Atrial and ventricular pacing after open heart surgery

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The effect of cardiac pacing, through a wide range of pacing rates, has been studied in 13 patients in stable sinus rhythm within 24 hours of operation. Using first atrial and then ventricular pacing, the cardiac rate was raised to 60% above control value. The effects of atrial and ventricular pacing were compared by observing the differences in the various parameters measured at corresponding paced heart rates. With atrial pacing there was no significant difference in the cardiac output, mean aortic pressure, mean left atrial pressure, or left ventricular work. During ventricular pacing there were significant falls in cardiac output (p<0.05), mean aortic pressure (p<0.01), and left ventricular work (p<0.01). There was a significant rise in mean left atrial pressure (p<0.01).

In this study atrial pacing had no significant effect on the parameters measured, through a wide range of cardiac rates. An inotropic effect resulting from increase of rate was not seen. Reported increases in cardiac output following pacing must therefore have resulted from correction of a dysrhythmia. Ventricular pacing incurs a definite haemodynamic penalty if used in patients with an intact atrioventricular conducting pathway. If atrial pacing is not available, this penalty must be balanced against the possible haemodynamic advantage of suppressing a dysrhythmia.

Harris et al. (1967) demonstrated the value of rate augmentation by cardiac pacing in the management of bradycardias and certain dysrhythmias following open heart surgery in man. By capturing the cardiac rhythm, pacing may increase the rate, suppress the dysrhythmia, and increase the cardiac output (Litwak et al., 1968; Frieson et al., 1968; Woodson, Frieson, and Starr, 1968; Nathan, Mundth, Buckley, and Austen, 1969). This haemodynamic improvement may be due either to the correction of the abnormal rhythm (Harris et al., 1967; Nathan et al., 1969) or to an intrinsic inotropic effect consequent upon increasing the heart rate (Covell et al., 1967; Sarnoff and Mitchell, 1961; Mitchell, Wallace, and Skinner, 1963; Litwak et al., 1968; Sonnenblick, Morrow, and Williams, 1966; Stein et al., 1966). This study was designed to test the latter possibility by measuring the effects of augmenting the rate by atrial pacing in stable postoperative patients with sinus rhythm.

Frieson et al. (1968) and Woodson et al. (1968) have demonstrated that for a given paced rate atrial pacing had more beneficial haemodynamic effects than ventricular pacing, in patients with sinus rhythm, in whom the atrioventricular conduction pathway was intact. The second object of these studies was to observe whether this difference occurred throughout a range of paced rates.

METHODS

Thirteen patients, 12 male and one female, whose ages ranged from 23 to 54 years, have been investigated. Aortic valve replacement with either a homograft (Ross, 1968) or a fascia lata valve (Ionescu et al., 1970) had been carried out. The condition of the patients was clinically stable; they were in sinus rhythm and the investigation was done within 24 hours of operation.

The techniques of measurement and the parameters measured are schematically represented in Figure 1.

CARDIAC OUTPUT Cardiac output was measured by the dye dilution technique (Balcon and Sowton, 1969; Bennet, Balcon, Hoy, and Sowton, 1970) using indocyanine green, which was injected into the right atrium through the central venous pressure catheter. Arterial blood was sampled from the radial artery (occasionally the femoral artery), passed through a cuvette, and analysed by a Gilford densitometer. After amplification the dye dilution curve was inscribed by a direct writing Honeywell recorder, and,
Demand pacing using a Devices Box (Type ‘E’ Serial No. 2990) was used with independent control of rate and the stimulus voltage. The rate will be described as a percentage rise above control value (Balcon and Sowton, 1969) rather than in absolute figures.

