THE PHASIC CHANGES IN CORONARY FLOW ESTABLISHED BY DIFFERENTIAL PRESSURE CURVES[1]

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The question whether coronary flow under normal conditions is reduced or even stopped during systole, or, on the other hand, undergoes a marked acceleration, is still in dispute. Records obtained by flow-recorders of different design give quite different answers (Anrep and various associates, 1; Hochrein and associates, 2; Wiggers and Cotton, 3b; Gregg, 4). This divergence of opinion is occasioned by the fact that neither the theoretical principles according to which reliable instruments should be designed nor the tests by which their efficiency can be judged have been as clearly formulated as in the case of pressure recorders. Consequently their construction is guided too largely by empirical trials while judgment as to their efficiency remains essentially a matter of opinion.

Since consecutive changes in flow are the resultant of differences in pressure which exist from moment to moment in the peripheral and central ends of a coronary ramus and since the pressure variations in the central end closely follow those in the aorta (Wiggers and Cotton, 3a), the variations of flow through the intramural vessels of the heart should be determinable by the difference between aortic and peripheral coronary pressures recorded by calibrated optical manometers.

The limiting physical conditions for such deductions are *a*, that the peripheral pressure changes in coronary branches are essentially due to the effects of ventricular contraction and not significantly to communication of pressure from collateral vessels, and *b*, that peripheral coronary pressures (P.C.P.) can be recorded accurately *both* as regards contour and magnitude. In a previous paper (5) we presented evidence that transference of pressure from collateral channels is of no material importance in territory supplied by the ramus descendens anterior. Evidence was also presented that efficient optical manometers reproduce with accurate con-

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tour and time relations the peripheral pressure changes, but do not register the maximal systolic resistance which the myocardium is capable of exerting. Consequently it was necessary to devise expedients for determining the true maximum and minimum pressures with reasonable accuracy. Furthermore, since some investigators have questioned the validity of observations made upon hearts which are separated from their natural nervous control and perfused with liquids other than the unaltered blood of the animal, it is important that these determinations be made under conditions to which such criticisms do not apply.

PROCEDURE. The aortic pressure was recorded as previously by a calibrated optical manometer. The main branch of the ramus descendens anterior was exposed for a distance of about a centimeter by grasping the surrounding sheath with four mosquito forceps, incising it in a frontal line with a sharp knife and laying back the sheath. In this way the greater portion of the nerves accompanying the vessel are spared. An electromagnetic compressor was applied to the isolated ramus, so that the normal blood supply was maintained except for brief intervals of clamping described below. A second optical manometer was inserted into a side branch of the main coronary vessel just below the region of preparation.

The peripheral diastolic pressure and contour of the P.C.P. pulses were determined as in our previous publication (5), i.e., by recording pressures from a lateral branch of the ramus descendens anterior, while the main vessel was clamped for 4 or 5 beats. Unless the heart is very slow, stabilization does not occur for several beats after occlusion (fig. 1, A). Hence the diastolic pressure should not be read on the first beat. It is immaterial which subsequent one is selected as the diastolic pressures differ only by 2 or 3 mm. Hg. We have arbitrarily selected the pressure level just preceding the second of such beats.

In order to determine the true peripheral systolic pressure, lateral pressure curves were similarly recorded while the coronary artery was clamped centrally for brief intervals at a frequency slightly different from that of the heart beat. In this way fluid could be trapped in the coronary system at various pressures up to aortic systolic. The level at which such a closed coronary vessel just held pressure at a constant level during such clamping was taken as the critical value for maximum systolic peripheral pressure under the circulatory conditions existing at the time. While the procedure was similar to that used by Anrep and Saalfeld (1d) the data obtained served quite a different purpose.

