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Calvin F. Kay, … , Harry F. Zinsser Jr., J. Malvern Benjamin Jr.

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Research Article

THE VALIDITY OF THE ELECTROKYMOGRAPHIC METHOD FOR MEASUREMENT OF DIAMETER CHANGE OF THE AORTA AND PULMONARY ARTERY DURING CIRCULATORY DISTURBANCE [1]

By CALVIN F. KAY, JAMES W. WOODS, JR., HARRY F. ZINSSER, JR., AND J. MALVERN BENJAMIN, JR.

(From the Robinette Foundation of the Hospital and the Moore School of Electrical Engineering, the University of Pennsylvania, Philadelphia)

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It has been demonstrated by Henny, Boone, Chamberlain, and their associates that border movements of the heart and great vessels may be recorded with the electrokymograph (1, 2, 4, 7). Confirmation of this work has been reported by others (3, 5, 6). Our studies were designed to investigate the accuracy with which movements of the aorta and pulmonary artery could be recorded with this instrument, and the significance of these movements as measures of dynamic cardiovascular changes during certain acute disturbances of the circulation. For these experiments, acute intrathoracic pressure variation was chosen as the implement for the production of circulatory disturbances. It is a procedure with which a new investigative method may be evaluated by comparison with observations of venous, arterial and pulmonary arterial blood pressure, cardiac output, roentgenokymography, and roentgenographic lung density in control studies or in the published reports of others.

The electrokymograph utilizes the roentgen ray beam of the fluoroscope. When the long axis of a small rectangle of fluoroscopic screen is placed perpendicular to the pulsating border of the heart or a great vessel, the average intensity of illumination of the whole rectangle will vary with the motion of the border. When movements of a great vessel are recorded, the dense shadow moves outward in systole to diminish the intensity and in diastole the reverse occurs. These changes in illumination, when viewed by a sensitive photoelectric tube, are converted to electrical impulses which are recorded by an electrocardiographic apparatus.

[1] The Heart Disease Demonstration Section of the U. S. Public Health Service provided the apparatus and part of the funds for this study.

METHODS

Recording methods

The electrokymographic equipment used in this study was of the type described in detail elsewhere (1, 2). An alternating current direct-writing recorder manufactured by the Brush Development Company (8) was used instead of an optically recording string galvanometer. A single stage of amplification was inserted between the photo multiplier tube and the filter.

The response characteristics of the recording apparatus were investigated as a preliminary procedure. To test the frequency response, a circular lead disc was eccentrically mounted on the shaft of a variable speed motor. The edge of the disc was interposed in the X-ray beam perpendicular to and directly in front of the rectangular slot of the phototube. As the disc revolved, a sine wave was recorded.[2] The recorded wave amplitudes were then measured at varying motor speeds. The results are shown in Table I.

TABLE I

Frequency response of recording instruments, expressed in terms of percentage of amplitude at one cycle per second

Frequency	% amplitude
1	100
2	89
3	79
4	70
5	62
6	55
7	49
8	43
9	38
10	33
15	20
20	13

In a second procedure, the response characteristics of the direct-writing Brush instrument were compared with those of optically recording Cambridge and Sanborn instruments. Using the same eccentric cam, rotated at speeds from 1 to 75 cycles per second, with the original filter and also other filters with which the loss at higher

[2] The approximation to a pure sine wave is close enough for practical purposes. The mathematical analysis of the wave form generated by the eccentric cam will be presented in a subsequent publication (20).

frequencies was much less than with the original filter, less than 2% variation was recorded at any time between these three instruments, recording simultaneously.

A third procedure was devised to test the relative sensitivity of the various portions of the phototube slot. For this purpose, the phototube output was directly coupled through a potentiometer to a Cambridge string electrocardiograph. A lead sheet was interposed immediately in front of and perpendicular to the phototube slot. Starting with the slot completely exposed, the lead sheet was moved in successive 2 mm. steps (from base toward free end of tube) until the slot was occluded. The magnitude of string deflection resulting from each 2 mm. movement of the lead sheet was then recorded and measured. The results are shown in Table II.

