A Study of the Central and Peripheral Arterial Pressure Pulse in Man
Correlation with Simultaneously Recorded Electrokymograms

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This communication describes and discusses the results obtained in studying central and peripheral arterial pressure pulses in man by means of retrograde aortic catheterization. The pressure pulse transformation and pulse transmission in the aorticbrachial system are presented and compared with comparable tracings reported in the dog. Several normal subjects and patients with hypertension, aortic regurgitation and coarctation of the aorta were studied. The genesis of the central pulse contour in aortic regurgitation is reconsidered. The changes in central pulse pressure, produced by the Valsalva-like maneuver, and ventricular premature systoles have been investigated, especially in their relationship to cycle length, end-diastolic volume and peripheral resistance. The technic of obtaining pulse pressure recordings by retrograde aortic catheterization is recommended for determining the location, size and dynamic significance of coarctation of the aorta. As an additional study, aortic densograms and border electrokymograms from the aortic arch and left ventricular border were correlated with simultaneously recorded central aortic pressure tracings.

Catheterization of the human aorta has been used recently for diagnostic purposes in retrograde angiocardiography. This experience prompted the study of cardiovascular hemodynamics by means of retrograde catheterization of the human aorta, a field which has been little explored. This report deals with some preliminary observations on the contour, transmission rate and transformation of the central aortic pressure pulse. As part of this investigation the central pressure pulse was correlated with the simultaneously recorded electrokymogram of the aorta.

Methods and Procedure

Simultaneous aortic catheterization and electrokymography were performed in patients for whom the information so gained was thought to be diagnostically important. They are listed in table 1 with their final clinical diagnosis: cases 2 and 3 were shown later not to have cardiovascular disease.

1. Aortic Catheterization. The brachial artery was identified just above the antecubital fossa; the skin overlying it was prepared in the usual fashion for sterile surgery and was anesthetized locally. Through a small transverse skin incision, the brachial artery and vein were identified and exposed by incising the lacerus fibrosus. The vessels were separated and two temporary No. 1 black silk loop ligatures placed around the artery about 1 cm. apart in such a manner that when the loose ends were elevated, the vessel was temporarily occluded. A test occlusion of the artery was then performed and signs of ischemia sought for peripherally. A longitudinal incision approximately 2 mm. in length was then made in the artery between the taut ligatures and a No. 4–6 French catheter was inserted into this opening. Hemostasis was completely maintained by means of the ligatures on the artery. Once in place the ligatures could be loosened, the artery sealing off the opening around the catheter. The catheter was then passed under constant fluoroscopic control into the arch of the aorta, its tip being oriented in a caudal direction. After withdrawal of the catheter at the end of the observations the incision was repaired, with the stay ligatures secure, using No. 00000 arterial silk as a simple, full thickness, continuous suture to obliterate the opening. Approximately four to six sutures were usually required for

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hemostasis. The stay ligatures were then removed and the skin closed. Penicillin was given over a 48 hour period postoperatively.

It was noted that the catheter passed more readily into the aorta when the left brachial artery was used. Following the procedure the radial pulse was permanently abolished in one case, moderately reduced in two, and only slightly reduced in the remaining four; in none of these cases was there any evidence of impaired circulation to the extremity. None of the seven cases exhibited any type of cardiac arrhythmia during the procedure, nor were there any untoward reactions.

2. **Electrokymography and Pulse Recording.** Records were obtained with the patient in the recumbent position holding his breath in midinspiration as in our other studies. Intraluminal pressures were recorded with an electromanometer. The kymographic apparatus employed was essentially that developed by Henny and Boone as modified by Luisada and co-workers and Grossman and Tiger.

Simultaneous recordings of lead II of the electrocardiogram, intra-arterial pressures and the electrokymogram were made with a four channel direct-writing polyoscillograph. The recording fidelity of the electrokymographic equipment has been described elsewhere. The galvanometer response to a square wave input (damping 71 per cent critical) was 95 per cent complete in 0.01 second. The time lag of an impulse through the catheter at 37 C. has been found to be 0.015 second. The electrokymograms were obtained with the slit of the pickup device positioned perpendicular to the border of the aorta and within 4 to 6 cm. of the catheter tip in recording border tracings. Densograms were recorded similarly, except that the slit was placed completely within the visible shadow of the aorta and parallel to the indwelling catheter.

