Editorial

British Heart Journal, 1976, 38, 433-442.

Transcutaneous aortovelography *A new window on the circulation?*

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Clinical need

Any quantitative description of a transport system is seriously deficient if it does not include information on the volume of flow carried. Yet perforce—such are the difficulties of obtaining a reliable index of blood flow—the management of a patient severely ill with a circulatory disorder is usually based on variables, like the arterial or pulmonary wedge pressure, etc. which have only a tenuous relation to systemic blood flow. While the experience and interpretative skill of the clinician can often make some allowance for the variable and non-linear relation which these variables bear to flow, this is an unsatisfactory situation.

Much effort has, therefore, gone into the development of techniques for the measurement of a haemodynamic index which closely and reliably reflects flow. Thus a number of methods for measuring cardiac output using gas uptake or indicator dilution techniques, or electromagnetic flowmeters have been developed; these are of great value when used with care and within their limitations, which unfortunately often affect their accuracy in low-output states (Prys-Roberts, 1969; Mills, 1972; Chamberlain, 1975). However, none of these methods is well suited for routine repeated bedside use in intensive and coronary care. Most are laborious: the invasive methods, in addition, cause discomfort to the patient and are not devoid of risk. Practical problems include the difficulty often experienced in introducing catheters into a shocked patient and the delay that may occur before measurements are available.

Two other techniques perhaps have something to offer in this field. The controversy about the estimation of stroke volume by *echocardiography* has now largely died down with the realization that plausible estimates can only be obtained in the

absence of dyskinesia (Gibson, 1973; Sweet *et al.*, 1975). Even when this can be excluded, the method is hardly suitable for repeated use in the wards, as reproducibility depends on a meticulous and time-consuming technique.

Thoracic impedance plethysmography (Baker *et al.*, 1971; Kubicek *et al.*, 1974) is still the subject of much controversy. Once the four electrode bands (which contribute to the stability of observations) have been applied, readings can be obtained in co-operative or passive patients during momentary interruptions of respiration. The product of the maximum rate of change of impedance and the ejection period (the latter often has to be obtained using the heart sounds) is interpreted as a measure of stroke volume. The correlations with cardiac output measurements which have been obtained in a number of studies have been variably adequate, but convincing correlations with changes in aortic blood velocity in individual patients were obtained in 6 out of 7 subjects in a pacing study (I. A. B. Brooksby and M. M. Webb-Peploe, 1976, personal communication). This technique appears to be useful for following changes in cardiac output in any one subject provided that cardiac and intrathoracic conditions do not deviate too far from normal. Rough estimates of absolute cardiac output may also be obtained under these circumstances. Little, however, has been published on practical experience with the method in intensive and coronary care. Serious problems might be expected in this area, because several extraneous factors likely to vary during treatment are capable of affecting the 'flow' signals. Some of these are the amount and distribution of extravascular fluid in the thorax (errors of -40% have been found by Baker and Denniston (1975) to arise on infusion of 100 ml saline into a dog's lung), the pulsatility and timing of venous

flow (Cross and Light, 1974), and the haematocrit. It is possible to allow for the error introduced by changes of haematocrit (Hill and Thompson, 1975), but only at the expense of operating convenience. Systemic and pulmonary blood pressures also might be expected to influence the signal by their effects on vascular elasticity (Becattini and della Corte, 1967), but no such effects appear to have been documented. The effects of abdominal distension are also unknown. In spite of the considerable effort which has gone into studying this technique, the precise origins of the impedance signal are still unclear, even in animals. Most of the signal probably derives from the pulsatile expansion of the major vessels, but in man the relative contributions from systemic arteries, the pulmonary circulation, and the major veins have not been established. The empirical nature of the measurement also hampers the systematic exploration of the limits of its validity (Light, 1976).