The results of this procedure are illustrated in figure 1, B in which advancing periods of clamping (0.3 sec.) are marked in 5 beats. By considering these from right to left ($1 \rightarrow 5$) their significance is better understood. In beat *1*, clamping began late in diastole and extended well into systole. After an abrupt fall during diastole, the pressure rises during

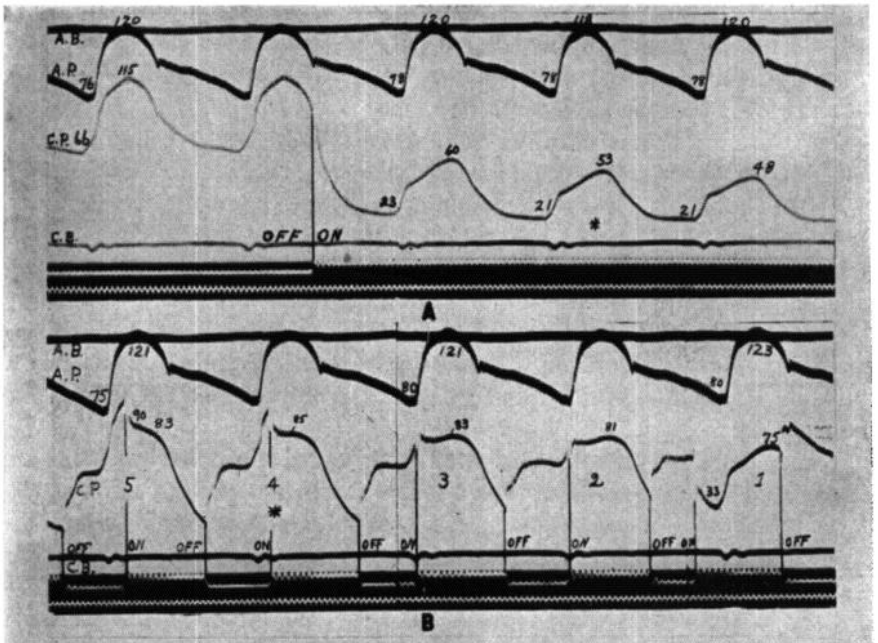

FIG. 1

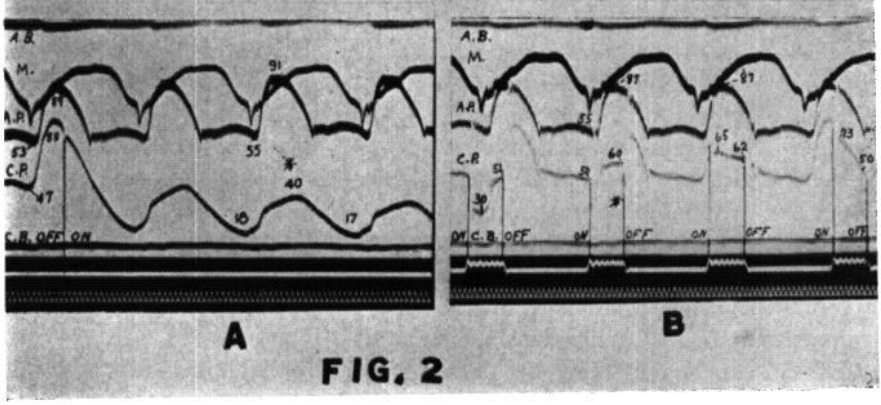

FIG. 2

Fig. 1. A, Record used for determining form of peripheral coronary pressure and the diastolic pressure. B, Record used for determining maximum systolic pressure by method of advancing short clampings. Description in text.

CP, coronary pressure; *AP*, aortic pressure; *CB*, coronary base line; *AB*, aortic base line. Periods of clamping in lower record denoted by "on" and "off." Time 0.02 second.

Fig. 2. Curves used for determining form and peripheral diastolic pressure (A) and systolic peripheral coronary pressures (B) under altered experimental conditions. Discussion in text. M, myogram; other lettering same as figure 1.

systole, reaching a height of 75 mm. The systolic part of the curve inscribed has the same form as that of a P.C.P. curve shown in figure 1, A. Although the beat develops from a relatively high diastolic pressure (33 mm. Hg) the systolic summit does not exceed 75 mm. Hg. In beats *2* and *3*, clamping occurred during the normal systolic rise of coronary pressure. In both instances a slight additional rise to 81 mm. and 83 mm. respectively occurs. In beat *4*, clamping took place after coronary pressure had exceeded such values. A pressure of 85 mm. is held during midsystole. In beat *5* the vessel was clamped nearer the systolic peak at 90 mm., but from this level it gradually declined to 83 mm. at the end of systole. Such a series of systolic clampings definitely shows that the maximum coronary resistance expressed in terms of peripheral coronary pressure is less than 90 mm. and greater than 83 mm.; in fact, it is about 85 mm. Hg.

