CRITICAL REVIEW

Transcutaneous Measurement of Arterial Flow Velocity with a Doppler Flowmeter in Normal Subjects and in Patients with Cardiac Dysfunction*

A. Benchimol, M.D., Antonio Pedraza, M.D., Leib Brener, M.D.,
Asher Buxbaum, M.D., Marvin R. Goldstein, M.D., and Jack Garlan

Telemetry of phasic arterial flow velocity in man has been made possible with the development of a Doppler ultrasonic flowmeter system by Franklin and associates1-7 and others.8-11 This technique has provided an opportunity to study abnormalities of peripheral arterial flow velocity in health and diseased states using probes inserted around the peripheral arteries on surgically exposed vessels.12-14 Further technical developments8,9 have demonstrated the potential usefulness of this technique to study arterial flow velocity of the unexposed vessel lying in close proximity to the skin. This approach has been used by Strandness and co-workers10,11 to study patients with occlusive arterial disease and by Ware and Laenger12 to determine blood pressure.

This report is a review of our experience with transcutaneous measurement of peripheral arterial flow velocity in normal subjects and in patients with cardiovascular diseases.

MATERIAL AND METHODS

One hundred seventy patients were studied. There were 37 normal subjects and 133 with various types of heart disease. Thirteen of the normal subjects had normal cardiovascular function as defined by right and left heart catheterization, indicator dilution curves and selective cineangiography. The remaining 24 normals who did not have cardiac catheterization were all employees of the hospital; all were asymptomatic and had no clinical or laboratory evidence of heart disease.

One hundred of the diseased group had the anatomic and physiologic diagnosis established by right and left heart catheterization, selective cineangiography and selective indicator dilution curves. The remaining 33 patients who were not subjected to cardiac hemodynamic studies had the usual clinical and laboratory evidence in keeping with their diseased states.

Fifteen patients of the diseased group were studied in the Coronary Care Unit where continuous monitoring of the transcutaneous arterial blood flow velocity was obtained. All had the clinical and laboratory evidence of acute myocardial infarction.

Flow velocity curves were obtained at rest in all patients. In 47 patients, measurements of arterial flow velocity were made during spontaneous or catheter induced cardiac rhythms; in 37, artificial atrial and ventricular pacing was employed using an external pacemaker connected to a bipolar catheter placed in the right heart chambers. In addition, recordings were made during administration of drugs (inhalation of amyl nitrite, intravenous administration of isoproterenol during coronary arteriography, before and after conversion of atrial fibrillation to sinus rhythm).

The arterial monitoring sites in the 170 patients included 138 examinations of brachial artery velocity at the level of the antecubital fossa, 26 of the dorsalis pedis artery and six of the radial artery at the wrist.

In 13 patients simultaneous measurements of brachial artery flow velocity were made with a transcutaneous probe positioned over the artery and a cuff probe placed around the surgically exposed artery. The transcutaneous probe was positioned immediately above or below the implanted probe. The surgical technique of implanting these probes in human beings has been previously described.13-15

Intracardiac and arterial pressures in patients subjected to cardiac catheterization were made with saline filled No. 7 or 8 end-lumen cardiac catheters using a strain gauge manometer (P23 Db Statham).

In all cases the transcutaneous arterial blood flow velocity was determined using the Doppler ultrasonic flowmeter.
telemetry system as developed by Franklin.1-6 The technique briefly consists of the use of a Doppler shift principle. A high frequency ultrasound (7 to 10 mega Hertz) is generated by a lead zirconate titanate piezoelectric crystal and coupled to the intact skin by a gel. The crystals are mounted on a plastic unit at about 45 degrees to the skin surface (Fig 1). Part of the sound is reflected or back-scattered and detected by a similar receiving crystal mounted on the same transducer. The reflected signal differs in frequency from the incident signal by a quantity which is proportional to the velocity of the target, in this case, blood cells. Thus, the frequency shift of the backscattered sound is proportional to the blood flow velocity. Detailed description of this technique has been previously described.1-6

Special precautions should be observed with the transcutaneous technique. A good coupling between the transducers and the area of the skin overlying the blood vessel wall must be obtained. This can be accomplished by using a commercially available gel (Aquasonic 100 Ultrasonic Transmission Gel, Parker Laboratories Corp.). After the transducer has been filled with the gel solution, pressure adhesive (Eastman 910 Pressure Adhesive, Eastman Kodak Company, Inc.) is placed on the inner surface of the plastic transducer to assure gluing the probe to the skin over the blood vessel. The area of the skin over the artery with maximal pulsatile excursion was used to center the probe. Once the probe had been fixed to the skin, motion of the patient interfered little with monitoring the arterial flow velocity.

