THE EFFECT OF CORONARY OCCLUSION ON MYOCARDIAL CONTRACTION

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Many students of the coronary circulation must have noted that the ventricular zone affected by ligating a large coronary branch not only appears cyanotic and dilated, but that it seems to alter in its mode of contraction. The detailed and sequential changes in contraction are not easily followed by the unaided eye and so far have not been recorded myographically. The reasons for this were the lack of an adequate and suitable myograph and a technic for the application of one to a limited ventricular surface so that records obtained represent, at least reasonably well, changes in muscle length and not predominantly artefacts due to position changes, thrusts and vibrations of the vigorously beating ventricle.

This communication concerns itself with descriptions of a technique and of a type of optical myograph suitable for such studies and an analysis of the changes in optical myograms which follow clamping of a large coronary vessel.

APPARATUS. After preliminary efforts to obtain satisfactory ventricular myograms with the segment myograph used by one of us (Wiggers, 1916) to study auricular contraction, it became obvious that in order to overcome the distortions produced by twists and thrusts of the beating ventricle an instrument was needed in which the movable lever arm operates in fixed bearings. A suitable myograph which retains the compactness, lightness and efficiency of the earlier form is illustrated in figure 1. The body of the instrument consists of a small receiving tambour, $E$, (2.5 cm. in diameter) from which a tube leads off at right angles for connection with an optical segment capsule. The lever arms which are of aluminum are spaced 1.5 cm. apart. The rigid arm, $A$, is attached solidly to the back of the tambour and the movable one, $B$, is pivoted in jewel bearings, $C$, as indicated in the insert sketch. The total weight of the myograph is only

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2 Fellow of the National Research Council.
10 grams, hence its attachment does not modify cardiac contraction. When the lever arms are securely stitched through the eyelets to the ventricular surface their approximation compresses the tambour rubber, D. The pressure changes thus created are transmitted to a Frank capsule optically so arranged that the recorded curve is upward in direction. The myograph arms must be firmly attached to the ventricular surface and exactly aligned in the direction of the superficial muscle fasciculi of the region studied. Twisting action still exerted on the myograph by the contractions of other fibers can generally be eliminated if the rubber tube connection on the lead-off tube is turned in one direction or the other until such motions are gone. To minimize the transference of finer vibrations, such as heart sounds, the tambour is covered with a relatively thick rubber dam (0.29 mm.) and the Frank capsule with a dam as heavy as the registration of curves of proper amplitude permits.

The attached myograph is suspended by an elastic band, the tension of which must be meticulously adjusted so that a light pull is exerted upon the stitches and underlying myocardium. With proper adjustment of the tension the up and down movements of the heart as a whole are not recorded. If the size of the ventricle changes, readjustments of tension can be made conveniently by means of a screw control at the upper end of the elastic suspension.

In addition the exposed heart is so adjusted in a pericardial cradle that the region selected for study moves as little as possible. The central anterior surfaces of both the left and right ventricles are obviously most suitable for recording good myograms, although even the extreme apex and basal regions with their extensive movements yield satisfactory records.

METHODS. Dogs 10 to 20 kilos in weight were anesthetized with morphine and sodium barbital and under mild artificial respiration the heart was exposed and rested in a pericardial cradle. In most experiments the ramus descendens anterior of the left coronary artery was isolated within 2 cm. of its origin in preparation for the application of a miniature clamp, and the myograph was stitched to the central anterior surface of the left ventricle. In a few experiments the right coronary artery
Fig. 2. Six segments from records showing A, control B, slow beat, C, D, E, evolving changes in left ventricular myogram during left coronary occlusion and F, recovery following release. Upper curve aortic pressure, lower, myograms. Time, 0.02 second. Further discussion in text. (Reduced.)
was isolated instead and the myograph similarly applied to the right anterior ventricular surface. In some experiments a second myograph was attached to a region of ventricle not supplied by the coronary vessel to be occluded.