TABLE II

Relative sensitivity of various portions on the receptor slot of the phototube

Motion in cm.	Recorded amplitude in cm.
0.0–0.2	0.05
0.2–0.4	0.45
0.4–0.6	0.50
0.6–0.8	0.60
0.8–1.0	0.60
1.0–1.2	0.65
1.2–1.4	0.65
1.4–1.6	0.55
1.6–1.8	0.50
1.8–2.0	0.45
2.0–2.2	0.20
2.2–2.4	0.05

From these studies the following conclusions are drawn: (*1*) If a sine wave of fixed magnitude is impressed upon the phototube, the recorded amplitude is diminished by 50% when the frequency is increased from 1/sec. to 7/sec. and by 75% when the frequency is in-

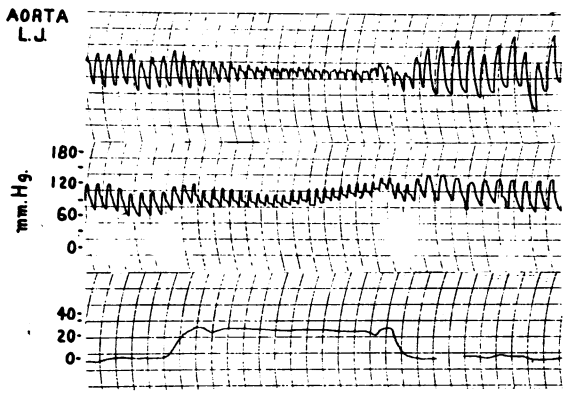

FIG. 1. SIMULTANEOUS RECORDINGS OF AORTIC KNOB PULSATIONS (UPPER), BRACHIAL ARTERIAL PRESSURE (MIDDLE), AND INTRABRONCHIAL PRESSURE (LOWER)

Subject No. 1. Camera speed 1 cm./sec.

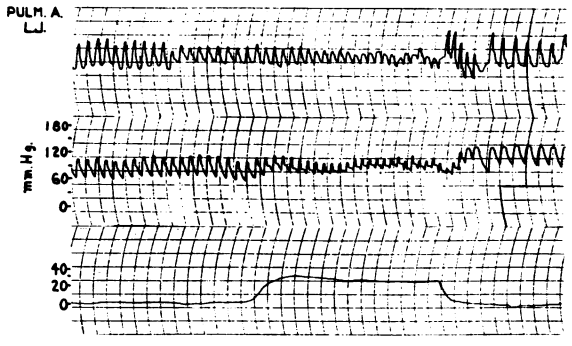

FIG. 2. PULMONARY ARTERY PULSATIONS (ABOVE), WITH BRACHIAL ARTERIAL PRESSURE (MIDDLE) AND INTRABRONCHIAL PRESSURE (LOWER)

Subject No. 1.

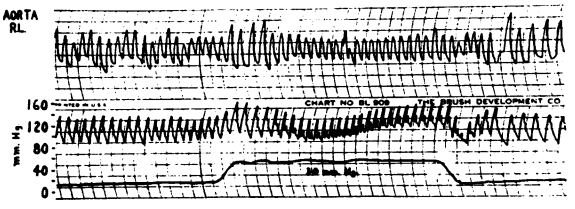

FIG. 3

Same as Figure 1. Subject No. 4. Intrabronchial pressure illustrated is an overlay of the actual recording.

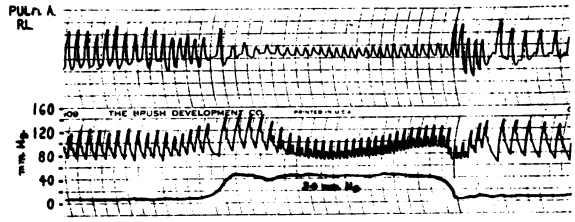

FIG. 4

Same as Figure 2. Subject No. 4.

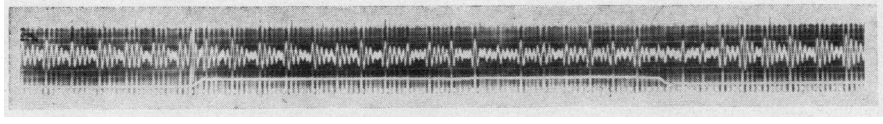

FIG. 5. BALLISTOCARDIOGRAPHIC (VERTICAL TYPE) RECORDING DURING THE STRAINING PROCEDURE

Intrabronchial pressure below. Subject No. 10.

creased from 1/sec. to 13/sec. (*2*) In response to any impulse having a sine wave frequency of from 1/sec. to 75/sec., the records obtained with the direct-writing Brush instrument are identical with those obtained with optically recording instruments. (*3*) The receptive slot of the photomultiplier tube is 2.4 cm. long. In the zone between 0.6 and 1.6 cm. from the tube-base end, the sensitivity is uniform with a maximum possible error of 15%. In the portion of the slot between 0.2 and 2.0 cm. the maximum possible error is 30%.

