THE PERIPHERAL PULSE WAVE: INFORMATION OVERLOOKED

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ABSTRACT. Although the waveform derived from a peripheral pulse monitor or pulse oximeter may resemble an arterial *pressure* waveform, it is in fact a visualization of blood volume change in transilluminated tissue caused by passage of blood: an indication of perfusion or blood *flow*. Most currently available pulse oximeters indicate this flow, but few display it in usable form. Since adequate tissue blood flow is a prerequisite for normal metabolic activity, it is a parameter that should merit a place in standard anesthesia or intensive care monitors. That the peripheral tissue blood flow is not routinely displayed may be in part due to the difficulty in quantifying data obtained: flow is not accurately measured as simply as pressure, even by invasive means. It is in the pattern of the waveform that beat-to-beat changes in stroke volume can be better seen than measured, or in the interaction of ventilation and circulation that tests general circulatory performance. The origin and interpretation of these changes are discussed and illustrated with examples. We indicate how new physiological tests of autonomic function and cardiac preload can be developed using pulse plethysmography. The importance and application of the Valsalva effect on the waveform is emphasized. This effect is particularly applicable for monitoring adequate fluid loading and the action of vasodilator drugs, which are both important in anesthesia. Differences between the arterial pulse pressure wave and tissue flow wave are discussed, as well as the cause of certain artifacts, including the wandering dicrotic notch.

KEY WORDS. Pulse oximetry, peripheral pulse wave, arterial pulse wave, hypovolemia, Valsalva maneuver, dicrotic notch, plethysmography.

INTRODUCTION

Pulse oximetry brought a major advance to patient monitoring in the 1980s, yet some of the most valuable data in the waveform signal are not used. By focusing on oxygen saturation, attention has been distracted from the pattern of the peripheral pulse wave, which provides a noninvasive window on several dynamic circulatory parameters. Indifferent displays of early pulse oximeters or the use of bargraphs also obscured any detail in the waveform. Although the pulse oximeter may indicate changes in perfusion for the tissues being illuminated, its waveform mimics an intraarterial pressure wave, despite its almost complete damping at the tissue level [1]. The essential difference is that the peripheral pulse monitor reads local tissue blood volume change in transilluminated tissue (caused by blood flow), which is an indirect indicator of upstream pressure only. As flow cannot be quantified as exactly as pressure, changes in the pattern of the waveform, rather than its dimensions, become of

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prime importance. We propose that such changes often provide more immediately relevant information on the state of the circulation during surgery than the ECG can. This discussion will deal briefly with the generation of wave patterns, their relation to function, and how and why patterns can change. Our goal is to stimulate discussion and inquiry into several issues that merit further clinical investigation. It is our belief that full interpretation of the data available from the peripheral pulse could lead to its becoming the most important single monitor for anesthesia, surpassing the ECG.

THE NATURE OF THE PERIPHERAL PULSE WAVE

Viewing the wave

The peripheral pulse wave is a mechanical event closely following an ECG complex, reflecting the interplay between left ventricular output and the capacitance of the vascular tree. An acceptable oxygen saturation and rate synchronization with the ECG confirm that the waveform is of a valid oximeter waveform [2]. The pulse oximeter waveform is derived from the infrared signal, which is influenced mainly but not exclusively by arterial blood: venous blood absorbs less light from the infrared spectrum.

As normally displayed (Figure 1), tissue distention by blood inflow during systole appears as an upgoing deflection, with return to baseline as blood runs off in diastole. In our clinical experience, the steepness of the slope of the inflow phase (dv/dt_{max}) may be used as an indicator of force of ventricular contraction (dp/dt) and the amplitude of the wave as an indicator of stroke volume (Figure 2).

In the descending runoff phase of arterial pressure and peripheral pulse wave is a dicrotic notch or incisura (Figure 1), classically attributed to closure of the aortic valve at the end of ventricular systole. Whether this interpretation is applicable in the peripheral waveform has been questioned (see below). For many years, we have used the vertical position of the incisura on the wave as an indicator of vasomotor tone: under most circumstances the notch descends to the baseline during increasing vasodilation and climbs towards the apex with vasoconstriction. The usefulness of this signal depends on comparison of successive traces.

The end diastolic baseline is stable, provided venous pressure is reasonably constant, when it indicates the basal minimum tissue perfusion. Raising and lowering the hand to which a pulse monitor is attached changes the baseline and with it the local venous, arterial, and their associated tissue perfusion pressures. The baseline is displaced upwards when the hand is below the heart, downwards when above the head (Figure 3).

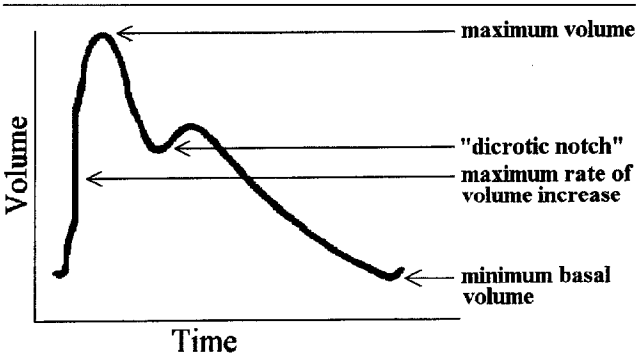

Fig 1. The normal pulse oximeter wave form. The anatomy of a standard plethysmographic wave, starting with the positive steep volume inflow due to ventricular systole, followed by the decline from peak light absorption after the maximum volume change has passed. Blood flow continues into the vascular bed during the latter phase when outflow exceeds inflow with a decreasing volume in the vascular bed.

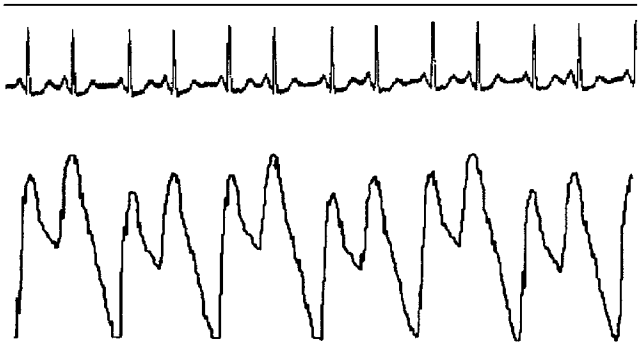

Fig 2. Pulsus bigeminus. The upper ECG tracing shows a bigeminy rhythm with a premature atrial contraction (PAC), a rhythm that may be difficult to identify on a monitor screen. The smaller wave caused by decreased preload from the premature beat is obvious on the oximeter trace. Since this wave has started from a raised baseline its peak is slightly higher.

