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THE CONTINUOUS MEASUREMENT OF ARTERIAL PULSE WAVE VELOCITY*†

GERSHON WELTMAN
NATO Fellow, Weizmann Institute of Science, Israel
GEORGE SULLIVAN and DALE BREDON
Spacelabs, Inc., Van Nuys, California, U.S.A.

Abstract—It has long been known that arterial pulse wave velocity (PWV) depends on arterial extensibility. Since extensibility is a function of arterial pressure, as well as of various pathological states, the measurement of PWV provides a promising approach to the indirect monitoring of the cardiovascular system. The present paper describes a Pulse Wave Velocity Computer designed to measure and record PWV in humans. Utilizing the EKG complex and a downstream pulse signal to define pulse-transit-time over a known arterial length, the computer calculates an individual velocity value for each emitted pulse, and generates a continuous, stepwise-analog record of the PWV. Some initial experimental work with human subjects is presented. Observations are reported for the PWV during rest, during deep breathing, and during execution of Valsalva maneuvers. In all cases the continuous PWV record appears to follow closely variations in arterial pressure, apparently adhering, in the Valsalva results, more to systolic than diastolic changes. Observed subject differences attest to the PWV's diagnostic value, and it is suggested that the continuous PWV may be a significant and useful circulatory indicant.

1. INTRODUCTION

EACH contraction of the heart produces a pressure pulse which travels outward over the arterial walls into the peripheral circulation. The pulse's propagation velocity can be expressed mathematically if certain simplifying assumptions are made (KING, 1950; MALCOLM, 1957). It is found that for short, uniform arterial lengths the pulse wave velocity (PWV) varies directly with the square root of the arterial modulus of elasticity, that is, inversely with the square root of the arterial extensibility. Extensibility does not remain constant in human arteries; it varies as a function of the following factors:

1. Age. As the artery ages, its gradual stiffening increases its elasticity and decreases its extensibility, raising the PWV. Data to document this change come from a number of sources (BRAMWELL, *et al* 1923; HALLOCK, 1950; and others), and indicate that arteries may be classified by their "apparent" as well as by their chronological age.
2. Pathological Condition. Various disease states affect the characteristics of the arterial wall, and accordingly the measured pulse wave velocity. Significant changes in PWV have been observed in arteriosclerosis by SANDS (1925), in peripheral cir-

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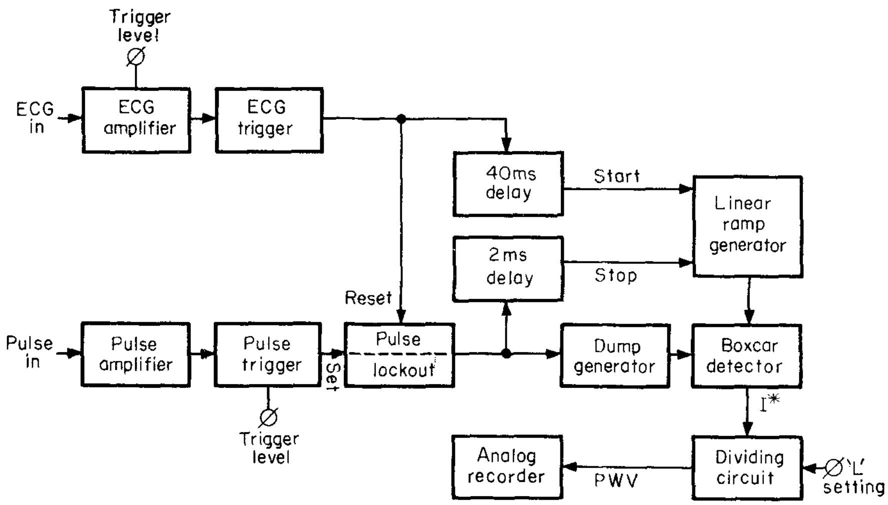

FIG. 1. Block diagram of the pulse wave velocity computer.

culatory disorders by LETTENBACH and STUCKI (1950), in coarctation of the aorta by JOHNSON (1950), and in diabetes by WOOLAM, *et al.* (1962), to name just a few pertinent studies.

3. Pressure. As transmural pressure increases, the artery is stretched and stiffened, and consequently the PWV is increased; as arterial pressure decreases, the relatively flaccid wall transmits a decreased PWV. Although this has been demonstrated theoretically by KING (1950), for excised arteries by BRAMWELL and HILL (1922*a, b*), and *in vivo* by BRAMWELL, *et al.* (1923*b*), HEMINGWAY, *et al.* (1928) and MATZE, *et al.* (1923), the relationship of internal pressure to PWV has not been exactly determined for long lengths of branched artery in human subjects. Studies relating PWV to mean pressure reveal a wide transmural pressure range within which the statistical dependence appears nearly linear, while there is some evidence of a higher correlation with systolic than with diastolic pressure (SANDS, 1925).