**Discussion of Results**

**A. Contour of Pressure Pulses**

1. **Central Aortic Pressure Pulses.** The normal pulse wave contour, a typical example of which (from case 2) is shown in figure 1, is essentially similar to that in the dog, showing the three fundamental changes of gradient (at points 3, 4, 5). By analogy with the pulse curves in the dog, the small vibrations 1 and 2 may be considered to be due to the contracting auricles and the bulging of the aortic valves respectively. In support of this genesis of vibration 1 may be cited its constant relationship,

whenever it appears, to the end of the P wave. Vibration 2, approximately 0.03 second in duration, is rather constant in appearance and immediately precedes the onset of the anacrotic limb. The deformation of the pulse contour at point 6 is probably the reflected dicrotic wave, and the form of the summit (point 4) may be due to a reflected wave superimposed upon the fundamental form.

In arterial hypertension, the characteristic contour seen is similar to that which occurs in the dog, with a high resistance systemic arterial circuit. Figure 2A is the aortic pulse of a rela-

![Fig. 1. Normal central aortic pressure pulse (case 2). Aortic pressure pulse curve above, lead II of electrocardiogram below, simultaneously recorded. Paper speed: 25 mm. per second. Time lines are 0.04 second apart. See text for discussion and meaning of numbered points.](image-url)

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† The pressure levels obtained in these cases are given in table 1.
1 on the anacrotic limb especially marked in the beat following a ventricular premature systole in figure 2B where it also occurs lower down on the curve. This early notch is also seen in experimental regurgitation. The cause for this phenomenon may be an orifice relatively restricted for the greatly increased systolic output. In aortic regurgitation the pulse shows a steep rise to an early and rather peaked summit, followed by a marked systolic collapse. In the absence of a clearly defined incisura, the beginning of diastole cannot be precisely determined, but the steep early diastolic collapse is

evident. In figure 2B where a change in gradient (point 2) probably marks the end of the incisura, this collapse can be most readily seen. The pressure declines more gradually during the remainder of diastole (but more so than is seen experimentally). The ventricular prema-

<table>
<thead>
<tr>
<th>Case</th>
<th>Diagnosis</th>
<th>Age</th>
<th>Sex</th>
<th>Central Aortic Pressure (Average)</th>
<th>Brachial Artery Pressure (Cuff Method)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Labile hypertension</td>
<td>32</td>
<td>female</td>
<td>135/70</td>
<td>120/70</td>
</tr>
<tr>
<td>2</td>
<td>Normal: no cardiovascular disease</td>
<td>29</td>
<td>female</td>
<td>110/75</td>
<td>115/75</td>
</tr>
<tr>
<td>3</td>
<td>Normal: no cardiovascular disease</td>
<td>31</td>
<td>male</td>
<td>120/65</td>
<td>110/70</td>
</tr>
<tr>
<td>4</td>
<td>Mild hypertension</td>
<td>53</td>
<td>female</td>
<td>155/100</td>
<td>160/90</td>
</tr>
<tr>
<td>5</td>
<td>Coarctation of the aorta</td>
<td>42</td>
<td>female</td>
<td>180/85</td>
<td>192/86</td>
</tr>
<tr>
<td>6</td>
<td>Essential hypertension. Arteriosclerotic heart disease</td>
<td>64</td>
<td>male</td>
<td>170/95</td>
<td>165/100</td>
</tr>
<tr>
<td>7</td>
<td>Aortic regurgitation</td>
<td>47</td>
<td>female</td>
<td>235/60</td>
<td>180/60</td>
</tr>
</tbody>
</table>

* Not recorded simultaneously with central aortic pressure.

Fig. 2A. Central aortic pressure pulse in hypertensive patient (case 6). B. Central aortic pressure pulse of patient with aortic regurgitation, showing ventricular premature systoles (case 7). Conventions as in figure 1, except recorded at double paper speed. See text for discussion and meaning of 1, 2 and 3 in segment B.
a large leak, which was in confirmation of other clinical data.