Investigative procedure

Ten healthy young adults were selected for this study. None had any symptoms or signs suggestive of heart disease or of pronounced vasomotor instability. The standard straining procedure, a modification of the Valsalva experiment, was performed in the sitting position. Braces were applied to the chest and shoulders to reduce extraneous movement. On command, breathing was arrested in a mid-respiratory position and the recording apparatus was set in motion. After a brief control period, the subject strained against a column of mercury to a pressure of 30 mm. This pressure was maintained for 15 seconds. He then relaxed for ten seconds before resuming respirations. The procedure was then repeated several times. The subject kept his glottis open throughout the experimental procedure.

Employing this standard procedure, electrokymographic tracings were recorded from the pulmonary artery and aortic knob. Intrapulmonic pressure was directly observed by the subject and operator, and recorded by a strain gauge and electrical recorder or capsule and optical recorder. Brachial artery pressure was recorded simultaneously through a Peterson catheter to a capacitance manometer (8, 9). In seven subjects, records were made with the vertical ballistocardiograph during the straining procedure. Representative tracings are shown in Figures 1–5.

In the electrokymographic records, an upward motion parallels outward motion of the great vessels during ejection; the downward curve follows the inward motion of the vessel during diastole. The intrapulmonic, arterial pressure, and ballistocardiographic curves are self explanatory.

RESULTS

The variations of pulse wave amplitude of the aorta and pulmonary artery, as recorded by the electrokymograph during and after the straining procedure, in each of the ten subjects, together with the results of control studies, are indicated in Table III.

Aortic pulsations

The pulse curves recorded from the aortic wall usually remained quite constant during the control period of respiratory arrest. A gradual decrease in amplitude began with the increase in intra-

TABLE III

Subject	Recording	Straining period			Post-straining period		
		4″	8″	12″	1″	4″	8″
No. 1	BP {BP	76	98	106	113	116	100
	BP {PP	81	57	57	67	119	114
	$\Delta p/\Delta d$	1.4	1.7		1.2	0.7	0.7
	EKY {AK	56	33	33	55	166	155
	EKY {PA	83	52	50	151	144	120
	Beats/second	1.6	2.0	2.2	1.4	1.1	1.2
No. 2	BP {BP	65	77	62	94	105	98
	BP {PP	93	87	57	50	100	93
	$\Delta p/\Delta d$	1.1	1.1		0.7	0.5	0.5
	EKY {AK	100	81	36	70	215	205
	EKY {PA	71	74	37	110	104	115
	Beats/second	2.2	2.9	3.0	3.0	0.8	1.1
No. 3	BP {BP	42	42	36	50	120	91
	BP {PP	52	45	56	47	122	141
	$\Delta p/\Delta d$	1.6	1.6		0.9	0.5	0.6
	EKY {AK	32	27	27	50	243	240
	EKY {PA	21	17	21	58	58	154
	Beats/second	2.5	1.2	3.0	3.0	1.3	1.6
No. 4	BP {BP	71	68	92	115	100	89
	BP {PP	147	90	95	90	126	114
	$\Delta p/\Delta d$	0.9	1.2		1.2	1.0	0.9
	EKY {AK	161	78	85	77	134	131
	EKY {PA	32	32	40	116	104	80
	Beats/second	1.1	1.4	1.6	1.5	0.8	0.9
No. 5	BP {BP	84	70	69	77	91	85
	BP {PP	100	77	77	70	95	95
	$\Delta p/\Delta d$*	1.1	3.0		2.8	3.2	1.5
	EKY {AK*	88	25	29	25	30	61
	EKY {PA	95	25	18	106	100	155
	Beats/second	1.3	1.3	1.6	2.0	1.0	1.2
No. 6	BP {BP	64	65	73	100	106	100
	BP {PP	80	60	60	60	100	113
	$\Delta p/\Delta d$	1.5	1.9		1.1	0.9	1.1
	EKY {AK	52	32	42	54	116	106
	EKY {PA	82	64	75	230	160	136
	Beats/second	1.4	1.7	2.0	1.9	1.1	1.0
No. 7	BP {BP	87	83	87	83	105	111
	BP {PP	100	50	50	50	89	115
	$\Delta p/\Delta d$	0.8	0.9		0.9	0.6	1.0
	EKY {AK	128	56	56	56	143	119
	EKY {PA	51	33	29	67	51	51
	Beats/second	2.4	2.4	2.5	2.3	1.6	1.3
No. 8	BP {BP	94	86	78	90	115	115
	BP {PP	100	78	78	61	100	144
	$\Delta p/\Delta d$*	1.5	1.5		1.0	2.0	2.7
	EKY {AK*	66	53	44	58	49	53
	EKY {PA	90	70	50	87	145	117
	Beats/second	1.7	1.9	2.0	2.0	1.2	1.5

* The procedure employed in obtaining these figures was technically faulty (see text).