Fluctuations in local venous pressure are continuous and significant: minor baseline variations always accompany ventilation. Some monitors so rapidly stabilize the baseline in order to confine the screen trace within its display channel that this element of the visible signal is degraded. Monitors often incorporate gain controls that slowly reset the wave size to fill the screen display, so making invalid anything more than short-term comparison of trace amplitudes. Nevertheless, the magnitude and duration of amplitude changes may possibly be developed as physiological tests of circulatory dynamics.

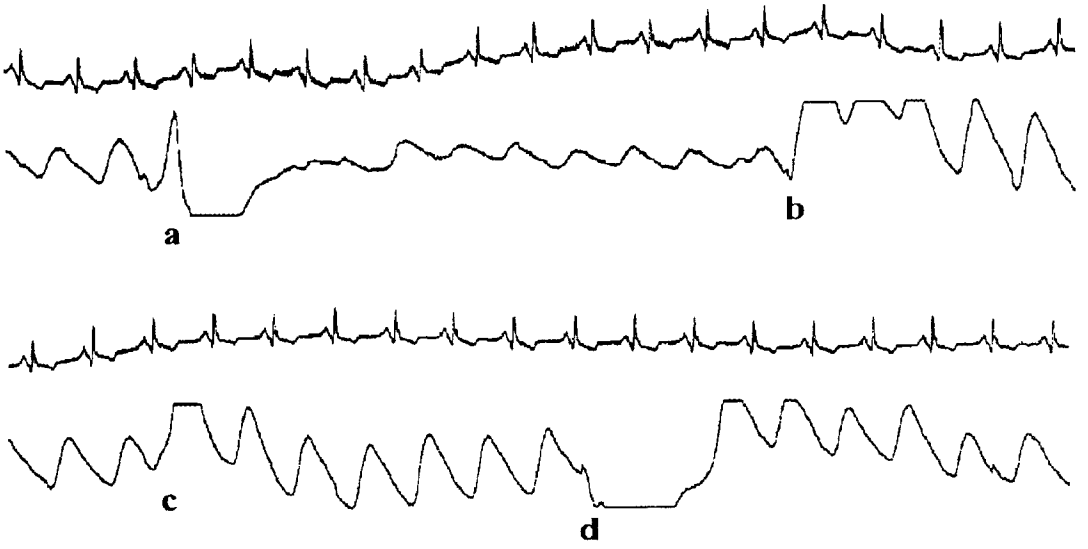

Fig 3. Constant position required for stable baseline. Changes in amplitude result from changing the balance between inflow produced by stroke volume, vascular capacitance, and outflow as modulated by peripheral vascular resistance. This can be mimicked by changing the transducer position relative to the right atrium, thereby altering local tissue venous hydrostatic pressure and resetting the wave form baseline. The maneuvers illustrate the normal waveform in an erect subject with transducer on a finger. Upper panel: (a) raising hand from heart level to above the head – local venous pressure falls, trace dips; (b) brought to heart level; note increase in amplitude due to residual vasodilation (remaining from period when hand was elevated). Lower panel: (c) hand lowered to knee height from heart level – venous pressure rises, trace displaced upwards (net inflow); note increase in amplitude; (d) raised to heart level. Note that the slow return to baseline of traces 2 and 4 seen in the middle of the screen is a monitor artifact that returns the trace to the baseline of its channel.

Origin of the typical waveform pattern

The runoff of an aliquot of blood (stroke volume) discharged into an elastic container (arterial tree) depends *inter alia* on the latter's compliance, on the pressure gradient, and on the adequacy of filling. Pronounced elasticity dampens peak pressures, prolongs the positive pressure phase, and thus extends perfusion time (runoff). The ideal waveform would show a broad peripheral flow wave with a large area under the curve, a short, steep inflow, followed by a 3 to 4 times longer outflow phase. Reduced filling or compensating vasoconstriction change the waveform to one with a narrow base, a brief, peaked plateau, and rapid runoff (Figure 4). In the periphery, where peripheral pulse monitors are usually placed, such a change tends to appear early since it is venomotor tone that responds first to fluid loss, limiting tissue perfusion gradients.

The peripheral pulse wave is generated by blood ejected from the heart during the opening of the aortic valve. The wave's form is shaped by this pressure interacting with complex elastic and reflective elements in the vascular tree. There are two components in the initial pressure: an initial pressure pulse so brief that flow is minimal, which travels at least at 15 times the velocity of the succeeding synchronous flow and pressure wave [1]. The faster propagation of the pressure pulse, outstripping its following arterial flow wave, leads to an increasing delay between the two towards the periphery. At tissue level both produce a pressure change: the brief pressure pulse produces no significant flow but may be

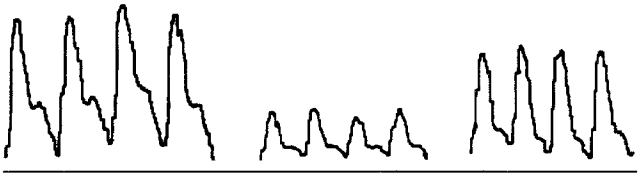

Fig 4. Changes in peripheral vascular tone during anesthesia. Examples of changes in amplitude commonly observed during routine anesthesia, the result of a balance between inflow driven by stroke volume, vascular capacitance, and outflow as modulated by peripheral resistance. These traces are from a single patient recorded over the short period between loss of consciousness with induction and the first surgical stimulus, applied before adequate depth had been achieved, as signaled by slight movement. A bolus of thiopental deepened anesthesia and restored peripheral pulse amplitude. Left panel – immediately after induction, showing the sudden vasodilation with loss of consciousness; center panel – skin incision in light anesthesia evokes rapid sympathetically mediated vasoconstriction with rise in blood pressure from a noxious input; right panel – deepening anesthesia with a thiopental bolus partially reverses vasoconstriction, lowers systolic blood pressure.

seen as conditioning the elastic vessel to accept imminent flow during the following extended pressure rise. This concept is discussed by Mostert and den Dunnen [3], who proposed it as one mechanism for inadequate peripheral circulation in advanced atherosclerosis: narrow arteries will impede a flow wave more than a pressure pulse. They tested this idea by restoring the usual phase difference between pulse and flow waves. In selected patients with advanced peripheral vascular disease, a boot plethysmograph was applied to the calf and foot of one leg (the other acting as control). Negative pulses, triggered by the ECG, were created just ahead of the flow wave and could produce dramatic pinking up of a long ischemic limb or a shock-like state following reperfusion of ischemic tissue. Their work merits further study. A fuller discussion of wave reflectance and variations in disease states is to be found in Nichols and O'Rourke [4]. In an electrical analogy the pulse wave is compared to an alternating current flowing through the parallel resistance and capacitance of peripheral conducting vessels. This type of simple AC filter produces a phase shift between voltage (the pressure wave) and current (the flow wave). Application of external negative pressure affects primarily the impedance. Changes in peripheral circulatory impedance alter sites of wave reflectance.