Most workers in this field have, therefore, found themselves driven back to obtaining such clues to blood flow, or changes in flow, as they can from a legion of indirect measurements and observations: blood pressure, central venous pressure, pulmonary wedge pressure, toe-to-core temperature difference, urine output and osmolality, blood gas analysis, and last but not least the patient's colour. As already noted, however, these measures are (with the partial exception of centrally-derived pulse pressures) strongly affected by other physiological variables. While this lack of specificity disqualifies them as reliable guides to body perfusion or heart action, many of them—when taken together with reliable information on systemic flow (or flow changes)—yield useful information on the other factors: the distribution of cardiac output, vascular tone, the adequacy of circulating blood volume, and ventricular filling. The limitations of some of the conventional haemodynamic measurements must, however, be borne in mind. For example, mean left atrial pressure does not necessarily represent left ventricular end-diastolic pressure (Braunwald and Frahm, 1961) and, in turn, pulmonary arterial wedge pressure sometimes is a poor indicator of mean left atrial pressure (Lozman *et al.*, 1974).

Information on central blood flow, therefore, is not only valuable in its own right, as reflecting pump function and the overall transport capability of the circulation, but it also allows more precise interpretation of the information obtained from the techniques in routine use in intensive care. Preferably, the measurement should be of phasic (instantaneous) flow because of the additional information then obtainable on the pattern of ventricular ejection and the integrity of aortic valve function. Ideally,

the flow should be indicated in absolute terms. The majority of clinical requirements would, however, be satisfied if the technique yielded a variable which in any one patient was proportional to flow. This would suffice to indicate his progress or the effect of treatment on his haemodynamic status.

The new technique

This is the background against which the new ultrasonic technique of transcutaneous aortovelocity (TAV), the subject of two papers in this issue (Sequeira *et al.*, 1976; Buchthal, Hanson, and Peisach, 1976), should be seen. It differs from echosonography in that what is measured is not the position of the reflecting interfaces (here the red blood cells), but the speed of their movement along the aortic arch—a variable closely related to blood flow. The principle employed, the Doppler effect, has a venerable history as a quantitative remote measurement method in fields as diverse as astronomy, aircraft control, and traffic law enforcement. In the medical sphere (Wells, 1969; Woodcock, 1975), it is in routine clinical use in a number of centres as a non-invasive means of observing fetal heart action and blood velocity waveforms in superficial vessels. Simple equipment lacking in directional resolution and linearity of response is commonly employed in the latter application (Light, 1972). Even so, much clinically useful information can be obtained when it is used within its limitations in relatively undemanding applications. The instrumentation developed for transcutaneous aortovelocity differs in that it retains the linearity and the potential for once-for-all-time calibration which are inherent features of the Doppler measurement principle. It also seems to have largely overcome problems associated with the intrathoracic position of the aorta, and its proximity to other vessels (Light and Cross, 1972; Light, 1974; Cross and Light, 1974).

The technique should allow the operator, who might be a nurse or technician, to measure blood velocity in the aortic arch in the majority of subjects. Multi-observer reproducibility trials in which Fraser *et al.* (1976) found the coefficients of variance to be less than 8 per cent suggest that inexperienced operators can obtain reproducible recordings of what—from theoretical considerations and circumstantial evidence—is likely to be the absolute value of 'mainstream' blood flow velocity in the proximal descending aorta. *This variable should be a useful near-quantitative index of relative systemic flow under most circumstances.*

Evidence for this is provided in one of the papers now published (Sequeira *et al.*, 1976).

Here it need be noted only that the observed blood velocity is linked to flow in the transverse aorta by two factors (and to cardiac output by three factors) which can, as a first approximation, be treated as constant in any one patient over a fair range of conditions. These are, the cross-sectional area of his aorta, and secondly, the transverse velocity profile in it. The third factor, the fraction of the cardiac output which flows into the transverse aorta, enters into the relation only when changes in cardiac output are to be deduced. When these factors remain exactly constant during serial measurements in any one subject, systemic blood flow will be exactly proportional to the indicated velocity, so that the latter is an exact index of what can be referred to as *relative flow*, i.e. the ratio of two serial blood flow values. The rationale for treating these factors as substantially constant under many practical conditions is not that they are truly invariable—for assuredly at least aortic dimensions and the distribution of cardiac output are not—but that under most clinical conditions their variations are sufficiently small compared with simultaneous changes in blood flow that velocity changes can represent changes in volume flow in a near-quantitative manner.