The derived parameters were obtained as follows:

- **Systolic ejection rate**
  
  \[
  \text{stroke volume (ml)} = \frac{\text{systolic ejection time (s)}}{\text{systolic ejection rate}}
  \]

  The stroke volume is obtained with knowledge of the cardiac output and the heart rate. The systolic ejection time is known from examination of the dynamic aortic pressure tracings (Sarnoff and Berglund, 1954). Systolic ejection rate is widely accepted as an index of the rate of fibre shortening of the myocardium (Balcon and Sowton, 1969; Levine et al., 1962) but some believe this is not so (Ross, Linhart, and Braunwald, 1965; Hamer, 1968; Dexter et al., 1951).

- **Left ventricular work**
  
  \[
  \text{LVW} = CO \times K(\text{MAP} - \text{LAP})
  \]

  where \( \text{LVW} \) = left ventricular work (kgm/min); \( \text{CO} \) = cardiac output (l./min); \( K \) = constant; \( \text{MAP} \) = mean aortic pressure (mmHg); and \( \text{LAP} \) = mean left atrial pressure (mmHg). Thus a change in the value of \( \text{LVW} \) is a composite reflection of changes in cardiac output, mean aortic pressure, and mean left atrial pressure.

  The investigation of each patient followed the same pattern (Fig. 2). A series of control observations was made and atrial pacing was then begun. The resting rate had usually been between 85/min and 95/min and the rhythm could be captured by atrial pacing with a rise in rate of less than 12.5%. The maximum rate was about 140/min in each case, representing a rise of up to 60%. Observations were also made at a paced rate in between these two. In each case a minimum of 15 minutes was allowed for stabilization in the altered circumstances (Litwak et al., 1968). After further control readings ventricular pacing was begun and observations were made at similar rates. A final set of control measurements was made.

### PLAN OF EACH STUDY

<table>
<thead>
<tr>
<th>Study</th>
<th>1 CONTROL</th>
<th>2 Minimum</th>
<th>3 Moderate (110–125)</th>
<th>4 Maximum (135–140)</th>
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</thead>
<tbody>
<tr>
<td>Atrial Pacing</td>
<td>3 Moderate (110–125)</td>
<td>20–40</td>
<td>45–60</td>
<td>&lt;12.5</td>
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<tr>
<td>Ventricular Pacing</td>
<td>6 Minimum</td>
<td>&lt;12.5</td>
<td>20–40</td>
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<tr>
<td>9 CONTROL</td>
<td>7 Moderate (110–125)</td>
<td>20–40</td>
<td>45–60</td>
<td>&lt;12.5</td>
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</table>
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ANALYSIS

To evaluate the haemodynamic effect of rate increase alone for each parameter measured, the results obtained at each rate during atrial pacing were compared with the control values. The effects of atrial and ventricular pacing were compared by observing the differences in the various parameters at corresponding paced heart rates. In order to demonstrate the underlying stability of the heart the control values measured before and after each type of pacing were also compared. All these differences were analysed using the paired t test and standard probability tables.

RESULTS

CONTROL VALUES The control observations for each parameter showed no significant change between measurements before and after pacing (Figs. 3 to 7).

MEAN AORTIC PRESSURE (Fig. 3) Atrial pacing resulted in a small rise in mean aortic pressure of up to 6 mmHg, which was not statistically significant. Ventricular pacing caused a significant fall of up to 9.7 mmHg (p<0.01). When ventricular pacing was discontinued the mean aortic pressure immediately returned to the higher control values. When the values obtained at corresponding rates during atrial and ventricular pacing were compared, the differences at the moderate and maximum rates were significant and were 13.1 mmHg (p<0.01) and 10.1 mmHg (p<0.05) respectively. The greatest difference was 7% of the control mean aortic pressure. At the minimum rate there was no significant difference either from the control values or between atrial and ventricular pacing.

CARDIAC OUTPUT (Fig. 4) There was no significant change in cardiac output during atrial pacing. During ventricular pacing there was a fall in cardiac output which reached significance (p<0.05) at the maximum rate; there was immediate recovery to the control value when ventricular pacing was withdrawn. When the values obtained at corresponding rates during atrial and ventricular pacing were compared, the differences at minimum, moderate, and maximum rates were all significant and were 0.48 l/min (p<0.05), 0.70 l/min (p<0.05), and 0.56 l/min (p<0.01) respectively. The greatest difference was 15% of the control cardiac output.