Ten healthy subjects, aged 20 to 30. Amplitude of electrokymographically recorded pulsations of the aortic knob (AK) and pulmonary artery (PA) during and after voluntary straining expressed in terms of percentage of control level. Diastolic pressure (DBP) and pulse pressure, simultaneously recorded from the brachial artery, are also expressed in terms of percentage of control level. Ratio of pulse pressure (Δp) to recorded amplitude of aortic pulsation (Δd) and beats/second are also shown.

TABLE III—*Continued*

Subject	Recording	Straining period			Post-straining period		
		4″	8″	12″	1″	4″	8″
No. 9	BP BP	78	67	72	90	96	85
	BP PP	91	82	78	71	107	107
	$\Delta p/\Delta d$	1.5	1.8		1.0	0.6	0.7
	EKY AK	61	44	53	74	182	158
	EKY PA	68	84	84	111	121	121
	Beats/second	1.5	1.7	1.9	1.9	1.7	1.4
No. 10	BP BP	80	68	96	104	105	101
	BP PP	94	77	58	110	135	135
	$\Delta p/\Delta d$	0.9	1.0		1.6	1.0	1.1
	EKY AK	100	75	75	69	137	118
	EKY PA	85	90	80	110	95	100
	Beats/second	1.2	1.4	1.6	1.7	1.2	1.2
Median all subjects	BP BP	78	69	76	92	106	99
	BP PP	94	77	59	64	104	114
	$\Delta p/\Delta d$	1.25	1.6		1.05	0.8	0.95
	EKY AK	77	49	43	57	140	125
	EKY PA	76	56	45	110	104	118
	Beats/second	1.7	1.8	2.1	1.9	1.1	1.25

bronchial pressure, and continued until straining ceased. Thereafter, the amplitude increased gradually for the first three or four beats, then rapidly increased to levels higher than in the control period. A pattern of this type was observed in eight of ten subjects. In two subjects (Nos. 5 and 8, see Table III), the amplitude after relaxation did not return to control levels. A technical error, to be discussed below, probably accounted for this.

Arterial pulse pressure

In measurements from the brachial artery, simultaneously recorded, the pulse pressure variations before, during, and after the straining procedure were, in general, parallel to the variations of aortic pulsation amplitude. The characteristic pattern of a fall in pulse pressure during the period of straining, a gradual rise after relaxation, followed by a wide pulse pressure in the later post-straining period, was observed in eight of the ten subjects. In the other two subjects (Nos. 2 and 5) the pattern was atypical only in that the amplitude in the post-straining period reached, but did not exceed, that of the control period.

Diastolic blood pressure

The diastolic blood pressure rose abruptly with the increase in intrabronchial pressure, and fell when straining ceased. This phenomenon was the result of the transmission to the recording apparatus of pressure derived from the activity of the thoracic and abdominal muscles in raising intrabronchial pressure, and had no direct relation to that component of the recorded pressure derived from the pumping action of the heart. Under these circumstances it is customary to designate the recorded pressure as the *gross* pressure. Subtraction of the intrabronchial pressure from the gross pressure gives the *net* pressure, or that component of the recorded pressure which is dependent upon the activity of the heart. Note that no such correction was necessary for the electrokymographic recordings, since the intrathoracic pressure changes were exerted equally upon both sides of the vessels.

The net diastolic pressure fell rapidly during the first few seconds of straining in all individuals. A considerable rise toward or even above the control level was observed in a few individuals in the late period of straining; in others the fall was sustained. The first beat or two after straining showed little change. A rise above the control level then occurred in eight of the ten subjects.

Pulse rate

From the onset of straining the pulse rate steadily increased. Tachycardia persisted for several beats after relaxation, followed by the abrupt onset of bradycardia.

Pulmonary artery pulsations

Electrokymographic tracings from the pulmonary artery during the period of straining followed the same general pattern as was recorded from the aorta. A reduction in amplitude occurred in all subjects. At the moment of relaxation an important difference was characteristic. A large increase in amplitude regularly appeared immediately upon the return of intrabronchial pressure to the atmospheric level. The later rise above the control level was neither as great nor as consistent as in the aortic recordings. The electrokymographic records from the pulmonary artery resemble the pressure curves recorded by Lauson, Bloomfield and Cournand (10) and by Cournand (11) from the pulmonary artery during a similar straining procedure.

