Patterns of Brachial Arterial Blood Flow in Conscious Human Subjects with and without Cardiac Dysfunction

By James H. Gault, M.D., John Ross, Jr., M.D., and Dean T. Mason, M.D.

It has not been practical in the past to study phasic arterial blood flow patterns in the extremities of intact, unanesthetized human subjects. Although some measurements of phasic blood flow have been made in anesthetized patients undergoing major surgical procedures, and the velocity of aortic blood flow has been computed in human subjects using the pressure gradient technique, these studies have provided only limited information concerning the intact peripheral circulation. Recent improvements in the design of electromagnetic flowmeter circuits, and the miniaturization of the flow transducers, suggested the possibility that this instrument might prove suitable for such measurements. The present study was undertaken to investigate the characteristics of phasic and instantaneous mean brachial arterial (BA) blood flow, measured with the electromagnetic flowmeter (EMF), in the extremities of patients undergoing cardiac catheterization. Phasic BA flow patterns were recorded in subjects without significant left heart disease, as well as in patients having various cardiac lesions. In addition, the patterns of mean BA blood flow in the resting state and the instantaneous changes in the mean flow during local and reflexly determined alterations in forearm vascular resistance (FVR) were investigated.

Methods

Thirty-five patients, ranging in age from 16 to 54 years, were studied at the time of cardiac catheterization, prior to diagnostic retrograde arterial catheterization of the left ventricle. The patients were studied in the supine position, in the postabsorptive state following premedication with sodium pentobarbital, 100 mg, intramuscularly. On the basis of clinical and hemodynamic findings, five patients were considered to have functional heart murmurs; one patient was studied after complete surgical correction of tetralogy of Fallot. Of the remaining 29 patients, three had aortic stenosis, one had combined aortic and mitral stenosis, nine had aortic regurgitation, six had mitral stenosis, three had mitral insufficiency, and seven had idiopathic hypertrophic subaortic stenosis.

Using 1% xylocaine local anesthesia, the right brachial artery (BA) was exposed in the antecubital fossa (fig. 1). The vessel was isolated and the adventitia removed for a distance of approximately 1 cm. The flow transducer (flow probe) was then positioned around the vessel; the probe size was selected to produce slight constriction of the artery, thereby allowing optimal contact with the vessel wall. It has previously been shown that if the constriction does not exceed 10% of the vessel diameter, no significant alteration in mean flow or the flow contour results; moreover, during reactive hyperemia, comparable percentage increases in flow measured simultaneously by this method and by plethysmography suggested that the degree of constriction was insignificant. The miniature electromagnetic flow transducers employed required no further exposure of the brachial artery than that necessary for performing the retrograde arterial catheterization, and the procedure was associated with no apparent additional hazard.

Zero flow was obtained by gradual inflation of a sphygmomanometer cuff on the upper arm to a suprasystolic pressure level. This method proved more satisfactory than occlusion of the vessel immediately proximal and distal to the probe, since with the limited exposure of the vessel employed, motion of the probe occurred with the latter maneuver and produced an unstable zero signal. In addition, collapse of the artery did not occur with gradual inflation of the cuff, presumably because of venous engorgement. Zero reference with this method was stable, provided that contact of the probe with the vessel wall was satisfactory.

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slightly underestimated. Mean BA flow was measured with a time constant of 0.5 second. The flowmeter output was amplified with a DC amplifier and recorded on a multichannel photographic recorder.† Phasic flow tracings were recorded at a paper speed of 100 mm/sec, and mean flow was recorded at 5 or 10 mm/sec. The lightweight iron-core circumferential flow probes‡ had internal diameters ranging from 1.5 to 4.0 mm.

Each flow probe was calibrated by placing it on a segment of canine femoral artery stripped of adventitia and suspended in a saline bath between plastic cannulae. The flow rate of heparinized blood was varied by adjusting the height of a reservoir, distention of the blood vessel being maintained by elevation of the outflow tubing to 20 or 30 cm. Volume flow was determined by timed collection. The calibrations for each probe were repeated on several different days under identical conditions, and an example of the reproducibility of these calibrations for a single probe is shown in figure 2. The maximum variation in the output signals for any flow probe employed ranged from ± 4.4 to ± 8.1%.

†Electronics for Medicine, White Plains, New York.
‡Models BL-1015, BL-1020, BL-1025, BL-1030, BL-1035, and BL-1040.

