Transcutaneous aortovelography

Potentially useful technique in management of critically ill patients

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Pilot evaluation of the clinical use of transcutaneous aortovelography has been undertaken and suggests that this technique will be useful in the assessment of left ventricular function in the acutely ill patient. Changes in waveform as a result of therapy or alteration in clinical condition can frequently be noted by critical visual assessment. Increasing clinical experience suggests that quantification will permit interpretation of less obvious changes in the recorded waveform. Transcutaneous aortovelography has the simplicity, speed, and safety afforded by a non-invasive technique.

The treatment of a critically ill patient necessitates the use of various measurements to serve as guide lines in patient management. These measurements should be reliable, easily obtainable, reproducible, and preferably non-invasive.

In the management of these patients a compromise has frequently to be reached between obtaining objective indices and initiating therapy based on clinical assessment. Rapid deterioration in the patient’s condition may necessitate ‘blind’ therapy which, if inappropriate, may lead to further deterioration.

There is a need for a rapid, reliable technique for the repeated measurement of left ventricular output, so that changes in output can be observed. Transcutaneous aortovelography may meet this requirement. This preliminary report describes the use of this technique in an Intensive Therapy Unit.

Instrumentation and basic therapy
The instrumentation and basic principles of the technique have been described in the preceding paper (Sequeira et al., 1976).

Method
The method is described in the preceding paper (Sequeira et al., 1976). Clinical experience has shown that the transducer usually has to point downwards and to the left in order to align the ultrasonic beam with blood flow in the transverse arch of the aorta. The controls which are constantly used during recording—recorder on/off, display intensity, and an event marker—are on a small, hand-held, remote control box so that one person can conveniently operate the instrument (Fig. 1).

A reproducible recording could be obtained in approximately 90 per cent of patients. Difficulties arose in patients with pulmonary emphysema and in those who were restless or could not lie flat.

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Records cannot be obtained from patients with tracheostomies or patients with surgical emphysema of the neck.

**Interpretation**

The outline of the aortic blood velocity signal describes the highest Doppler shifts received at any time, corresponding to the maximum aortic blood velocity. Even if other vessels are within the beam, no vessel in this area normally contains receding blood flowing with such high velocities in a direction so closely aligned with the ultrasonic beam. Venous signals can usually be readily distinguished by their low Doppler shifts and diastolic timing.

Changes in the volume flow entering the descending aorta can be calculated from changes in maximum aortic velocities on the assumptions that the cross-sectional area of the vessel, its velocity profile, and the proportion of blood supplying the head, upper limbs, and coronary arteries (all of which is lost to measurement) remains constant between observations. Initially we found that visual assessment of the record gave useful sequential information (Fig. 2), but a quantitative analysis of the records is now being made to facilitate interpretation of less obvious waveform changes (Fig. 3). By fitting straight lines to the leading and trailing edges of the record, several measurements can be made. The height (Vxp) of the constructed triangle measures peak velocity, and the length of the base (tx) of the triangle ejection time. The area (A) gives an index of stroke volume, and the quantity V is the time-averaged or mean maximum velocity and is expressed in cm/s. Fig. 3 shows how these calculations are made. When respiratory variations are present, the record is analysed over a number of respiratory cycles. So far, these calculations have been made in a few patients only.

**Subjects studied**

Over a period of 4 months recordings were obtained from 68 subjects, 21 of whom were healthy volunteers. A total of 47 patients were studied (Table).
Fig. 5 Myocardial infarction. Transcutaneous aortovelograph recordings in two patients on admission to hospital.

Results

Normal subjects

Records obtained from the 21 healthy subjects gave some impression of the range of normal appearances (Fig. 4). Clearly more subjects are required in order to assess the range of normal values obtained and their statistical distribution. In our initial study we were primarily concerned with analysis of sequential changes in the same patient and not with comparison of data between different patients.

Myocardial infarction

All 21 patients studied had electrocardiographic evidence of a recent myocardial infarction. Patients

<table>
<thead>
<tr>
<th>Category</th>
<th>No. of patients studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial infarction</td>
<td>21</td>
</tr>
<tr>
<td>‘Shock’</td>
<td></td>
</tr>
<tr>
<td>Hypovolaemic</td>
<td>6</td>
</tr>
<tr>
<td>Septic</td>
<td>7</td>
</tr>
<tr>
<td>Drug overdose</td>
<td>5</td>
</tr>
<tr>
<td>Drug response</td>
<td>3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>5</td>
</tr>
</tbody>
</table>

with no clinical evidence of myocardial dysfunction other than a fourth heart sound had transcutaneous aortovelography records which were not obviously abnormal. Clinical evidence of deterioration in

Fig. 6 Transcutaneous aortovelograph recordings in a patient with complete heart block showing effect of endocardial pacing at different rates.
FIG. 7 Effect of endocardial pacing at different rates on time-averaged mean velocity calculated from recordings in Fig. 6

FIG. 9 Observations, including time averaged mean velocity ($\bar{V}$), obtained from patient admitted with hypovolaemic shock (numbers on graph of $\bar{V}$ refer to transcutaneous aortovelo graph traces shown in Fig. 8)