The flowmeter audio signal, intracardiac and arterial pressures and the analogue record of the flow velocity curves were continuously monitored and recorded in a multichannel tape recorder (Sanborn Model No. 3900) and on a light beam oscillograph recorder (Electronics for Medicine, Model DR 12) at various paper speeds and time lines.

RESULTS AND COMMENTS

Normal Pattern of Arterial Flow Velocity

The analogue record of the transcutaneous brachial artery flow velocity with this technique consists of a major systolic wave related to ventricular systole. This is a high frequency signal whose frequency shift is in the range of 0 to 4,000 cycles per second or 0 to 4 kilo Hertz (K Hz) which corresponds to a calculated blood flow velocity in the range of 0 to 40 cm/sec. The peak flow velocity is slightly smaller in the radial and dorsalis pedis arteries as compared with the brachial artery (Fig 2). This systolic wave represents forward flow (ie, peripherally directed) and correlates well in timing with intraarterial pressure curves obtained from the same site. Its onset follows the beginning of the QRS complex of the lead II of the electrocardiogram by approximately 0.12 second at the brachial artery, 0.16 second at the radial artery and 0.20...
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A secondary wave follows the primary or anterograde wave and its nature is poorly defined. This secondary wave occurs in early diastole as determined from the second heart sound of the phonocardiogram. It coincides in time with the dicrotic wave of the arterial pressure curve recorded from the same site. The peak amplitude of this wave is approximately one-third to one-fourth of the peak of the primary systolic wave, and is rounded with relatively slow rise and decline. Arterial retrograde (ie, centrally directed) flow seems to be the probable source for this wave. The wave form of primary systolic wave was nearly identical for the brachial, radial and dorsalis pedis artery flow velocity curves (Fig 2).

The wave form of the flow velocity curve obtained with the transcutaneous probe and with the probe inserted around the artery showed nearly identical contour (Fig 1). Pharmacologic agents (Fig 3), arrhythmias, exercise and other maneuvers changed the wave form and amplitude of the transcutaneous and implanted flow velocity tracings in virtually the same manner.

Cardiac Arrhythmias

Atrial extrasystoles caused a variable peak flow velocity which was dependent on the preceding diastolic interval (Fig 4). Extrasystoles occurring very early in diastole, ie, 0.36 sec. or less after the previous sinus beat, did not result in any significant measurable flow velocity. Compensatory pause did not occur and the peak of the postextrasystolic beat indicating the end of mechanical systole (Fig 2).
was about 20 percent higher than the usual sinus beat.

In a ventricular extrasystole the peak arterial flow velocity was small and correlated well with its timing during the cardiac cycle (Fig 5). The shorter the interval between the preceding sinus beat and the extrasystolic beat the smaller the peak flow velocity; the area under the curve of the postextrasystolic beat was 30 percent to 40 percent higher than the normal sinus beat. This postextrasystolic increase in flow velocity was a common occurrence during this arrhythmia.

The technique appeared to be particularly useful in identifying atrial and ventricular extrasystolic beats which were associated with a measurable arterial flow velocity, an index to the mechanical efficiency of each ventricular systole (Fig 6).

In patients with atrial fibrillation peak flow velocity varied from beat to beat (Fig 7). The peak velocity and the area under the curve of each beat was directly proportional to cycle length, inversely proportional to the magnitude of peak flow velocity of the preceding beat, and followed the variations in the arterial pulse pressure recorded from the same site. This variation in beat to beat peak flow velocity was particularly apparent when the ventricular rate was greater than 120 to 140 beats/min. Conversion of atrial fibrillation to sinus rhythm resulted in a more uniform peak flow velocity for each beat but the peak flow was essentially identical to that before conversion, providing the heart rate did not change appreciably (Fig 7). Spontaneous or artificially induced atrial tachycardia reduced peak flow velocity; the magnitude of this decrease was directly proportional to the heart rate. Regularity of the rhythm, uniform area under the curve, nearly identical peak flow velocity from beat to beat were important in recognizing atrial tachycardias (Fig 8) and differentiating it from ventricular tachycardia (Fig 9, 10). Peak flow velocity was higher during atrial tachycardia than during ventricular tachycardia at the same rate. When conduction through
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M.M. - 60 F. - MITRAL INSUFFICIENCY

Figure 6. Lead II of the electrocardiogram and dorsalis pedis artery flow velocity in a 60-year-old woman with mitral insufficiency. The patient's rhythm is atrial fibrillation. Note the presence of multiple ventricular extrasystoles and a short run of ventricular tachycardia resulting in marked decrease in peak flow velocity. Peak flow velocity during atrial fibrillation varies from beat to beat which is related to the diastolic filling period.

