Journal of Physiology (1993), **460**, *pp.* 641–655
With 8 figures
Printed in Great Britain

FLUCTUATIONS IN BLOOD FLOW TO ACRAL SKIN IN HUMANS: CONNECTION WITH HEART RATE AND BLOOD PRESSURE VARIABILITY

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(*Received 10 February 1992*)

SUMMARY

1. Spontaneous fluctuations in blood flow in arteries supplying acral skin were investigated with Doppler ultrasound in human subjects. Finger blood pressure, heart rate (HR) and cardiac output were measured simultaneously and non-invasively.

2. Synchronous fluctuations in flow were found in arteries supplying the hands and feet. The fluctuations were larger and more rapid than the flow variations which have been demonstrated with other methods. The magnitude of the total flow fluctuations in the hands and feet was estimated to be 5–10% of cardiac output in resting subjects. This range of flow fluctuations is made possible by spontaneous opening and closing of skin arteriovenous anastomoses (AVAs).

3. The fluctuations in skin blood flow were accompanied by inverse fluctuations in mean blood pressure (MAP). The power spectra of skin vascular conductance and MAP both contained maximum intensity at low frequencies, below 0·15 Hz, with high coherence.

4. The central circulatory events connected with the skin blood flow fluctuations were calculated from the experimental data with the use of transfer function analysis. There was a rise in HR, cardiac output and MAP starting 1–4 s before a cutaneous vasoconstriction. This indicates that the HR and MAP responses are not only passive effects of changes in peripheral resistance, but are the result of a simultaneous activation of the peripheral vascular and cardiac efferent branches of the autonomic nervous system. The HR and MAP responses are then modified, probably by baroreceptor activation.

INTRODUCTION

A characteristic pattern of large, spontaneous fluctuations in blood flow to acral skin has been described in human subjects by several authors (Freeman, 1935; Burton, 1939; Abramson & Katzenstein, 1941; Coffman, 1972; Thoresen & Walløe, 1980). These fluctuations are most pronounced in a thermoneutral environment and are greatly reduced at high and low environmental temperatures. The characteristic flow pattern has been found only in the feet, hands, toes and

fingers, not in the leg, forearm, or in blood vessels supplying the skin of the trunk (Abramson & Ferris, 1939; Abramson & Katzenstein, 1941; Thoresen & Walløe, 1980). The blood flow fluctuations are abolished by sympathectomy and epidural anaesthesia, which confine the fluctuations to innervated vessels (Freeman, 1935; Janbu, 1989). Burton (1939) and Thoresen & Walløe (1980) assumed that the fluctuations were caused by synchronous opening and closing of skin arteriovenous anastomoses (AVAs). The AVAs in human skin are located in the skin of the fingers and toes, the nail beds, the palm of the hands and the sole of the feet and possibly in the earlobe (Grant & Bland, 1931; Prichard & Daniel, 1956). They are densely innervated with sympathetic vasoconstrictor fibres (Sherman, 1963; Böck, 1980).

The work of Coffman (1972) supports the assumption that the blood flow fluctuations in acral skin are caused by flow variations through skin AVAs. He measured nutritional blood flow in the fingertip using $Na^{131}I$ and total fingertip blood flow by venous occlusion plethsymography, and demonstrated large, rapid fluctuations in fingertip blood flow despite a stable nutritional flow. Nutritional blood flow in the finger was estimated to be only 10–20% of total finger blood flow, the rest passing through AVAs.

Burton (1939) noted characteristic heart rate and blood pressure changes occurring simultaneously with the blood flow fluctuations in the fingers and toes. A connection between the fluctuations in acral skin blood flow and spontaneous HR and blood pressure variability was also suggested by Kitney & Rompelman (1980), but the exact nature of this connection remains to be investigated.

In conscious sheep, the total proportion of cardiac output passing through hindleg skin AVAs, measured with the microsphere technique, increases from 1 to 10% on exposure to a warm environment (Hales, Fawcett, Bennet & Needham, 1978). In man, however, the proportion of cardiac output passing through skin AVAs is not known.

Thus, the aim of the present study was to use Doppler ultrasound to investigate in greater detail the magnitude of the blood flow fluctuations in arteries supplying skin with AVAs, and the degree of correlation between the fluctuations in individual arteries. In addition, the connection between these blood flow fluctuations and simultaneous spontaneous beat-by-beat fluctuations in HR and MAP has been investigated by transfer function analysis.

METHODS

Subjects

Eleven volunteers (4 males, 7 females) aged 20–51 years were investigated. They were all healthy without signs or symptoms of cardiovascular or neurological disease. The subjects were lightly clothed, supine, and the surrounding temperature was within the thermoneutral range (24–30 °C). They were allowed to stabilize for at least 20 min before the start of the recordings. All subjects gave their informed consent before the start of the experiments.

Procedures

Blood velocity measurements were made in the ulnar, radial, dorsalis pedis and tibialis posterior arteries using a stand-alone Doppler instrument and a colour-flow mapper (SD-100 and CFM-750, both instruments manufactured by Vingmed Sound, Horten, Norway). The Doppler unit is identical in both instruments. The instantaneous intensity-averaged mean velocity was calculated

by the instruments and fed to the same Apricot XI computer (ACT Computers, Birmingham, England), together with a three-lead ECG signal, for on-line collection of data. The mean velocity was integrated for each R–R interval.