In connection with such optical myograms, pressure pulses from either the aorta or the left ventricle were recorded in the usual manner by means of calibrated optical manometers. After satisfactory control records had been obtained the isolated coronary vessel was securely clamped and synchronous optical records were taken either continuously on slowly moving paper or at frequent intervals after occlusion on rapidly moving paper. In a similar fashion the effects of decompression of the coronary artery were also studied after varying intervals of occlusion. During

![Image](http://ajplegacy.physiology.org/)

**Fig. 3.** Four segments of records showing A, control B, C, D, evolving changes of right ventricular myogram (middle) and unaltered character of left ventricular myogram (upper) following ligation of right coronary artery. Lower curve, aortic pressure. (Reduced.)

several experiments, ventricular fibrillation occurred either before or just after the release of the clamp. In a large proportion of such accidents fibrillation was abrogated and a perfectly normal beat reestablished by applying electrodes to the heart and sending brief counter-shocks of an A-C current directly through it, as described by Hooker, Kouwenhoven and Langworthy (1933).

**Characteristics of the normal ventricular myogram.** Myograms recorded from different accessible regions of the right and left ventricle are essentially alike. Typical tracings from the left ventricle are shown in figure 2, A and B, in which curves with rapid and slow rates of beat are illustrated. Other examples are shown in figure 3 (upper curve) and in curve A of figure 4.

During the isometric contraction phase (indicated by lines $1-2$) the
myograms show two characteristic deformations, i.e., either a steep drop followed by several small oscillations or a transient sharp positive spike. Frequently less conspicuous oscillations or dips characterize this phase. All of these must be considered as artefacts which we have not succeeded in eliminating.

Precisely with the onset of ejection (line 2) the myogram at first rises steeply (2-a) and then more gradually to a summit near or at the end of systole (line 3). This portion of the curve corresponds to the auxotonic shortening process. The summit generally persists during isometric relaxation (fig. 2, B and F) or sometimes a further small elevation follows the incisura which is an artefact (fig. 4, A). With the onset of ventricular filling the curve declines rather rapidly to a basic diastolic level. In cycles with a long diastole (fig. 2, B) a slight gradual after-stretching occurs during diastasis.

Changes in myograms following occlusion of ramus descendens anterior. Continuous records taken after occlusion show a series of evolving changes in the contour of the contraction curve leading in about a minute to its complete inversion during systolic ejection (fig. 2, E). The detailed evolution is shown in curves of figure 4. Although the myograms alter from beat to beat they may for convenience be described as of three categories, viz., 1, an initial type characterized by smaller amplitude and decreased duration of contraction (fig. 2, C; fig. 4, B-F); 2, transitional types (fig. 2, D; fig. 4, G, H, I) indicating progressive decrease in shortening and a struggle between the forces causing shortening and those tending to lengthen the fibers, and 3, frankly inverted myograms (fig. 2, E; fig. 4, J-K). A detailed study of myograms recorded immediately following occlusion shows in addition to a decreasing amplitude of contraction a concomitant shortening of the period of contraction of the affected muscle (2-b) as illustrated in A, C and D of figure 2. The duration of systolic ejection also decreases during this interval and usually remains reduced until some time after frank inversion of the myogram has occurred when it may again come to equal the initial duration.

Analysis of the inverted myogram (fig. 2, E; fig. 4, J-K) brings out several significant changes in time relations. The chief abrupt drop of the curve,
indicating expansion of the ischemic muscle, occurs during the isometric rise of intraventricular pressure and prior to the rise of aortic pressure. During systolic ejection (2–3) a slight degree of muscle shortening generally occurs; the curve is rarely an inverted image of the intraventricular pressure summit. Precisely synchronous with the incisura of the aortic pressure curve (3) and the rapid fall of intraventricular pressure during isometric relaxation the curve rises sharply to the diastolic level with a subsequent gradual decline as the ventricle fills during diastole.