FIG. 8 Sequential transcutaneous aortovelo graph recordings taken from a patient with hypovolaemic shock and treated by volume replacement
1. Admission

2. Isoprenaline infusion for 1½ hours

3. 24 hours after admission. No isoprenaline

FIG. 10 Sequential transcutaneous aortovelograph recordings taken from patient admitted with amylobarbitone overdose

Cardiac output (a falling blood pressure, tachycardia, and peripheral vasoconstriction) was associated with a record showing a reduced peak and mean velocity and shortened ejection time (Fig. 5). Clinical improvement in cardiovascular status was also associated with signs of improvement in the transcutaneous aortovelogram, shown by rising peak velocity and longer ejection time. Further study is clearly required in order to establish whether transcutaneous aortovelography might be of value in the initial haemodynamic assessment of patients admitted with acute myocardial infarction and whether changes in sequential recordings might give a guide to prognosis.

Complete heart block associated with myocardial infarction

Two patients were admitted with complete heart block. Transcutaneous aortovelograph recordings obtained at different pacing rates in one of these patients are shown in Fig. 6. The results are presented by plotting the endocardial pacing rate against the mean velocity ($\bar{V}$) (Fig. 7). The mean velocity was found to be maximum at a pacing rate of 80/min. Measurements made from transcutaneous aortovelograph recordings at different heart rates in patients paced endocardially may make it possible to select the optimum pacing rate for that individual.

FIG. 11 Amylobarbitone overdose. Graph showing progress and response to therapy (numbers on graph of $\bar{V}$ refer to transcutaneous aortovelograph traces shown in Fig. 10)
‘Shock’

Serial recordings were obtained in shocked patients from the time of admission to the Intensive Therapy Unit; additional records were made when a drug or manoeuvre was expected to affect the haemodynamic status of the patient. Whenever possible appearances were interpreted in the light of changes in pulse rate, blood pressure, central venous pressure, urine output, and skin-core temperature differences.

**Hypovolaemic shock**

Recordings from patients admitted in hypovolaemic shock consistently showed a low peak and mean velocity, short ejection time, and small area. Fig. 8 shows the sequential changes in transcutaneous aortovelo-graph recordings from a patient admitted with shock and treated by fluid replacement. Fig. 9 shows the various observations made during the treatment of shock. The systolic blood pressure was unrecordable on admission and the mean velocity recorded by transcutaneous aortovelo-graphy was extremely low. Fluid therapy (combination of purified protein fraction and crystalloids) was begun on the basis of these findings and of evidence of fluid loss over the previous week. A central venous pressure line was not established until an hour later, but the measured central venous pressure was then less than zero (mid-thorax reference level) after intravenous infusion 800 ml in one hour. With increasing volumes of fluid infused, there was a steady increase in mean aortic velocity, central venous pressure, and blood pressure; the skin-core temperature difference decreased as peripheral perfusion improved. Records from patients with myocardial failure showed a similar appearance to those from patients in hypovolaemic shock, but these patients usually had a raised central venous pressure and did not show improvement after a fluid challenge. On the other hand, mean aortic velocity rapidly increased with a fluid challenge in hypovolaemic patients and this was associated with a simultaneous increase in central venous pressure.

**Septic shock**

In this condition, transcutaneous aortovelo-graph appearances are variable. Interpretation of these records will require detailed measurements and correlation with other cardiovascular indices in order to determine whether the technique will prove of value in the management of this condition.

**Drug overdose and drug response**

Fig. 10 shows sequential transcutaneous aortovelo-graph recordings taken from a patient admitted with an amyllobarbitone overdose. Fig. 11 shows the various observations made. At the time of admission, the central venous pressure was high, the pulse rate was 150/min, and the blood pressure unrecordable. The mean aortic blood velocity was low. These findings were consistent with the diagnosis of ‘shock’ secondary to a fall in cardiac output, presumably a result of the myocardial suppressant effect of amyllobarbitone. In view of these changes an isoprenaline infusion was started. This was followed by a fall in the central venous pressure, a rising blood pressure, a steady increase in urine output, and a dramatic increase in the mean aortic blood velocity. It was possible to regulate the dosage of isoprenaline infused in order to maintain an improved but submaximal mean velocity and a pulse rate of 110/min or less.

**Conclusion**

Our limited experience suggests that sequential transcutaneous aortovelo-graph recordings in conjunction with other clinical indices may be of value in the management of hypovolaemic shock and response to fluid therapy, and as an indicator of the effect of a drug on the cardiovascular system. They may also prove to be of particular value in the assessment of the therapeutic effect of inotropic agents and agents producing peripheral vasodilatation, so that a titrated dose can then be administered intravenously.

Transcutaneous aortovelo-graphy has the advantage over many other techniques for assessment of cardiovascular function being non-invasive. Recordings can be obtained from approximately 90 per cent of critically ill patients and sequential observations made with little patient discomfort. A pilot evaluation suggests that this technique provides useful information on cardiovascular status, and that serial records can give a guide to progress and response to therapy. Simple quantitative analysis of the tracings is likely to increase their value.

**Reference**