It may be added parenthetically that a survey of all our records obtained under a great variety of conditions has never revealed an instance in which such maximum resistance ever approximated aortic systolic determined from simultaneous aortic pressure curves.

Critique of method. The question arises whether values for systolic P.C.P. so established truly represent the full magnitude of the pressure developed in the intramural vessels of the left ventricle. Various sources of error were considered and tested. The possibility that leaks in the manometer system might account for the drop of pressures when vessels were clamped at the systolic pressure peak was checked in each experiment by clamping the vessel both centrally and peripherally to the side branch and in some cases by introduction of dyes into the whole system. The area of distribution of the ramus descendens anterior precluded the existence of branches to the auricle such as Anrep and Saalfeld encountered in using the circumflex ramus. That a "runoff" occurred by arterial anastomoses with the left ventricle or other arterial branches (coronary or extracardiac) is improbable since the "holding" pressure was less than that in other arterial branches or the ventricle. Indeed, in a previous communication (5) we analyzed particularly the reverse possibility, viz., that pressure transmission may occur from arterial collaterals, but we could find no evidence of significant functional anastomoses in normal hearts. To check the possibility that tributaries of the ramus descendens anterior to the right ventricle may play a part in reducing the actual maximal pressure, a heart was selected in which several large branches of ramus descendens anterior appeared to be directed toward the right ventricle. Clamping of all these branches was, however, without effect upon the form of the P.C.P. curve or upon the maximal peripheral systolic pressure established by the method of advancing short clampings. Therefore no other conclusion could be drawn than that the pressure exerted upon intramural

vessels is definitely less than that created in the left ventricular cavity. Such relationships are physically understandable. The ventricular walls during contraction cannot be regarded as a semi-liquid substance in which pressures spread equally in all directions, but are more comparable to a laminated elastic ball under internal pressure in which the tensions are unequally distributed. While the theory of distribution of elastic stresses in such globular structures has not been reduced to mathematical terms, sufficient is known to square our observations with the physical possibilities. At the time of maximal contraction, the tension at any point within the myocardium can be resolved into three vectors. Two of these have a direction perpendicular to equatorial planes at right angles to each other and the direction of the third is parallel to the radius of the circumference.

The tension (F per unit area) developed perpendicular to either equatorial plane is that which tends to separate the two halves of the spherical shell containing fluid under pressure. It is presumably equal at every point and may be roughly calculated by the formula

$$F = \frac{PR^2}{R_1^2 - R^2}$$

in which P is the tension per unit area exerted upon the internal surface, R is the radius of the cavity of the sphere and R_1 is the radius to the outside wall. Assuming a reasonable capacity and thickness of ventricular wall during contraction it can be calculated that the tension per unit area is less in either equatorial plane than that exerted upon the internal surface, i.e., than intraventricular pressure.

The magnitude of the third vector (parallel to the radius) is not the same at all points. It is greatest in the internal layers and becomes progressively less toward the exterior where it is zero. In other words, a gradient occurs from the interior to the exterior, much as would be the case if the pressures could be measured between numerous layers of a large series of thin rubber balloons inflated from the interior and jointly bearing the pressure. Consequently the mean tension developed parallel to the radius is necessarily less than that exerted upon the inner surface, i.e., than intraventricular pressure. Actually the distribution of elastic stresses is probably far more complicated and we merely offer this physical analysis as a tentative explanation of our experimental facts.