**TABLE 1**

<table>
<thead>
<tr>
<th>EXP. NO.</th>
<th>DURATION OF LIGATION</th>
<th>TIME INTERVAL BETWEEN LIGATION AND FAILURE</th>
<th>RECOVERY</th>
<th>TIME INTERVAL BETWEEN RELEASE AND RECOVERY</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1 min. 40 sec.</td>
<td>50 sec.</td>
<td>Yes</td>
<td>45 sec.</td>
<td>Without fibrillation</td>
</tr>
<tr>
<td>2</td>
<td>2 min. 20 sec.</td>
<td>50 sec.</td>
<td>Yes</td>
<td>30 sec.</td>
<td>Without fibrillation</td>
</tr>
<tr>
<td>3</td>
<td>2 min. 30 sec.</td>
<td>35 sec.</td>
<td>No</td>
<td></td>
<td>Fibrillation without revival</td>
</tr>
<tr>
<td>4</td>
<td>2 min. 30 sec.</td>
<td>20 sec.</td>
<td>No</td>
<td></td>
<td>Fibrillation without revival</td>
</tr>
<tr>
<td>5</td>
<td>3 min. 75 sec.</td>
<td>No</td>
<td></td>
<td></td>
<td>Fibrillation without revival</td>
</tr>
<tr>
<td>6</td>
<td>3 min. 150 sec.</td>
<td>No</td>
<td></td>
<td></td>
<td>Fibrillation without revival</td>
</tr>
<tr>
<td>7</td>
<td>4 min. 55 sec.</td>
<td>Yes</td>
<td>11 min.</td>
<td></td>
<td>Fibrillation revived</td>
</tr>
<tr>
<td>8</td>
<td>4 min. 90 sec.</td>
<td>Yes</td>
<td>1 min.</td>
<td></td>
<td>Without fibrillation</td>
</tr>
<tr>
<td>9</td>
<td>4 min. 44 sec.</td>
<td>70 sec.</td>
<td>Yes</td>
<td>14 min.</td>
<td>Fibrillation revived</td>
</tr>
<tr>
<td>10</td>
<td>10 min. 40 sec.</td>
<td>Yes</td>
<td>5 min.</td>
<td>30 sec.</td>
<td>Fibrillation revived</td>
</tr>
<tr>
<td>11</td>
<td>23 min. 65 sec.</td>
<td>Partial</td>
<td></td>
<td>8 min.</td>
<td>Fibrillation revived</td>
</tr>
<tr>
<td>12</td>
<td>45 min. 150 sec.</td>
<td>No</td>
<td></td>
<td>Heart beat for 2 hours after release</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>46 min. 80 sec.</td>
<td>No</td>
<td></td>
<td>Fibrillation without revival</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>1 hr. 31 sec.</td>
<td>No</td>
<td></td>
<td>Fibrillation 14 minutes after release</td>
<td></td>
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</tbody>
</table>

These changes persist until the ventricle fibrillates or stops in a hypodynamic state as is usual in most acute experimental occlusions. In some instances, however, as in the series reported by Orias (1932) an effective ventricular action continued for 1 to 1½ hour despite the constant lack of any significant contraction in the ischemic zone.

When in such experiments the coronary clamp is released after a short period of closure, a reversed set of evolving changes occurs leading rapidly to a complete restoration of normal contractions in the zone (fig. 2-F).
A determination of the maximal duration of ventricular ischemia compatible with such recovery requires a much larger series of observations than are at our disposal at present, although the data of table 1 indicate that after intervals of 23 minutes or greater, recovery was only partial or absent even an hour later.

The excitability and conductivity of the ischemic area. The absence of contraction in the ischemic zone may be due either to a depression of the contractility of the affected muscle or to a failure of impulses to reach it because of a loss of conductivity and/or irritability. That these latter functions are not essentially disturbed was demonstrated by the following experiments:

Myographic records were simultaneously recorded from the right and left ventricles. The anterior descending ramus of the left coronary was ligated and after several minutes when the myogram of the left ventricle showed distinct inversion the heart was stopped by vagal stimulation and the ischemic zone stimulated with rhythmic break induction shocks of a strength which had previously been found just adequate to excite the normal ventricle. With this stimulation impulses spread in the usual manner to the right and left ventricles and caused their contraction, although each elevation of left intraventricular pressure continued to expand the ischemic area. This demonstrates that the functions of contractility can be seriously impaired by anoxia while the properties of irritability and conductivity are essentially unchanged.