An estimate of the likely error introduced by the assumption of constant aortic cross-section can be derived from Goldberg's large series of echosonic *in vivo* measurements of diameter changes with pulse pressure in the proximal part of the human aortic arch (Goldberg, 1971). Typical diameter changes of 7 per cent were observed. These suggest that when a fall in blood flow of, say, from 5 to 2.5 litres per minute is accompanied by a fall in systolic blood pressure by 45 mmHg (6.0 kPa), the observed blood velocity will typically fall to 58 per cent of its previous value compared with the 50 per cent fall in flow. Greater divergence between flow and velocity ratios will occur in patients with highly elastic vessels and for greater falls in blood pressure, but, otherwise flow changes will be more accurately reflected by velocity changes.

The transverse velocity profile within any individual aorta would be expected to remain constant as long as the architecture of the proximal aorta is not altered. Supportive evidence is discussed in Sequeira *et al.* (1976).

While the accuracy with which flow changes in the transverse aorta can be deduced from velocity changes will thus depend on the circumstances, the errors involved are—with one exception (pathological deformation of the proximal aorta between measurements)—likely to be small (perhaps trivial)

compared with those which may be involved in deducing flow changes from the indirect measurements (BP, central venous pressure, PA wedge pressure, AV oxygen difference) which are in common use.

Experimentally, as reported in this issue, good proportionality was found between measurements by transcutaneous aortovelography and measurements of blood velocity and flow by invasive techniques, under clinical conditions. Comparisons with acetylene uptake studies, made with Dr. Brotherhood, suggest that proportionality also extends to changes with exercise (Light, 1974). Other workers have reported similar findings with closely related techniques (Mackay and Hechtman, 1975; Huntsman *et al.*, 1975). They confirm also that recordings can be simply and quickly obtained in the majority of patients and that the technique is compatible with the routines of clinical care. Preservation in the recording of the full spectral information helps to indicate when a transducer placed on the supra-sternal notch of the supine patient is properly aimed towards the aortic arch. The power levels of ultrasound involved (less than 50 mW/cm²) are most unlikely to present any safety hazard (Curzen, 1972; Ulrich, 1974; Wells, 1974).

The recordings of instantaneous (phasic) blood velocity, which are obtained by transcutaneous aortovelography, yield not only an approximate index of time-averaged flow, but also allow the manner of ejection of blood from the left ventricle to be observed. As discussed below, additional information which can be obtained from the velocity wave form about variables such as flow period, early systolic acceleration, peak velocity, and the duration of any turbulence present, is likely to be of clinical value.

What are the *disadvantages* of the technique? While reproducibility has been shown to be good and there is circumstantial evidence that the technique indicates a particular blood flow velocity with reasonable accuracy, we suggest that only *relative volume flow* should be deduced from this at present. Calculations of *absolute volume flow*, using independently derived information on aortic diameter, involve assumptions that (a) the transverse velocity profile is perfectly flat, and (b) the aortic cross-section is circular. Our findings suggest that such calculations of absolute flow sometimes yield values which are too inaccurate for anything much more demanding than the differentiation of hyperdynamic from hypodynamic conditions. When knowledge of absolute cardiac output is essential, a single 'calibration' measurement on the particular patient by another technique is required.

The *reliability* of this remote-sensing technique

FIG. 1 Non-invasive aortic blood velocity recordings by transcutaneous aortovelocityography, taken at various pacing rates during diagnostic catheterization of two patients with suspected ischaemic heart disease. Phasic blood velocity during systole is indicated by the outline of the dark complexes. The velocity (vertical) and time (horizontal) scales are identical in all recordings.