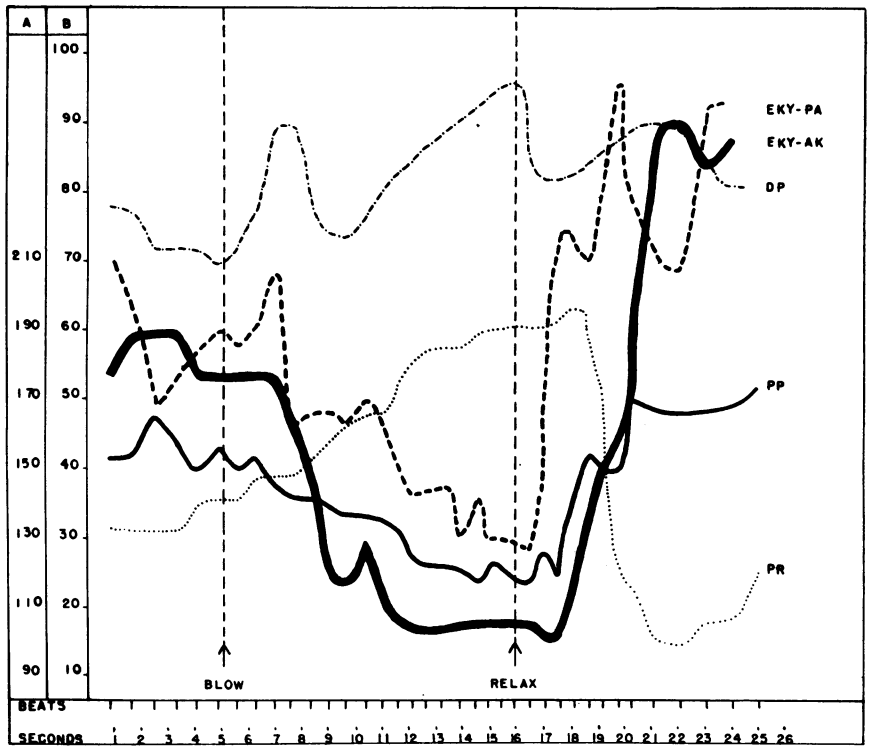

FIG. 6

Subject No. 1. Beat-by-beat amplitude of recorded pulsations of the aortic knob (AK) in mm. (scale B), simultaneously recorded with pulse pressure (PP) and gross diastolic pressure (DBP) in mm. Hg (scale B) and pulse rate (PR) in beats/minute (scale A). Amplitude of pulsations from the pulmonary artery (PA) during a repetition of the straining procedure is shown to illustrate especially the characteristic response in the immediate post-straining period.

Beat-by-beat graphic records from one subject of the magnitude of aortic pulsation with reference to simultaneously recorded pulse pressure, diastolic pressure, and intrathoracic pressure are shown in Figure 6. A similar record of the magnitude of pulmonary artery pulsation of the same individual during a repetition of the straining procedure is included especially to illustrate the major difference between aortic and pulmonary artery response in the immediate post-straining period.

Ballistocardiographic records

Most of the ballistocardiographic records of stroke volume in the sitting position during straining were technically unsatisfactory. The muscular activity necessary to increase voluntarily intrathoracic pressure invariably introduced artefacts into the records. A reduction of complex amplitude was occasionally discernible, followed by large complexes when straining ceased (Figure 5). Application of the Starr formula for determination of stroke volume (12) was rarely possible. Previous ballistocardiographic studies of the stroke volume during straining (13–15) are not pertinent to this problem since the subjects were in the recumbent posture, whereas our subjects were in the sitting posture.

Analysis of results

Stroke change in relative diameter of the aorta and pulmonary artery could be accurately measured by the electrokymographic method if (*1*) the recorded curves bore a direct, linear relation to the actual movements of the vessel wall, and (*2*) if the pulsatile change in position of the vessel wall were a true measure of change in vessel diameter. From the data available it is

possible to estimate the degree to which we have approached this ideal.

The relation of recorded curves to vessel wall movements

The response characteristics of the recording apparatus introduced the characteristic imperfections of overdamping. From a study of the frequency response, shown in Table I, it is apparent that this factor must have had an important influence upon recorded wave shape. For example, it was shown that at a frequency of 10/sec., a sine wave of fixed magnitude is recorded only 33% as large as at frequency of 1/sec. If the wave manifested in the dicrotic notch were a sine wave with duration of 0.1 sec., the recorded amplitude would be 33% of that which should have been recorded. Since the wave is actually complex, and usually of shorter duration than 0.1 sec., the magnitude of recorded error is even greater. Other components of the wave pattern are undoubtedly at frequencies of 20/sec. or more. Thus, the finer details of wave shape were smoothed out in the recorded curves and the peaks of the major components were flattened. Some of the differences in wave shape between the recorded aortic curves and the brachial pulse curves (*e.g.:* curves during the late straining period in Figure 1) cannot be explained on the basis of overdamping of the apparatus. Actual differences in wave forms of the two vessels must have been a factor of appreciable importance.

