Constancy of pressure-rate product in pacing-induced angina pectoris

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Constancy of the pressure-rate product (heart rate × systolic blood pressure) or the triple product (heart rate × systolic blood pressure × ejection time) at the onset of exercise-induced pain has been observed in patients with exertional angina (Robinson, 1967; Redwood et al., 1971). Both ‘products’ reflect myocardial oxygen consumption (MVO₂). The duration of the ejection time is now thought to be of minor importance in estimating MVO₂. The tension-time index has been found to correlate less well with MVO₂ than the pressure-rate product or just the heart rate (Monroe, 1964; Kitamura et al., 1972; Sheffield and Roitman, 1973; Nelson et al., 1974).

It has been shown that with either exercise or atrial pacing patients develop ischaemic manifestations at a constant tension-time index (Sowton et al., 1967; Bahler and Macleod, 1971) or pressure-rate product (O’Brien et al., 1969). We have further investigated the constancy of the pressure-rate product in patients who developed anginal pain with rapid right atrial pacing.

Patients and methods

Twelve patients were studied. All had developed anginal chest pain in response to rapid right atrial pacing performed by the usual technique (Sowton et al., 1967; Kelemen et al., 1973). Atropine was given to three patients to reverse second degree atrioventricular block occurring at lower heart rates (Cokkinos et al., 1973). All were premedicated with 5 to 10 mg diazepam intramuscularly. Blood pressure during the test was directly recorded in eight patients through an indwelling needle in the brachial artery and measured by a cuff sphygmomanometer in the remaining four patients. The pressure-rate product at the onset of pain was calculated in mmHg/min (Sheffield and Roitman, 1973) (1 mmHg/min=0.133 kPa/min).

After pacing in the initial test had been stopped an intravenous infusion of angiotensin (2.5 mg in 1 litre normal saline) was started together with continuous monitoring of the blood pressure. When the systolic pressure had increased by 20 mmHg (2.7 kPa) or more rapid right atrial pacing was repeated and the pressure-rate product at the time of anginal pain was noted. After the angiotensin infusion had been stopped and the blood pressure had returned to initial levels pacing was repeated and the pressure-rate product at the onset of anginal pain recorded again. The heart rate, blood pressure, and pressure-rate product at the time of anginal pain before, during, and after angiotensin administration were compared by the paired t test.

Informed consent for the addition of an angiotensin infusion to the usual procedure of diagnostic rapid atrial pacing was obtained from all patients.

Results

Angiotensin produced the desired increase in systolic blood pressure in all 12 patients (see Table). The cardiac frequency at which each patient developed chest pain was significantly higher before and after the effect of the angiotensin wore off and lower during the infusion (P<0.001). Thus, though there was a large rise in systolic pressure during angiotensin infusion, the fall in heart rate
TABLE  Heart rate, blood pressure, and pressure-rate product at onset of anginal pain in patients subjected to right atrial pacing before, during, and after angiotensin infusion

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>HR (min)</th>
<th>BP (mmHg)</th>
<th>PRP (mmHg/min)</th>
<th>BP (mmHg)</th>
<th>PRP (mmHg/min)</th>
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<tr>
<td></td>
<td></td>
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<td>Before angiotensin</td>
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<td>During angiotensin</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>(kPa)</td>
<td>(kPa×10⁶/min)</td>
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<td>18-96</td>
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<td>165/110</td>
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Mean (± SEM)

- A vs B: Δ34.7 ± 3.31 (systolic) ± 2.99 (0.10)
- B vs C: Δ37.1 ± 3.36 (systolic) ± 2.44 ± 0.09
- P < 0.001

A vs C

*Ischaemic ST segment changes during pain.
Abbreviations: HR = heart rate; BP = blood pressure; PRP = pressure-rate product; SEM = standard error of mean; SED = standard error of difference; Δ = difference.

Discussion

Our results support the hypothesis that there is a constant myocardial oxygen consumption for each patient at which angina will develop. Each of our patients experienced anginal pain at a constant pressure-rate product regardless of whether this was produced by a relatively greater increase of the heart rate or of the systolic blood pressure.

We chose atrial pacing and angiotensin infusion...
Pressure-rate product in pacing-induced angina pectoris

for manipulating the pressure-rate product for various reasons. Rapid pacing, in contrast to muscular exercise (Piggott et al., 1971), does not produce any significant change in adrenergic activity provided diazepam is given to prevent undue anxiety (Voridis et al., 1974). Nevertheless, Nelson et al. (1974) found that the pressure-rate product correlated very well with MVO₂ in exercising patients. We chose angiotensin for raising the blood pressure because, in contrast to sympathomimetic pressor amines, it does not alter myocardial contractility in man (Ross and Braunwald, 1964; Perloff, Binnion, and DeLeon, 1967), though Koch-Weser (1964) and Fowler and Holmes (1964) have described a positive inotropic effect in animals. Moreover, venous tone, venous return, and heart volume, which are considered to influence MVO₂ (Simaan, 1974), are only minimally affected by angiotensin (Rose et al., 1962). Patients exercising after nitroglycerin develop anginal pain at a higher pressure-rate product than controls, presumably because of reduced ventricular dimensions, which lower the MVO₂. Conversely, after propranolol the pressure-rate product at the onset of angina is reduced, possibly as the result of increased ventricular size, which tends to augment MVO₂ (Wolfson, Phillips, and Schecter, 1970; Detry and Bruce, 1971; Goldstein and Epstein, 1973; Nelson et al., 1974).

Angiotensin has been found to increase coronary arterial resistance (Fowler and Holmes, 1964) but Perloff et al. (1967) believed this action to be of no practical significance. Angiotensin has also been used to evaluate contractility in patients with coronary artery disease without any evidence of coronary constrictive effect (Parmley et al., 1975). Anginal chest pain was taken as the criterion of a positive response to atrial pacing. It has been repeatedly stressed that ST segment changes are not specific enough, especially at high atrial pacing rates (Kelemen et al., 1973; Rios and Hurwitz, 1974). Nevertheless, 10 of our 12 patients developed such changes coincidentally with the pain.

Our observations suggested that in addition to other possible haemodynamic modifications drugs preventing increases in blood pressure during exercise may be expected to ameliorate exertional angina.

References


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FIG. Case 1. Anginal pain and ischaemic ST changes occur at about the same pressure-rate product before and after angiotensin administration.
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