The respiratory interaction

Changes in intrathoracic pressure during the respiratory cycle also displace the baseline and change the amplitude of the peripheral pulse wave train, primarily by modulating central venous pressure.

During normal *spontaneous breathing*, an 8- to 10-second peripheral pulse trace shows a slow phasic respiratory wave (Figure 5) that shapes the envelope of the cardiac pulsations. Subatmospheric pressure during spontaneous inspiration draws air and blood together into the lungs: blood is drawn from the vena cava into the right heart and then on into the expanding pulmonary vascular bed. A minor decrease in peripheral venous pressure ensues. Simultaneously, left ventricular output briefly decreases for one or two heart beats as blood accumulates in the pulmonary circuit – the origin of the classic *pulsus paradoxus*. Thereafter, expiratory pressure improves flow to the left heart, increasing stroke volume, peripheral pulse flow, pulse amplitude, and peripheral venous pressure. Although the intrathoracic pressure changes are minor, the effect is consistently seen in a peripheral pulse trace.

The same effect is more prominent during *positive pressure ventilation* except that inspiration is now driven by positive pressure that raises peripheral venous pressure

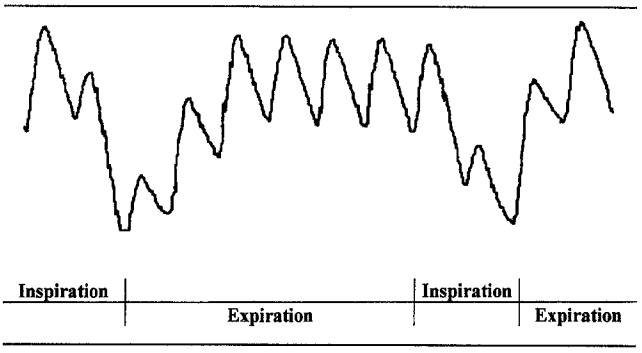

Fig 5. Spontaneous respiration (slow rate). Spontaneous respiration which has been slowed by the use of narcotics: expiration and inspiration are as indicated. Note the immediate decrease in amplitude and baseline drop at the start of inspiration (in contrast to positive pressure ventilation – see text). Note: the indicator of inspiration/expiration has been shifted to eliminate any wave transmission delay.

by damming back venous return to the right heart, and so tends to drive the peripheral pulse baseline upwards. Simultaneously, and very briefly, blood forced from the low-pressure pulmonary vascular bed increases return to the left heart [5] and stroke volume. This is quickly followed by a decrease in cardiac output as venous return into the central circulation drops off. Peripheral arterial and venous constrictor responses to compensate for decreasing cardiac output are triggered by low pressure receptors in the great veins and heart. The extent of the peripheral pulse baseline fluctuation depends on the state of filling of the peripheral vascular bed, the intrathoracic pressure, peripheral vasoconstrictor activity, and on the central blood volume that is driven to the left heart to augment its output. This whole cycle, repeated every few seconds with each breath, is easily followed on the peripheral pulse wave.

General anesthesia often involves intermittent positive pressure ventilation (IPPV), with its respiratory imprint on the peripheral pulse wave. In fact, the vasodilatation and damped vasomotor response, often a feature of general anesthesia, will accentuate fluctuations that follow intrathoracic pressure changes. The peripheral pulse waveform, thus, can be a sensitive indicator of changing circulatory dynamics during surgery. For instance, in our clinical experience, we have found that early hypovolemia may be reflected in an exaggerated respiratory wave before other more "classic" signs of decreased urine flow, tachycardia, or hypotension appear. The changes in the respiratory wave of the peripheral pulse must be read in phase with intrathoracic pressures: spontaneous inspiration lowers central venous pressure and increases venous return; positive pressure inspiration increases venous pressure and reduces cardiac output. IPPV postpones maximum venous inflow to late expiration, in effect, a

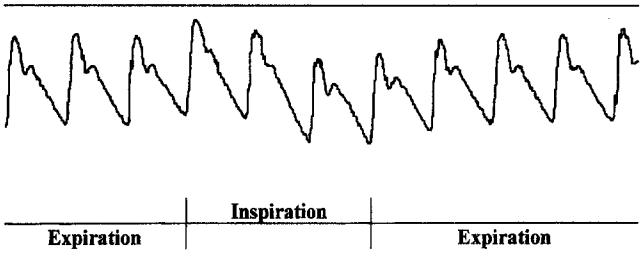

Fig 6. The respiratory wave during positive pressure ventilation. The pulse oximeter trace shows the influence of positive pressure ventilation. Ventilation was interrupted and the pulse oximeter wave form allowed to stabilize. A single ventilator breath was administered. Note the increased amplitude of the wave form at the start of inspiration (see text for explanation) followed by a decrease in the amplitude and baseline with subsequent recovery.

miniature Valsalva maneuver. However, the stroke volume of the first two to three heartbeats at the start of IPPV inspiration may increase (Figure 6) as the pressure in the great veins increases. The change in baseline and amplitude is opposite to that seen during spontaneous breathing. Four factors contribute to the brief, increased left ventricular stroke volume and pulse oximeter amplitude:

- increased preload as blood is forced from lungs to the left heart;
- direct physical compression of the left heart by the lungs;
- decreased afterload of the left ventricle [5];
- decreased right ventricular volume with septal shift to the right augments left ventricular filling by increasing the left ventricular compliance.

