A NON-INVASIVE TECHNIQUE OF COMPARING MYOCARDIAL PERFORMANCE FOLLOWING EPIDURAL BLOCKADE AND VASOPRESSOR THERAPY

A. L. FORREST, J. I. M. LAWSON AND P. E. OTTON

SUMMARY

The duration of electromechanical systole (Q→S₂), left ventricular ejection time (LVET) and the pre-ejection period (PEP) were determined, non-invasively, in a series of twenty patients before and after lumbar epidural block. The Q→S₂ interval and the PEP lengthened after epidural block. Restoration of the arterial pressure to control levels was achieved with intravenous methoxamine (mean dose 3 mg) in ten patients, or ephedrine (mean dose 13.5 mg) in the remainder. Methoxamine caused a further lengthening of PEP while ephedrine significantly shortened PEP. The influence of heart rate, afterload and preload on the systolic time intervals is discussed.

Interest in non-invasive techniques of assessing cardiovascular function has grown since Katz and Feil (1923) established the method of recording simultaneously the heart sounds, central arterial pulse and the electrocardiogram (E.C.G.) to define the intervals in the cardiac cycle. Using the first (S₁) and second (S₂) heart sounds to denote the beginning and the end of systole and by subtracting the left ventricular ejection time (LVET) from the S₁-S₂ interval, they observed that this difference, the isometric contraction time (ICT) (fig. 1), was an index of the rapidity of ventricular contraction.

Kumar and Spodick (1970) found that the most reliable external measurement of ICT was the interval between the upstroke of the carotid pulse (suitably corrected for pulse transmission delay time) and the beginning of the systolic wave upstroke of the apex cardiogram.

Recently, measurement of the pre-ejection period (PEP) has become popular (Weissler, Harris and Schoenfeld, 1969; Diamant and Killip, 1970; Blackburn et al., 1972; Armstrong, Lewis and Gotsman, 1973). The pre-ejection period is obtained by subtracting LVET from Q→S₂ interval (fig. 2). Major changes in left ventricular end-diastolic pressure (preload) and aortic diastolic pressure (afterload) influence the PEP (Metzger et al., 1970; Talley, Meyer and McNay, 1971; Martin et al., 1971; Blackburn et al., 1973), while Prys-Roberts et al. (1972) found, in the anaesthetized dog, that PEP

FIG. 1. Simultaneous recordings of E.C.G. phonocardiogram and carotid pulse wave at paper speed of 100 mm/sec. Isometric contraction time (I.C.T., msec) is obtained by subtracting left ventricular ejection time (L.V.E.T., msec.) from the time interval between first (S₁) and second (S₂) heart sounds.

\[
I.C.T. = (S₁→S₂) - E.T.
\]


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correlated poorly with their index of myocardial contractility, the ratio
\[
\frac{\text{max LV dp/dt}}{\text{IP}}
\]
(Weissler, Harris and Schoenfeld 1969) showed a close correlation between the ratio PEP/LVET and cardiac output and stroke volume, while Reitan et al. (1972) demonstrated in the dog that the inverse square of PEP correlates with peak ascending aortic blood flow acceleration.

Arterial hypotension following epidural analgesia usually responds to change in posture and the rapid administration of intravenous fluid, but occasionally a vasoactive drug is indicated. The purpose of this study was to investigate the changes occurring in the pre-ejection period and its components and derivatives during lumbar epidural analgesia, and the effectiveness of intravenous methoxamine or ephedrine in restoring these measurements to the values occurring before the nerve block.

**Technique of comparing myocardial performance**

Seventeen females and three males, aged between 17 and 84 years, scheduled for lower abdominal surgery under lumbar epidural analgesia were studied. Their general health was good and the informed consent of each patient was obtained. Seventeen patients were premedicated with papaveretum 20 mg and hyoscine 0.4 mg i.m. 2 hours before the procedure, while three older patients received half of these doses. A slow intravenous infusion of Hartmann's solution was established, and an epidural catheter was introduced through the 2nd or 3rd lumbar interspace.

Standard lead II e.c.g. was monitored, heart sounds were recorded by an SEM 4/84 contact microphone (SE Laboratories) placed over the praecordium, and the arterial pressure waveform was obtained from a modified pressure transducer (4/82, SE Laboratories) hand-held over the external carotid artery in the neck. All records were made on an "Emma" System six-channel recorder at paper speeds of 100 mm/sec. Arterial pressure was measured by the auscultatory method.