The dependence of PWV on internal pressure suggests an attack on the as yet unsolved problem of continuously and indirectly monitoring arterial blood pressure: that is, use externally determined PWV to infer internal events. Previous investigators, handicapped by prohibitively slow data reduction techniques, produced virtually no information on PWV as a dynamic circulatory variable. WELTMAN (1959) obtained some initial continuous PWV recordings with rather crude experimental equipment. They seemed to confirm the validity of this approach. His preliminary work has been extended by the construction of a compact, sensitive, and more reliable Pulse Wave Velocity Computer, and by the testing of the computer on a larger subject group.

2. THE PULSE WAVE VELOCITY COMPUTER

The present computer determines PWV, defined as

$$\text{PWV} = L/I \text{ (m/sec)}$$

where:

L = the arterial length between the aortic arch and peripheral pulse transducer, estimated with the aid of anatomical landmarks. (WELTMAN, 1959.)

I = the time interval between ventricular ejection from the heart and pulse arrival at the peripheral transducer, measured as the interval between detection of the

"R" wave of the ECG complex and the peripheral pulse, and corrected for the average 40 ms expulsion delay, which SANDS (1923) found to be relatively independent of heartrate.

All physiological information is secured by external sensors. Standard leads are used to obtain the ECG signals. A PWV value is computed for every heartbeat, and these values combined to produce an analog record representing PWV as a continuous time-function.

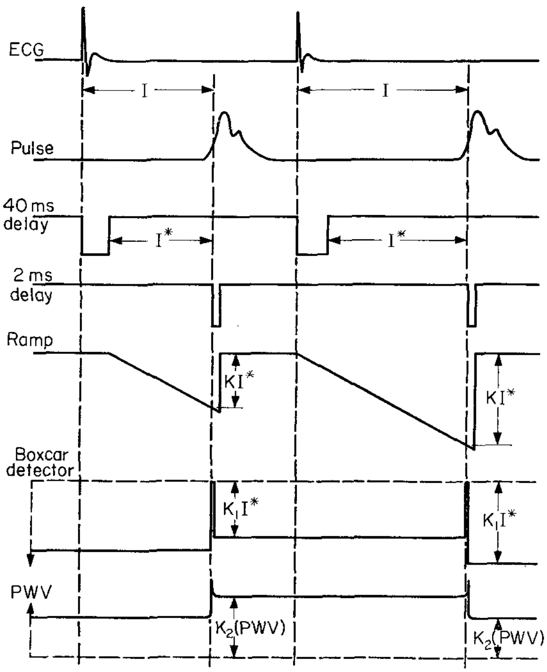

FIG. 2. Waveforms and timing relations within the pulse wave velocity computer.

At present, a crystal microphone which differentiates the pressure waveform is used to detect the peripheral pulse. The peripheral transducer is a crucial component, as PWV measurement in active subjects is limited by the transducer's ability to reject movement artifact. Development of better arterial-rider (PRESSMAN and NEWGARD, 1962) or photoelectric transducers may help the future situation, but currently available units of both types function poorly on the unstabilized limb.

A schematic diagram of the computer is presented in Fig. 1. The ECG and pulse signals form the primary inputs. Both are amplified, then shaped by trigger circuits with individually adjustable trigger levels. Activation of the ECG trigger initiates the generation of a linear voltage-ramp, after a 40 msec expulsion-correction delay. Subsequent arrival of the peripheral pulse signal simultaneously activates a lockout circuit (which prevents double triggering) and a unit called the dump generator. The dump generator instantaneously brings the voltage on a storage circuit, the boxcar detector, to zero. The boxcar detector then quickly recharges to the voltage level of the ramp (which now represents I), and holds this level. The ramp generator is reset to zero after a two msec delay, and awaits the next ECG signal, which opens the peripheral pulse lockout and reinitiates the cycle.

Thus the output of the boxcar detector is a continuous stepwise analog representation of I, interrupted only for short time intervals. The analog divider circuit, formed by placing a diode quarter-square multiplier in the feedback loop of an operational amplifier, uses this voltage to form the quotient L/I. (A voltage proportional to the constant L is set into the computer by means of a ten-turn potentiometer). The computer is calibrated by using an external delay circuit to supply start and stop pulses at variable known intervals. Operation of the computer is illustrated by the waveforms and timing relations shown in Fig. 2.

An analog voltage linearly proportional to PWV appears at the computer output, and may be fed to an oscilloscope, a direct-writing rerecorder, or a tape recorder. The voltage reflects the stepwise character of the boxcar detector output, but the limited frequency response of most recording instruments smooths the reset discontinuity—leaving only a small spike useful for identifying individual heartbeats. Retention of this stepwise response insures that the computer exactly follows changes in circulatory dynamics, which may occur even between individual heartbeats. Because of leakage in the final storage capacitor, the PWV records of this initial study took on a sawtooth appearance. A better capacitor was obtained, and this characteristic has now been eliminated.