Stroke volume is boosted briefly by the extracardiac positive intrathoracic pressure – the pressure unloading of the left ventricle – until reduced filling of the left heart cuts back its output (Figure 6). This may not be seen in the failing heart, where the reduced venous return may restore ventricular dimension to a more favorable plot on the Starling curve [6].

Respiratory fluctuations of the peripheral pulse wave, therefore, reflect a balance between two pressures in a closed chest cavity: those driving venous return and those producing ventilation. High airway pressures, along with low venous return pressures, create the largest respiratory fluctuations. Thus, by establishing constant ventilator settings during anesthesia, optimal conditions for interpretation of fluid loading from the respiratory wave are produced. The effect of controlled ventilation on the *arterial pressure wave* during blood loss has been described by Perel [7], but no formal studies of the *pulse oximeter "flow" waveform* during hemorrhage are yet available.

TRANSDUCING THE PERIPHERAL SIGNAL

The peripheral pulse signal is pressure-dependent, but is essentially generated by a change in tissue perfusion or blood inflow. Thus, detecting the signal is best done by optical means that follow changes in tissue perfusion rather than transduce pressure. Perfusion pressures of peripheral tissue flow are small, highly damped [1], and thus easily distorted by mechanical devices.

Types of peripheral pulse sensors

The original sensor for peripheral blood flow was a plethysmograph that measured volume changes in a whole digit, *using volume change per unit time as the flow signal*, and thus operated as a pressure sensor of a closed volume. While these original sensors gave accurate measurement, they were bulky, subject to movement artifact, and therefore, seldom used as clinical monitors. The tension of the attachment is important for consistent accuracy since the transducer must neither be displaced by the pressure change it senses, nor dampen the underlying capillary flow. Such sensors are unsuitable for pulse oximetry.

Various other sensor types have been developed that either detect local tissue pressure fluctuations – microphone capsules often serve as sensitive transducers for the purpose – or changes in tissue transmission or reflectance of various wavelengths of visible or infrared light. For a more in-depth discussion, see Wukitsch et al. [8] or Severinghaus and Kelleher [2].

The present standard design, essential for pulse oximetry, uses the transmission or reflectance of red and infrared light to detect changes in both peripheral tissue volume and blood oxygen carriage, and, by reasonable inference, changes in blood flow responsible for a volume change. An advantage of such a sensor is that it can be applied to skin without retaining pressure, generally to either a finger or an earlobe. This is not a pressure sensor, but an inflow/outflow or perfusion sensor. The distinction needs emphasis since peripheral flow need not necessarily follow driving pressure. A low pressure into a low impedance vascular bed will give a high flow signal. Contrast this with a high pressure applied to a vasoconstricted vascular bed, which a pressure sensor could interpret differently from a flow sensor. A knowledge of the type of sensor in use is thus essential for interpretation of the peripheral waveform. This discussion will deal mainly with patterns derived from optical flow sensors.

Positioning the sensor

As noted previously, the peripheral pulse wave is a complex derived from the ejection of stroke volume into the aorta. The two components are an initial pressure "shock" wave rapidly transmitted to the periphery, followed by a slower flow wave. The different transmission speeds of the two waves result in an increasing phase shift towards the periphery of the vascular tree and thus may change the nature of the transduced signal. The pressure wave may in fact reach the periphery before the flow wave and be reflected back to interact with the flow signal picked up by a sensor [1, 9, 10]. If the time interval between the two is appropriate, the pressure wave promotes flow by influencing the impedance, making these phase shifts significant [11]. In a single subject, differences are likely between peripheral pulse signals from the earlobe [12], the finger, and the toe [13]. When comparing changing waveform patterns, choose a single site for the transducer. Again, note that present peripheral pulse monitors do not offer an absolute measurement of flow: interpretation of changing waveform patterns is used in other forms of clinical monitoring and is emphasized here.

INTERPRETING CHANGES IN THE PULSE WAVE

Amplitude

As already indicated, the waveform provides visualization of local tissue perfusion that results from the interaction of left ventricular stroke volume and peripheral vascular resistance. An increase in this resistance (for instance, the IV injection of an α-agonist) will result in a decreasing peripheral flow, and a concomitant reduction of the elastance of the arterial tree may dramatically reduce the peripheral pulse amplitude (Figure 4). The goal of peripheral vasoconstriction is primarily to redirect flow to priority areas to which a flow sensor is unlikely to be coupled. Delayed refill of the nailbed after compression has long been used as a reliable clinical sign of peripheral vasoconstriction; a flow detector such as the oximeter can be expected to function similarly. This is a field for further investigation. Marked beat-to-beat variations in amplitude result from changes in stroke volume; longer-term changes more often signal changes in autonomic tone or fluid loading, provided automatic gain controls in the monitor are inoperative.

Significance of increased amplitude (Figure 4)

A large amplitude wave in the underlying bed indicates local vasodilation by a direct vascular action such as histamine release or decreased vasomotor tone as might result from deep anesthesia, ganglion block, or increased body temperature. Provided some minimum pressure is met to maintain flow and vessel patency, arterial pressure changes need not correlate closely with the volume of passage because compliance can vary. A large pulse wave may indicate an increased tissue demand, a restoration of normal blood volume, or a paralysis of regulation. In very general terms a full peripheral pulse (large amplitude waveform) is a valuable indicator of adequate whole body perfusion.

Significance of decreased amplitude

Compression of the artery supplying the sensor site, as might occur under a sphygmomanometer cuff, will reduce pulse volume amplitude, as will hypovolemia, which causes blood flow to be redirected to essential organs.

Specific circumstances during anesthesia include: vasoconstriction upon lightening of anesthesia, the response to body core cooling, the slow depletion of blood volume by fluid loss into the third space, hemodilution, or poor blood loss control. We believe an increase or decrease in peripheral pulse amplitude should be regarded as a sensitive rather than a specific indicator of circulatory change. The clinical value depends on correlation with other signs: with suspected fluid depletion – this would come from an exaggerated respiratory wave, decreased urine output, slow peripheral capillary refill, and, later, hypotension. In general, at a given low blood pressure, a large pulse amplitude (indicating vasodilation and increased perfusion) is probably more desirable than a low amplitude pulse, implying vasoconstriction and decreased perfusion.

Premature cardiac beats are necessarily associated with a decreased stroke volume and are reflected as such in the peripheral pulse following limited diastolic filling of the ventricle (Figure 2).