For the *comparison* of relative amplitudes of recorded waves during the various periods of the straining procedure, overdamping of the degree indicated in Table I should not be expected to introduce a large discrepancy. The duration of the ejection phase of systole in the normal individual with a pulse rate of 1/sec. is 0.25 sec. (16). The curve approximates a half sine wave, therefore the sine wave duration would be 0.5 sec. or a frequency of 2.0/sec. By a similar calculation, the sine wave frequency of systolic ejection at a pulse rate of 2/sec. is 3.0/sec. Although figures are not given, by extrapolation of the curves, the frequency at a pulse rate of 3/sec. is calculated at about 4.0/sec. By calculation from data provided in Table I, it is found that a loss of 19% in recorded amplitude results from a frequency change of this magnitude. Insofar as the wave patterns of systolic ejection differ in shape from a half sine wave, the loss would be somewhat greater. Since systole ends before the inscription of the last portion of the half sine wave, the loss would be somewhat less. It seems justified to conclude that in these studies, the additional loss in recorded amplitude resulting from the increased pulse rate itself was not greater than 30%, and that amplitude changes of this magnitude, or even of considerably less than this magnitude when pulse rate was taken into consideration, could be considered significant.

In Table II it was shown that the sensitivity of the middle third of the phototube slot was approximately linear. It was usually easy to so place the subject that the shadow of the vessel wall remained in this zone throughout the straining procedure. Hence, non-linearity of the receptor apparatus was not ordinarily an important source of error. Reference was made above to atypical aortic response patterns in subjects Nos. 5 and 8 (see Table III). In reexamining these tracings, it was found that the individual wave patterns were distinctly bizarre in form, with long, flat crests. This pattern was not observed in any other individual studied. It was subsequently demonstrated that similar waves could be produced by allowing the shadow of the pulsating vessel to extend in systole beyond the sensitive portion of the receptor slot. The unusual results in these two individuals are therefore attributed to this technical error. Such faulty technique should be readily avoided and easily recognized when it occurs.

The relation of vessel wall movements to stroke change in diameter

This problem is difficult to subject to objective, quantitative study. Presumptive evidence is offered in several observations. If vessel expansion and contraction were equal in all diameters and the center of the vessel remained fixed, as in Figure 7A, motion of the wall would be in exact proportion to the change in vessel diameter. Even if one side of the vessel were fixed, as in 7B, the same would hold true. If, however, the center of the vessel moved erratically, as in 7C, movement of the vessel wall would be a very inexact measure of the diameter change. Several

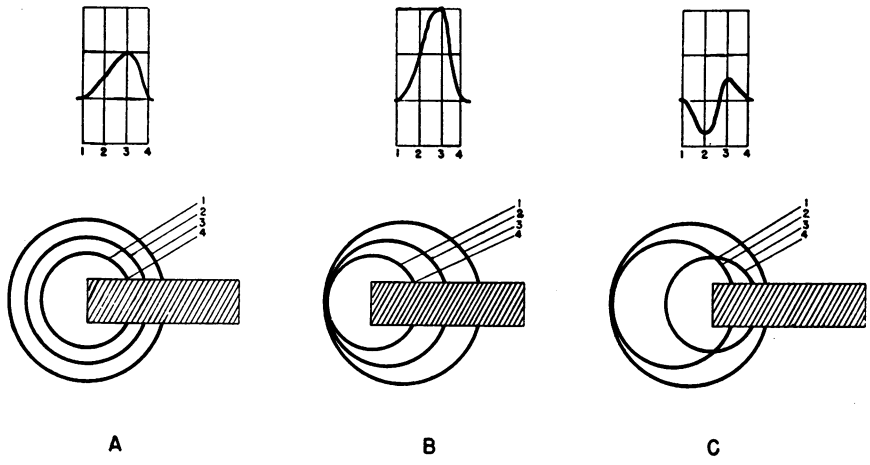

FIG. 7. THE EFFECTS OF LATERAL DISPLACEMENT OF A VESSEL UPON THE CONTOUR OF RECORDED CURVES (UPPER FIGURES)

The cross-hatched rectangle represents the slot of the phototube. Numbers indicate position of the vessel wall at various moments of the pulse cycle.