The Doppler instruments were operated in pulsed mode at 10 MHz (SD 100) and 6 MHz (CFM-750). Different frequencies were used to avoid interference problems. The radial and ulnar arteries were investigated approximately 3 cm proximal to the wrist, the dorsalis pedis artery was

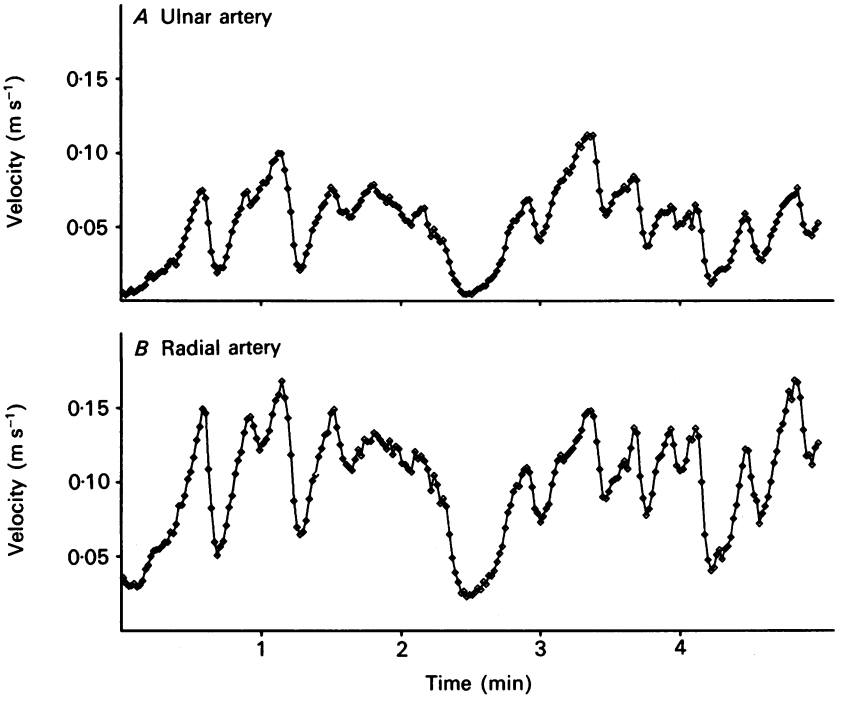

Fig. 1. Simultaneous 5 min samples of a 20 min velocity recording from the right ulnar (*A*) and radial (*B*) arteries. The points on the curves represent time-averaged velocity of individual cardiac cycles.

investigated on the dorsum of the foot approximately 3 cm distal to the ankle joint at the location where it is most easily palpable, and the tibialis posterior artery was investigated as it runs behind the medial malleolus. Two arteries were investigated simultaneously in each experiment. All arteries were investigated at sites where they are known to run parallel to the skin surface. The Doppler probes were hand-held and the angle of insonication was 45 deg. The measurements from the radial and dorsalis pedis arteries were used for further calculations.

Cardiac output was estimated using the method described by Eriksen & Walløe (1990). A 2 MHz transducer was held at the suprasternal notch, measuring blood velocity in the aorta just above the semilunar valves. The maximum velocity of the jet at the location of the sample volume was used as a measure of the velocity across the aortic ring, where the velocity profile is thought to be rectangular. The computer calculates cardiac output from the average systolic velocity of each R–R interval and the diameter of the aortic orifice, which was measured with 2-dimensional ultrasound imaging from a parasternal location.

Instantaneous blood pressure values were obtained with a non-invasive device which continuously measures the arterial blood pressure in a finger (Ohmeda 2300 FINAPRES, Madison, WI, USA). The instrument measures finger blood pressure according to the Peñáz principle. The FINAPRES has been compared with intra-arterial and Dinamap monitoring (Wesseling, Settels, Van der Hoeven, Nijboer, Butjin & Dorlas, 1985; Imholz, Van Montfrans, Settels, Van der Hoeven,

Karemaker & Wieling, 1988; Imholz, Settels, Van der Meiracker, Wesseling & Wieling, 1990; Parati, Casadei, Groppelli, Di Renzo & Mancia, 1989) and good correlation has been found. The instantaneous blood pressure data were fed to the computer and the MAP for each R–R interval was calculated by numerical integration and analysed together with the velocity and ECG signals.