Area under the curve

In each peripheral pulse wave, the area under the curve (AUC) (Figure 7) is an indicator of the volume of blood in the tissue scanned by the transducer. This will be influenced by the stroke volume, modified by changes in local vasomotor tone. The wave has three chief divisions:

1. A (ventricular) systolic phase corresponding to the ascending limb as the stroke volume first fills the

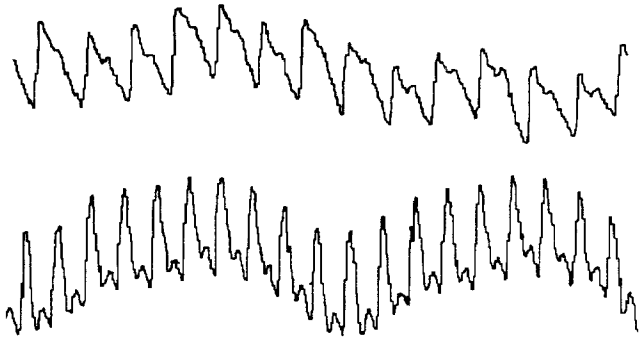

Fig 7. Area under the curve. The area under the curve is an indication of and proportional to the stroke volume. The upper panel shows a higher stroke volume indicated by a larger area under the curve compared to the lower panel. Both traces are from the same patient. The lower panel shows four signs of vasodilation: 1. larger amplitude; 2. smaller area under the curve, due in part to the tachycardia; 3. lower dicrotic notch; 4. larger baseline fluctuation due to ventilation – this could also be an indication of low preload (low CVP) or high inspiratory (ventilator) pressure.

arterial tree and then peripheral tissues. It is normally steep and brief.

2. A combined diastolic flow-through and runoff phase as blood stored during systole in elastance vessels continues to flow forward as the pressure plateaus and falls. The capacitance of these vessels, influenced by vasomotor control, and the ability of the blood volume to fill them, will make this phase the most variable of the three, usually several times longer than phase 1. Diastolic flow is the first affected by tachycardia and blood loss.
3. A baseline, often brief, during which blood inflow will be matched by blood outflow in the tissue area illuminated by the light path of the oximeter.

With optimum systolic filling, the runoff phase will be prolonged as perfusion pressure drops more slowly. The dicrotic notch normally remains midway in the descending limb. The waveform is broad-based and rounded (Figure 8).

With a degree of vasodilation such that filling is sub-optimal, the broad-based rounded waveform may either be preserved or become peaked, and the dicrotic notch moves towards the baseline. Figure 9 was recorded from a patient gradually vasodilating as the level of volatile anesthesia was deepened, accompanied by marked venodilation and acceleration of capillary refill.

Hypovolemia and subsequent vasoconstriction will produce a narrow-based, low-amplitude, peaked waveform with a rising dicrotic notch: filling of the arterial bed is incomplete and, thus, runoff is faster. The height of the wave may vary according to the placement of the transducer. The more peripheral the site – for example, a finger rather than an earlobe – the greater the likelihood of vasoconstriction both from volume loss and heat loss.

Fig 8. Optimal filling. This pulse oximeter trace shows optimal filling indicated by large amplitudes and areas under the curve as well as a clear dicrotic notch placed approximately in the middle of the descending limb. The 3 panels are from the same patient with deepening levels of anesthesia from left to right, indicated by the beginning downward displacement of the dicrotic notch.

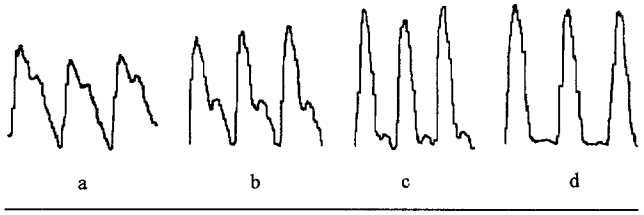

Fig 9. The position of the dicrotic notch. Reading from left to right the dicrotic notch position follows stages of increasing vasodilation due to deepening levels of isoflurane anesthesia. (a) A notch high on the outflow curve indicates vasoconstriction – a tight fit of blood volume to intravascular volume whereby vasoconstriction has reduced the compliance of the arterial tree; (b) A mid position on the outflow curve is normal; (c) A position low on the outflow curve is a feature of vasodilation that has increased compliance. A notch at this position does not indicate aortic valve closure but pressure wave reflectance (see text). (d) Virtual disappearance of the dicrotic notch and the appearance of a true basal perfusion rate. The dicrotic wave may be hidden in the following upstroke. The traces are all from the same patient. Heart rate and amplitude have increased with the vasodilation as could be expected from a lowered peripheral resistance.

The dicrotic notch (Figure 9)

As previously noted, the position of the incisura or dicrotic notch on the descending section of the pressure or pulse trace can be used as an indicator of vasomotor tone. Reduction in vasomotor tone is characteristic of the induction of most general anesthetics. As vasodilation develops, peripheral veins dilate, the pulse signal increases, and the notch descends towards the diastolic pressure baseline. We have also found this to be a useful sign of effective dosing of sodium nitroprusside, nitroglycerin, or ganglion blocker during stable anesthesia: these drugs may produce no further decrease in blood

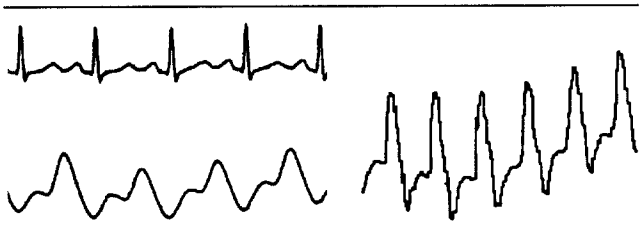

Fig 10. Systolic ascent of a dicrotic notch. The panels show 2 different printouts from different monitors and from different patients. The dicrotic notches are seen to encroach upon the ascending (systolic) limb of the following wave form. We believe this is not an artifact but an indication of extreme vasodilation.

pressure if the incisura is on the baseline, only a delayed recovery.

However, when the dicrotic notch moves further from the diastolic pressure base into the systolic section of the next wave (Figure 10), some explanation other than aortic valve closure is needed. Distance from the root of the aorta is pertinent [1, 4]. As an intraarterial cardiac catheter is slowly withdrawn from the aortic valve to the periphery, the distinct early diastolic incisura of aortic valve closure is slowly replaced by another dicrotic wave element later in diastole [14, 15]. The nature of this other wave is not clear, but could be related to reflection back from the periphery of the more rapid pressure wave [9]. Reflection of the pressure wave is an explanation of a higher peripheral arterial than aortic root pressure: flow and pressure waves summate at the transducer site [1, 10]. The Soliton theory of wave propagation may be applicable here, but is beyond the ambit of this paper [16].