Although all aspects of the wave form (including early systolic acceleration and the duration of systolic flow) are affected by pacing rate in the two patients, the peak velocity and the area of the complex (an index of stroke volume) show the most obvious differences. The transient response to changes in heart rate (not illustrated) also differed in the two patients. It is hoped that similar studies of the response to non-invasive interventions may contribute to the practical assessment of left heart function. (From studies conducted with Dr. R. F. Sequeira.)

has been extensively investigated (Light, 1974; Light *et al.*, 1976). The apparently complex grey-scale spectral recording, which is provided (Fig. 1), allows the wanted blood velocity information to be distinguished from artefacts. Results appear to be trustworthy whenever the appearance of the aortic velocity outline satisfies some simple criteria (Light, 1976). Dubious readings, which are occasionally obtained, can thus be recognized and discarded. The need for inspection of the record,

however, implies that wholly automatic data extraction is not feasible at present. The bedside quantification of edited recordings is nevertheless conveniently achieved by the use of mechanical aids or a free-stylus planimeter (Light, Lowe, and Rosenthal, 1974b) developed for this purpose.

Adequate signals cannot be obtained from the body surface in patients in whom ultrasonic access is obstructed by tracheostomies, or surgical emphysema, and also from a small proportion of other

subjects. (An alternative approach, by an oesophageal probe, may then be appropriate: Olson and Cooke, 1974; Duck, Hodson, and Tomlin, 1974.) In a further proportion of subjects, from whom only barely adequate signals are obtained, patience and experience are required to produce usable recordings.

Finally, mention should be made of two major practical disadvantages: (1) Because relatively complex signal processing is involved, the instrument will be more expensive than, say, an electrocardiograph; one instrument, however, can cover a ward or department. (2) The technique and the variables observed are relatively novel and the relevant published material is therefore sparse: aortic blood velocity observations have hitherto been largely restricted to sedated cardiac patients with the type and severity of disease that warrants catheterization. However, as the technique is non-invasive and relatively simple, a clinician can rapidly familiarize himself with it and the interpretation of the data obtained.

Potential value of transcutaneous aortovelocity

It is possible to foresee ways in which this new technique will add to our understanding of the behaviour of the circulation in health and disease. The observation that some hypotensive agents produce predominantly a reduction of peripheral resistance and others a reduction of cardiac output was not unexpected (Birkenhäger *et al.*, 1975; Light *et al.*, 1976). On the other hand new questions will be asked, and new perspectives gained.

For example, clinically useful information may be obtained by analysis of various aspects of the velocity wave form in states of depressed left ventricular function (Hanson and Buchthal, 1976). Pathological changes are not confined to the slope of the leading edge of the systolic complex, representing early systolic acceleration (dV/dt), which is an index of left ventricular 'contractility'. Major changes also take place in other aspects of the wave form (e.g. peak velocity, deceleration, and ratio of systolic to diastolic period). It became apparent that much of what has been established by work on anaesthetized animals is not directly applicable to unsedated or lightly sedated man, for reasons that have been summarized by Vatner and Braunwald (1975). A pilot examination of what can be deduced from the waveform was conducted with an eye on the pressing practical problem of assessing left ventricular function in patients. This has led to a closer definition of the possibilities and to the appreciation that another

clinically important variable might be measured—an index of the neurocirculatory status of the critically ill patient.

In general it is necessary to distinguish between various circulatory operating 'regimens' which impose differing constraints on the regulatory mechanisms—whether the patient does or does not possess some cardiac reserve, and whether left heart function or venous return governs cardiac output. (The transient response to a sudden stimulus—observable only by a beat-by-beat method—should allow differentiation between the latter alternatives: a greater short-term enhancement of stroke volume may, for example, be produced when dilated pulmonary veins hold a reservoir of blood.) The heart normally possesses considerable functional reserve, so that the distinction between actual functional state and the potential functional capability is important—hence the widespread use of exercise and other stress tests. Whenever this functional reserve is positive, extrapolation from an observation of functional state to an estimate of capability would require precise information about the factors, including the intensity of autonomic nervous stimulation and the biochemical milieu, which define the inotropic state at the time of observation. Neither this sort of information, nor the knowledge of how to carry out the extrapolation, is at present available. Some such information may, however, become available from inspection of the wave form, if the suggested interpretation of the wave form changes illustrated in Fig. 2 applies also when some cardiac reserve is present.