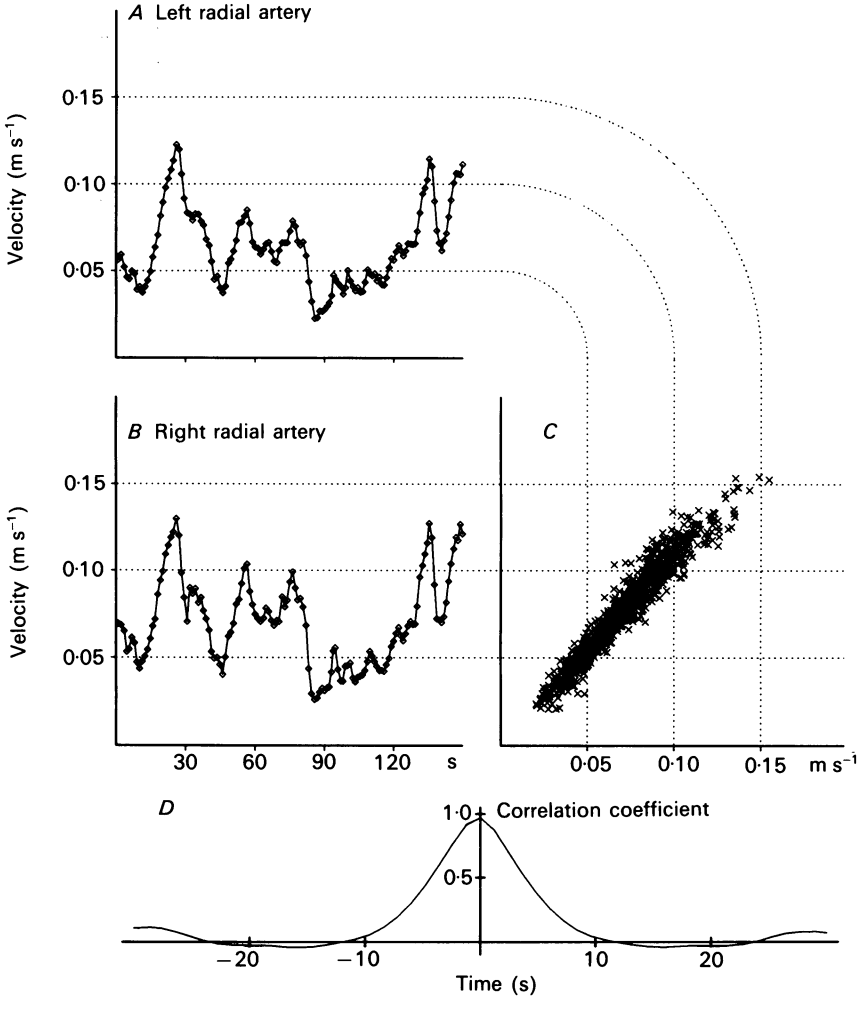

Fig. 2. Simultaneous 2·5 min samples of a velocity recording from the left (*A*) and right (*B*) radial artery and a scatterplot from the total 20 min recording (*C*). The *y*-axes of *A* and *B* are projected on to the *x*- and *y*-axis of the scatterplot. $r = 0{\cdot}935$ ($P < 0{\cdot}001$). *D* correlation between the velocity values as a function of the time lag (in s) between the individual measurements. The correlation is best when the lag is 0.

All data series were from 20 min uninterrupted recordings. The recording of blood pressure started at least 10 min before the start of the data collection. Because of the statistical analysis method chosen, the servo-adjust function of the FINAPRES device was turned off during data collection. Some of the blood pressure recordings were continued for 2 min after data collection with the servo-self adjust function turned on to verify that no changes in measured MAP could be attributed to the lack of the servo-self-adjust function.

To establish whether the velocity fluctuations could be explained by diameter variations, the

internal diameter of the radial artery and the blood velocity in the artery were measured simultaneously by the duplex scanner mode of a colour-flow mapper (CFM-750). The internal diameter of the artery was measured repeatedly at the same measurement site during the systolic pulse wave and in diastole, without removing the probe between the measurements.

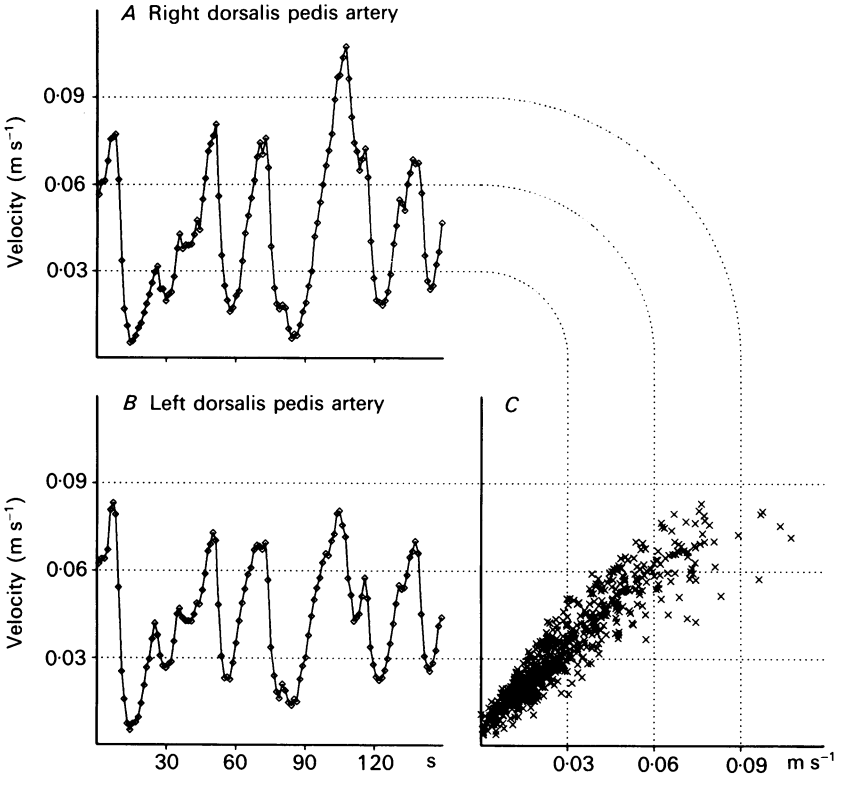

Fig. 3. Simultaneous 2·5 min samples of a velocity recording from the right (*A*) and left (*B*) dorsalis pedis artery. *C*, scatterplot of the total 15 min recording. $r = 0{\cdot}966$ ($P < 0{\cdot}001$).

Mathematical and statistical analysis

The fluctuations in velocity, HR and MAP were analysed using a statistical time series analysis programme (BMDP 1T). Before calculations, the signals were converted into equidistant time samples by interpolation. The power spectra were obtained by fast Fourier transform (FFT) and analysed using a band-width of 0·01 Hz. All signals were averaged and sampled on a beat-to-beat basis. Since the sampling process was carried out at approximately 1 Hz, frequency components in the underlying biological signals below 0·5 Hz are correctly reproduced, while frequencies above 0·5 Hz will give rise to unwanted frequency components due to aliasing. The power spectra of the signals contained very little intensity at frequencies above the respiratory frequency at approximately 0·25 Hz, making the presence of major signal components due to aliasing unlikely.