From clinical observation, a dicrotic notch delayed to the ascending or systolic portion of the following pulse wave appears more likely with near maximal vasodilation, a raised cardiac output, and tachycardia. If the pressure wave is reflected at the interface of conductance and resistance vessels, vasodilation would delay its return from the periphery. This may explain why a shortened diastolic period and vasodilation could shift the dicrotic notch to the next early systole.

The double dicrotic notch

A double dicrotic notch can sometimes be observed in both arterial pressure and tissue flow waves (Figure 11). There is controversy over whether, in catheter-manometer pressure monitoring systems, this is not an artifact of inappropriate system resonances to one of the signals [17]. In pulse oximeters, peripheral pulse transducer system resonance is not an issue, frequency response may be higher, so that what is measured is a true reflection of

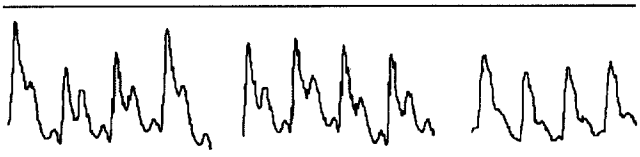

Fig 11. Double dicrotic notch. The panels are all from the same patient. The first two show double dicrotic notches influenced by ventilation. The third panel shows vasodilation with the second notch fusing with the next wave form (see text for explanation).

volume change. While the explanation is obscure, there is the documented link [9, 18] with a hyperdynamic circulation. It may be that during vasodilation with a higher than normal dp/dt, strong pressure waves are reflected back via short (brachial) and long (femoral) artery pathways to reach a finger transducer at different times. However, Shelley [19] considers these double waves to be venous pressure waves originating as a, c, or v waves in the right heart. Alternatively, pulsation transmitted from a hyperdynamic artery to its venae comitans might change the tissue perfusion pressure gradient to cause a brief dicrotic flow rise and fall.

Valsalva's maneuver – testing circulatory competence

Valsalva's maneuver [20] as a means of testing respiratory and circulatory interaction is simply performed by forced expiration after full inspiration against a closed glottis or against a measured resistance with a leak, such as a wind instrument or a column of mercury offering a resistance of between 20 and 40 mmHg. A 15- to 30-second expiration time is usual, but may be impossible for those patients with compromised respiratory or circulatory systems. During this brief period, the circulatory responses are mediated by mechanical factors and the evoked sympathetic response. Changes in vagal tone do not appear to be involved, but there is evidence of an interplay between vagal and sympathetic tone [21]. The pattern of change and recovery of central venous pressure, arterial pressure, and heart rate have long been used as a dynamic test of circulation [6]. The reader will gain a clearer understanding of the maneuver by performing it while attached to a pulse oximeter and noting the extreme distortion of the wave. In the present context, the relevance of this maneuver is as an illustration of the effects that IPPV might have in patients in anesthesia and intensive care units. It can assist in the interpretation of peripheral pulse changes. IPPV, PEEP, expulsive efforts during labor [22], and cough transients [6] are variations that extend the concepts of Valsalva's 18th century treatise.

Three response patterns to the Valsalva maneuver are

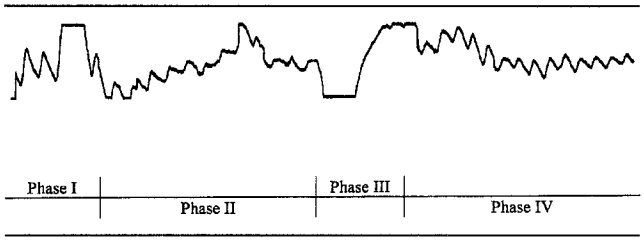

Fig 12. A Valsalva maneuver. Recording from a spontaneously breathing erect subject during a period of forced expiration against a closed glottis. (a) Phase I: start of raised intrathoracic pressure with rise of venous pressure masking any increased stroke volume. (b) Phase II: monitor readjusts baseline and shows falling output with diminished pulsations (stroke volume) which slowly recover. (c) Phase III: sudden release of intrathoracic pressure results in fall in local venous pressure, and displacement downwards of trace. (d) Phase IV: followed by pressure overshoot with increasing pulsations, and return to normal pulsations and level baseline

described that depend on the fillingz of the vascular bed, the capacity for vasomotor response, and the integrity of the pump. The circulatory variable may be *normovolemia*, *hypovolemia*, or *hypervolemia/cardiac failure*. Four phases are classically distinguished during the individual maneuver [23]. Responses are most accurately followed by rapid response, extrathoracic, intraarterial pressure monitoring: the peripheral pulse is a good noninvasive alternative (Figure 12).

Normovolemia

Normovolemia is the standard response with a normal blood volume, intact vasomotor responses, and a healthy heart. Phase I: a transient (± 2 second or 2 to 3 heartbeats) blood pressure rise with bradycardia [24–27] as intrathoracic pressure is increased, of amplitude proportional to the increase in intrathoracic pressure (the four reasons are described above). Phase II: a decreasing output and blood pressure as the intrathoracic pressure decreases venous return and throttles flow through the intrathoracic space to the left heart. There is a rise in pulmonary vascular resistance that is not sympathetically mediated and the net pressure within the right atrium and the directly linked great thoracic veins is little affected [27]. However, left heart stroke volume, output, mean arterial pressure, and pulse pressure fall; heart rate and systemic vascular resistance rise. Reflex venoconstriction tends to restore venous return and reflex arterial constriction tends to maintain the blood pressure, usually at a lower level. Phase III: with sudden airway pressure release blood pressure drops briefly and heart rate increases by a mechanism opposite to that in Phase I. This is rapidly followed in Phase IV, upon the return to ambient airway pressure, by a sudden surge of systemic and pulmonary return to both the right and left heart. This increased return produces a pressure and flow overshoot with an associated baroceptor reflex-induced decrease in heart rate. Stabilization of heart rate and blood pressure may take longer than the maneuver itself. Many of these changes are easily followed with a Swan-Ganz catheter during right heart monitoring with IPPV or PEEP. Phase I and III are responses to sudden mechanical change; Phases II and IV are the consequent evoked reactive autonomic responses mediated by high pressure baroceptors in the carotid sinus and ascending aortic arch, possibly in synchrony with low pressure receptors in the pulmonary circuit and atria. These baroceptors are all mechanoreceptors that sense transmural pressure. Only the carotid sinus is outside the pressurized area so that it may be mainly receptors within the thorax, sensing a pressure drop (decreased transmural pressure), that are chiefly responsible for the autonomic response. This concept appears not to have been fully investigated. The major autonomic effects are seen as changes in pulse rate and also as variations in systemic vascular resistance, which are both reflected in the pulse waveform.