Differential pressure plots. It should be apparent that two such records as are shown in figure 1, A and B, when taken in rapid succession supply all the data for construction of differential pressure curves. The reconstruction illustrated by curves of figure 3 was carried out as follows: The beat denoted by * in figure 1 A, and the corresponding aortic pressure curve were enlarged by optical projection upon cross-section millimeter paper

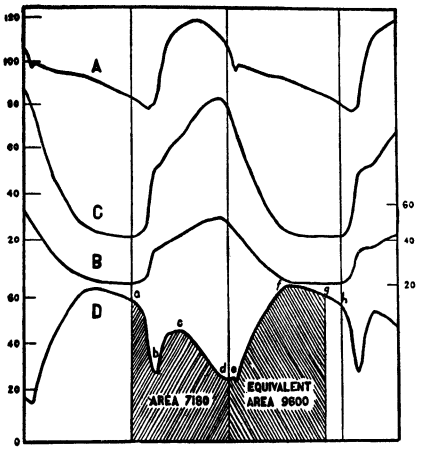

FIG. 3

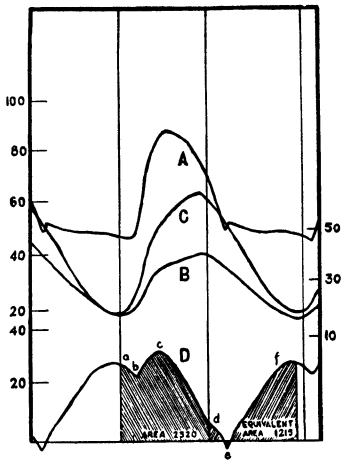

FIG. 4

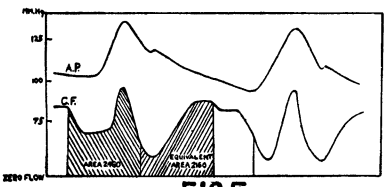

FIG. 5

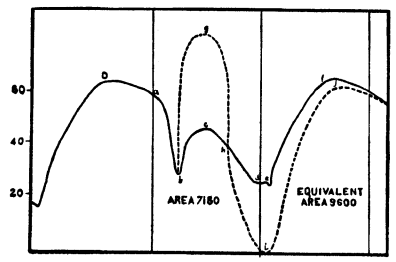

FIG. 6

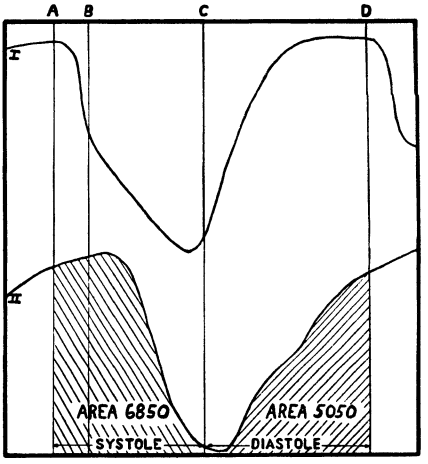

FIG. 7

Fig. 3. Reconstructions from curves of figure 1 showing relative volume flows and velocity in intramural coronary vessels during systole and diastole. A, aortic pressure; B, peripheral coronary pressure; C, same, reconstructed with true ordinate values; D, velocity curve. Shaded areas, volume flow with inscribed numbers indicating planimeter measurements of areas on original enlarged graphs. Ordinates, millimeter Hg; abscissa, time. Discussion in text.

Fig. 4. Reconstruction graphs from curves of figure 2; lettering same as in figure 3. Discussion in text.

Fig. 5. Reconstruction of flow curves from Anrep, Davis and Volhard 1c, figure 8, arranged to read from left to right. Discussion in text. *AP*, aortic pressure; *CF*, coronary flow.

Fig. 6. Comparison of intramural coronary velocities (curve *D-a-c-e-f*) redrawn from figure 3 and possible inflow velocities into coronary artery (curve *b-g-i-j*). Discussion in text.