The prototype computer is shown in Fig. 3. It utilized tube circuitry and several commercial subassemblies. An optimally designed unit would be smaller, perhaps through the use of transistor circuitry, include a small oscilloscope tube for monitoring waveforms and setting trigger levels, and provide an associated meter-readout of PWV.*

A major advantage of PWV as an indication of circulatory events is that unlike plethysmographic and similar measures, it involves no inherent baseline–drift problems, being tied to a stable timebase within the Pulse Wave Velocity Computer. This means that even though artifacts may intermittantly disrupt a record, reception of a good ECG-pulse signal pair produces "true" data regardless of when the signals are received, and what the state of other physiological variables.

The potential value of the continuous PWV record has been recognized by other investigators as well as by the present authors. SALISBURY and WICHMANN (1962) have suggested using artificial pulses, rather than the natural arterial events, to define pulse-transit-time. Their system would impress a square-wave pressure pulse at the brachial artery, and detect this same pulse as it moved to the wrist. The advantage is that the pulse shape and approximate arrival time is known, thus optimal filtering can be applied to reject noise signals. The danger is that the impressed pulses, travelling upstream as well as downstream, will themselves affect the circulatory system's regulatory mechanisms.

In the present system, the sharply-rising leading edges of the R wave and the differentiated pulse signal defined pulse-travel-time. The triggering level itself was about half way up the respective waveforms. Bacause the baselines were stable and the transit-time long (about 250 msec) compared to the average signal rise times, simple triggering errors due to changes in wave shape were judged to be inconsequential. The error due to the time discrepancy between R wave detection and actual pulse ejection is another matter. The 40 msec expulsion delay selected for this study is, of course, only an average value; and while it might increase the mean accuracy of determination for a large group, it does little for the precision of measurement on an individual subject, or where exercise, disease, etc., are primary experimental variables. In fact, the Russians BAYEVSKY and GAZENKO (1963) report that a change in electromechanical delay was one of the more interesting, and unexplained, concomitants of cosmonaut Titov's exposure to weightlessness.

The measurement ambiguity could be avoided by using two peripheral sites to define pulse-transit-time; the present equipment could be readily adapted to this technique. However, since the peripheral transducer is the main trouble spot in achieving artifact-free recording from active subjects, it would seem that the introduction of two such transducers would double our problems here, while in addition accentuating the effect of pulse-shape variations and decreasing the advantage now gained by using long arterial lengths. The transduction of ventricular contraction at a site over the heart appears to be a more reasonable approach. We are currently studying the use of phonocardiography, employing sharply-tuned microphones, for this purpose. Perhaps the time-corrected ECG signal could be employed when convenience is the dominant experimental criterion, and a more precise expulsion indicator substituted when the constancy of the electromechanical delay is suspect.

3. EXPERIMENTAL INVESTIGATION

Seven normal subjects were used to examine PWV response during relaxation, deep breathing, a Valsalva maneuver, and the same maneuver following deep breathing. The subjects reclined during experimentation, and the peripheral sensor was placed on the radial pulse. Unfor-

* Computers of this type are commercially available from Spacelabs, Inc.

FIG. 3. The prototype pulse wave velocity computer.

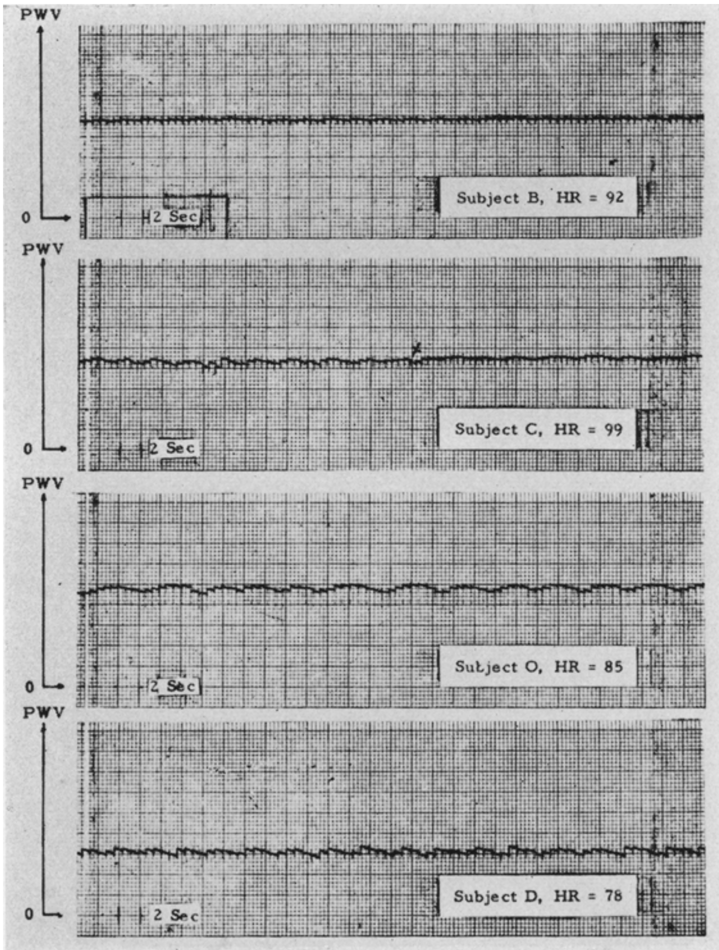

FIG. 4. PWV records obtained from four subjects during supine relaxation. Anatomical measurements were not uniformly taken; thus the records shown, while linearly proportional to true PWV are not calibrated absolutely. WELTMAN (1959) reported the similarly-obtained mean resting PWV to be about 4·5 m/sec over this pathway.