In patients with spontaneous, catheter or pacemaker induced ventricular tachycardia, peak arterial flow velocity and the area under the flow velocity curve were significantly reduced; the diastolic flow component was increased. The flow pattern in this situation was nearly continuous throughout the cardiac cycle, presumably as a result of the short diastolic period. In addition, there was marked variation in beat-to-beat peak flow velocity, particularly noticeable at rates above 150/min. Of particular significance was the fact that in pacemaker induced ventricular tachycardia, regular electrocardiographic rhythm was present at the same time that monitoring of the peripheral flow velocity showed marked variation in peak flow velocity of individual heart beat, suggesting marked impairment of peripheral perfusion. It is noteworthy to emphasize that approximately 50 percent of the "regular electrocardiographic beats" resulted in no measurable arterial flow velocity. Termination of ventricular tachycardia (spontaneous or induced by manipulation of the catheter in the cardiac chambers) was abrupt and usually preceded by a beat with a higher peak flow velocity than the other beats recorded during this arrhythmia.

Complete Heart Block

In patients with complete heart block there was moderate variation in beat-to-beat peak flow velocity which was directly related to the timing of atrial contraction during the cardiac cycle (Fig 11).

Ventricular Fibrillation

One patient developed ventricular fibrillation

R.P. - 25 M. - MILD AORTIC STENOSIS

Figure 7. Lead II of the electrocardiogram and dorsalis pedis artery flow velocity in a 25-year-old man with mild aortic stenosis. The tracings on the left were taken during a short episode of atrial fibrillation. The one on the right is a continuous recording of spontaneous conversion of atrial fibrillation to sinus rhythm. Peak flow velocity after conversion to sinus rhythm is only slightly higher than during atrial fibrillation.
Figure 8. Dorsalis pedis artery flow velocity and lead II of the electrocardiogram in a 52-year-old woman with patent ductus arteriosus. The tracing on the left was taken during an episode of atrial tachycardia and the one on the right during sinus rhythm and bigeminal rhythm. During bigeminal rhythm there is an alteration in peak flow velocity. The beats with lower peaks are the ones originating in an ectopic ventricular focus.

while being monitored. This record is illustrated in Figure 12. With onset of this arrhythmia, there was an immediate cessation of flow velocity which persisted as long as this arrhythmia continued. Defibrillation with a countershock resulted in restoration of flow velocity pattern a few beats after correction of the arrhythmia.

Coronary Arteriography

Injection of contrast agent into the coronary arteries for the purpose of obtaining coronary arteriograms resulted in uniform bradycardia and, in the majority of cases, no cardiac activity was recorded for several seconds. There was no measurable flow velocity during bradycardia and/or cardiac arrest as illustrated in Figure 13.

Aortic Valvular Disease

The wave form of the transcutaneous arterial flow velocity in aortic valvular stenosis showed the following abnormalities of the primary wave: slow rise of the ascending limb, decreased peak flow velocity, prolonged and round peak, increased area under the curve (Fig 14).

In patients with aortic insufficiency, the abnormalities of the primary systolic wave included: rapid ascending limb, sharp peak, rapid fall-off, increased area under the curve. A large secondary wave with a slow fall-off was common.

Three patients with idiopathic hypertrophic subaortic stenosis demonstrated a rapid ascending limb, increased area under the curve and a late secondary peak in mid or late ventricular systole. The diastolic wave was of a normal size and configuration. These findings were most pronounced during administration of isoproterenol or following an artificially induced ventricular extrasystole.

Coronary Care Units

Monitoring of the transcutaneous dorsalis pedis artery blood flow velocity was obtained in the Coronary Care Units in 15 patients with acute myocardial infarction. A short run of ventricular tachycardia was recorded and during this arrhythmia there is a significant decrease in peak flow velocity.

Figure 9. Lead II of the electrocardiogram and dorsalis pedis artery flow velocity in a 60-year-old man with acute myocardial infarction.
cardiac infarction and in patients with pulmonary edema. Measurements have been continued for periods of as long as 72 hours without evidence of untoward effects. Audible monitoring of the flow velocity signal with a portable transistorized FM tuner made possible for the nurse to detect these arrhythmias from a distance. Two patients developed pulsus alternans despite the presence of regular electrocardiographic rhythm (Fig 15). In both cases the presence of pulsus alternans preceded the onset of severe left ventricular failure by 30 to 60 minutes.

**FIGURE 10.** Lead II of the electrocardiogram, brachial artery flow velocity and aortic pressure in a 62-year-old man with coronary artery disease. The tracings were taken during ventricular pacing at increased rate of pacing. Note a progressive decrease in flow velocity which becomes irregular and markedly reduced at rates of 160 beats/min.