A gated sine-wave electromagnetic flowmeter* was employed. In order to minimize background noise, a frequency response level of 20 cps was used in recording phasic flow tracings. This level of damping caused an 8% amplitude attenuation with an electrical sine-wave input of 5 cps superimposed on the meter carrier frequency.14 Since at normal heart rates the significant amplitude-contributory waves in the peripheral arterial flow tracing have a frequency of 5 cps or less,6 the peak systolic flow values reported are only

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*Model BL-310, Biotronex Laboratories, Silver Spring, Maryland.

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Figure 1

Exposure of the brachial artery and placement of the flow transducer. The incision, which is made at the level of the skin crease in the antecubital fossa, is shown in relation to the course of the brachial artery and its relations in the diagram on the left. (1). The lacertus fibrosus is exposed by blunt dissection; retraction of the lacertus will then usually serve to expose the underlying structures, including the brachial artery, its venae comites, and the median nerve. Sometimes it is necessary to make a small transverse incision at the radial margin of the lacertus to obtain adequate exposure of the artery. (2). The artery is then isolated, suspended between rubber tapes, and stripped of areolar tissue and adventitia. (3). The transducer is then positioned on the vessel, and the plastic insert or key (shown attached to the transducer in 2) is placed in the slot on the transducer. The ground wire is sutured to the subcutaneous tissue and the vessel returned to its bed.

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Figure 2

Calibration curves for one of the flow probes employed; the deflection of the flow signal in millimeters is plotted on the ordinate, measured flow on the abscissa. The symbols correspond with different dates on which calibration curves were determined. The points on each curve represent the average of at least two determinations at a given flow rate, and the insert shows the determinations for a single, representative calibration.

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Phasic and mean right BA blood flow patterns at rest and during interventions were recorded simultaneously with left BA pressure, measured continuously by means of an indwelling Cournand needle at the level of the antecubital fossa. Forearm vascular resistance was then computed as

\[
\text{mean left BA pressure, mm Hg} \\
\text{mean right BA flow, ml/min}
\]

Arterial pressure tracings were recorded using a Statham P23D pressure transducer, connected to the arterial needle with nylon tubing. The mean arterial pressure was measured with a time constant of 2.5 seconds. Phasic and mean BA blood flow were measured in the resting state in all patients, and in eight patients mean BA flow was determined before and after exclusion of the hand from the forearm circulation by inflation of a sphygmomanometer cuff at the wrist. Muscular exercise of the forearm consisted of clenching the fist forcefully in a sustained or rhythmic manner for periods of 20 to 75 seconds. For the Valsalva maneuver, the patient expired forcibly into a tube connected with a pressure manometer, expiratory pressure being maintained at 40 mm Hg for 20 seconds. Carotid occlusion was performed by manual compression of one or both carotid arteries at the base of the neck, and carotid sinus stimulation consisted of gentle massage of the right carotid bulb. Several sham carotid sinus manipulations were performed in each patient prior to the study of carotid sinus reflexes and consisted of manual compression of an area adjacent to the carotid bulb. The effects of local xylocaine infiltration and surgical exposure of the right brachial artery on resting blood flow and forearm vascular reflex connections were examined in six patients. In these studies, plethysmographic measurements of right forearm blood flow were performed before and during immersion of the left hand in ice water for 1 minute; this sequence was repeated both before and after the surgical exposure.

**Results**

The clinical diagnoses, the hemodynamic findings at cardiac catheterization, and the BA flow values in the resting state are summarized in table 1. The levels of peak and mean BA flow bore no obvious relation to the presence or absence of cardiac disease, to the cardiac output, or to the stroke volume.

**Phasic Brachial Arterial Blood Flow Patterns**

**Subjects Without Significant Left Ventricular Disease**

Phasic BA flow in the resting state was recorded in six patients (group I, table 1) who exhibited no left ventricular dysfunction at cardiac catheterization. Typical phasic BA flow contours in three of these patients are illustrated in figures 3, 4, and 5. Peak flow occurred within 0.10 second after the onset of systolic flow, the peak flow levels ranging from 1.39 to 6.41 ml/sec and averaging 3.64 ml/sec (table 1). Peak flow was followed by

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**Figure 3**

*Phasic brachial arterial flow patterns in a patient without left heart disease. Instantaneous B.A. flow, in ml/sec, is shown on the vertical axis. The arrow indicates the brachial arterial (B.A.) pressure pulse, adjacent to which are shown the systolic and diastolic arterial pressures. (Left panel). The hand is included in the forearm circulation. (Right panel). Same patient: the hand has been excluded by inflation of a wrist cuff.*