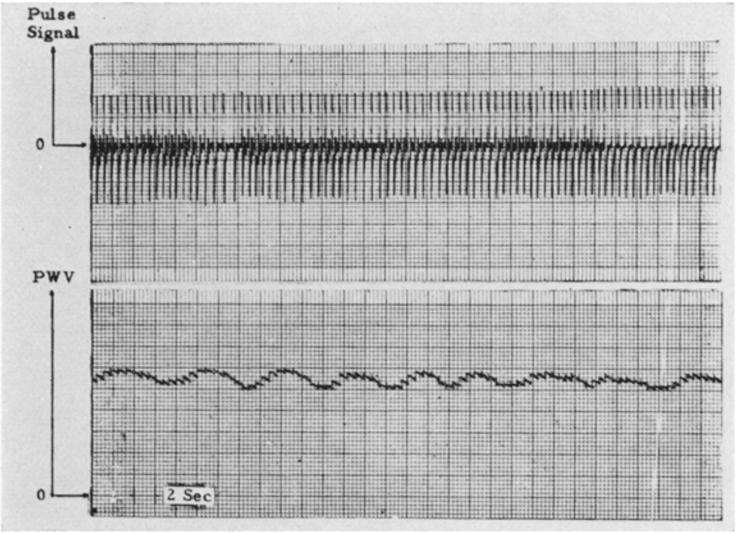

FIG. 5. Peripheral pulse signal and PWV during deep breathing (heart rate about 87 beats/min).

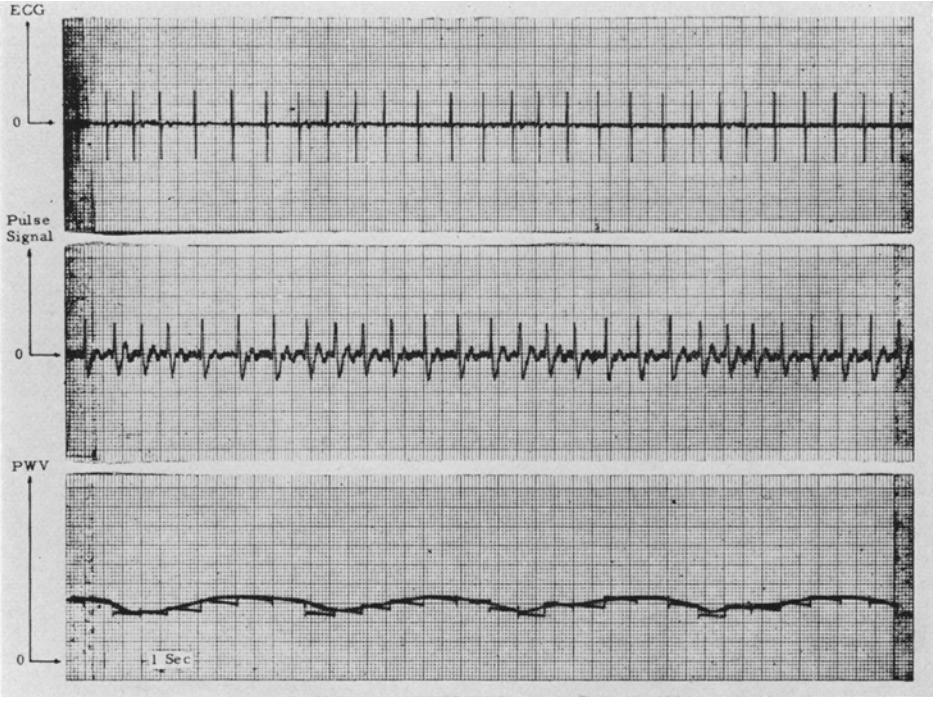

FIG. 6. Changes in the heart rate and amplitude of the EKG, pulse signal, and PWV during hyperventilation.