**DISCUSSION**

It appears clear that this technique is of value in the study of peripheral arterial circulation in man under a variety of conditions, particularly because of important hemodynamic data can be obtained atraumatically, rapidly and on a continuous basis.

The alterations in flow velocity wave form in patients with aortic valvular stenosis, aortic insufficiency and idiopathic hypertrophic subaortic stenosis appear to be of important diagnostic value, although the mechanisms responsible for the alteration in these conditions is not completely un-
In aortic valvular stenosis, mechanical obstruction may slow blood flow velocity in the early part of systole and cause a delay in the ascending limb of the flow curve. The increased duration of mechanical systole observed in this condition may be responsible for the increased area of the curve. In hypertrophic subaortic stenosis, initial ventricular ejection is unobstructed and the outflow tract of the left ventricle empties rapidly, but as ventricular systole progresses, the obstruction becomes important causing a delay in the emptying of the body of the left ventricle. In aortic insufficiency, the velocity of the ventricular ejection is fast and stroke volume is large, resulting in a rapid rise of
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The flow velocity curve\textsuperscript{17,18} to a high peak. The increase in the secondary wave may reflect increased retrograde flow resulting from regurgitation across the aortic valve. However, accurate recognition of retrograde flow must await development of a directional Doppler flowmeter.

This technique appears to be valuable in the assessment of cardiac performance during arrhythmias such as atrial and ventricular tachycardias, extrasystoles, etc. The marked abnormalities in peripheral arterial blood flow velocity during ventricular tachycardia indicate the severity of the hemodynamic abnormalities caused by this arrhythmia, particularly pronounced in patients with severe myocardial disease.\textsuperscript{19}

This technique seems to be useful as a monitoring device in the coronary care units, intensive care units, and operating rooms where cardiac arrhythmias, ventricular failure and shock are common occurrences. It is clear that this technique is not intended to replace the electrocardiogram as a monitoring tool but we feel that it will supplement the monitoring value of the electrocardiogram for several reasons: (1) the flowmeter signal is stable, the patient motion produces little artifact; (2) the flowmeter signal has not been significantly influenced by somatic tremor or 60 cycle interference; (3) monitoring of the arterial flow velocity provides indication of the peripheral hemodynamic status, while the electrocardiogram simply reflects the existence of an electrical signal which, although regular, may or may not be associated with effective mechanical pumping; (4) monitoring of the mechanical beat is useful in detecting the appearance of pulsus alternans, usually an indication of severe impairment of left ventricular function. It is of particular interest to note that in two patients who developed pulsus alternans detected with this technique, pulmonary edema followed within one hour. No suggestion of this was apparent by routine electrocardiographic monitoring. The implications here are that a predictive clinical status may be obtained prior to the catastrophic condition. The information thus obtained may permit the clinician the opportunity of initiating appropriate prophylactic therapy; (5) the fact that monitoring of the audio signal can be telemetered offers a unique advantage over other monitoring techniques in the coronary care unit. The nurses in charge of the patient may monitor the heart beat by ear from a distance and need not observe the monitoring oscilloscope. Audio mon-

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itoring of the flow velocity signal is probably su-
perior to the monitoring of the electrocardiographic “beeper” signal because it provides an indication of the quality of flow signal.

Limitations of the Technique

In our experience the most important and com-
mon problems have been: (1) position of the probe—change in contour of the flow velocity rec-
ord will occur as a result of displacement of the probe from the area over the blood vessel. How-
ever, if the area of the skin overlying the blood vessel is properly scanned and the area reflecting maximal audio signal is selected, then the audible signal and the analogue record are reproducible and stable on the same patient from day to day and on a group of patients with the same diseased state; (2) volume flow—the transcutaneous measurement of blood velocity does not provide a quantitative evaluation of volume flow. This is due to the fact that the diameter of the blood vessel under study is not known and, in addition, one does not know the exact angle that the incident ultrasound signal makes with the blood flow axis. Thus, the measurements obtained with this technique only reflect blood flow velocity in a given segment of the arterial circulation and its relative changes. However, the relative changes in flow velocity in the same patient or in a group of patients with the same disease state has proved to be useful, meaningful and valuable measurement; (3) central hemodynamics—measurement of peripheral arterial flow velocity is not a direct expression of stroke volume. The vessels accessible for this type of study are peripheral arteries which are under the influence of a number of neural, humoral and local factors which regulate the peripheral arterial flow distribution. It is concluded that this approach is useful to study the influence of pathologic states on the peripheral arterial flow velocity in man and as monitoring devices in intensive and coronary care units.

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REFERENCES


Reprint requests: Dr. Benchimol, 1033 East McDowell, Phoenix, 85006

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