Using continuous 10–20 min recordings, conductance was calculated beat-by-beat as averaged radial artery velocity divided by averaged MAP. Periodograms of conductance and the dependent variables were calculated by fast Fourier transform (FFT) using the sampling process described above. The power spectrum of the conductance recording and the cross-spectra of the dependent variables *versus* conductance were all smoothed with a bandwidth of 0·01 Hz. The frequency domain transfer functions were found by dividing the individual cross-spectra with the conductance power spectrum, and the time-domain transfer functions (impulse responses) were then found by

inverse FFT. The calculations were performed as described by Berger, Saul & Cohen (1989) and Challis & Kitney (1991). The impulse responses were scaled and integrated with respect to time, yielding the step responses of heart rate and MAP to an instantaneous drop in conductance from the average value of the whole recording to zero, and of infinite duration.

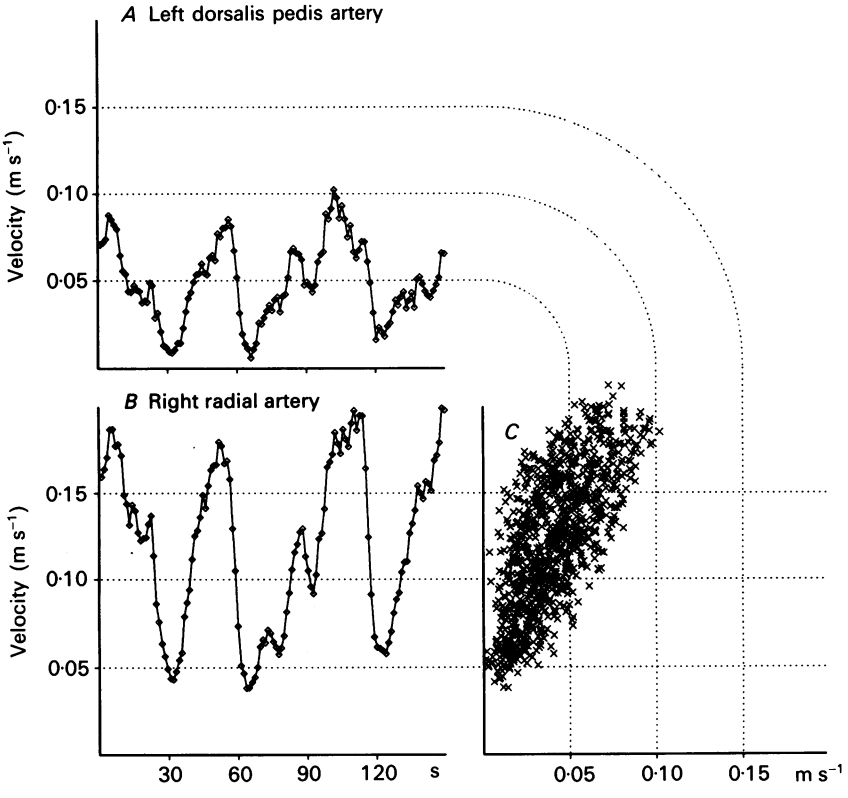

Fig. 4. Simultaneous 2·5 min samples of a velocity recording from the left dorsalis pedis artery (*A*) and right radial artery (*B*). *C*, scatterplot of the total 20 min recording. $r = 0{\cdot}754$ ($P < 0{\cdot}001$).

RESULTS

Velocity measurements

Spontaneous fluctuations in velocity were found in all the arteries investigated. The amplitude of the fluctuations was large, the maximum velocity was generally 5–10 times the minimum velocity. Figure 1 shows a representative 5 min segment of a simultaneous recording from the right radial and ulnar artery, demonstrating the characteristic large velocity fluctuations. The similarity between the velocity fluctuations in the two arteries indicates synchronous flow fluctuations through the AVAs in the hand.

When simultaneously obtained velocity recordings were compared, correlation between corresponding velocity values in the two arteries was high in all experiments (Table 2). Two-and-a-half minute representative sections of a recording from the right and left radial arteries are displayed in Fig. 2*A–B*. Figure 2*C* shows a scatterplot of the corresponding beat-by-beat averaged velocities in the two arteries

TABLE 1. Blood velocity and internal diameter in the radial artery measured simultaneously with a combined 2-dimensional and Doppler ultrasound probe during diastole

Subject	Velocity (m s^{-1})	Internal diameter (cm)
1	0·03	0·25
	0·15	0·25
2	0·04	0·27
	0·15	0·26
3	0·01	0·22
	0·11	0·22
4	0·03	0·28
	0·14	0·28

The velocity values fluctuated widely without corresponding changes in vessel diameter.

TABLE 2. Correlation between velocity fluctuations in the radial and dorsalis pedis arteries and between conductance in the radial artery vascular bed and MAP

	No. of expts	No. of subjects	Correlation coefficient		Lag y (s)
			Median	Range	
Two radial arteries	7	6	0·92	0·87–0·97	0
Two dorsalis pedis arteries	7	6	0·89	0·80–0·94	0
Radial (x) and dorsalis pedis (y) artery	13	7	0·73	0·64–0·87	−3 to 0
Conductance (x) and MAP (y)	12	6	−0·60	−0·78 to −0·34	1–4

The median value and range of the correlation coefficient are given together with the range of the time lag (s) between the independent variable (x) and the dependent variable (y) which gave optimal correlation in the individual recordings. In four of the thirteen recordings, velocity fluctuations in the radial artery preceded fluctuations in the dorsalis pedis artery by 2–3 s. The MAP fluctuations preceded the conductance fluctuations by 1–4 s in all recordings.

from the total 20 min recording. The correlation between the velocities measured in the two vessels is highly significant ($P < 0·001$). Figure 2*D* shows the correlation coefficient as a function of the time lag between the individual velocity values from the two arteries. The highest correlation was found when the lag was zero, indicating synchronous fluctuations in velocity.