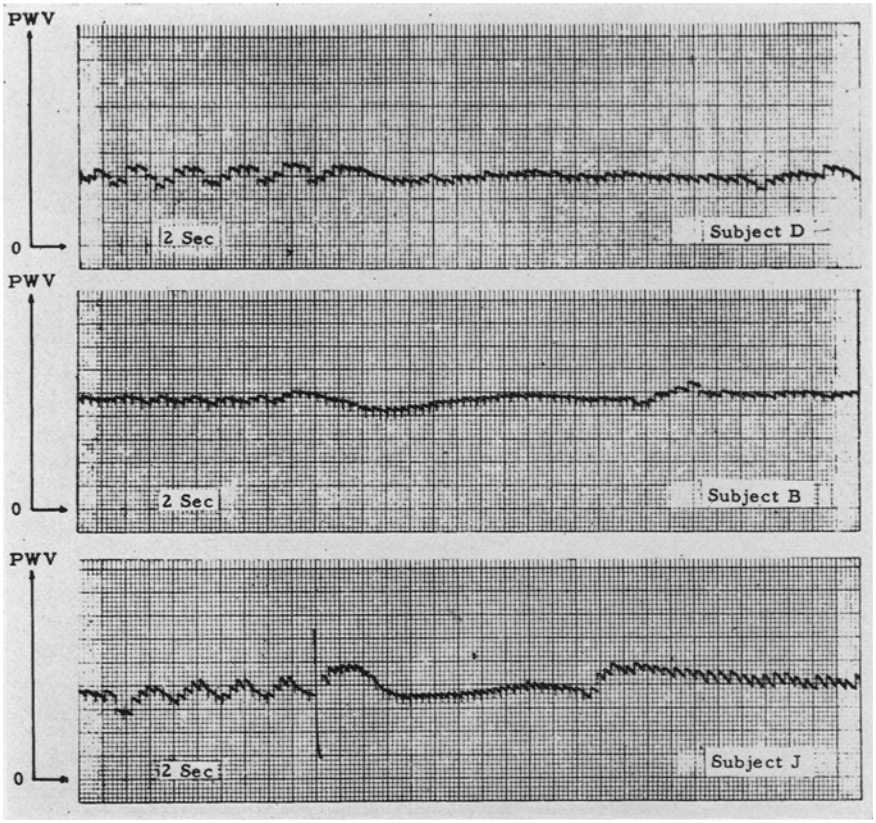

FIG. 10. PWV response to the prolonged valsalva maneuver following hyperventilation.

tunately, neither respiratory phase nor true arterial pressure were recorded during the subject runs, so we have had to work backwards, using indirect evidence to relate PWV variations first to respiratory events and then to known concurrent blood pressure events. The results are summarized below.

3.1. *Relaxation*

Previous work by HICKSON and MCSWINEY (1924*a* and *b*) and WELTMAN (1959) has characterized the PWV as extremely stable during relaxation. This was again shown by the present study. Representative individual records are reproduced in Fig. 4. The periodic fluctuations in PWV, most evident in Subject O's record, are related to the circulatory events during the respiratory cycle. They demonstrate both the observed differences among subjects and the sensitivity of the PWV record. Note, for example, the distinct decrease in the PWV variations midway through C's record; at this time he dozed and his breathing became shallower.

3.2. *Deep breathing*

The respiratory variations noted in the PWV records during relaxation were accentuated when the subjects hyperventilated. This effect is well shown in Fig. 5, which includes the tracings of the peripheral pulse channel, and was obtained during a period of deep, slow breathing, normal heart rate, and high PWV. High paper-speed records, such as that of Fig. 6, were used to infer the relation of PWV to arterial pressure during the respiratory cycle. Figure 7 graphs heart rate (obtained from the EKG tracings of the previous record) simultaneously with PWV.

BARD (1961) has documented the variation in heart rate during respiration, so that we could associate inspiration with its marked rise, and expiration with its sustained slowing and subsequent recovery. Looking then at the PWV record, we found that it apparently followed the accepted movements of the arterial pressure: a brief drop during the beginning of inspiration, a return, and a maximum early in expiration. PWV was thus indirectly correlated with blood pressure during respiration, as it had previously been directly correlated by HICKSON and MCSWINEY (1924*a*). But since both systolic and diastolic pressures react similarly during respiration, no differential relationship could be assigned.

3.3. *Valsalva maneuver*

A Valsalva maneuver is essentially a static exhaling against a closed glottis, involving strong contractions of the stomach musculature. When this maneuver follows relaxation, a recognizable

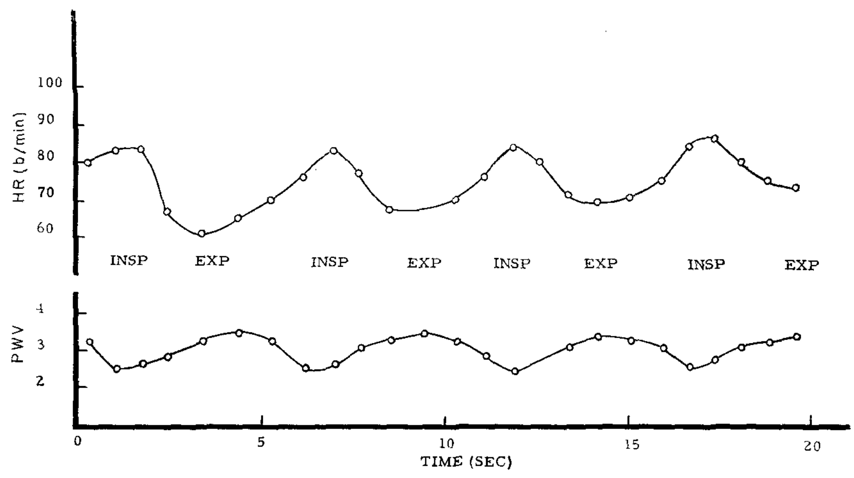

FIG. 7. The simultaneous variation of heart rate and PWV during hyperventilation.