We would therefore suggest that if valid deductions of left ventricular capability are to be based on haemodynamic measurements, observations of the response to such interventions as exercise, change of posture, non-invasive sequestration and controlled release of part of the circulating blood volume, etc. will be required, rather than single measurements (Light, Cross, and Hansen, 1974a). This proposition has not yet been seriously examined, but observations of the wide individual variations in the response of the aortic velocity waveform to changes in heart rate with pacing (Fig. 1) suggest that some progress may be possible along these lines. This reasoning also leads to another hypothesis, that the interpretation of the observations might be simpler in patients who were known to have inadequate cardiac output at rest, because the problems associated with the existence of a positive functional reserve would then presumably be absent.

As it is generally accepted that one result of impaired left ventricular function, whether this is the result of diffuse myocardial involvement, a focal lesion, uncoordinated excitation, or inadequate

FIG. 2 A diagrammatic representation of the time-course of aortic blood velocity, as seen in patients with gross impairment of circulatory function, with suggested interpretation. The index of central blood flow obtained from transcutaneous aortovelocity recordings is the time-average of the indicated phasic blood velocity (dashed line). Different ways in which this may be depressed are illustrated by the blood velocity waveforms (b) and (c). The deviation from what might be a typical normal waveform at the corresponding heart rate is also indicated.

If it is verified that waveform (b), expressing the primary depression of left heart function, is indeed transformed by sympathetic stimulation into waveform (c), as commonly seen after severe myocardial infarction or in hypovolaemic shock, it may prove possible to derive indices from aspects of the waveform which separately indicate the degree of primary myocardial depression and of reactive sympathetic stimulation. Note that the early systolic acceleration (dV/dt) of aortic blood velocity in waveform (c) is paradoxically high only when compared with normal subjects at rest. A more appropriate comparison might be with normals in a state of increased sympathetic tone.

filling pressure, is a reduction in contractility, we expected to find low values for the early systolic acceleration (dV/dt) under these circumstances. A wide range of observations on patients with severely depressed haemodynamic status in intensive and coronary care, however, showed that the majority had raised values of dV/dt compared with normals at rest. Associated findings were that the duration of systolic flow was much shorter than normal for the particular heart rate, peak velocity was reduced, and the area under the velocity waveform, an index of stroke volume, was grossly depressed. Fig. 2c shows the wave form commonly seen in these patients.

A possible explanation for these findings is that

the observed increase in dV/dt and shortening of the ejection period is a response to the sympathetic stimulation which is known often to accompany inadequate output. It is suggested that the primary circulatory depression results in the waveform of Fig. 2b and that increased sympathetic stimulation not only increases the heart rate, but converts what would otherwise be a pathologically slow but sustained myocardial contraction into an apparently vigorous but disproportionately brief movement. Some support for this view comes from instances when the expected primary depression of dV/dt (Fig. 2b) has in fact been seen: these include subjects under deep halothane anaesthesia and some with diabetic neuropathy. Depression of dV/dt has

also been seen in catheterization studies on patients with ischaemic heart disease (Bennett *et al.*, 1974; Jewitt *et al.*, 1974). It will be interesting to establish whether the broad difference between these observations on one hand, and those in intensive and coronary care units on the other, is the result of differences in the severity of the haemodynamic disturbance, acuteness of its onset, the sedation used, or other factors (Shillingford and Thomas, 1967).

The high observed dV/dt values may also in part be caused by an abnormally low afterload, i.e. low peripheral resistance, in many hypotensive intensive care patients. Reduced afterload would not account, however, for the shortening of systole, and it probably contributes only little to the 'paradoxical' increase in dV/dt (van den Bos *et al.*, 1973). The relatively intense reflected pressure wave from the periphery, which is found in vasoconstricted patients (Mills *et al.*, 1970; McDonald, 1974), and is an indirect result of increased sympathetic activity, may contribute to the shortening of systole but is not thought to be a major factor: in many hypotensive patients systole is indeed over before this reflected wave arrives at the measurement site.