Wiggers has explained the rapid systolic collapse in aortic regurgitation as the consequence of a redistribution of ejection so that most of the blood is discharged during the first half of the ejection phase. In addition, he has emphasized that the high systolic pressure reached at the peak contributes to the sharp drop. In our patient, the almost identical, though miniature, contour of the pulse curve recorded in the ventricular premature systole (fig. 2B) is difficult to explain on the above basis, since it resembles the contour of the sinus beats closely. The anacrotic limb is less steep, the pulse pressure smaller, the systolic peak level lower, and it does not clearly show the anacrotic notch described (point 1, fig. 3). But the systolic (point 3, fig. 2B) and early diastolic collapse segments show a striking similarity to the corresponding portion of the sinus beat, with only slight differences in gradient. Since the absolute level of blood pressure and the initial ventricular volume (and hence the rate of ejection) of the premature contraction are decidedly less than the sinus beat, and yet the rapid collapse occurs, factors other than, or in addition to, those postulated by Wiggers (in the animal) must be operative in its genesis.

When recordings were made in a patient with a coarctation of the aorta of mild degree (case 5, figs. 3A and 3B), several changes occurred in the form of the aortic pressure pulse both above and below the region of coarctation. Above the coarcted area, the pulse contour shows only minor changes from the normal which are probably due to hypertension. (Other effects of coarctation on the pulse contour which might be expected are absent, probably because of the mild degree of the stenosis.) Just beyond the area of coarctation the changes in pulse form (compare fig. 3B with fig. 3A) consist of: (1) a decidedly less steep anacrotic limb rising protractedly to a late summit; (2) a distinct vibration appearing less than halfway up the anacrotic limb; (3) coarse systolic vibrations of rather low frequency (about 50 per second) on and at the summit of the curve; (4) a slight diminution of the pulse pressure, the systolic pressure falling more than the diastolic. These alterations in the pressure pulse are similar to those seen beyond the stenosis in dogs in whom the aorta was narrowed just above the aortic ring. In these animal experiments the more marked the degree of stenosis, the sooner vibration 1 (fig. 3B) appeared on the anacrotic limb, the more reduced was the anacrotic gradient preceding the vibration, the smaller were the vibrations at the summit, and the more marked the systolic pressure drop, the diastolic level remaining essentially unchanged. Judging by these criteria, the contour of the poststenotic pressure pulse indicates a narrowing of mild degree, a circumstance suspected on clinical grounds. The onset of the anacrotic limb of the poststenotic pulse curve precedes that recorded proximally by about 0.03 second. The pressure rise immediately following anacrotic halt coincides with the steep rise of the anacrotic limb of the prestenotic pressure pulse (as it is propagated peripherally). The mechanism responsible for the apparent accelerated onset of the poststenotic pulse wave is obscure, but may be related to the collateral circulation which enters below the stenosis. The dicrotic

![Graph](http://circ.ahajournals.org/)

**Fig. 3.** Central aortic pressure pulse above (A) and below (B) the site of coarctation (case 5). B was recorded within a few seconds of A. Conventions as in figure 2. I in segment B indicates the anacrotic notch. Discussed in text.
notch becomes larger in amplitude and shorter in duration distal to the coarctation. The femoral pulse contour in human coarctation of the aorta has recently been studied.\textsuperscript{15}

The recording of pressure pulses above and, if possible, below the site of coarctation can be used both qualitatively and quantitatively in the diagnosis of human coarctation, using the criteria discussed above to localize it precisely.

![Blood Pressure Graphs](http://circ.ahajournals.org/)

**Fig. 4.** Central aortic pulse tracing during Valsalva maneuver in case 6. Beginning, middle and end of maneuver are in segments 1, 2 and 3. Note electrocardiograph turned off before last pulse in segment 1. End of maneuver is marked in segment 3. Note ventricular premature systole with no evidence of its activity in the pulse tracing in segment 3. Conventions as in figure 1. Discussed in text.

and determine its size and dynamic significance. This should provide more reliable data than routine methods now employed in the consideration of surgical intervention. It may also prove of value in determining the nature of hypertension encountered in this anomaly. The subject has recently been analyzed in detail by Gupta and Wiggers.\textsuperscript{16}