observations suggest that lateral displacement of the entire vessel was not a major factor in the pulse waves recorded. (*a*) Respiratory movement undoubtedly causes lateral movement, but this is slow, tending to cause displacement of the entire baseline, which is continuously compensated by the time constant of the apparatus and hence does not greatly influence the amplitude or shape of the individual complexes. In our studies respiration was arrested during the recording period. (*b*) Rocking motions of the base of the heart might be expected to cause rhythmic lateral displacements of the great vessels. In assessing this factor, a comparison of curves from the left side of the aortic knob with those from the right side of the ascending aorta is of little value, since the differences between the records can evidently be accounted for by the effects of superior venacaval pulsations upon the records from the ascending aorta (6, 7). However, the curves recorded from the aortic knob and from the pulmonary artery invariably show changes in amplitude which correspond qualitatively and to a considerable degree quantitatively with changes in pulse pressure in the respective vessels. If lateral displacement were an important factor, wave motions completely unrelated to pressure changes would be expected. Furthermore, pulsations of large amplitude were regularly recorded from the pulmonary artery in the immediate post-straining period, during which aortic pulsations were very small. Hence, the motions of the two vessels appear to be independent, each related to the pressure change within the respective vessel, and not a function of a rocking motion of the base of the heart.

In view of these observations, it seems reasonable to conclude that the electrokymographic tracings recorded from the pulmonary artery and aortic knob provide a measure of the relative diameter changes of these vessels resulting from changes in intrinsic vascular pressure. It is obvious that changes in pulse pressure (Δp) should result in changes in aortic cross sectional diameter (Δd). In our studies this was found to be true, but the ratio of pulse pressure to aortic pulsation magnitude ($\Delta p/\Delta d$) varied by as much as 50% in the separate beats of a single individual during a straining procedure (see Table III).

The relation of aortic volume change (Δv) to pressure change (Δp) has been demonstrated by Hallock and Benson (16) in studies of excised human aortas. In individuals of the age group of our subjects (20–30), $\Delta p/\Delta v = k$ where pressures are in the physiologic range. The expression $(\Delta v/\Delta p)V = k$, as employed by Wiggers (17), is essentially the same, since V is a fixed value for each individual tested: namely, the aortic volume at a specified mean pressure.

Katz and his associates (18) found considerable discrepancy between the pressure-volume relationships of the aortas of intact, living dogs

as compared with the same aortas after death. They injected diodrast directly into the aortas and computed aortic volume from the X-ray shadows of films taken at varying levels of blood pressure. Wiggers and Wégria (19) measured changes in aortic diameter directly, with a clamp aortagraph. As in our studies, $\Delta p/\Delta d$ relationships in the separate beats of a single subject showed considerable variation. An inspection of their illustrations shows $\Delta p/\Delta d$ to be 1.0 and 1.4 in beats (y and y of Figure 4, Wiggers and Wégria [19]) of the same approximate pulse pressure and diastolic blood pressures, measured by the technique which we have employed. One would not expect pressure-diameter relationships to exactly parallel pressure-volume relationships. A rough estimate of aortic diameter change to aortic volume change may be deduced as follows:

(*1*) The relation of aortic diameter change to aortic cross sectional area change approaches linearity. If aortic diameter were 2.0 cm. at a given diastolic pressure, and a unit of systolic pressure increment increased the diameter to 2.1 cm., the resulting increase in aortic area is from 1.0π cm.^{2} to 1.1025π cm.^{2}. An area change of ten times this magnitude (1.0π cm.^{2} to 2.025π cm.^{2}) is accomplished with a diameter change to 2.846 cm. Therefore, within the physiologic range specified, diameter change is a linear function of area change within the degree of error in the equation $.1/1 = .846/10$, or about 15%.

(*2*) Area change would be a linear function of volume change if lengthening were not a factor, but the latter is actually of relatively minor importance. Thus, if in increasing the aortic area from 1.0π cm.^{2} to 2.0π cm.^{2}, the aorta were increased in length from 100 cm. to 106 cm., area would be a linear function of volume within the margin of error in the equation $100/1 = 212/2$, or about 6%.

(*3*) Thus, change in aortic diameter (Δd) appears to be a reasonable approximate linear function of change in aortic volume (Δv). Thus, if $\Delta p/\Delta v = k$, then $\Delta p/\Delta d = k$ should be reasonably accurate.