Fig. 7. Comparison of coronary intramural flow curves when artery is perfused under constant pressure. Curve I, our curve determined from curve C, figure 3; curve II, curve of Anrep, et al., 1a, fig. 4. Both curves redrawn to same coördinate scales. *A-B*, isometric contraction; *B-C*, ejection; *C-D*, diastole.

and redrawn. The P.C.P. curve recorded by a calibrated manometer, varying slightly in sensitivity from the aortic manometer, was first brought to the same scale as the aortic and then the true P.C.P. curve was constructed by increasing the ordinate values of the whole curve so as to bring the maximum pressure to the systolic level established in figure 1 B (85 mm. Hg). In actual practice the two steps were combined in one operation and are illustrated by curve C of figure 3.

The pressure differences between the aortic pressure curve (A) and the true peripheral resistance curve (C), when again plotted with respect to zero yield curve D. We consider this to represent the coronary velocity curve. It should be emphasized that it actually depicts the *velocity of the "runoff"* from the coronary arterial system and does not correspond in every detail with the *velocity of inflow* into the coronary orifice which is affected in addition by changes in capacity as coronary pressures increase or decrease. Since sources for the "runoff" other than intramural capillaries seemed to have been excluded by our previous studies, such curves represent the velocity of intramural flow. By drawing vertical lines such as *a*, *e*, *g*, to the zero base line and measuring with a planimeter the areas so bounded, values are obtained which, when compared, express reasonably well the *relative volumes* flowing during different periods of the heart cycle. Such comparisons are not quite exact owing to slight differences in the size of vessels in different phases of the cycle; but the error so incurred is within the range of accuracy in redrawing and plotting such curves.

The phasic variations of coronary flow under natural conditions. Curve D of figure 3 is submitted as exemplifying changes of flow through the ramus descendens anterior under natural conditions. The contour of the aortic pressure pulses and the ordinate values for diastolic and systolic pressure are within normal ranges. Unfortunately the heart rate in our experiments was generally higher than we wished—in this case 107 per minute.

An analysis of the velocity curve (D) shows that coronary flow is sharply reduced during isometric contraction (*a–b*), increases sharply during the early half of systolic ejection (*b–c*), reaches a maximum with the summit of aortic pressure (*c*) and then decreases considerably during the latter half of systolic ejection (*d*). During isometric relaxation the flow accelerates, reaching a maximum (*f*) in mid-diastole despite the gradual decline of aortic pressure. Following this the flow remains large but decreases gradually; in fact it essentially follows the diastolic pressure gradient. Grossly described, two distinct accelerations occur: a first (*c*) during systolic ejection, and a second (*f*) during diastole. The positive wave representing the first acceleration is situated in a systolic valley and the maximum velocity (*c*) is less than that at any diastolic point subsequent to isometric relaxation. Such relationships indicate that velocity of coronary flow is definitely decreased by contraction; but it is never zero.

Measurement by a planimeter of the areas under curve D representing systole (*a–d*) and diastole (*e–h*) yields an S/D ratio of $\frac{7180}{11460}$ or $\frac{1}{1.6}$. Similar comparison of the systolic area with an equivalent area of early diastole yields an S/D ratio of $\frac{7180}{9600}$ or $\frac{1}{1.34}$, thus indicating that the *volume flow* during systole is 75 per cent of that during an equal time interval of early diastole. It has generally been recognized that an increase in coronary peripheral resistance during systole accounts for the decreased systolic volume flow but a pressure curve such as is illustrated by curve C in figure 3 emphasizes the additional fact that the resistance decreases about as gradually during diastole as it increases during systole. In this way gradually diminishing resistance is offered to flow until resistance reaches a diastolic level. In other words, contraction operates to decrease flow during the early portion of diastole as well as during systole.

By methods just described it has been found that similar phasic variation and relative magnitudes of volume flow during systole and diastole also occur in the left circumflex ramus. Thus in one experiment the ratio of flow during systole and an equivalent portion of early diastole was $\frac{1}{1.35}$.