2. Valsalva Maneuver. In two instances the central arterial pulse was recorded while the patient exhaled against a positive pressure of about 30 mm. Hg. Figure 4 shows representative sections from the beginning (strip 1), middle (strip 2) and end (strip 3) of the maneuver in one case. The blood pressure changed from 170/90 mm. Hg at the onset to 63/50 at the middle and 94/50 at the end of the maneuver. The ejection phases measure 0.27, 0.17 and 0.27 second for the corresponding cycles.\textsuperscript{*} The cycle lengths in these instances are 0.80, 0.68 and 0.66 second, respectively. The contour of the pulse is altered decidedly during the Valsalva maneuver: the anacrotic limb begins 0.04 second later and rises less steeply; the summit is

\begin{itemize}
  \item [\textsuperscript{*}]: The ejection phase was measured from the beginning of the rise of the anacrotic limb to the end of the incisura. While this period includes the protodiastolic phase, it has a sharper definition and serves the purpose of a relative measure of change.
\end{itemize}
more flattened and a prominent dicrotic wave appears. The circulatory response to the Valsalva maneuver in various conditions is considered in detail elsewhere.17

3. Premature Beats. During the occurrence of ventricular premature systoles in the electrocardiogram, in case 6, the record of the pulse wave (fig. 5) illustrates several unusual circumstances: (1) mechanical evidence of the premature contraction is absent in the central pulse (see also fig. 6); (2) the ejection phase of the beat following the compensatory pause (that is, after a mechanical cycle length of 0.80 second) is almost identical with that of the normal cycle of 0.40 second (see below); (3) the systolic pressures during and the diastolic pressures at the end of the beats are almost identical whether the beat follows a double or a single cycle interval; (4) the dicrotic limb declines very gradually and continuously during the long pause, until the next mechanical systole. Unfortunately information about the ejection of the right side of the heart during the “dropped” mechanical beat of the left side is not available.

4. Changes in Duration of Ejection Phase. Based on detailed animal experiments, Wiggers and Katz18 have described three primary influences on the ventricular ejection phase:
   (a) Increased initial volume (without change in heart rate and blood pressure) prolongs the ejection phase.
   (b) Increased resistance to emptying (constant heart rate and relatively unchanged controlled blood pressure), produced by clamping the aorta at the level of the diaphragm, results in a shorter ejection phase in normal hearts. They found that the hypodynamic heart subjected to increased resistance to its emptying...
CENTRAL AND PERIPHERAL ARTERIAL PRESSURE

could not increase the velocity of ejection so that the systolic discharge was reduced, diastolic distension increased and thus the ejection period ultimately actually lengthened.

(c) Heart rate leads to no definite and constant relationship between the duration of consecutive ejection periods and that of their preceeding diastole. This last seems to hold true for the human heart also, at least on occasion (see fig. 7), inasmuch as the ejection phase is of the same duration during the normal cycle as it is following the compensatory pause during which the mechanical cycle is equal to twice the normal duration.

From the records of the Valsalva experiments it is seen that the length of the ejection phase varies directly with the height of the blood pressure. Though seemingly at variance with the experimental data in animals, it is explained by the fact that the blood pressure changes are determined by the filling of the left heart. Thus the reduced filling of the left heart causes a drop in blood pressure and at the same time by reducing the initial (end-diastolic) volume, shortens the ejection phase. As filling of the left ventricle increases again and contributes to the blood pressure rise, initial volume rises and the ejection phase is lengthened again in accordance with experimental data.

B. Transmission of the Central Pulse to the Periphery

The transmission time and velocity have been extensively investigated with volume pulses and it has been found that the arterial pulse wave velocity varies directly with the rigidity of the arterial wall, traveling more slowly in progressing from the central (elastic) vessels to the peripheral (muscular) arteries. It also varies directly with the intravascular pressure and the degree of smooth muscle contractions of its wall. However, data on pulse velocity in man from the aorta peripherally have been lacking.*

* While this manuscript was being prepared, an abstract appeared (T. G. Schnabel, W. J. Rashkind...
Using the peak of the R wave of the electrocardiogram, measuring its distance from the rise of the anacrotic limb of the corresponding pressure pulse centrally and peripherally and dividing their difference by the distance measured with the indwelling catheter, the pulse velocity was calculated in three patients.* Table 2 gives the values obtained. The speeds found confirm the striking difference in pulse wave velocity in the central as contrasted with the peripheral blood vessels. Planimetry shows no significant change in mean pressure between the central and peripheral pulses in cases 3 and 5. In case 7, the central aortic and brachial artery pressure measurements (about 30 seconds apart) were quite different and there had been a marked reduction in mean pressure also; apparently a partial occlusion of the catheter had occurred in the interval.