PWV response pattern results. Figure 8 illustrates this pattern, and includes a simultaneous heart rate record. Immediately after initiation of straining the PWV rises momentarily, then drops markedly, and slowly returns to rest level. The heart rate begins to rise, and remains elevated during the straining period, but the magnitude of the pulse signal decreases. When the breath is expelled and breathing resumed, the PWV first dips, then increases rapidly at the same time that the heartrate falls sharply, and the pulse wave signal returns to rest level or greater. This condition of slow, powerful beats in combination with a relatively high PWV holds for about 30 sec to 1 min, gradually returning to pre-stress conditions. There is no appreciable change in the "R" wave amplitude. Detailed analysis of the ECG complex has not been attempted; although there is some evidence that changes do occur (SHAFTERL, 1960), they do not appear important to our timing conventions.

The diversity of individual response is illustrated by the records reproduced in Fig. 9. Subject D exhibits no response during the straining period, Subject B demonstrates the characteristic pattern, while Subject J shows a standard, but disorganized, response.

Prior hyperventilation strengthens the PWV response pattern. This is clearly seen in Fig. 10, which presents records for the same three subjects. A slight pattern is now discernible in D's record, B remains unchanged, and J's response is now strong and clear.

Virtually the same sequence of events has now been observed in continuous PWV records obtained from about 15 subjects. The apparent correspondence of this pattern to arterial pressure events is illustrated by Fig. 11, which presents some typical arterial pressure contours

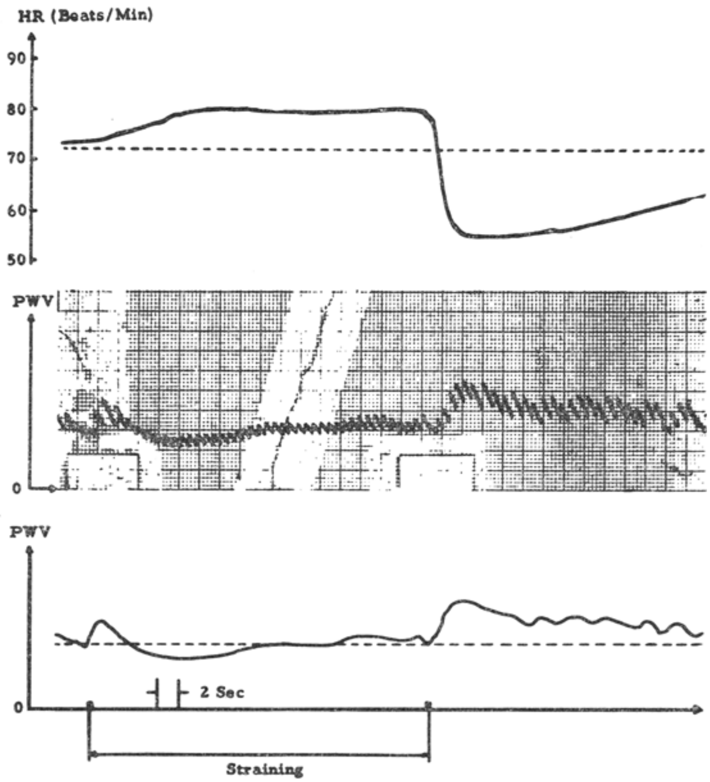

FIG. 8. The typical pattern of heart rate and PWV response during the prolonged valsalva maneuver.

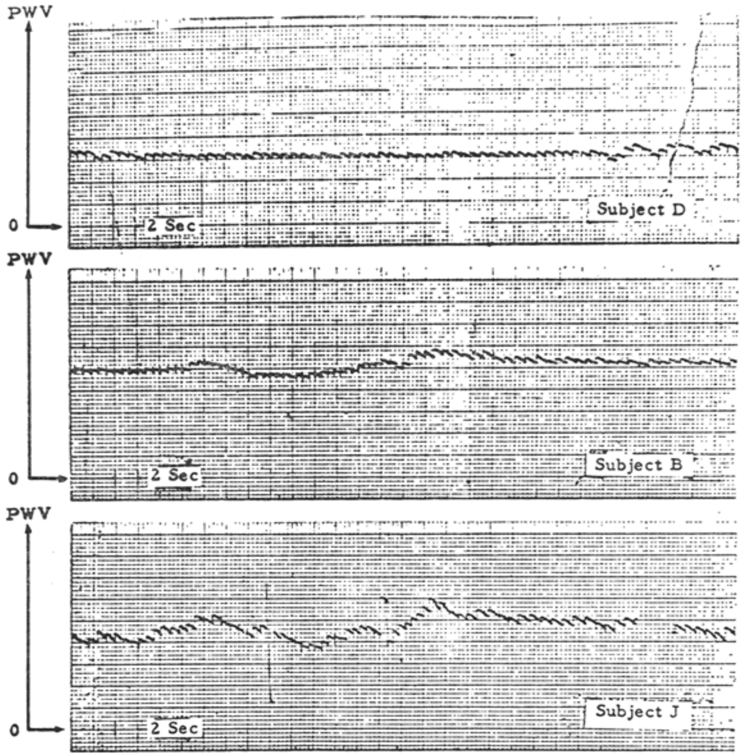

FIG. 9. PWV response to the prolonged valsalva maneuver following normal breathing.