The phasic relations under altered dynamic conditions. Thus far we have emphasized that under essentially normal circulatory conditions the systolic volume flow approximates but never quite equals that in early diastole. In 1931 Hochrein (2) advanced the conception that the maximal flow may shift variously between systole and diastole under different circulatory conditions. While not subscribing to his experimental proof, our analyses indicate that the flow might be maximal in systole under circulatory conditions in which diastolic pressure is relatively low and pulse pressure large. Experimental proof that this can occur is offered in the curves reproduced in figure 2 and their reconstruction in figure 4. The differential pressure curve (D) obtained shows that the first systolic wave, *c*, reaches a higher peak than the subsequent diastolic maximum at *f*. Measurement of equivalent areas gives an S/D ratio of $\frac{2320}{1215}$ or $\frac{1}{0.52}$, i.e., the systolic volume flow is almost twice as large as the diastolic. Such experiments indicate that our current conceptions must be revised by admitting that dynamic conditions *can be created experimentally* in which the maximum volume flow shifts from diastole to systole.

Discussion. Our present analyses of flow through the intramural vessels under normal conditions depicted in figure 3 accord with observations of Wiggers and Cotton (3a) that lateral pressure curves give no evidence of decrease in coronary flow except during the period of isometric contraction when an extra preliminary oscillation occurs. The flow curves pre-

dicted by Wiggers and Cotton (3b) as a result of flow measurements under declining pressures also compare quite favorably. Even the sharp early systolic decrease in velocity missed by all previous observers was predicted. The investigators failed however to recognize the abruptness and extent of the systolic acceleration of flow and also the extent of its decrease during late systole.

Our interpretations of the systolic and diastolic coronary flow under natural conditions agree in part with those of Anrep and his associates; in several major particulars however our views are different. In our opinion these differences are partly attributable to use of different methods but many are merely a matter of interpretation. The cause of these differences, real and apparent, must be discussed, for no research is complete unless an effort is made to harmonize discrepancies. We are in fundamental agreement with Anrep and his colleagues 1, that ventricular contraction increases intramural resistance; 2, that *normally* the coronary volume flow is not greater during systole than diastole as claimed by German investigators, and 3, that, consequently, the beat of the ventricle mechanically reduces the minute volume flowing through the coronary vessels under otherwise identical conditions.

We feel however that the magnitude of the systolic reduction in flow was greatly over-estimated. In the first place distinctions between velocity and volume flow must be made. Our own curves show distinctly an initial sharp decrease in velocity followed by a subsequent increase which never attains the maximum found during late diastole. But despite these fluctuations, measurement of the areas beneath the curves shows that the volume flow during systole is still about 75 per cent of that during an equivalent interval of diastole.

If, as in figure 5, similar areas are measured under velocity curves published by Anrep, Davis and Volhard it is found that the *systolic inflow* into the coronary artery is even greater during systole than during an equivalent interval of diastole. Such curves are not quite comparable to our own, however. The systolic peak rises to a level higher than that of diastole owing to the fact that a component attributable to systolic expansion of the vessel is included. We cannot agree however that this systolic inflow volume is entirely determined by the elastic expansion of vessels. It is our impression that the clever experiments designed by these investigators to support such an argument may not have been as crucial as they believed. Thus they found that the systolic inflow is equal to normal even when coronary vessels are blocked by lycopodium. It must be recalled however that occluded vessels may accommodate more blood under the same systolic pressure in a main artery than when flow is unimpeded. This can easily be shown by placing a carotid artery in a plethysmograph and clamping the vessel peripherally.

An examination of the records published by Anrep, Davis and Volhard, one of which is redrawn in figure 5, gives, we believe, explicit evidence that a marked systolic intramural flow must have taken place. If the systolic part of flow curves were due wholly to capacity changes, then the curve should drop to a zero line at the point of maximum arterial expansion, i.e., at the peak of aortic pressure. *Since this is not the case,* but on the contrary the velocity never equals zero at any point, this must signify that some fluid was also moved through the intramural vessels during systole.