LEFT ATRIAL PRESSURE (Fig. 5) During atrial pacing there was no significant change in mean

FIG. 3. Effect of pacing on mean aortic blood pressure (9 patients).

FIG. 4. Effect of pacing on cardiac output (mean of 11 patients).

FIG. 5. Effect of pacing on mean left atrial pressure (9 patients).
left atrial pressure. Ventricular pacing caused significant elevation of the mean left atrial pressure at all rates. When the effects of atrial and ventricular pacing at corresponding rates were compared, there were significant differences of 3.73 \( (p<0.01) \), 3.85 \( (p<0.01) \), and 3.27 \( (p<0.001) \) mmHg at the minimum, moderate, and maximum rates. These differences were up to 50\% of the control pressure.

**LEFT VENTRICULAR WORK**  (Fig. 6) Atrial pacing caused no significant change in left ventricular work. Ventricular pacing caused significant falls at moderate and maximum paced rates. The difference between the effects of atrial and ventricular pacing was significant at each rate: 0.96 kgm/min \( (p<0.05) \), 2.17 kgm/min \( (p<0.01) \), and 1.40 kgm/min \( (p<0.001) \) at minimum, moderate, and maximum rates respectively. These differences were up to 38\% of the control value.

**SYSTOLIC EJECTION RATE**  (Fig. 7) Atrial pacing caused a fall in systolic ejection rate which became significant \( (p<0.05) \) at the maximum rate only. Ventricular pacing caused a fall of 37 ml/s \( (p<0.01) \) at the moderate rate and 55 ml/s \( (p<0.01) \) at the maximum rate. The differences between the effects of atrial and ventricular pacing were small and reached statistical significance only at the maximum rate \( (p<0.05) \) with a difference of 16 ml/s.

**DISCUSSION**

Bradycardia occurring after open heart surgery may lead to reduced cardiac output and predispose to dysrhythmias (Harris et al., 1967; Litwak et al., 1968; Nathan et al., 1969): dysrhythmias will likewise cause a fall in cardiac output and may lead to potentially fatal ventricular dysrhythmias (Litwak et al., 1968; Hoffman et al., 1969). When there is a cause such as hypoxia, hypokalaemia, altered acid-base status, digoxin intoxication, or inadequate coronary perfusion (Harris et al., 1967; Litwak et al., 1968; Hoffman et al., 1969), therapy should be directed towards its correction. When there is no identifiable cause empirical therapy may be used, either with a pharmacological agent such as procainamide (Friedberg, 1966; Leading article, *British Medical Journal*, 1970a) or lignocaine (Friedberg, 1966; Binnion, Murtagh, Pollock, and Fletcher, 1969), or with a physical agent such as pacing (Harris et al., 1967). By capturing the cardiac rhythm, pacing may suppress the dysrhythmia, augment the rate, and increase the cardiac output (Harris et al., 1967; Litwak et al., 1968). Friesen et al. (1968) and Woodson et al. (1968) recorded rises of up to 20–30\% in cardiac output using atrial pacing. When nodal rhythm was captured by atrial pacing a rise of up to 60\% was found (Friesen et al., 1968).
basis for the latter possibility is found in the work of Sarnoff and Mitchell (1961) and other reports. They have shown that an increase in rate causes an increase in ventricular contractility in both animals (Sarnoff and Mitchell, 1961; Covell et al., 1967; Mitchell et al., 1963) and in normal men (Stein et al., 1966; Benchimol and Ligget, 1966), as indicated by a rise in tension-time index or a fall in left ventricular end-diastolic pressure. Similar findings are reported by Balcon and Sowton (1969) who increased the rate by atrial pacing in patients with ischaemic heart disease. However, the increased myocardial contractility observed by these authors was not accompanied by an improvement in performance, as judged by such haemodynamic criteria as, for example, the cardiac output.