If the interpretation of the waveform put forward in Fig. 2 indeed allows differentiation between the primary deficit and the reflex reaction, this should contribute to an assessment of left ventricular state as well as providing an indicator of pathological 'stress' (Bushman, 1973). An increase in early systolic acceleration without concomitant increase in an abnormally low ratio of systolic to diastolic period might thus be an indicator of increased sympathetic activity. Some observations suggest that this type of change is an unfavourable sign (Hanson and Buchthal, 1976). Any estimate of depression of myocardial function thus obtained is likely, however, to be very approximate. The observed similarity of velocity wave forms in severe hypovolaemia and in severe myocardial ischaemia suggests that further differentiation between extrinsic and intrinsic causes of myocardial depression is unlikely to be feasible on the basis of static observations alone. Observation of the effect of interventions, such as trials of specific therapeutic measures, will again be required. For example, while adequate volume replenishment will restore blood velocity to normal if the *sole* cause of depressed function is hypovolaemia, the response to blood volume adjustment will be less in subjects with cardiac lesions. Presumably these changes are the result of adjustment of the atrial pressures to allow the heart to operate at the maximum of whichever ventricular function curve is limiting the cardiac output.

Some other observations of pathophysiological interest concern the *distribution of cardiac output and abnormal venous flow patterns*. When normal cardiac output is restored after a period of relative ischaemia, relaxation of the peripheral vasoconstriction is not necessarily immediate. Normal or hyperdynamic conditions may be seen in the descending aorta under these circumstances for perhaps two hours before limb perfusion returns to normal. A possible explanation is that the phenomenon of reactive hyperaemia, familiar in the peripheral circulation, may also occur in the central circulation and for a time predominate. Signals from venous flow, probably from the innominate vein, are often also incidentally observed. They can be resolved from aortic signals as they normally appear in a different shade of grey and have a different wave form, which is elongated, with relatively low Doppler shifts and strong respiratory variation. In occasional patients with serious circulatory impairment, however, a very different venous flow pattern is seen (Cross and Light, 1974): this resembles arterial flow in having near-triangular wave form, very high peak velocities (equalling or exceeding those in aortic flow), but is diastolic in timing. The circumstances associated with this phenomenon, which probably accounts for the diastolic peak sometimes seen on thoracic impedance plethysmograph recordings (Kubicek *et al.*, 1974), still have to be elucidated.

Applications to patient assessment and management

While the above are examples of unfamiliar phenomena seen through this new 'window on the circulation', most observations are readily interpreted. This applies particularly to the (usually simultaneous) increases in peak velocity, flow duration, and time-averaged (mean) velocity, which reflect an improvement in body perfusion. Even in such instances, however, more sophisticated analysis of the data may well give more detailed information of clinical value about the state of cardiac function and the circulatory system. Taken together with the measurements conventionally used in the care of the critically ill patient, non-invasively acquired aortic blood velocity data should give a fuller picture of the changing haemodynamic status than has hitherto been readily available. In particular, the patient's immediate response to various therapeutic interventions should be a guide to optimum management in many situations during intensive care. Thus, if blood flow is seen to respond positively to a trial infusion of fluid (Bradley, Jenkins, and Branthwaite, 1970;

Loeb *et al.*, 1973) and the central venous pressure does not rise excessively, volume expansion may be continued until the optimum loading, which will be signalled by a falling-off in improvement, is approached. Further improvement of cardiac output may then be achieved by the monitored use of inotropic and/or afterload-reducing agents (Wyse, Gibson, and Branthwaite, 1974; Hood, 1975). Such closely supervised therapy might be expected to yield better results than these measures used singly or blind. In other patients, the technique may be valuable to check on the effects of ventilator or pacemaker adjustments.

The cardiologist, in addition, should find the wave form abnormalities seen in aortic valve disease and hypertrophic obstructive cardiomyopathy of interest (Light, 1974). Visualization of blood velocity in the pulmonary artery, which can (with somewhat greater difficulty) be carried out in children by the same instrument (Cross, Light, and Sequeira, 1974; Cross and Light, 1974), may assist the paediatrician in the non-invasive diagnosis of congenital defects.