Hypovolemia

Whenever the fit of circulating volume to the vascular bed capacity is poor, as with blood loss, an inadequate autonomic response to such loss [28], or with sympathetic paralysis, compensation for raised intrathoracic pressure by peripheral vasoconstriction is diminished and the Valsalva maneuver can produce rapid circulatory collapse with slow recovery [6]. In our clinical experience of severe hypovolemic blood loss, we have found the Phase I response to be minimal or inverted, Phase II compensation reduced or lost, the Phase III after drop accentuated, and Phase IV prolonged with a slow return to the pretest blood pressure without overshoot.

Hypervolemia and pump failure

Fluid overload or cardiac failure increase the central blood volume. Such circumstances may benefit from a raised intrathoracic pressure that provides the *vis a tergo* to supplement the force of left ventricular contraction and so the left ventricular output, while at the same time limiting venous return until the excess central volume is relocated to the periphery. Assisted emptying of a distended left ventricle may ultimately improve contractility. Phase I and Phase II may often maintain a systolic pressure greater than normal, which declined during

Table 1. Summary of the main clinically useful diagnostic signs in the peripheral pulse (PP) wave form assuming stable cardiac function

Wave component	Significance	Decrease caused by	Increase caused by
Amplitude	Cardiac output Volume loading Vasomotor tone	Hypothermia Fluid or blood loss Arterial obstruction	Hyperthermia Fluid loading Vasodilators with fluid loading
Area under the curve	Indicative of left ventricular stroke volume	As above	As above
Respiratory wave modulation of the peripheral pulse wave form baseline	Indicator of fluid loading: pattern depends on spontaneous or IPPV Mediated primarily through changes in central venous return to right heart	Decreases in superimposed respiratory wave amplitude indicate adequate match between circulating volume and vascular bed	IPPV: Downward deviation with inspiration indicates hypovolemia IPPV: Upward deviation with inspiration indicates fluid overload Spontaneous breathing: changes follow opposite pattern to IPPV
Dicrotic notch position	Indicator of vasomotor tone	Descends towards wave form baseline with peripheral vasodilation	Ascends towards wave peak with peripheral vasoconstriction
Double dicrotic notch	Indicator of vasomotor tone and pressure wave reflection speed	Normally absent from standard trace	Presence indicates hyperdynamic circulation and significant pressure wave reflection
Valsalva maneuver: applying positive intrathoracic pressure for ±15 sec	Indicator of fluid loading and effectiveness of autonomic responses	Decrease PP wave amplitude with dehydration, fluid or blood loss, vasomotor paralysis	Sustained increase of PP wave amplitude with fluid overload or congestive cardiac failure

Phase III and Phase IV to pretest levels [6, 29]. We postulate that the peripheral pulse monitor will reflect this as an increase in amplitude – this requires repeated study with new technology.

While these changes are described in terms of changes of the blood volume to circulation fit, other variables in the pump and vasomotor control may operate to confuse the picture. Increased intrathoracic pressure, whether by IPPV or Valsalva, briefly throttles back the circulation. A normal heart handles the accumulated preload load on pressure release; the failing heart may not, as reflected by a delayed recovery from Phase III hypotension to Phase IV.

The obvious variable that must be controlled in order to interpret the pulse wave changes is ventilator delivery. Thus, the tidal volume, inspiration-to-expiration time ratio, and level and length of the end-inspiratory plateau should be constant for meaningful interpretation of peripheral pulse changes.

In a preoperative test that has long been used for general circulatory competence, changes in blood pressure during and following the application of a Valsalva maneuver are followed, with or without foot-down tilt. Rapid stabilization of the blood pressure during the application of a constant intrathoracic pressure is taken to indicate an ability to compensate for blood loss and postural change during light anesthesia. Failure would indicate the need for checking blood volume, cardiac reserve, and peripheral neuropathy. Rapid tracking of the response by non-invasive means over a short period of 60 to 90 seconds is ideally achieved with peripheral pulse monitoring.

Baseline width (Figure 13)

Comparing the width of the baseline with the width between the two flow peaks on either side gives an indication of the filling of the vascular bed for the prevailing vasomotor status. A rapid diastolic runoff with a relatively long baseline may indicate inadequate stroke volume (Figure 13).

CLINICAL APPLICATIONS

Most clinical applications of peripheral pulse monitoring are summarized in Table 1. Figure 2 illustrates the effect during dysrhythmia (bigeminy) on peripheral pulse am-

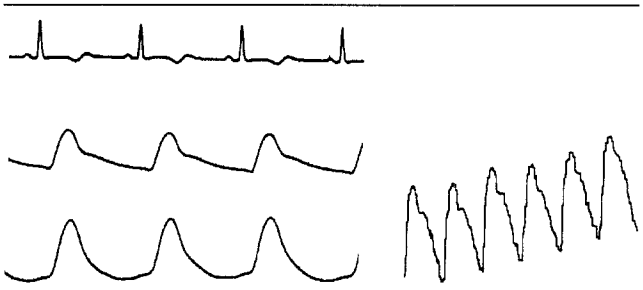

Fig 13. Baseline width. The baseline width is an indication of the adequacy of the stroke volume in matching vascular capacitance for a given state of peripheral vascular tone. The left panel shows from top to bottom: ECG, arterial pressure, and a plethysmograph. Note: a relatively small area under the curve of the plethysmograph wave form, which lasts less than 50% of the period and early return to basal peripheral volume. The right panel (from a different patient) shows greater peripheral perfusion (lasting 100% of the period) with adequate balance of stroke volume to arterial compliance.