Measurements from the right and left dorsalis pedis artery, and from one radial and one dorsalis pedis artery, are similarly presented in Figs 3 and 4. All the recordings from the arteries investigated showed large velocity fluctuations and high correlation between the corresponding velocity values in each pair of recordings. In four of the recordings from one radial and one dorsalis pedis artery, the cross-correlation studies indicated that the velocity fluctuations in the radial artery preceded the fluctuations in the dorsalis pedis artery by 2–3 s. In the remaining nine recordings from one hand and one foot, the cross-correlation studies showed that the fluctuations were synchronous.

Diameter measurements

Simultaneous internal diameter and Doppler velocity measurements were made in the radial artery of four subjects (Table 1). All subjects showed the typical

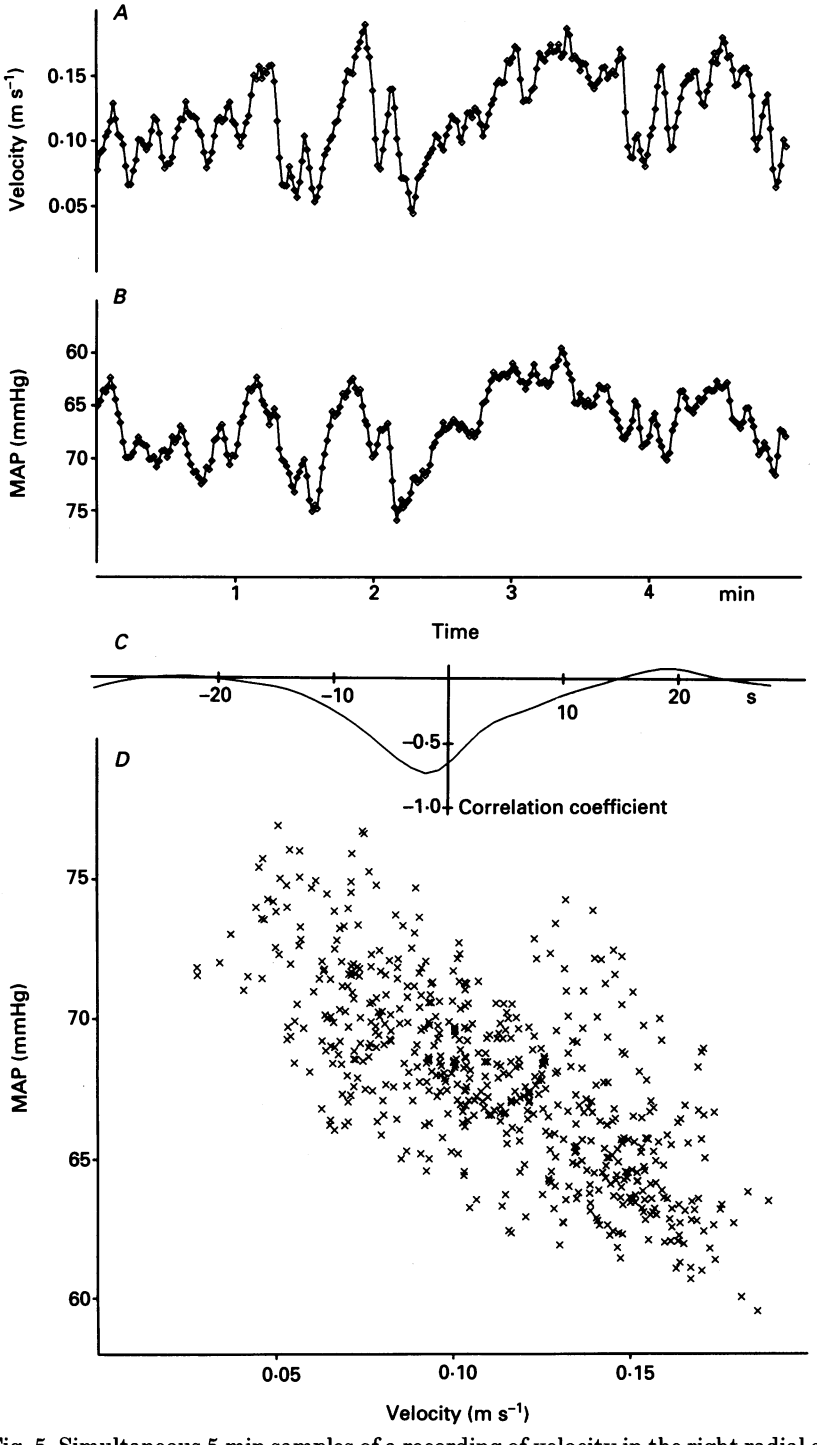

Fig. 5. Simultaneous 5 min samples of a recording of velocity in the right radial artery (*A*) and MAP (*B*) measured in the left third finger. The MAP axis is inverted. *D*, scatterplot of the relationship between velocity in the radial artery and MAP. The plot is based on

fluctuating velocity pattern during the measurements. The variation in internal diameter from systole to diastole was less than 0·02 cm. There were no long term diameter variations that could be attributed to the large velocity fluctuations observed.