From the investigations of others, cited above, a roughly linear relationship between change of pressure and change of diameter should have been expected in our subjects. It will be noted that the value of $\Delta p/\Delta d$ is consistently relatively low during the late post-straining period and high during the period of straining. The pulse rate is seen to parallel the values of $\Delta p/\Delta d$. As shown above, the recorded wave amplitude is seen to bear a direct relationship to the pulse rate because of the technical characteristics of the recording apparatus. This technical defect exerts an influence in the direction of the results. The range in results of $\pm$ 50% in the ratio $\Delta p/\Delta d = k$ is beyond the estimated maximum of 30% error in the response characteristics of the apparatus. A description of certain alterations of the instrument which have resulted in improved linearity of frequency response is now in preparation (20). With such a correction, and with allowances for the differences in wave forms in the aorta as compared with the brachial artery, it seems probable that the variations of $\Delta p/\Delta d$ ratio which we have recorded during acute circulatory disturbance reasonably approximate the actual pressure-volume relationships in the aortas of the individuals studied.

Discussion of the physiologic response to intrathoracic pressure change

The effects of transient increase of intrathoracic pressure, produced by voluntary straining, upon cardiovascular dynamics have been studied intensively by many methods (10, 11, 13, 21–29). The differential pressures in the various heart chambers and great vessels, stroke volume, the distribution of blood mass, and the pulse rate have been shown to be influenced. Interpretation of these data has led to the following conclusions. Increase in intrathoracic pressure is accompanied by a comparable increase in intra-abdominal pressure and results in: (*a*) An immediate reduction of blood return to the heart from the periphery, with resulting increase in peripheral venous pressure. The blood return from the abdominal viscera is little if at all impeded. (*b*) The right heart output is consequently reduced with resulting fall in net pressure in the right ventricle and pulmonary artery. (*c*) The pulmonary blood reservoir is progressively exhausted with reduction in opacity to roentgen rays and subsequent reduction of blood return to the left heart chambers. This is followed by (*d*) reduced stroke volume of the left ventricle and by reduced pulse pressure and net systolic and diastolic pressure in the systemic circulation with reflex peripheral vasoconstriction and tachycardia. The return of intrathoracic pressure to the atmospheric level is accompanied by a rush of blood into the thoraco-abdominal cavity and right auricle, a sudden marked increase in right heart

output and in pulmonary artery pulse pressure and blood pressure, restoration of the pulmonary blood volume and blood return to the left heart, and restoration of stroke volume, pulse pressure, and systemic blood pressure. In the post-straining period a transient hypertension is normally observed as the result of normal or above-normal cardiac output and persisting peripheral vasoconstriction. A sudden bradycardia regularly occurs as the aortic reflexes respond to this stimulus. All of the cardiovascular functions characteristically return to normal within one or two minutes.

From a careful examination of electrokymographic records alone, most of these physiologic disturbances can be deduced.

SUMMARY

1. Pulsatile movements of the aorta and pulmonary artery were electrokymographically recorded before, during, and after increased intrathoracic pressure produced by voluntary straining. The records obtained were compared with simultaneously recorded brachial artery pressure and with the reported observations of pulmonary artery pressure by others. Ballistocardiography as a control procedure was found to be unsatisfactory.

2. The response characteristics of the electrokymographic apparatus were studied as a preliminary procedure. The sensitivity of the various zones of the receptive slot of the phototube was found to be approximately linear. The frequency response of the apparatus resulted in the characteristic wave shape deformities of an overdamped system. During the various phases of the straining procedure, comparative recorded wave amplitude was sufficiently influenced by this technical factor to necessitate a correction of results for pulse rate.

3. A measure of relative stroke change in diameter of the aorta and pulmonary artery was recorded. Qualitatively these changes were respectively parallel to coincident changes in pulse pressure in the brachial artery in control studies and pulmonary artery in the reported observations of others.

4. The recorded amplitude of aortic pulsations, after correction for pulse rate, bore a quantitative relationship to pulse pressure approximating that which would be expected between aortic diameter change and pulse pressure.

CONCLUSIONS

From a study of the movements of the aorta and pulmonary artery by the electrokymographic method, most of the cardiovascular disturbances produced by straining may be deduced. Although the measurements are quantitative only in a relative sense, the dynamic changes in the circulation are clearly recorded. This method may provide a useful substitute for direct manometric measurements with catheters in studies in which absolute quantitative measurements of intraaortic and intrapulmonic artery pressures are not necessary. It is a rapid, painless, and easily performed procedure suited to the study of the effects of a cardiovascular stimulus upon each of a large group of subjects. Certain limitations of the value of this method, imposed by the response characteristics of the apparatus in its present form, have been described. From the accuracy with which the nature of the cardiovascular disturbances produced by straining may be deduced from the electrokymographic records alone, the possible application of this method to other studies is implied.

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