Total electromechanical systole (EMS) was measured in msec as the interval from the Q wave of the e.c.g. to the initial high frequency component of the second heart sound, Q→S₂ (fig. 2). Left ventricular ejection time (LVET) was measured from the onset of the rapid upstroke portion of the carotid arterial pulse to the trough of the incisura of the dicrotic notch. Each component was derived as the mean of ten consecutive complexes, the PEP (msec) being the difference between Q→S₂ and LVET. Heart rate was calculated from the R-R interval of the e.c.g. Calculations were made of \(1/\text{PEP}^2\), expressed as units of reciprocal square seconds (S⁻²), and of the ratio PEP/LVET. Mean arterial pressure was derived from the expression:

\[
\text{mean arterial pressure} = \text{systolic pressure} + (2 \times \text{diastolic pressure})/3.
\]

Following recording of measurements in the resting state, 2% lignocaine hydrochloride solution without adrenaline, in a volume calculated to produce a block extending to the costal margin, was injected through an epidural catheter inserted in the lumbar region. Fifteen minutes later the segmental height of the epidural block was determined by the loss of sensation to pinprick, and further recordings were made. Ten patients (Group I) were then given methoxamine i.v. and the remain-
ting ten (Group II) received ephedrine i.v., in both
instances by increments until the arterial pressure
was restored as close to the resting value as possible.

**RESULTS**

The ten patients in Group I were in the age range
33–85 years (mean 57 years), while those in Group
II were in the range 15–57 years (mean 44 years).
The epidural block reached a mean segmental
height of T5 in Group I and T6 in Group II, and
was achieved with mean doses of 270 mg lignocaine
and 370 mg lignocaine respectively.

**Arterial pressure.**

A significant decrease in arterial pressure followed
epidural block (P<0.01) (tables I and II). The
average dose of methoxamine and ephedrine
required to restore the arterial pressure to pre-
epidural levels was 3 mg and 13.5 mg respectively.

**Heart rate.**

Epidural blockade was accompanied by a decrease
in heart rate (tables I and II). Methoxamine caused
a further significant decrease in heart rate (P<0.01),
while ephedrine restored the blood pressure with
little alteration in heart rate.

**Systolic time intervals.**

Regression analysis showed that the Q→S₂ and
LVET intervals varied inversely and significantly
(P<0.01) with heart rate (fig. 3). The relationship
between heart rate and PEP (fig. 3) was found to
be not statistically significant, a finding in agreement
with Leighton et al. (1971). Exceptions to this find-
ing occurred following epidural blockade when the
PEP varied significantly with heart rate (Group I:
\(r=-0.705, t=2.815\); Group II: \(r=-0.803, t=3.813\)).

The pre-ejection period lengthened following
epidural block mainly because of a prolongation of
electromechanical systole (Q→S₂ interval) (tables I
and II). Methoxamine caused a further lengthening
following epidural block, while ephedrine significantly
shortened the pre-ejection period (P<0.05).

The value for the index \(1/\text{PEP}^2\) decreased

**TABLE I. Mean values (±SEM) from 10 patients of measurements: (a) before epidural blockade; (b) after epidural blockade; (c) after intravenous methoxamine.**

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Q→S₂ (msec)</th>
<th>LVET (msec)</th>
<th>PEP (msec)</th>
<th>1/\text{PEP}² x 10⁻⁶</th>
<th>PEP/LVET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before epidural</td>
<td>74.6 ± 6.38</td>
<td>91.5 ± 4.19</td>
<td>399.10 ± 14.07</td>
<td>294.43 ± 11.47</td>
<td>104.67 ± 4.26</td>
<td>98.84 ± 8.27</td>
<td>0.355</td>
</tr>
<tr>
<td>After epidural</td>
<td>61.5 ± 4.17</td>
<td>65.6 ± 5.84</td>
<td>424.46 ± 8.20</td>
<td>306.21 ± 6.44</td>
<td>118.25 ± 3.49</td>
<td>75.86 ± 4.94</td>
<td>0.378</td>
</tr>
<tr>
<td>After methoxamine</td>
<td>51.4 ± 3.68</td>
<td>100.7 ± 7.79</td>
<td>458.95 ± 10.65</td>
<td>337.00 ± 8.37</td>
<td>121.95 ± 4.31</td>
<td>69.31 ± 4.45</td>
<td>0.363</td>
</tr>
</tbody>
</table>

**TABLES II. Mean values (±SEM) from 10 patients (Group II): (a) before epidural blockade; (b) after epidural blockade; (c) after intravenous ephedrine.**