Blood pressure, heart rate and cardiac output

In eight subjects, simultaneous recordings were made of MAP in the left third finger and blood velocity in the right radial artery. A negative correlation between corresponding velocity and MAP values was found in all experiments (Table 2). Figure 5*A* shows a representative 5 min sample of such a recording. For ease of interpretation, the vertical scale of the MAP tracing is inverted. There are large fluctuations in MAP with a peak-to-trough magnitude of 15 mmHg. The median value of the beat-by-beat MAP fluctuations in all twelve experiments was found after removal of trends and longer term variations by high-pass filtering the signal with a cut-off frequency of 0·005 Hz. The amplitude was then calculated as the difference between the 0·1 fractile and the 0·9 fractile of the sample value distributions of this filtered signal. The median value of the amplitude of the MAP fluctuations was 8·2 mmHg (range 6·6–11·2).

The scatterplot in Fig. 5*D* shows the data from 10 min of the recording in Fig. 5*A*. The plot is made with the MAP values lagged 2 s in relation to the corresponding velocity values, as this was shown to give the largest negative correlation (Fig. 5*C*).

Power spectral analysis of conductance and MAP fluctuations in all experiments revealed maximum intensity in the frequency range 0·001–0·15 Hz, with steadily declining intensity towards higher frequencies (Fig. 6*A* and *B*). The coherence between the signals was high in this frequency range (Fig. 6*C*). The MAP power spectrum in the figure shows a prominent mid-frequency or Mayer wave at 0·1 Hz.

The real-time beat-by-beat recordings of conductance in the radial artery vascular bed, HR and MAP indicated a connection between conductance fluctuations and blood pressure and heart rate variability (Fig. 7*A–C*). The figure shows 30 s simultaneous real-time beat-by-beat recordings of conductance in the radial artery vascular bed, HR and MAP. HR and MAP started to rise 2–3 s before the start of the vasoconstriction. The increase in HR was followed by a decrease and a subsequent return to near the original rate, while conductance was still low. MAP also decreased in parallel with the decrease in HR, and gradually returned to its previous value.

The results of transfer function analysis of the relation between HR, MAP and conductance confirmed the observations from the real-time recordings. Figure 7*D* is based on the same experiment as Fig. 7*A–C*, but the calculations are made with the use of data from the whole 20 min recording. The figure shows the calculated response in HR and MAP to a step in conductance from the average value of the whole recording to zero. Approximately 5 s before a steplike drop in conductance, HR rises, there is a decline in HR at about the time of the vasoconstriction, and a

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a 10 min recording. The MAP values are lagged 2 s with respect to the corresponding velocity values, as this was shown to give the best correlation between the signals (*C*). $r = 0{\cdot}706$ ($P < 0{\cdot}001$).

subsequent return to near the control values during the next 5–10 s. MAP rises in parallel with the rise in HR, declines slightly, but stays elevated while conductance is zero. The predictive value of the result of the transfer function analysis was tested by feeding the real-time conductance recording in Fig. 7*A* through a digital filter

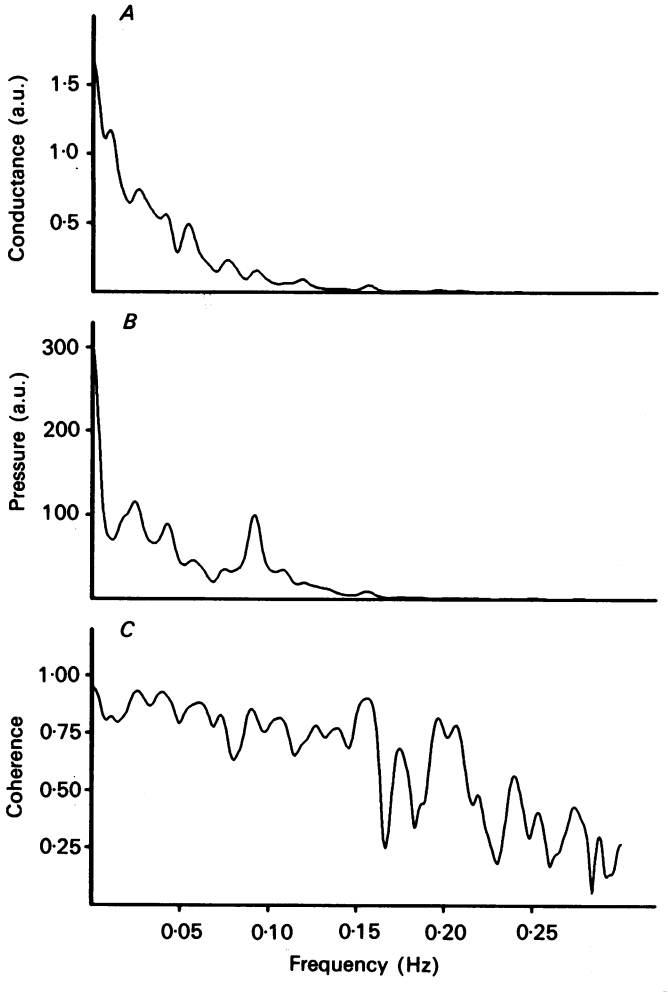

Fig. 6. Power spectra of beat-by-beat fluctuations in skin vascular conductance (*A*) and MAP (*B*) from one of the experiments. Both spectra show maximal intensity at frequencies below 0·15 Hz with high coherence (*C*). Note the prominent mid-frequency wave at 0·1 Hz in the MAP spectrum. a.u., arbitrary units.

with a step response for MAP as shown in Fig. 7*F*. The result of this is indicated by the dotted lines in Fig. 7*F*.