*Circulation, Volume XXXIV, November 1966*
### Table 1

**Cardiac Catheterization Findings and Data Obtained with the Electromagnetic Flowmeter**

<table>
<thead>
<tr>
<th>Group &amp; patients</th>
<th>Number</th>
<th>Diagnosis</th>
<th>HR</th>
<th>CI</th>
<th>BA pr. (mm Hg)</th>
<th>Fr. gradients (mm Hg)</th>
<th>Mean (ml/min)</th>
<th>Peak systolic (ml/sect)</th>
<th>End-diastolic (ml/sec)</th>
<th>HR*</th>
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<td>I.</td>
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†Denotes patients in atrial fibrillation; and † denotes patients in atrial flutter.

Abbreviations: MS = mitral stenosis; MI = mitral insufficiency; LV dis. = left ventricular disease; AS = aortic stenosis; AI = aortic insufficiency; IHSS = idiopathic hypertrophic subaortic stenosis; HR = heart rate at time of cardiac output determination; HR* = heart rate at time of brachial arterial flow study; BA pr. = brachial arterial pressure; S/D; LA-LV = left atrial-left ventricular pressure gradient at end diastole; and LV-BA = left ventricular-brachial arterial peak systolic pressure gradient.
BRACHIAL ARTERIAL BLOOD FLOW

Figure 4

Modifications in the phasic brachial arterial (B.A.) flow tracings in patients with cardiac disease. (A). Patient W.S. (control) had no left heart disease; patient J.C. had severe aortic stenosis. Abbreviations as in figure 3. (B). Tracings from patients with moderate and severe aortic regurgitation. (C). Tracings from patients with severe mitral stenosis and pure mitral regurgitation. The heart rate in patient A.F. was 98/min.

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Phasic brachial arterial flow tracings in patients with idiopathic hypertrophic subaortic stenosis (I.H.S.S.). Patient L.G. (control) was studied following complete correction of tetralogy of Fallot, and had no left heart dysfunction. Patient C.G., postoperative I.H.S.S. with mild residual left ventricular outflow tract obstruction, exhibits an early, rapid midsystolic flow decline with protracted late systolic backflow. Patient B.S. had I.H.S.S. with severe obstruction to left ventricular outflow; not the bifid contour of both flow and pressure pulses. Abbreviations as in figure 3.

Patients with Cardiac Disease

It has been observed previously in animals,17, 18 and in man,6 that the contour of the peripheral arterial flow pulse can be substantially altered by changes in the peripheral vascular resistance. As pointed out above, changes in the phasic flow contour were observed in the present study during inflation of a cuff at the wrist, emphasizing the potential effects of local factors on the characteristics of the phasic flow pulse. However, despite differences in FVR, specific alterations of the phasic flow contour were noted with certain cardiac lesions, particularly those affecting left ventricular ejection.

Aortic Stenosis

In three of the four patients with moderate to severe obstruction at the aortic valve (group II, table 1), a delayed rise in systolic flow rate was observed, peak flow being reached in from 0.12 to 0.26 sec. As shown in figure 4A, this delay in achieving peak systolic flow was more striking than the associated slow rise in the arterial pressure pulse. One patient, A.C.,
whose LV-BA pressure gradient was 59 mm Hg (table 1), exhibited a normal time-to-peak flow velocity (0.08 to 0.10 sec).

Aortic Regurgitation

Phasic BA flow contours were recorded in nine patients with predominant aortic regurgitation. In three of the patients, the regurgitation was mild to moderate, and in six it was severe (group II, table 1). Figure 4B shows typical BA flow tracings recorded under these two circumstances. With severe regurgitation, the dicrotic wave was absent, and a rapid decline in flow was observed throughout diastole; however, retrograde flow was noted in only two patients during late diastole. In three of the patients who exhibited no retrograde flow, it could be elicited by inflation of the wrist cuff.