observed by other investigators in normal subjects during the prolonged Valsalva maneuver. The initial arterial pressure rise reflects the momentary effect, within the heart and large vessels, of the suddenly raised intrathoracic pressure; the subsequent fall follows an uncompensated decrease in venous return. Compensation in the form of perpheral vasoconstriction and an increased heart rate causes a pressure return if straining is prolonged. As straining is released, there is first an arterial pressure drop which again reflects the change in intra-thoracic pressure, and then a gradually diminishing "overshoot" phase, during which the heart beats powerfully into the still-constricted peripheral circulation, raising pressures well above rest levels. As shown, small variations in this general response appear among different normal subjects. One notices, however, that the systolic response, whether large or small, always adheres to the PWV pattern described. The diastolic response, on the other hand, though occasionally approaching this pattern, frequently exhibits characteristics (such as a marked increase over rest level during straining, and only a small, short overshoot after release), not seen in our collected data. A tentative hypothesis, then, is that the PWV is following systolic rather than mean or diastolic arterial pressure.

4. CONCLUSIONS

Initial testing of the Pulse Wave Velocity Computer was satisfactory from the electronics standpoint. A small experimental study produced continuous PWV records of good quality, showing both stability during rest and sensitivity of response. The regular reaction to the Valsalva maneuver, when contrasted to PWV stability under equilibrium conditions, indicated that we were observing real changes in a circulatory variable. Both the deep breathing and the Valsalva results evidenced a correlation of PWV with arterial blood pressure, but this was demonstrated indirectly rather than rigorously. Clearly, simultaneous recording of PWV and directly-measured pressure over a variety of conditions is necessary before truly significant correlations can be assigned.

Methods of calibration for individual subjects would have to be devised in order that PWV be used to infer arterial blood pressure. This process appears possible, although perhaps tedious and probably rather imprecise. One wonders if it is necessary. In PWV we have a stable measure, apparently indicative of pressure variations in short-term circulatory episodes, but also revealing changes in arterial-wall condition over longer time-periods, and permitting comparison either among subjects or between arterial pathways in one subject. In addition to the diagnostic comparisons previously cited, PWV reportedly also shows promise in assessing the functional state of persons exposed to chronic radiation (DENISOVA, 1962). We have demonstrated a marked and individually varying response to the Valsalva maneuver; other investigators, such as OLIVER and HULTGREN (1961) and ROSENBERG, *et al.* (1958), have linked observed circulatory response to this maneuver to various pathological conditions.

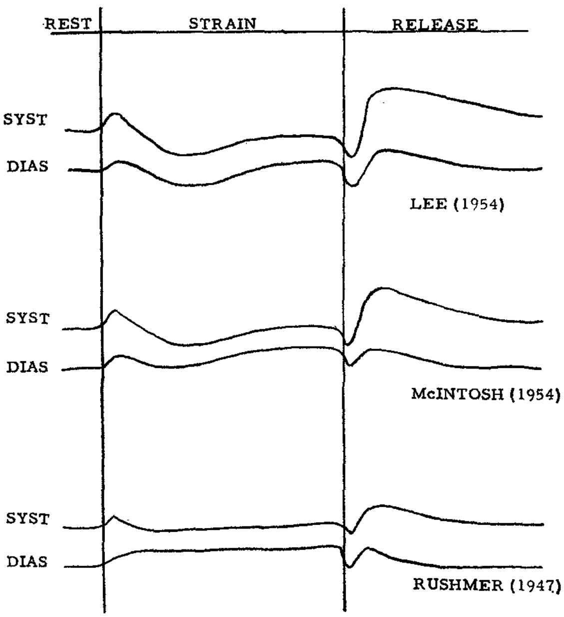

FIG. 11. Some typical arterial pressure responses to the prolonged valsalva maneuver.

Since the clinical, indirectly-obtained blood pressure is in itself only a defined variable, it might be worthwhile to neglect strict calibration in favour of extensive determination of normative and abnormal PWV levels and response characteristics. As SULLIVAN, *et al.* (1962) suggest, the continuous PWV, recorded along with a few other easily-measured variables, could then be used to monitor cardiovascular dynamics indirectly but reliably, and in fact might provide more information then does the largely homeostatic blood pressure. Interpretation of PWV need not depend on quantification in blood pressure term, but could be referenced, as clinical blood pressure is now, to a body of exploratory studies. At any rate, the inability of the robot arm-cuff monitors to cycle fast enough to provide truly continuous readings, a failing shared even by the more comfortable digital sphygmomanometers, justifies the continuation of research on the PWV as the only currently promising means of continuous indirect circulatory recording in the freely moving subject.