C. Transformation of the Central Pressure Pulse

This has been extensively studied in the dog10, 11, 21 ever since Otto Frank developed an adequate manometer with which he could give conclusive evidence for discrepancies in pulse form in different portions of the arterial tree.20 We had occasion to observe the transformation of the intravascularly recorded central aortic pulse in the transmission to the brachial artery in four adult men. Figure 6 shows pressure pulse curves recorded from the arch of the aorta, the axillary artery and the brachial artery within 15 seconds of each other in a normal young adult, superimposed to utilize the same time and pressure scales. The surface areas of each of these curves are practically identical. In figure 7A, B and C are recorded curves from the same individual in the same areas at another time at a slower speed. The following changes are seen: (1) the onset of the anacrotic limb occurs later as the pulse is transmitted peripherally; (2) starting somewhere in the axillary artery, the systolic pressure becomes progressively higher; (3) the anacrotic gradient change gradually disappears along its transmission in the axillary artery, until the anacrotic limb rises smoothly and more steeply to the systolic peak; (4) beyond the first recorded position in the aortic arch, all systolic peaks occur at the same time in relationship to the peak of the R wave of the electrocardiogram (preceding the centrally recorded systolic peak); (5) the inesura, prominent centrally, is gradually lost and is replaced by a diastolic dip ending in the dicrotic notch and followed by a dicrotic wave. Both the dicrotic notch and wave become more pronounced peripherally. In the one case of coarctation of the aorta (fig. 7D, E and F) the fundamental central form of the pulse is essentially unaltered in its transmission peripherally down the blood vessels of the upper limb.

In considering the transformation of the pulse it would seem that the observation of Hamilton and Dow22 about the existence of a standing wave in the aortocofemoral system of

<table>
<thead>
<tr>
<th>Case</th>
<th>Measured Distance</th>
<th>Over-all Pulse Velocity</th>
<th>Pulse Velocity of Central Portion*</th>
<th>Pulse Velocity of Peripheral Portion*</th>
<th>Comparison of Planimetry Area of 3 Pulses†</th>
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<tr>
<td>4</td>
<td>44</td>
<td>8.9</td>
<td>7.3</td>
<td>11.0</td>
<td>Unchanged</td>
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<tr>
<td>5</td>
<td>42</td>
<td>7.5</td>
<td>5.6</td>
<td>11.5</td>
<td>Unchanged</td>
</tr>
<tr>
<td>7</td>
<td>45</td>
<td>11.2</td>
<td>9.6</td>
<td>16.0</td>
<td>Markedly changed</td>
</tr>
</tbody>
</table>

* Central portion is taken from aortic arch to axillary artery, peripheral portion from axillary artery to brachial artery just above antecubital fossa.
† Planimetry area refers to area under pulse curves obtained from (a) aortic arch, (b) axillary artery and (c) brachial artery. The change in area in case 7 is attributed to progressive damping of the curve as the lumen of catheter narrowed by elot.
Fig. 8A. Simultaneous recordings of central aortic pressure pulse (upper), densogram of aorta (middle) and lead II of electrocardiogram (lower). Recorded at double paper speed. B. Simultaneous recordings of central aortic pressure pulse (upper), border tracings of the aorta (middle) and lead II of the electrocardiogram (lower). Segments A and B are both from case 7. Recorded at double paper speed. Discussed in text.

Fig. 9A. Simultaneous curves of central aortic pressure pulse (upper), densogram of aorta (middle) and lead II of electrocardiogram (lower). B. Simultaneous curves of central aortic pressure (upper) border tracing of the aorta (middle) and lead II of electrocardiogram (lower). Both tracings are from case 3. Recorded at double paper speed. Discussed in text.
the dog also applies essentially to the aorto-
brachial system of man, with its node some-
where in the axillary artery. No effort has been
made to utilize the subtraction curve to evalu-
ate the distortion factor in addition to the
standing wave, as recently described by Alex-
ander.

D. Correlation of Central Arterial Pulse Pressure
with Simultaneously Recorded Electrokymo-
gram

A previous report from this department has
dealt with the comparison of right ventricular,
auricular and pulmonary artery pressures with
the electrokymogram. In the present study of
four cases we have attempted to define the
significance of the electrokymogram of the
aorta and left ventricle by means of the intra-
luminal aortic pressure curves in man. Electro-
kymographic tracings were obtained from the
descending portion of the arch of the aorta and
compared with the aortic pressure curve with
reference to the rise of the anacrotic limb and
the incisura, as a measure of the ejection phase
of the left ventricle.