Alterations have been observed previously in aortic and femoral arterial flow tracings of anesthetized patients undergoing cardiac operations. In patients with severe aortic regurgitation, marked retrograde flow always occurred; in the femoral artery, retrograde flow was greatest in early diastole and then decreased progressively. The findings in the present study indicate that in the presence of severe aortic regurgitation, retrograde flow is much less common in the brachial artery than in the femoral artery and that it is predominant during the opposite phase of diastole. Inflation of a cuff at the wrist induced retrograde BA flow in several patients with aortic regurgitation, and perhaps more experience with this maneuver will indicate whether or not the extent of this backflow can be correlated with the severity of valvular regurgitation.

Idiopathic Hypertrophic Subaortic Stenosis (IHSS)

Six of the seven patients with IHSS had intraventricular pressure gradients ranging from 21 to 135 mm Hg (group III, table 1); one exhibited no obstruction at rest, but developed a pressure gradient during provocative maneuvers. Two patients with small intraventricular pressure gradients (20 and 21 mm Hg) exhibited flow patterns indistinguishable from normal. In one patient with a moderate intraventricular pressure gradient (37 mm Hg), and in one patient (G.C., fig. 5), who exhibited no gradient at rest, a sharp early decline in systolic flow occurred while in those with large pressure gradients (75, 115, and 135 mm Hg), the systolic BA flow pulses were bifid (B.S., fig. 5, and V.S., fig. 6). This bifid systolic flow pulse in patients with IHSS and large intraventricular pressure gradients is presumed to be responsible for the percussion and tidal waves commonly observed in the peripheral arterial pressure pulses of these patients. It seems likely that this flow contour reflects rapid, early ejection of blood from the left ventricle followed by a markedly reduced ejection rate in late systole, findings documented in the ascending aorta by the computed pressure gradient technique and by means of electromagnetic flowmeter recordings from the aorta at operation. In the latter study, simultaneous measurements of aortic and left ventricular pressures indicated that the rapid, early deceleration of flow was coincident with the development of the intraventricular pressure gradient.

Phasic BA flow tracings were recorded during premature ventricular contractions in three patients with IHSS, all of whom exhibited an abnormal response in the BA pulse pressure in the beat following a premature beat. In contrast to the normal pattern (fig. 6, upper panel), in each of these three patients a diminution in the peak and total systolic BA flow accompanied the decline in arterial pulse pressure during the first postextrasystolic beat (fig. 6, lower panel).

Mitral Valve Disease

The phasic BA flow tracings recorded in six patients with moderate to severe mitral stenosis (group IV, table 1) were indistinguishable from the normal pattern (fig. 4C, patient J.G.). In three patients with severe mitral regurgitation (group IV, table 1), the rate of decline of flow in midsystole to late systole was somewhat more rapid than that observed in patients without left-heart disease (fig. 4C, patient A.F.). This finding is consistent with observations that such patients may not be able to sustain ventricular ejection
in late systole; however, it is also possible that the relatively rapid heart rates in these patients contributed to this alteration in the flow contour.

**Mean Brachial Arterial Blood Flow Patterns**

**Mean BA Flow in the Resting State**

The mean BA flow, measured in 31 patients at rest, varied widely, ranging from 22 to 185 ml/min and averaging 73.7 ml/min. There was no obvious relation between the level of mean flow and the cardiac index, and stroke volume, or the nature of the heart disease (table 1). In eight patients, exclusion of the hand from the forearm circulation by inflation of the wrist cuff resulted in decreases in mean BA flow that ranged from 33.3 to 72.7% and averaged 56.2%.

Three types of variation in the level of mean BA flow were observed in the resting state; in some patients each of these patterns was observed at different times during the study. The three patterns consisted of:

1. In 24 patients, periodic fluctuations in mean arterial pressure were accompanied by directionally similar variations in mean BA flow. This pattern, which was noted intermittently, occurred with a sinus arrhythmia in two patients (fig. 7A), but was not associated with changes in heart rate in the remaining 22 patients. The flow variations exhibited a period ranging from 10 to 20 seconds. These

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**Figure 6**

Phasic brachial arterial flow pulses recorded during ventricular extrasystoles. (Upper panel): Patient L.G. had no left heart dysfunction and exhibited an increase in brachial artery pulse pressure (BA) and in peak and total systolic flow in the first postextrasystolic beat. (Lower panel): Patient V.S. had idiopathic hypertrophic subaortic stenosis (I.H.S.S) with severe obstruction, and exhibited a decrease in both peak and total systolic flow and a decline in BA pulse pressure in the first extrasystolic beat.
cyclical variations in mean BA pressure presumably resulted from periodic alterations in the cardiac output, or from cyclical changes in the vascular resistance of areas other than the forearm.