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UN PROCEDÉ CONTINU DE MESURE DE VITESSE DE L'ONDE PULSATOIRE

Sommaire—Il est connu depuis longtemps que la Vitesse d'Onde Pulsatoire (PWV) dépend de l'extensibilité artérielle. L'extensibilité étant une fonction de la pression artérielle ainsi que de différents facteurs pathologiques, la mesure de la PWV constitue une approche intéressante du contrôle automatique indirect du système cardiovasculaire.

La présente étude décrit un système pour mesurer la PWV chez l'homme au moyen d'un calculateur. Ce calculateur utilise le complexe ECG et le signal du flux pulsatoire descendant pour définir la durée de son transit sur une longueur artérielle donnée et calculer la vitesse individuelle de chaque pulsation, en obtenant ainsi un enregistrement analogique progressif continu de la PWV. Quelques exemples des premières expériences sur sujets humains sont rapportés, ainsi que des observations relatives à la PWV, faites en état de repos, d'hyperpnée et au cours de l'épreuve respiratoire Valsalva. Dans tous les cas étudiés, l'enregistrement continu de la PWV semble suivre de près les variations de la pression artérielle. Au cours des épreuves Valsalva, les variations de la courbe enregistrée se rapprochent apparemment davantage des changements systoliques que diastoliques. Les différences subjectives observées attestent de la validité diagnostique de la PWV qui pourrait devenir ainsi un facteur d'utilité notable dans la détection d'incidents circulatoires.

DIE KONTINUIERLICHE MESSUNG DER ARTERIELLEN PULSWELLENGESCHWINDIGKEIT

Zusammenfassung—Seit langem ist bekannt, daß die arterielle Pulswellengeschwindigkeit (PWV) von der Elastizität der Arterien abhängt. Da die Dehnfähigkeit der Arterien sowohl vom Arteriendruck, wie auch von verschiedenen pathologischen Bedingungen abhängt, liefert die PWV-Messung eine vielversprechende Methode zur indirekten Kontrolle des Herzgefäßsystems. Die vorliegende Studie beschreibt einen Elektronenrechner für die Aufzeichnung und Messung der Pulswellengeschwindigkeit bei Menschen. Unter Verwendung des EKG-Komplexes und eines distallaufenden Pulses zur Bestimmung der Pulslaufzeit über eine bekannte Arterienlänge errechnet der Elektronenrechner für jeden Pulsschlag einen individuellen Geschwindigkeitswert und liefert dadurch eine stufenweise Analog-Aufzeichnung der PWV. Es wird über einige Versuche an Menschen berichtet. Die Beobachtungen erstrecken sich auf PWV während Ruhe, während Tiefatmen und während Valsalva-Atmungen. In allen Fällen scheint die PWV-Aufzeichnung den Veränderungen des Arteriendrucks sehr genau zu folgen. Bei den Valsalva-Ergebnissen scheint die Aufzeichnung mehr den Systole-als den Diastoleveränderungen zu entsprechen. An Testpersonen festgestellte Unterschiede beweisen den diagnostischen Wert der PWV. Die kontinuierlichen PWV-Registrierungen werden als nützliches Indikationsmittel bei Kreislaufphänomenen empfohlen.

НЕПРЕРЫВНОЕ ИЗМЕРЕНИЕ СКОРОСТИ РАСПРОСТРАНЕНИЯ АРТЕРИАЛЬНОЙ ПУЛЬСОВОЙ ВОЛНЫ

Резюме — Давно известно, что скорость распространения артериальной пульсовой волны (СПВ) зависит от растяжения артерии. Так как растяжимость является функцией артериального давления, а также и многих патологических факторов, измерение СПВ дает возможность непрямого контролирования системы кровообращения. В настоящей работе описано вычислительное устройство для измерения и регистрации у человека скорости распространения артериальной пульсовой волны. Используется ЭКГ комплекс и пульсовой сигнал в нижнем конце артерии для определения времени распространения пульсовой волны по части артериального русла известной длины; вычислительное устройство подсчитывает индивидуальную величину скорости для каждого пульсового колебания и ведет непрерывную шагообразно-аналоговую запись СПВ. Начата экспериментальная работа на людях. Сообщается о наблюдениях СПВ в покое, при глубоком дыхании и во время проведения пробы Вальсальвы. Во всех случаях видно, что непрерывно зарегистрированная СПВ строго следует за изменениями кровяного давления. Установлено, что при пробе Вальсальвы изменения в диастоле большие, чем в систоле. Наблюдаемые индивидуальные различия свидетельствуют о диагностической ценности СПВ. Высказывается предположение, что непрерывная регистрация СПВ может иметь значение и быть полезной в установлении циркуляторных изменений.