ABSTRACTS

INTERACTION OF FREQUENCY OF CONTRACTION AND PRELOAD ON THE PERFORMANCE OF ISOMETRIC KITTEN PAPILLARY MUSCLES

Initial fiber length and heart rate are important intrinsic determinants of cardiac muscle performance. To date the effect of their interaction has not been considered. To study the interaction of the length-tension and frequency-force relationships, peak developed tension (F), peak first derivative of tension development (dF/dt), time to peak tension (TP), and time to decline to ½ of peak developed tension (TP/2) were plotted as functions of a series of increments of frequency ranging from 0.2 to 2.4 contractions/sec at several different preloads for 15 kitten papillary muscles (mean cross-sectional area 0.550.4 mm²). The stimulation frequencies at which the F and dF/dt curves peaked and subsequently declined and the slopes of the curves of the above 4 indices of performance over a given range of frequencies were compared for each pair of preloads in each muscle. At high preloads F and dF/dt reached peaks at significantly lower frequencies than at corresponding low preloads; similarly, dF/dt rose to its peak and TP declined at significantly greater rates. Once F peaked it declined at a consistently greater rate at the high preload than at the low preload. Post-control results returned toward those of the precontrol experiments. Hypoxia as the mechanism was excluded by data from 7 additional muscles at 2 levels of oxygenation which demonstrated the converse of the above hemodynamic changes. These data indicate that preload determines whether a given increment in frequency produces a positive or negative inotropic effect and define the interaction of these two intrinsic variables on performance.

HEMOLYSIS IN THE STARR-EDWARDS AORTIC PROSTHESES
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This study was to compare the degree of hemolysis in the various models of the Starr-Edwards (S.E.) aortic prostheses. Patients seen in our post-operative follow-up clinic over the last 18 months who had only aortic prostheses and no significant paravalvular leakage were studied. Hematocrits (Hct), serum haptoglobin, lactic dehydrogenase, glutamic oxalacetic transaminase levels and reticulocyte counts were obtained. In addition the regular use of iron therapy was noted. Patients were grouped as having non cloth covered valves namely Series 1000, 1200 and 1260 - Group I. Cloth covered valves were Series 2320 - Group II and the modified valve Series 2310 and 2320 - Group III. The results are noted in the table.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Hct</th>
<th>Mean LDH</th>
<th>Fe Rx</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>44</td>
<td>128</td>
<td>0</td>
<td>21</td>
</tr>
<tr>
<td>II</td>
<td>37</td>
<td>512</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>III</td>
<td>39</td>
<td>263</td>
<td>5</td>
<td>38</td>
</tr>
</tbody>
</table>

The differences between the groups for iron therapy is significant (P<0.01). Group I differed significantly from Group II and III both in terms of LDH and Hctm (F<0.01); however there was insufficient evidence to detect a difference between Groups II and III either in LDH or Hctm values. Patients receiving iron therapy all had hematocrits in excess of 30%. Patients who received iron therapy all had hematocrits in excess of 30%.

CLINICAL AND ELECTROPHYSIOLOGIC SIGNIFICANCE OF BUNDLE BRANCH BLOCK (BBB) IN ACUTE MYOCARDIAL INFARCTION (AMI)
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In order to assess atrioventricular (AV) conduction in BBB occurring in AMI, Bundle of His (HH) electrograms were recorded during pacemaker insertion or withdrawal. Twelve patients (pts) with right bundle branch block (RBBB) and two with left bundle branch block (LBBB) were observed. All 2 pts (with RBBB) had narrow QRS complexes prior to AMI. All studies were carried out during sinus rhythm. Ten of 12 pts with RBBB and both with LBBB had abnormal His-Purkinje (HP) conduction (50 to 135 msec). All had normal AV nodal conduction. The response to atrial pacing at rates in AMI. In all pts of 6 pts restudied 5 to 90 days later had abnormal HP conduction, including 3 pts who had lost their BBB. Nine pts expired and 5 survived. Five of 6 pts who died at the same levels (20-60 mmHg) up to 2 hours. Changes in hemodynamics were primarily related to changes in the slope of phase 4 depolarization. Maximum diastolic and threshold potentials were not affected. No other electrophysiological property was immediately responsive to changes in pO2. Only after HP fibers showed signs of deterioration in hypoxia such as loss of resting potential, was enhanced automaticity manifested. In conclusion, in RBBB the properties of automaticity and contractility are closely linked to pO2 tension. Such dependence may be one electrophysiological mechanism for hypoxic arrest in vivo.

THE EFFECT OF HYPOXIA ON AUTOMATICITY AND CONTRACTILITY IN CANINE PURKINJE FIBERS
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Previous studies in vivo and in vitro have indicated that electrical properties of Purkinje (P) tissue are unaffected by low O2 levels for long periods of time. After several hours of hypoxia, enhanced automaticity has been reported. To re-evaluate the relation between p02 and v cell function we used isolated P strands attached to a force transducer, under .3-.6 gms of tension, as well as preparations of the entire endocardial surface of the ventricles pinned and superfused with Tyrode's solution at 37°C. O2 tension was varied from 600-20 mmHg while pH and pCO2 were kept constant. Within 5 minutes after changing the gas mixture, the rate of firing and isometric contractile tension changed in direct relation to the change in O2 tension. Over the full range of pO2 values the range of frequencies was 40-8 beats/min. The full range of isometric tension was 100-30%. Changes in automaticity were most marked at pO2 values less than 150 mmHg. No appreciable change in isometric tension was seen at low p02 levels. Only after pO2 fibers showed signs of deterioration in hypoxia such as 1000 of resting potential, was enhanced automaticity manifested. In conclusion, in P fibers the properties of automaticity and contractility are closely linked to pO2 tension. Such dependence may be one electrophysiological mechanism for hypoxic arrest in vivo.