In our study the principal criteria were cardiac output, left atrial pressure, aortic pressure, and left ventricular work. During atrial pacing we found these parameters to remain essentially stable with increasing rate, as did Sarnoff and Mitchell (1961), Stein et al. (1966), and Balcon and Sowton (1969). It is possible that with more refined techniques, such as measurement of acceleration in the ascending aorta, a change in contractility could be detected. However, it is difficult to imagine how a change, so small that it is not reflected in the haemodynamic criteria that we measured, could be of much practical value. This study therefore suggests that the improved haemodynamic status which occurs when atrial pacing is used to control dysrhythmias is due to correction of the rhythm and restoration of efficient cardiac action and not to any inotropic effect of rate augmentation.

COMPARISON OF ATRIAL AND VENTRICULAR PACING

In patients in whom the atroventricular conduction pathways are intact, pacing may be achieved by either atrial or ventricular stimulation. Many studies have been carried out comparing the results of the two methods in different circumstances. Mitchell, Gilmore, and Sarnoff (1962) and Gilmore, Sarnoff, Mitchell, and Linden (1963), in carefully controlled experiments in dogs, demonstrated that ventricular function was significantly depressed by ventricular pacing, as compared with atrial pacing. Benchimol, Ellis, and Dimond (1965) studied normal human subjects and were unable to demonstrate any significant difference in the haemodynamic parameters, except that there was a greater fall in systolic ejection rate with ventricular pacing. In patients with heart disease, both Benchimol et al. (1965) and Samet, Bernstein, Levine, and Lopez (1965) showed that cardiac performance was more efficient with atrial pacing. Previous studies with bradycardias and dysrhythmias following open heart surgery have shown that with a modest rise in rate to a level not exceeding 115-120/min atrial pacing may achieve a rise in cardiac output of up to 30%, while ventricular pacing in the same circumstances may cause a fall in cardiac output of 10-20% (Frieson et al., 1968; Woodson et al., 1968). In the present studies the subject did not have a dysrhythmia and with atrial pacing the cardiac output was not increased, but with ventricular pacing it fell by 17%. Whereas in the previous studies these observations were made at one paced rate only, this report shows that these effects may be observed at different rates up to 140/min, and the difference becomes more marked at higher heart rates. This pattern was seen consistently in all the haemodynamic parameters observed. It should be stressed that with atrial pacing there is no haemodynamic penalty in increasing the rate to 140/min. This is consistent with the findings of Balcon and Sowton (1969), Benchimol and Ligget (1966), Ross et al. (1965), and Wright, Fabian, and Epstein (1970), but Kloster et al. (1966) and Benchimol et al. (1965) state that in patients with heart failure the optimum rate lies between 110 and 120/min.

REASONS FOR DIFFERENCE

The reasons for this difference have been a recurring subject for discussion in recent years. The atrium functions as a reservoir whether atrial systole occurs or not. Synchronized atrial systole, however, enables the atrium to function as a supplemental pump, achieving a left ventricular end-diastolic pressure which is greater than the mean left atrial pressure; this is important with regard to the end-diastolic state of myocardial fibres and ventricular contractility, and also to the retrograde effect of mean left atrial pressure on pulmonary venous circulation (Braunwald, 1964). Atrial contraction is also considered to contribute to efficient closure of the atroventricular valves (Braunwald, 1964; Leading article, British Medical Journal, 1970b), and in ventricular pacing large 'v' waves occur in the left atrial pressure recordings, suggesting some mitral insufficiency (Gilmore et al., 1963; Mitchell et al., 1962; Samet et al., 1965). Finally, where excitation occurs via normal pathways a coordinated ventricular contraction results, while if excitation is initiated at an ectopic site where a ventricular electrode is implanted, the resultant contraction is not coordinated (Gilmore et al., 1963). The healthy heart is assumed to have sufficient reserves of function to compensate for these
factors when ventricular pacing is carried out in normal subjects (Leading article, British Medical Journal, 1970b).

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