In four subjects, cardiac output and velocity in the radial artery were measured simultaneously. Figure 8 shows the calculated average response in cardiac output to a step in skin vascular conductance similar to that in Fig. 7*D*, based on the four individual recordings. The time relation and direction of the cardiac output changes

was similar in all subjects, but the magnitude of the response differed somewhat. The variations in cardiac output associated with a steplike drop in conductance closely parallel the changes in HR. Two to three seconds before the conductance dropped, there was an increase in cardiac output of nearly 1 l min^{-1}, followed by a sharp

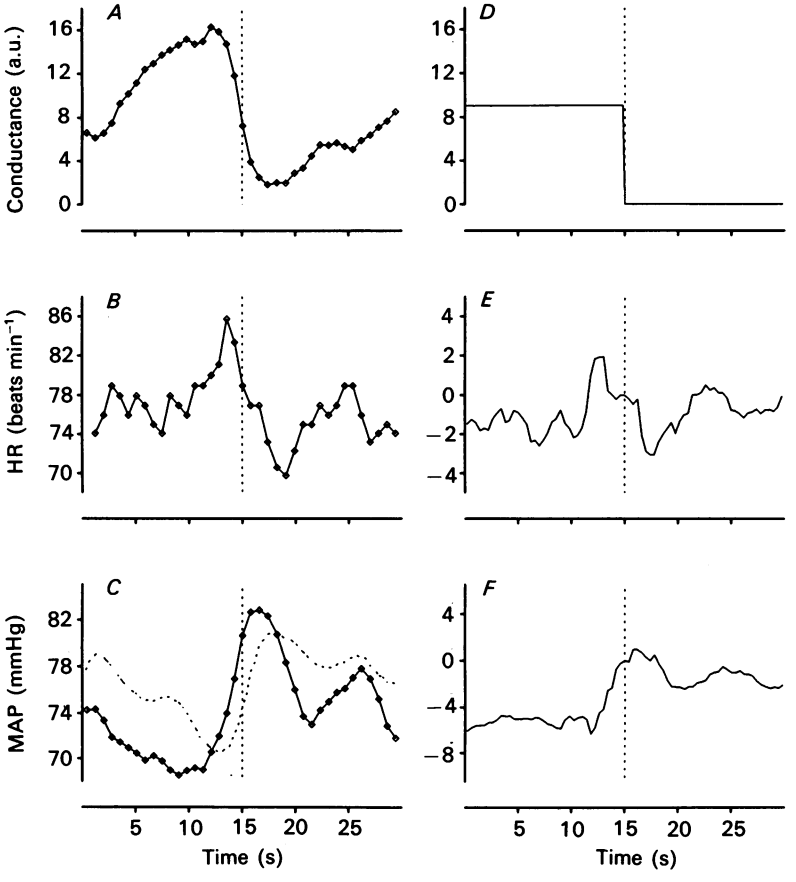

Fig. 7. A simultaneous real-time beat-by-beat recording of peripheral vascular conductance (*A*), HR (*B*) and MAP (*C*). The conductance values are derived from beat-by-beat velocity and MAP values. The plot consists of 30 s of a 20 min recording. The vertical dotted line marks the mid-point of a spontaneous vasoconstriction. *D–F*, the time-domain representation of a transfer function analysis of MAP and HR with respect to skin vascular conductance. The data are taken from the total 20 min continuous recording, which is sampled in real time in *A–C*. The response to a step in conductance from the long term average value to zero (*D*) has been calculated. *E* shows the HR response, and *F* the MAP response. A prediction of the real-time MAP response, based on the calculated step response (*F*) and the actual conductance recording (*A*) is given as a dotted curve in the lower left-hand panel.

decline of approximately 1·5 l min^{-1} and successively a return to near previous values.

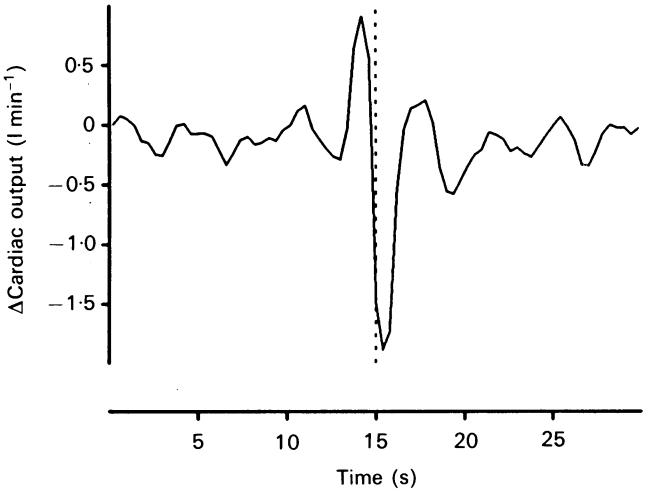

Fig. 8. The time-domain representation of a transfer function analysis of cardiac output with respect to skin vascular conductance using the same procedure as in Fig. 7. The figure shows the average of the results in four subjects.

DISCUSSION

The study confirms the occurrence of large, spontaneous, synchronous blood flow fluctuations in acral skin, and the magnitude and location of the fluctuations supports the assumption that they are caused by rhythmic synchronous opening and closing of skin AVAs. If one assumes that the hands and feet are each supplied by two arteries with an internal diameter of about 2 mm, the sum of the flow fluctuations in the vessels supplying these skin areas is of the order of 250–500 ml min^{-1}, corresponding to 5–10 % of the cardiac output of resting subjects.