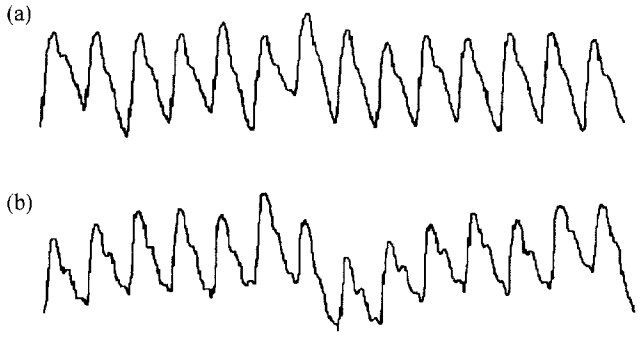

Fig 14. The effect of head up tilt. Respiratory fluctuation of the peripheral pulse waveform of a patient before and after head up tilting to 20°. (a) Patient supine – IPPV – minor respiratory fluctuation. (b) Head up tilt – IPPV – large respiratory fluctuations indicating lowered cardiac filling pressures caused by peripheral pooling – a generally useful sign of reduced venous return.

plitude that follows changing ventricular loading. Three further uses of special interest in anesthesia appear below.

Fluid loss and overload (Figure 14)

Changing waveforms of the peripheral pulse can be anticipated with changes in fluid loading, sensitized by the effects of general or spinal anesthesia. With fluid loss by hemorrhage, third space accumulation, or tissue drainage, the respiratory wave is accentuated (Figures 6 and 7) and reduced peripheral flow may lead to loss of signal tracking [30]. The nadir comes shortly after peak inspiration with IPPV, or before the peak of a spontaneous inspiration, and is accentuated during IPPV. Rapid fluid loading flattens the respiratory wave; overloading may reverse its relation to the phases of respiration. These effects are easily demonstrated clinically when the differences between IPPV and apnea become pronounced.

Monitoring vasodilator drugs

The intravenous vasodilators used in anesthesia act rapidly to produce a misfit between circulating volume and vascular bed. Three immediate effects are seen: 1) the dicrotic notch descends towards baseline; 2) if the original waveform was broad and rounded, it may become narrow and peaked; and 3) the outflow section of the wave may descend *below the baseline* before it returns to the "normal" basal flow that precedes the systolic phase (Figures 9 and 10). Adequate fluid loading will restore the broad waveform and descent to normal baseline without a change in the position of the dicrotic notch.

Peripheral neuropathy

Circulatory homeostasis during sudden postural change requires an intact vasomotor response, making this a good preanesthetic indicator of peripheral neuropathy that may cause unstable blood pressure during general anesthesia. The test, carried out on patients placed on a tilting table, must follow rapidly the changes in blood pressure/peripheral flow over not more than 5 minutes. For this the peripheral pulse waveform is a superior detector as a noninvasive, beat-to-beat detector of change.

As pointed out earlier, any circumstance in which cardiovascular reflexes are compromised will reduce the ability to handle postural change or Valsalva's maneuver, or a demand for raised physical work. Circulatory compromise may be discovered in high spinal anesthesia, early cardiac failure, Guillain-Barré syndrome, peripheral neuritis, severe blood loss, or with certain vasodilator or β-sympathetic blockers [6, 26, 28, 31]. A hypotensive response to a small dose of a vasodilator premedication has long been used as an indicator for fluid loading before anesthetic induction.

Light anesthesia and awareness

Depth of anesthesia can be correlated to sympathetic activity, which is depressed by deeper levels than light surgical anesthesia [32]. The amplitude of the peripheral pulse oximeter waveform and the position of the dicrotic notch appear to be sensitive indicators of the level of catecholamine stimulation. A pressor response with de-

creased peripheral pulse amplitude, usually with an elevation of the dicrotic notch, is a signal of sympathetic activity, an early indicator of lightening anesthesia, with the attendant risk of awareness [33] (Fig 4).

Hypotension and the pulse oximeter waveform

The sole purpose of the systemic circulation is to provide adequate gas exchange, nutrition, and waste product removal for body cells, the ultimate controllers of the circulation. Supply is fed to exchanging tissues close to ambient pressure; the distribution is controlled by varying resistance in supply pathways. The peripheral pulse waveform may be an indicator of adequate supply in two ways: by minor changes in oxygen saturation or in the waveform itself. Two waveform pattern extremes are seen: the full, rounded wave descending slowly to baseline or the thin, peaked wave with a long baseline. The former suggests luxurious perfusion, vasodilation, and correct fluid loading; the latter a cutback of supply by vasoconstriction to manage hypotension, hypovolemia, or failing cardiac output. Perfusion may be adequate with either, depending on supply pressure, but is less likely in the latter case. An accepted principle is to perfuse at lower rather than higher systolic pressures, thereby reducing afterload and cardiac work. In the supine patient, with no hydrostatic pressure difference between either end of the body, a low pressure with full peripheral volume flow is to be preferred. The peripheral pulse and blood pressure are the guide to such management, but when hypotension is deliberately maintained, other checks of organ function must be added – ECG, EEG, and urine flow.

Emergence from anesthesia

It has long been recognized that the high-pressure intrathoracic transients generated by coughing can lead to depression of systemic blood pressure by mechanisms essentially similar to those already discussed [6, 29]. Such transients are most likely during anesthesia on extubation, a time when monitoring may be reduced and significant hypotension goes undetected. At such times, the pulse oximeter may detect such decreases. Its role here remains to be examined.

CONCLUSION

The waveform from a peripheral pulse monitor or pulse oximeter offers a valuable, noninvasive diagnostic tool for circulatory dynamics. Because the data are presented in the form of a wave in which only time, not amplitude, can be meaningfully measured, interpretation is based on waveform pattern changes rather than on absolute measurements. These changes may be followed over a passage of time or they may be produced acutely by circulatory challenge such as Valsalva's maneuver, sudden position change, intravenous vasodilators, or the effect of intermittent positive pressure breathing. Several of the phenomena described in this paper can be developed into new tests for cardiorespiratory interactions.

APPENDIX

The waveforms displayed in the figures were recorded on Hewlett-Packard Co. (Waltham, MA) Merlin or North American Dräger (Telford, PA) Mk 4 monitors using Nellcor D-25 optical transducers. Some of the longer traces have been horizontally digitally compressed to fit. We used the Hewlett-Packard monitors after changing from Signal Quality Indicator (SQI) Mode to Perfusion Mode operation to improve the display of baseline shifts. A standard paper speed of 25 mm/sec was run with the HP Merlin monitor; 12.5 mm/sec with the NAD Mk 4 monitor.

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