown in figures 2, 3, and 4.

In figure 2 (patient 6) **strip 1** shows atrial fibrillation with ventricular escape beats and idioventricular rhythm. A total of 30 mEq. of potassium was administered over a period of 54 minutes. The rate of injection varied but a rapid infusion was impossible because of severe local pain whenever an attempt was made to infuse the solution rapidly. **Strips 2 and 3** recorded 25 minutes after the experiment began, show no essential change except that the T waves were somewhat taller. At the end of infusion (strips 4–7) the T waves were upright and peaked. There was marked depression of A-V nodal conduction and long periods elapsed during which, on the basis of the control tracing, one would expect ventricular escape beats to appear. However, the ventricular escape beats did not occur, indicating depression of ectopic focus by potassium. Thirty-five minutes after infusion was stopped (strips 8–10) the T waves were much lower and rhythm seen in **strip 1** was again present.

In figure 3 (patient 4) the control tracing shows atrial fibrillation with a ventricular rate of 75 to 90 and a run of ventricular bigeminy. Serum potassium before infusion was 5.5 mEq. A total of 30 mEq. of potassium was infused over a period of 75 minutes. The infusion was begun at a rapid rate. Within 4 minutes (strip B) the ventricular bigeminy disappeared with simultaneous marked decrease of conduction through the A-V node. A serum potassium obtained at this moment was 4.0 mEq. Because of pain at site of the needle the infusion was stopped and within 4 minutes the bigeminy reappeared (strip C). Reinstitution once again of a rapid infusion resulted in prompt elimination of the bigeminy and appearance of complete A-V dissociation with a ventricular
rate of 37 beats per minute. The serum potassium level during the A-V dissociation was 4.7 mEq.

Figure 4 (patient 7) shows prior to the infusion (strip A) atrial fibrillation and ventricular bigeminy with the third and sixth premature beats differing in appearance from the others. The complex marked $X$ is inverted and differs from the dominant complexes marked $Y$. Similar complexes appear in strips $B$, $C$, $D$, and $E$. These beats ($X$) are most likely A-V nodal in origin with aberrant conduction, although their origin below the bifurcation of the bundle of His cannot be ruled out. That the latter may be true is suggested by appearance of complexes marked $Z$, which may represent fusion of $X$ and $Y$. If this is the case, then the origin of $X$ must be in ventricles rather than in the A-V node. In strip $B$ the $X$ beats were more frequent, and in strip $C$ they represent the dominant rhythm with resultant complete A-V dissociation and A-V nodal or idioventricular rhythm and ventricular bigeminy. The ventricular premature beats disappeared in strip $D$ leaving the A-V node or idioventricular pacemaker in complete control of the ventricles. The ventricular rate of 60 beats per minute is more in keeping with A-V nodal pacemaker rather than one originating below the bifurcation of bundle of His. Strip $E$

taken 30 minutes after infusion was discontinued again shows atrial fibrillation with reappearance of the ventricular bigeminy observed during the control period. This experiment stresses again that the disappearance of ventricular bigeminy occurs parallel with marked depression of conduction through the A-V node.

Changes in $T$ Waves and Serum Potassium. Only 3 patients (nos. 2, 5, 6) showed significant change in the amplitude of the $T$ waves. The expected rise in serum potassium at the time the infusion was exerting its maximal effect on the ventricular rate or the cardiac rhythm was not observed. To the contrary, of the 13 patients in whom a satisfactory specimen was obtained during administration of the potassium, 10 showed a serum potassium content lower than that recorded prior to start of the infusion. The drop of potassium level varied from 0.2 to 1.5 mEq., with an average drop for the 10 patients of 0.75 mEq.

Discussion

It is extremely difficult to be certain that a given arrhythmia is due to digitalis and not a result of other factors, even if the patient is receiving digitalis. There are many changes in individuals with heart disease that may give rise to arrhythmias such as are commonly seen in digitalis intoxication. For
example, paroxysmal atrial tachycardia with block, in patients treated with digitalis, is frequently assumed to be a sign of digitalis intoxication. Such a cause-and-effect relation is often difficult to prove. In Barker's recent and rather comprehensive review of this arrhythmia one is struck by the paucity of cases due to digitalis, but to the contrary many were abolished by digitalis. The uncertainty that in any given patient digitalis is responsible for the arrhythmia in question is exemplified by our observations. In all our cases the A-V nodal or ectopic ventricular arrhythmias appeared while the patients were under observation and increasing doses of digitalis were being administered. Consequently one can assume that the arrhythmias observed before the infusion of potassium in each case were in some way related to digitalis. If, however, the electrocardiographic changes recorded during our experiments were studied without prior knowledge of administration of potassium, the resultant interpretation would most likely be misleading, because the decrease of conduction through the A-V node, appearance of A-V nodal escape beats, A-V nodal rhythm, and complete A-V dissociation would most certainly suggest to the electrocardiographer increased digitalis effect or digitalis intoxication.

It seems reasonable to assume that if potassium has an effect opposite to digitalis, administration of the former should increase conduction via the A-V node and thus increase the ventricular rate and at the same time abolish the A-V nodal escape beat and escape rhythm. Furthermore, should this occur with concomitant disappearance of ventricular ectopic beats, then one could conjecture that the effect of digitalis was to some extent negated by potassium. Our results, however, failed to bear out this assumption. The ventricular ectopic beats did disappear but with parallel depression of A-V conduction. The latter was manifest by complete A-V dissociation and appearance of an independent ventricular pacemaker. The subsidiary pacemaker was probably A-V nodal in origin with aberrant conduction in one case (fig. 4). This aberrancy, however, cannot be differentiated from beats originating below the bifurcation of the His bundle.