We have attempted to show by the dotted part of the curve in figure 6 the maximum extent to which our own curves of velocity might have been modified if the elastic accommodative changes during ejection had been added. Such superposition depicts at a glance how inflow velocities and outflow velocities of a coronary vessel may differ in phase without affecting the total volume flow per beat. This superimposed curve was obtained by assuming a flow of 0.3 cc. per beat and calculating the additional "probable increase" in capacity (determined from previously published volume-elasticity curves) at the peak of aortic pressure, then spreading the curves so that the area gained under *b–g–h* is subtracted by *h–i–j*. Such curves resemble the coronary inflow curves of Hochrein and associates even more than those of Anrep, Davis and Volhard. They emphasize how misleading interpretations of intramural flow based upon changes in velocity at a coronary orifice may become.

The flow at constant perfusion pressures. Since for technical reasons most of the studies on coronary flow require perfusion of the vessels with blood or oxygenated saline solution under a *constant* pressure and since Anrep and his associates are inclined to regard flow curves obtained under these conditions as most trustworthy it seems advisable to compare their curves with those predictable from curves of P.C.P. redrawn to a true ordinate scale. If in relation to curve C, figure 3, we assume a constant perfusion pressure above the maximum P.C.P. the flow curve should be represented by an inversion of this curve. Such a curve, together with a copy of the flow curve of Anrep et al. (1a) are redrawn in figure 7. The essential differences are 1, an earlier retardation of flow during systole, and 2, an earlier acceleration and a quicker return to a maximum in diastole in our curves. In brief, if the curve II were advanced 0.02 to 0.04 second it would show a general correspondence to our own. These differences, though apparently minor, are nevertheless important.

If we were likewise to determine the flow curves at progressively lower perfusion pressures from curve C, figure 3, it is obvious that the initial retardation would *always begin* with the isometric contraction and not progressively earlier as claimed by Anrep and Häusler (1b). Since we have shown that the early retardation of flow is due to a rise of myocardial

tension during isometric contraction it is difficult to conceive of any factual reason why the time of such retardation should be a function of the perfusion pressure. Anrep and Häusler's results are more probably due to progressing hypodynamic states associated with slower development of intraventricular and myocardial tensions combined with a natural lag of the flow-recording apparatus as a whole.

As regards the magnitude of the decrease in flow during systole when the vessels are perfused at constant pressures both curves of figure 7 show that the velocity of flow diminishes rapidly. According to our evidence systolic flow is never arrested, even momentarily, unless perfusion pressures are used which are below the maximal peripheral coronary pressures (e.g., 85 mm. Hg in the record under discussion). According to the curves of Anrep and his various associates complete arrest can occur momentarily somewhere toward the end of systole, even when perfusion pressures 50 mm. above aortic-systolic are employed. The fact that at most a momentary arrest occurs and that a considerable volume flow is nevertheless maintained is obvious when the areas determined by such curves are measured. As shown in figure 7, the systolic volume flow is even greater than during an equivalent period of early diastole (ratio 1.36:1.0). This fact which was most certainly evident to Anrep and his various associates was never strongly emphasized; on the contrary, readers of his articles who have no clear appreciation of the graphic evidence gain the impression that complete cessation of flow occurs for all or a large part of systole. Thus in their last article (1d) the phraseology again appears, "the blood supply to the heart *during systole* is negligible." It is regrettable that such unfortunate phraseologies have been repeatedly copied and transcribed by writers when in fact the actual records show that the volume flow during systole is as large as during early diastole when the vessels are perfused under constant pressures.

SUMMARY

Under experimental conditions which permit retention of a natural blood supply under pulsatile pressure and which spare the innervation of blood vessels, we recorded the form and magnitude of the central coronary pressure (aortic pressure) and by special expedients, detailed in the text, also the contour and magnitude of the peripheral coronary pressure. The differential pressure curve constructed from these quantitated records gives a *velocity* curve of intramural flow and the areas beneath different portions of such a curve with zero pressure as a base line allow comparison of the relative *volume flows* during various portions of the heart cycle.

Such curves show that under normal conditions the velocity of intramural flow is abruptly decreased during isometric contraction; the flow

accelerates while aortic pressure rises to its summit, again to be retarded during the last portion of systole. A great acceleration occurs during isometric relaxation and a maximum velocity is attained at about the time the ventricles begin to fill during diastole. During the remainder of diastole, the velocity gradually decreases, the degree of retardation at the end of the cycle depending upon duration of diastole and the gradient of the drop in aortic pressure.