2. In six patients, periodic variations in mean arterial pressure were associated with directionally opposite fluctuations in mean BA flow. This phenomenon occurred transiently after reflex alterations induced by carotid sinus manipulations and the Valsalva maneuver, and also spontaneously (fig. 7B). These flow alterations were not associated with a change in heart rate. The period of the variations ranged from 10 to 20 seconds. The occurrence of this pattern suggests that the forearm was participating in spontaneous, cyclical variations in the general systemic vascular resistance.

3. In four patients, large increases or decreases in mean BA flow occurred without apparent pattern (fig. 7C). In these patients, heart rate and arterial pressure remained constant during the random flow variations. These changes could have been neurally mediated through the cutaneous vascular bed responsible for body temperature regulation,26-28 or they could have resulted from alterations in the skeletal muscle or cutaneous vascular beds consequent to emotional stress.28-31 It has been reported that blood flow in the hand is particularly variable and may fluctuate by 20% or more several times a minute.32 Since inflation of the wrist cuff greatly reduced or eliminated spontaneous fluctuations in mean BA flow, it is likely that they were due largely to changes in the primarily cutaneous bed of the hand. Indeed, all of the three types of flow variation were reduced and sometimes eliminated by excluding the hand from the forearm circulation.

Effects of Plethysmographic Measurements on Mean BA Flow

Observations were made in six patients in whom forearm blood flow was recorded by means of a Whitney mercury gauge plethysmograph, using a technique described previously,15,16 and simultaneously by the EMF, the probe being positioned on the artery distal to the venous occluding cuff. From 5 to 8 seconds after inflation of the cuff to 30 mm Hg, a consistent decrease in mean BA flow occurred; this decline in mean BA flow was always accompanied by a change in slope of the plethysmographic tracing (fig. 8, panel A). When the hand was excluded from the forearm circulation, the change in slope of

Figure 7
the plethysmographic tracing and the accompanying decline in mean BA flow occurred much later and were small (fig. 8, panel B). Thus, the initial slope of the plethysmographic tracing, as has previously been reported, appears to reflect accurately the rate of BA inflow; moreover, with low rates of inflow it appears possible to make reliable plethysmographic measurements continuously for periods as long as 20 seconds.

**Mean BA Flow and FVR During Forearm Exercise**

Mean right BA flow was determined in eight patients during muscular exercise of the right forearm. During sustained muscular contractions, a marked decrease in flow was observed consistently, and in several patients complete cessation of flow occurred transiently (fig. 9A). During sustained contractions, lasting from 20 to 30 seconds, flow always remained below control levels. With release of
the contraction, maximum postcontraction hyperemia was achieved within 5 seconds, the flow increases ranging from 102 to 138% of control levels. With rhythmic contractions, reductions in BA flow also occurred during each contraction, as shown in high speed tracing reproduced in figure 9B; however, as the rhythmic exercise was continued, mean BA flow gradually increased above control levels. Following rhythmic forearm exercise of 50 to 60 seconds' duration, the maximum flow increase during postcontraction hyperemia ranged from 49 to 133% of control levels.

It has been reported that blood flow to the exercising human forearm or calf muscles can be reduced during the exercise period.44–48 However, the accuracy of plethysmographic estimations of arterial inflow during exercise is uncertain since the volume of the exercising extremity may be diminished by expulsion of venous blood past the occluding cuff.46 The present findings reveal that even brief, submaximal contractions of the forearm muscles cause striking mechanical interference with blood flow and that maximum forearm contractions produce actual cessation of arterial inflow; both of these effects presumably result from the high intramuscular pressure or from mechanical “nipping” of larger arteries.

Mean BA Blood Flow During Reflex Interventions

The possibility was considered that interference with basal blood flow and the reflex