The only conclusion that could be drawn from these observations was that the observed effect of potassium on ventricular rate and ectopic beats must have been one of depression of the myocardium and the A-V node. This depression accounted for disappearance of ventricular ectopic beats, decrease of A-V nodal conduction, and appearance of complete A-V dissociation. Presence or absence of ventricular bigeminy, degree of depression of A-V nodal conduction, and the rate of A-V nodal discharge in presence of A-V dissociation depended largely on the rate of infusion of potassium. In some patients we were able, almost at will, to control the disappearance and reappearance of ventricular bigeminy as well as the degree of depression of the A-V node. The rapidity of changes during the experiment and the reappearance, in less than 30 minutes after infusion was stopped, of the arrhythmias present during the control periods can be best explained on the nonspecific depressing effect of potassium during the infusion. In many cases reported in the literature the dramatic and prompt response to potassium of the arrhythmias occurring in patients under digitalis therapy must have been due to this nonspecific depressing effect. The mechanism of observed alterations is difficult to ascertain. It is unlikely for reasons listed below that the pronounced effect observed in some of our patients could be due entirely to rise in serum potassium. These reasons are (1) the amount injected during any period of "rapid infusion" that resulted in heart block was very small; (2) we were unable to demonstrate any rise in serum potassium at time of maximum depression of conduction but to the contrary in 10 patients there was lowering of the serum level; (3) in only 2 patients did the T-wave change parallel the disturbance of rhythm, but in neither were these changes striking. It is probable, as was pointed out by Wiggers, that the concentration delivered to the heart was greater than that present in the general circulation.

The only evidence suggesting opposite
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The effect of potassium and digitalis is the late rise in ventricular rate in 8 patients and speeding of A-V nodal rhythm in patient 14. The increased rate of conduction through the A-V node following administration of potassium may have been due to inhibition of the digitalis effect on the node. If this assumption were correct, then administration of atropine at this point, by blocking the vagal effect on the A-V node, should further enhance conduction through the node with a rise of ventricular rate above that observed when atropine was given prior to infusion of potassium. The anticipated speeding of ventricular rate was not observed. Another interesting observation was the relative ease with which complete A-V dissociation was produced in our patients and yet such a state has never been observed in patients with normal sinus rhythm.

It seems that further study of the effect of potassium in atrial fibrillation is needed. Use of larger amounts of potassium and atropine coupled with careful documentation of the rate of infusion and time relation of the observed electrocardiographic changes may clarify the relative significance of the nonspecific myocardial effect of potassium and "antagonism" to digitalis.

**Summary**

The effect of potassium on the A-V nodal conduction and ectopic ventricular beats in patients with atrial fibrillation treated with digitalis was studied.

The disappearance of ventricular ectopic beats with parallel depression of conduction through the A-V node indicates that the early dramatic response of arrhythmia to intravenous potassium is probably due to a non-specific myocardial depression.

In all our cases the ventricular ectopic beats present during control period reappeared promptly when infusion was stopped.

The presence or absence of ventricular premature contractions, depression of A-V nodal conduction and the rate of discharge of the A-V nodal pacemaker could often be controlled by merely speeding or slowing the rate of infusion of potassium.

The need for further study of this problem in patients with atrial fibrillation is stressed.

**Acknowledgment**

We wish to thank Dr. K. G. Kohlsteadt for his continuous support and encouragement and Drs. B. L. Martz and O. M. Helmer for making available to us the laboratory facilities of the Lilly Laboratories for Clinical Research at the Indianapolis General Hospital.

**Summario in Interlingua**

Esseva studiata le effecto de kalium super le conduction del nodo atrio-ventricular e super le ectopic pulsos ventricular in patientes con fibrillation atrial tractate con digitalis.

Le disparition del ectopic pulsos ventricular con un depression parallel del conduction a transverso le nodo atrio-ventricular indica que le precoce e frappante responsa de arrhythmia al administration intravenose de kalium es probabilmente le resultato de un nonspecific depression myocardial.

In omne nostre casos le ectopic pulsos ventricular que esseva presente durante le periodo de controlo reapareeva promptemente quando le infusion esseva arrestate.

Le presentia o absentia de prematuri contractiones ventricular, le depression del conduction per le nodo atrio-ventricular, e le mesura del discarga in le pacemaker del nodo atrio-ventricular esseva regulabile in multe casos per simplemente accelerar o renvaler le infusion de kalium.

Es sublineate le necessitate de studios additional de iste problema in patientes con fibrillation atrial.

**References**


These 2 enzymes (and transaminase as well) are widely distributed throughout body cells, the former participating in the citric acid cycle and the latter being concerned with glycolysis. Measurement of the plasma level of either enzyme is easy to carry out, although the result in the case of malic dehydrogenase may not be entirely specific. In 18 patients with acute myocardial infarction but not in shock, the plasma activity of all 3 enzymes was abnormally elevated, the peak level of each being reached at about 24 hours after onset of pain. In 14 patients with a variety of liver diseases the plasma activity of all 3 enzymes was elevated in 10 instances, that of 2 of the 3 enzymes was increased in 3 instances, and the activity of all 3 was normal in 1 patient (who had portal cirrhosis). The greatest activity of phosphohexose isomerase and of transaminase was found in patients with infectious hepatitis, whereas peak values of malic dehydrogenase were found in individuals having myocardial infarction. There was fair parallelism in the levels of activity of the 3 enzymes.

Rogers
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