The changing resistance within the ventricular walls and the variations of aortic pressure during each cardiac cycle constitute continually opposing forces which determine the velocity of flow from moment to moment; but one or the other dominates at different times during each heart beat. The rapidly increasing resistance previous to the systolic elevation of aortic pressure accounts for the brusque retardation of early systole. Similarly the rapid decrease in myocardial resistance accounts for the prompt acceleration of diastole flow before ventricular filling begins. The secondary acceleration during systole as well as the continued high velocity during most of diastole are dominated by the aortic pressure.

A study of our curves shows that these fluctuations in *velocity of flow* during the cardiac cycle give no information regarding the *volume flow* during systole and equivalent periods of diastole. *Actual comparisons of such volume flows show that the myocardium receives approximately three-fourths as much blood during systole as during an equivalent interval of early diastole, but of course much less than during the entire period of diastole on account of its greater length.* Under exceptional experimental conditions characterized by relatively low diastolic pressures and large pulse pressures the systolic volume flow may even exceed that of equivalent periods in diastole.

Neither the phasic velocity nor volume flow *from* the larger coronary rami into and through the capillaries of the myocardium correspond exactly with the velocity or volume flow *into* the mouth of a coronary artery, for as Anrep, Davis and Volhard correctly emphasized the rate and volume of inflow are modified by changes in the capacity of the coronary vessels as the arterial pressure increases or decreases. It is therefore impossible as Hochrein and Keller have done to predict systolic and diastolic variations in intramural volume flow from velocity curves obtained at the mouth of an artery. The curves of figure 6 show how greatly the curves may differ. On the other hand, we have presented evidence of our own and point out suggestive evidence in curves published by Anrep, Davis and Volhard which indicates that the increased volume entering the coronary system during systole is not retained until it is moved onward during subsequent diastole; on the contrary a part of this systolic increase is moved forward through intramural capillaries during the period of systolic ejection.

An analysis of the changes in peripheral coronary resistance indicates that, contrary to general belief, the tension exerted by the myocardial contraction upon the intramural vessels is never as great as aortic systolic pressure. Consequently it is our belief that when coronary vessels are perfused under constant pressures, the flow is also not arrested—even momentarily—unless perfusion pressures very definitely below aortic-systolic are employed. Even in such curves, as in records presented by Anrep and his associates, the evidence is clear-cut that the *volume flow* during systole is quite as large as during an equivalent period of early diastole. *Physiologically it is more important to stress the maintenance of a good systolic volume-flow than the decreasing velocity and possible momentary arrest toward the end of systole, when vessels are perfused at constant pressures.*

Any interpretation or statement regarding coronary flow which implies that the myocardium receives only a negligible supply of blood during contraction —regardless of whether constant or pulsatile pressures are operating—is contrary to all hemodynamic evidence, that submitted by others as well as ourselves.

REFERENCES

(1a) ANREP, G. V., E. W. H. CRUICKSHANK, A. C. DOWNING AND A. SUBBA RAU. Heart **14**: 111, 1927.
(1b) ANREP, G. V. AND H. HÄUSLER. J. Physiol. **65**: 357, 1928.
(1c) ANREP, G. V., J. C. DAVIS AND E. VOLHARD. J. Physiol. **73**: 405, 1931.
(1d) ANREP, G. V. AND E. v. SAALFELD. J. Physiol. **79**: 317, 1933.
(2) HOCHREIN, M. Der Coronarkreislauf. Julius Springer, Berlin, 1932.
(3a) WIGGERS, C. J. AND F. S. COTTON. This Journal **106**: 9, 1933.
(3b) WIGGERS, C. J. AND F. S. COTTON. This Journal **106**: 597, 1933.
(4) GREGG, D. E. This Journal **109**: 44, 1934.
(5) GREGG, D. E., H. D. GREEN AND C. J. WIGGERS. This Journal **112**: 362, 1935.