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**Figure 9**

Effect of forearm muscular exercise on mean brachial arterial blood flow. Abbreviations as in figure 7. (A). During a forceful, sustained hand grip, mean BA flow initially decreases, then rises slowly, exceeding control levels only on release of the contraction. (B). During rhythmic hand grips, shown at a more rapid paper speed, mean BA flow declines abruptly with each contraction (arrows), rising progressively above the control level upon release of successive contractions.
connections to the forearm might result from the instillation of local anesthetic and surgical exposure of the brachial artery. Therefore, a determination was made of the effects of the surgical procedure on resting forearm blood flow measured by the plethysmograph. The integrity of the reflex connections was also tested by studying the effects on this flow of a cold pressor test, performed before and after the surgical procedure. In six patients, right forearm blood flow, measured plethysmographically, averaged 2.90 ml/100 g/min (range 0.83 to 5.65 ml/100 g/min) in the control state, while following exposure of the right BA the average value was 2.87 ml/100 g/min (range 1.16 to 5.18 ml/100 g/min). The response to immersion of the left hand in ice water, as indicated by the maximum increase in FVR, was somewhat less following exposure of the artery, the increases averaging 94% compared with 150% before exposure of the artery. However, the induced rise in systemic arterial pressure was less during the second cold pressor test, and the average absolute decrease in forearm blood flow was 46.1% before and 39.9% after exposure of the artery, suggesting that the reflex connections to the right forearm vascular bed had remained essentially intact.

**Carotid Sinus Reflexes**

Fourteen determinations of mean BA flow and FVR during unilateral or bilateral carotid arterial compression were made in five patients. In 11 studies, in which the hand was included in the forearm circulation, mean BA flow declined by 15.2 to 44.6% (average 31.6%), while FVR increased by 21.1 to 94.3% (average 53.4%) (fig. 10A). In each instance, a response in pressure and flow was apparent within 3 to 4 seconds after the onset of compression, and the peak responses occurred in 7.5 to 12.0 seconds. During these alterations, small increases in heart rate were also noted. Thus, marked reflex vasoconstriction in the forearm was induced rapidly and consistently by carotid arterial compression. Sham carotid arterial occlusions were performed in each patient by compression of the neck adjacent to the carotid artery and resulted in negligible changes in BA flow.

Manual stimulation of the right carotid sinus was performed eight times in four patients. Evidences of a reflex effect were apparent within 3 seconds, transient slowing of the heart rate (average 29.4%) being accompanied by a fall in mean BA pressure (average 12.1%). Initially, mean BA flow decreased and in six instances (fig. 10B) this decline appeared to be passive, that is, proportional to the fall in arterial pressure. In two instances, a disproportionately greater decline in BA flow occurred. Subsequently, while arterial pressure remained low, the BA flow increased in all eight studies (fig. 10B).

Because of the initial fall in systemic arterial pressure and the concomitant passive reduction of flow, the effects of carotid sinus stimulation are clearly more difficult to interpret than those following carotid arterial compression. For example, a reflex increase in flow to the skeletal muscle bed could have been masked by a larger passive decrease in flow through the hand. However, the secondary increase in flow which was observed, while arterial pressure remained low, strongly suggests that active vasodilatation accompanied carotid sinus stimulation. Whether or not this flow increase occurred in the skeletal muscle vascular bed remains uncertain, although it has been reported that no change in the vascular resistance of the hand occurs with carotid sinus manipulations.²⁹ The early onset of the secondary flow increase suggests that it was not due to autoregulation in the skeletal muscle bed.⁴⁰ These observations on the effects of carotid sinus stimulation and carotid arterial occlusion are consistent with a recent report indicating that the human forearm vascular bed participates in systemic baroreceptor reflexes.¹¹

**The Valsalva Maneuver**

Vasoconstriction, localized to the skeletal muscle bed, is known to occur in the forearm during the period immediately following a Valsalva maneuver.⁴² In the present study, it was possible to measure mean BA
Mean brachial arterial flow during reflexly induced changes in forearm vascular resistance. Abbreviations as in figure 7. (A). The effects of right (RT.) carotid arterial occlusion, maintained for 6 seconds. Mean right BA flow declines within 3 seconds; the concomitant increase in mean arterial pressure (mean L.B.A.) indicates that vasoconstriction has occurred. (B). The effects of right carotid sinus stimulation (C.S.S.). Slowing of the heart rate and a decrease in mean arterial pressure are apparent in 2 seconds; mean BA flow initially declines, then increases to exceed control levels. (C). Effects of the Valsalva maneuver, intrathoracic pressure being maintained at 40 mm Hg during the bracketed 20 second period (see text).