Experimental technique

As long as the diameter of the vessel is constant and the total cross-section is illuminated, blood velocity as measured by our method is proportional to blood flow. The diameter changes in the radial artery showed only very small variations related to the pulse wave, which were of the same order of magnitude as the resolution of the ultrasound instrument. No diameter variations were detected corresponding to the long term velocity fluctuations observed, and it is therefore a valid assumption that the large velocity fluctuations represent proportionally large variations in flow. Fluctuations in blood flow to the fingers and toes were also described by Burton (1939) and other authors using venous occlusion plethysmography, but this method has the disadvantage of a poor time resolution. During a rapid vasoconstriction, the time from maximum to minimum velocity in our study was frequently found to be only four or five heart beats, which is below the time resolution of the

plethysmograph. The plethysmograph is also unable to measure very high or very low flow values (Abramson, 1967). The amplitude of the large flow variations may therefore have been underestimated and rapid flow changes have remained undetected with this method.

We used the FINAPRES device for measuring blood pressure. The measured variations in finger blood pressure are not necessarily representative of changes taking place more centrally in the circulatory system. Moreover, the mean and diastolic blood pressures in the finger arteries are somewhat lower than in the upper arm, due to flow resistance in the arm, and this pressure drop is less during vasoconstriction when flow in the hand is low (Wesseling *et al.* 1985). However, the fluctuations in finger MAP in our experiments were too large to be accounted for by the passive effect of vasoconstriction or vasodilatation in the peripheral vessels. Indeed, Imholz *et al.* (1988, 1990) showed that the FINAPRES faithfully reproduced beat-by-beat intra-arterial blood pressure variations in the brachial artery during a control period, during a Vasalva manoeuvre which is known to cause large variations in peripheral blood flow, and during orthostatic stress. The correlation was best for MAP, and slightly less close for the systolic and diastolic values.

Synchrony of the blood flow fluctuations

The close correlation between the fluctuations in skin blood flow in the hands and feet indicated synchronous activation of the sympathetic efferent nerves supplying skin vessels in different parts of the body. Synchronous spontaneous bursts of activity in sympathetic nerves to skin in the hands and feet have been detected by microneurography (Bini, Hagbarth, Hynninen & Wallin, 1980). In our studies, the correlation was slightly better when results from one pair of radial or dorsalis pedis arteries were compared. The correlation between velocity fluctuations in the hand and foot was slightly lower, but still highly significant. In four of thirteen experiments, the cross-correlation studies showed maximum correlation when the velocity values in the radial artery were lagged 2–3 s in relation to the corresponding velocity values in the dorsalis pedis artery. Bini *et al.* (1980) found a time lag of about 0·3 s between the bursts led off from the median and peroneal nerves during spontaneous neural activity and after electrical skin stimulation. The time lag, which is due to the conduction velocity in the efferent sympathetic nerves, is too small to influence our estimated correlation coefficient and cannot explain the observed time lag in our experiments.

Connection with heart rate and blood pressure variability

Our study demonstrated large spontaneous fluctuations in MAP with a median peak-to-trough difference of 8·2 mmHg and with a significant negative correlation to velocity fluctuations in the radial artery. The power spectra of skin vascular conductance and MAP were highly coherent at frequencies below 0·15 Hz. This includes the mid-frequency peak or Mayer wave in the MAP spectrum around 0·1 Hz, which has been related to the frequency response of the baroreceptor reflex (Hyndman, Kitney & Sayers, 1971). Thus, our findings suggest a causal relationship between skin vascular conductance and MAP fluctuations, both at the Mayer wave frequency and for lower frequencies.

Microneurographic investigations of human skin nerves (Hagbarth, Hallin, Hongell, Torebjørk & Wallin, 1972) have failed to demonstrate any correlation between spontaneous activity in sympathetic nerves and variations in blood pressure. However, these studies involved nerves containing a mixture of vasoconstrictor and sudomotor fibres, and the authors did not differentiate between skin areas with and without AVAs.

The presence of slow, rhythmic variations in peripheral resistance and HR, connected with temperature regulation, has been described by other authors (Hyndman *et al.* 1971; Kitney & Rompelman, 1980). These authors explain the relationship in terms of a non-linear, oscillating control system. In our opinion, the relationship between acral skin vascular conductance, MAP and HR can be described as a linear relationship.

The real-time recordings and the time-domain representation of the transfer function analysis indicate synchronous rhythmic autonomic nervous action on the sino-atrial node and the skin AVAs, resulting in increased HR, constriction of the AVAs and an increase in MAP. In terms of the transfer function relationship, the autonomic rhythm may be considered as the true independent variable, which causes both the changes in skin vascular conductance, MAP and HR. This rhythmic activity of the skin AVAs may play a role in the long term regulation of body temperature.

The time lag between the HR and AVA responses was too large to be accounted for just by the conduction velocity in the sympathetic nerves. However, it could be related to the fact that the AVAs in the skin are innervated by sympathetic nerve fibres, with no evidence of a parasympathetic innervation, whereas parasympathetic nerve activity strongly influences heart rate in resting humans; cardiac responses to parasympathetic stimulation begin after a shorter latency (< 500 ms) than the response to sympathetic stimulation (1–3 s), and this is due in part to different mechanisms for release, action, and removal of transmitters at the nerve terminals (Salata & Zipes, 1991). It seems likely that the rise in MAP caused by the increased HR and decreased skin vascular conductance elicits a baroreflex response, which in turn reduces HR, in many instances to below the control value (Fig. 7).

The spontaneous fluctuations in blood flow through acral skin in a thermoneutral environment are thus shown to have a significant impact on the blood pressure and heart rate variability in human subjects.

We would like to thank Jarlis Wesche, Ola Bergslien and Karin Toska for valuable help with the recordings. The work was supported by the Norwegian Council on Cardiovascular Diseases.

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