Possible future developments

The information provided by transcutaneous aortovelocity complements that given by echocardiography; it supplements the pictures of intracardiac structure and local function (Gibson and Brown, 1976; Weissler, 1974) obtained by the latter by providing a measure of *overall* left heart function. For diagnostic purposes, these two non-invasive ultrasound techniques may be joined by a third—a range-gated narrow-beam Doppler technique for examining the timing and nature (laminar or disturbed) of local flow through the heart valves (Baker and Johnson, 1975; Angelsen, Aaslid, and Brubakk, 1975). Such instruments for sensing highly localized blood motion may indeed be incorporated in 'duplex' echocardiography units. However, because of the increased difficulty in obtaining reproducible data on aortic flow velocities with highly selective instruments, the latter are unlikely to compete with transcutaneous aortovelocity for serial observations of relative systemic flow. (Complexity of operation and difficulties of access also appear to preclude the transcutaneous application to the deep thoracic vessels of the multi-gate pulsed Doppler techniques (Histand, Miller, and McLeod, 1973; Doroit *et al.*, 1976) now being developed for the measurement of absolute flow.)

Techniques derived from transcutaneous aortovelocity, but with different (simplified) signal processing and/or display methods, are being

investigated in several centres (Huntsman *et al.*, 1975). Thus Boughner has shown that in expert hands limited objectives (the quantification of aortic and mitral regurgitation) can be achieved with simpler signal processing (Boughner and Nicol, 1974; Boughner, 1975). Such variants, however, must be distinguished from the parent technique, as there is much evidence to suggest that 'simplification' is attended by sacrifices in one or more important features of transcutaneous aortovelocity: reliability of serial flow estimations, suitability for use by non-specialist personnel, and speed of data acquisition. It is probable that greater operational convenience than has already been attained by transcutaneous aortovelocity can be achieved, but more complex signal processing will be required if the present visual data extraction from a spectral display is to be replaced without loss of reliability by the use of, for example, an analogue output which directly indicates aortic blood velocity.

Conclusion

In summary, transcutaneous aortovelocity is a non-invasive ultrasound technique, based on a well-understood quantitative principle, for recording the phasic mainstream blood velocity in the human transverse aorta. Reproducibility studies suggest that velocity changes exceeding 10 per cent can be detected with greater than 90 per cent confidence. Serial velocity measurements appear to give a useful near-quantitative measure of changes in blood flow in the central circulation and also to indicate some of the characteristics of left ventricular ejection on a beat-by-beat basis.

Sufficient laboratory and clinical experience has now been gathered for an early assessment of its clinical potential: clues to the state of the patient are given by several aspects of the instantaneous velocity wave form. Serious hyperdynamic and hypodynamic conditions appear to be indicated by abnormalities of scale, and most lesions of the left ventricular outflow tract are marked by characteristic irregularities. Transcutaneous aortovelocity is likely to prove a simple way of quantifying regurgitation (which is indicated on a bidirectional display) and for reviewing prosthetic valve function during follow-up. The range of abnormal wave forms seen in severe circulatory depression is believed to reflect the degree of sympathetic response to the primary functional disturbance, and illustrates the importance of examining the whole of the systolic velocity wave form. Observation of the effect of diagnostic or therapeutic interventions is likely to give further useful information in assessment of patients. Greater flexibility in patient

management and a closer approach to optimal therapy may result from the immediate and continuing feedback of data relating to the patient's haemodynamic response, which is conveniently made available by transcutaneous aortovelocity.

It is a rash man who attempts to set into perspective a technique which he has helped to develop. The danger of prejudice is great, and I cannot hope to have escaped it entirely. It is an even rasher man, who as a non-clinician, also engages in some of the more controversial issues of cardiology. I hope, however, that these observations will contribute to the debate. I am most grateful to the many clinicians upon whose experience I have drawn, notably Drs. G. C. Hanson and D. C. White, who have shared and discussed their findings with me, to Professor E. N. Terry, Dr. H. L. Green, Dr. J. G. Jones, and Mr. H. S. Wolff for critical comments on this manuscript, and to Mr. G. Cross and other colleagues for their technical help. The responsibility for misinterpretations and for the more speculative elements in the above discussion is however mine alone.

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