Discussion

In interpreting the various reflex interventions described above, it is recognized that the “post Valsalva overshoot” could be the residual effect from the marked vasoconstriction which occurs during the maneuver; in some patients flow actually fell to zero immediately upon release of the maneuver. In addition, it is possible that contraction of the forearm muscles during the Valsalva maneuver contributed directly to the fall in flow, since, as demonstrated above, BA flow is quite sensitive to the mechanical influence of muscle contraction.
complexity of the vascular bed served by the brachial artery influences greatly both the quantitative and directional changes in FVR. Thus, the extent of reflex changes in FVR must depend not only on the relative distribution of BA flow just prior to the intervention, but also on the magnitude of changes in flow through forearm beds such as the skin of the hand or forearm, which may not participate in the reflex, but respond passively to accompanying alterations in systemic arterial pressure. The distribution of BA flow when the hand is excluded from the circulation is not yet known. It is possible that under this condition the brachial artery supplies a relatively larger proportion of flow to skeletal muscles and less to the skin than when forearm blood flow is measured by the plethysmograph; this important problem will require clarification if the complex reflex effects described above are to be completely understood.

Summary

The patterns of phasic and instantaneous mean brachial arterial blood flow at rest, and during various interventions, were studied in 35 conscious human subjects. Flow was measured with an electromagnetic flowmeter applied to the brachial artery (BA) prior to retrograde arterial catheterization of the left ventricle.

The patterns of phasic BA flow in six patients without left-heart disease were compared with those of patients having a variety of cardiac lesions. In three patients with aortic stenosis, a prolonged upslope and a delayed systolic flow peak were noted, while in five patients with severe aortic regurgitation, a progressive decline in flow occurred throughout diastole; retrograde diastolic flow occurred infrequently at end diastole, but was elicited in three patients by inflation of a wrist cuff. Among seven patients with idiopathic hypertrophic subaortic stenosis, those with large pressure gradients across the left ventricular outflow tract exhibited a bifid systolic brachial arterial flow pulse and an abnormal response in brachial arterial flow during the beat following a premature ventricular contraction. In patients with mitral stenosis, the flow pulse was normal, while in three patients with mitral regurgitation, the period during which systolic flow occurred was abbreviated.

Mean brachial arterial blood flow averaged 72.7 ml/min, and neither peak nor mean flow levels bore a relation to the cardiac lesion, the stroke volume, or the cardiac index. All patients exhibited spontaneous variations in resting brachial arterial flow. Usually the fluctuations were in phase with periodic fluctuations in mean arterial pressure, although in some patients periodic fluctuations in arterial pressure and brachial arterial flow occurred in opposite directions, and in other patients random flow changes were observed.

Contractions of the forearm muscles were found to cause striking mechanical limitation of instantaneous brachial arterial flow. During maximal contraction of the forearm muscles, actual cessation of flow occurred.

Prior to investigating the influence of reflex interventions on brachial arterial flow, studies were performed to determine whether or not local anesthesia and surgical exposure of the brachial artery altered resting flow in forearm bed or the reflex connections to the forearm. Plethysmographic determinations of the resting forearm blood flow, and of the response to a cold pressor test, before and after exposure of the brachial artery, indicated that resting flow was unchanged and that reflex activity was essentially intact. Carotid arterial compression in five patients resulted in a prompt increase in forearm vascular resistance (average 53.4%). The response to manual stimulation of the carotid sinus in four patients suggested that reflex forearm vasodilatation was rapidly induced. In six patients studied during the course of a 20-second Valsalva maneuver, marked reflex vasoconstriction occurred approximately 10 seconds after the onset of the maneuver.

The present technique has permitted the first description of the normal pattern of phasic brachial arterial flow in unanesthetized human subjects, as well as the alterations in phasic flow contour accompanying various cardiac
lesions. In addition, it has allowed investigation of rapid, transient alterations in peripheral arterial blood flow not previously accessible to measurement.

References
of blood vessels in the resting hand and forearm to various stimuli. Amer Heart J 19: 541, 1940.


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On Style

Style, in its finest sense, is the last acquirement of the educated mind; it is also the most useful. . . Style is the ultimate morality of mind . . . with style the end is attained without side issues, without raising undesirable inflammations. With style you attain your end and nothing but your end. With style the effect of your activity is calculable, and foresight is the last gift of gods to men. With style your power is increased, for your mind is not distracted with irrelevancies, and you are more likely to attain your object. Now style is the exclusive privilege of the expert.—ALFRED NORTH WHITEHEAD: The Aims of Education and Other Essays. New York, Macmillan Co., 1959, p. 19.
Patterns of Brachial Arterial Blood Flow in Conscious Human Subjects with and without Cardiac Dysfunction
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