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Q→S₂ (msec)</th>
<th>LVET (msec)</th>
<th>PEP (msec)</th>
<th>1/\text{PEP}² x 10⁻⁶</th>
<th>PEP/LVET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before epidural</td>
<td>68.5 ± 6.32</td>
<td>93.8 ± 3.88</td>
<td>409.5 ± 12.65</td>
<td>307.64 ± 10.85</td>
<td>101.85 ± 3.17</td>
<td>98.78 ± 5.70</td>
<td>0.332</td>
</tr>
<tr>
<td>After epidural</td>
<td>66.1 ± 6.11</td>
<td>73.1 ± 3.58</td>
<td>412.25 ± 13.12</td>
<td>305.25 ± 10.42</td>
<td>107.00 ± 3.22</td>
<td>89.49 ± 5.43</td>
<td>0.349</td>
</tr>
<tr>
<td>After ephedrine</td>
<td>66.4 ± 6.99</td>
<td>91.4 ± 4.07</td>
<td>397.71 ± 12.87</td>
<td>305.32 ± 11.93</td>
<td>92.38 ± 2.75</td>
<td>119.30 ± 6.83</td>
<td>0.302</td>
</tr>
</tbody>
</table>
following epidural blockade. The difference after methoxamine or ephedrine is shown in figure 4. Methoxamine was accompanied by a further decrease in this index, the difference from the resting level being significant (P<0.005). Ephedrine reversed the effect of epidural block. There was a significant difference between the mean values in the methoxamine and ephedrine groups (P<0.001).

The ratio PEP/LVET was increased following epidural blockade. A further slight decrease occurred after methoxamine (fig. 5) while ephedrine produced a significant improvement as compared with the resting value. This difference between the effects of methoxamine and ephedrine on the ratio PEP/LVET was significant (P<0.005).

**DISCUSSION**

Shaver et al. (1968) showed that all phases of left ventricular systole were lengthened in association with an increase in arterial pressure following injection of methoxamine in subjects with normal arterial pressure, while Talley, Meyer and McNay (1971) noted that large increases in aortic diastolic pressure in dogs caused a small increase in the duration of the pre-ejection period. Recognizing this afterload effect on the left ventricular systolic time intervals, and to allow comparison between ephedrine and methoxamine, we attempted to restore arterial pressure to as near pre-epidural levels as possible with each drug.

Arterial pressure decreased following epidural block, while the Q→S₂ interval and the pre-ejection period lengthened. Methoxamine caused a further increase in Q→S₂ interval and PEP but ephedrine significantly shortened these indices. Methoxamine raised the diastolic pressure more than ephedrine.
FIG. 4. Changes in 1/P.E.P.² from pre-epidural values (baseline). 1/P.E.P.² was reduced after epidural block. The difference between methoxamine and ephedrine is highly significant (P<0.001).

FIG. 5. Changes in P.E.P./L.V.E.T. from pre-epidural values (baseline). This index increased following epidural block. The difference between methoxamine and ephedrine is significant (P<0.005).

but the difference in diastolic pressure between the pre-epidural state and after methoxamine was not significant.

Heart rate slowed after epidural blockade. A further slowing followed the administration of methoxamine, but the lengthening of Q→S₂ and PEP were shown by co-variance analysis (fig. 3) to be independent of this change in rate. Little alteration in heart rate occurred after ephedrine, a finding in agreement with the work of Stephen, Lees and Scott (1969).

A reduction in filling pressure, or preload, of the left ventricle may cause an increase in the PEP. Blackburn et al. (1973) showed that an increase in the pre-ejection period occurred when intrathoracic pressure was increased during the Valsalva procedure and Stafford et al. (1967) noted that the PEP/LVET ratio increased during the peripheral pooling of venous blood with venous occlusion tourniquets. As both PEP and PEP/LVET ratio increased with epidural block in our series, it is reasonable to suppose that, in an epidural block extending to the mid-thoracic region, this was caused by a reduction in filling pressure. The fact that methoxamine, in restoring blood pressure, did not significantly improve PEP, 1/PEP² or the PEP/LVET ratio may be associated with its poor vasoconstricting properties (Udhoji, Weil and Sambhi, 1964) and its failure to increase venous return. The significant shortening of PEP after ephedrine may reflect its ability to increase cardiac output and venous return (Stephen, Lees and Scott, 1969).

Karim (1965) demonstrated that methoxamine has a mild beta-adrenergic blocking effect, and Smith and Whitcher (1967) noted depression of cardiac output and left ventricular work. The prolongation of PEP following methoxamine in our study could have resulted from a deficient rate of myocardial force development in early systole with a consequent decrease in the rate of increase of intraventricular pressure throughout the pre-ejection period, while the significant reduction in 1/PEP² suggests that the maximum initial velocity of the ventricular muscle was slowed. The beta-adrenergic stimulating action of ephedrine, as noted by Ward et al. (1966), could be responsible for the significant shortening in Q→S₂ interval and PEP and the improvement in 1/PEP². This effect may be beneficial when upward extension of a lumbar epidural block to involve the cardiac sympathetic nerves adds impaired myocardial contractility to peripheral vasomotor paralysis (McLean et al., 1967; Otton and Wilson, 1966).

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REFERENCES

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