1. Timing. In three of four cases the onset
of the anacrotic limb of the aortic densogram
precedes the corresponding point on the aortic
pressure curve by an average of 0.03 second,
range 0.01 to 0.05 second. In the fourth case,
the onset of the rise of the aortic densogram
follows that of the pressure curve by 0.02 sec-
ond (fig. 9A, points A).

In all four cases the onset of the anacrotic
limb of the aortic border tracing follows the
pressure curve by an average of 0.02 sec-
ond (fig. 9A, points A).

Accurate determination of the ejection phase
is impossible because none of the aortic border
electrokymograms or aortic densograms has a
constant or measurable incisura or notch.

2. Contour. In three of the four cases the
contour of the densograms over the aortic area
is surprisingly similar to the pressure pulse
of this vessel (figs. 8A and 9A); of course dif-
fences between the two curves exist. The
descending limb of the electrokymogram fol-

dows that of the pressure curve somewhat more
faithfully than does the ascending limb.

Figures 8B and 9B, recorded from the same
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patients, are border tracings from corresponding locations. The dissimilarity between the descending limb of the aortic border electrokymograms and that of the aortic pressure pulse is apparent. The anacrotic limb of the pressure pulse, however, is more faithfully reproduced in the electrokymogram.

Figure 10.1 and B are electrokymographic recordings from the lower and middle portions of the left ventricular border approximately 4 cm. apart, correlated with aortic pressure curves and lead II of the electrocardiogram. The striking resemblance of the ventricular electrokymogram to the ventricular volume curve of the dog is again noted. However, the marked variations between the curves only 4 cm. apart on the left ventricle border is evident.

The onset of ejection in the left ventricular electrokymograms recorded in figure 10.1 is approximately 0.05 second later than in figure 10B when compared with the beginning of the rise of the anacrotic limb of the aortic pressure pulse. Other phases defy accurate measurement because of indefinable endpoints. Rapid changes of gradient which occur on the pressure pulse have no constant relationship to similar changes on border electrokymograms of the left ventricle. It is thus demonstrated again that the electrokymogram of the left ventricle represents an unpredictable and often distorted picture of volume changes within this chamber.

The densograms from the aorta in three of four cases follows strikingly the contour of the central arterial pulse during varying dynamic states. Border electrokymograms from similar areas of the aorta are considerably less faithful in this regard.

From this analysis and that previously reported it would appear that with further refinement of the electrokymographic apparatus with respect to standardization and frequency response the densograms of the aorta and pulmonary artery may prove a valuable adjunct in the diagnosis of cardiovascular disorders as reflected in these vessels.

**SUMMARY**

1. Retrograde catheterization of the aorta via the brachial artery affords a feasible method of studying directly the magnitude and contour of the arterial pressure pulse in man.

2. That this may contribute significantly to a keener insight into human cardiodynamics in health and disease was first suggested from the voluminous data comparably collected in the animal.

3. The procedure, performed by a well-trained team, is no more hazardous than right heart catheterization. Complications such as cardiac arrhythmias and pain were not encountered in any of the seven cases in the present series. With careful arterial suturing and adequate sterile precautions little difficulty in preserving the radial pulse is encountered.

4. The contour of the aortic pressure pulse in normal man and in patients with hypertension, aortic regurgitation and coarctation of the aorta resemble closely comparable tracings reported in the dog. The genesis of the central pulse contour in aortic regurgitation is reconsidered.

5. The effects of cycle length, initial volume and peripheral resistance in man, especially in the presence of ventricular premature systoles and of the Valsalva maneuver, are discussed on the basis of recorded central pressure pulses.

6. The determination of the location, size, and dynamic significance of coarctation of the aorta with this technic is advocated.

7. The pulse wave transmission velocity is measured in various parts of the aorticobrachial system and the transformation of the central pulse contour along this path is described and compared with data established from animal experiments.

8. Aortic pressure pulses are compared with simultaneously recorded volume changes by means of aortic densograms and border electrokymograms from the aortic arch and left ventricle border. The advantages and limitations of the electrokymograms in the determination of the onset of ejection and in reflecting volume changes are discussed.

**ACKNOWLEDGMENT**

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