The ineffectiveness of perfusions with Locke's or Tyrode's solutions. In an effort to evaluate the minimal oxygen supply capable of sustaining muscular contraction it was proposed to perfuse the descending ramus of the left coronary artery with fully oxygenated Locke's or Tyrode's solutions and then by progressive gradual decreases in oxygen pressures and simultaneous time flow measurements to calculate the minimal oxygen requirement necessary to sustain contraction. Since no difficulty was encountered by us in maintaining beats in the hearts of small dogs perfused by the Langendorff method with the same solutions and since various workers in this laboratory had shown that fibrillation and hypodynamic failure so common in dogs after coronary ligation could be prevented by such perfusion even at low pressures the plan seemed perfectly feasible. It immediately became apparent, however, that even at the highest oxygen and perfusion pressure compatible with experimental methods (200 mm. Hg above atmospheric pressure) such solutions are certainly incapable of maintaining sufficient contraction in the perfused zone to cause a shortening with the heart performing work under natural conditions. The myographic curves from these zones invariably resembled those of figure 2, E. Since it can be shown by simple calculations that such solutions even under these high O₂ tensions cannot absorb more than 2.5 volumes per cent of
oxygen, coronary flow cannot be made great enough to supply sufficient oxygen for supporting efficient contractions in the normally working heart. Further studies employing pure hemoglobin solutions will be required to determine the minimal oxygen requirements.

The responses of the right ventricle during ischemia. Whether the contractile failure will eventually prove to result from exhaustion of and a failure to resynthesize phosphocreatine, from accumulation of lactic acid, from decrease in pH, or less probably from failure in oxidation of lactic acid there can be no doubt but that anoxia due to an inadequacy of collateral blood supply is the tangible factor. Inasmuch as the Thebesian and other communications are anatomically more extensive in the right heart and since its total metabolic requirements are less, the question arises whether a similar prompt reduction and failure of efficient contraction occurs after occlusion of the right coronary artery.

To study this question the right coronary artery was isolated and compressed and the effects on myograms recorded from both right and left ventricles were compared. Such experiments illustrated by 4 segments of records in figure 3 demonstrated that changes identical with those described in the case of the left ventricle are certain to follow occlusion of the right coronary artery, although the time required for complete reversal was regularly somewhat longer (up to 3 or 5 minutes). In these records the upper curves are myograms from the left ventricle, the middle curves, myograms from the right ventricle, and the lower, an aortic pressure curve. The right myograms in segment B, C and D show respectively the initial depressed, the transitional and the frankly inverted characteristics, while the character of the left ventricular myogram remains unaltered. Apparently, the advantages gained by the better collateral communications of the right coronary system with the ventricle are largely offset by the fact that they necessarily carry less completely oxygenated blood than do the main vessels.

Discussion. The interesting and somewhat surprising discovery that approximately one minute after coronary occlusion the contractile force in an ischemic area is either abrogated or certainly so feeble that the ischemic muscle stretches instead of shortens during systole and in proportion to the elevation of ventricular pressure, has many implications of importance to clinical medicine and experimental physiology. Of these we shall discuss a few:

1. Our results demonstrate more convincingly than direct circulatory studies the functional inadequacy of anatomically described collateral branches to ventricular muscle. Furthermore, the numerous instances of infarction and cicatrization found postmortem after coronary occlusion in humans and in dogs (experimentally produced) indicate that circulatory conditions are not dissimilar in these hearts. Consequently our observa-
tions strongly suggest that if an extensive collateral circulation has not developed prior to a total occlusion, the muscle in the zone affected is not likely to survive.  

2. Since the muscle fibers in the affected zone promptly undergo periodic stretching instead of shortening, the thought arises that such mechanical factors, rather than chemical, as commonly postulated, may be the ultimate stimulus to the sensory nerves and so account for the immediate intense pain associated with the occlusion. Upon such an assumption, the hitherto unexplained benefits of pressure lowering drugs would be clarified since, by lowering the maximum intraventricular pressure, the degree of the periodic stretching would be reduced. This possibility is worthy of more extensive investigation.

3. Experimentally our observations supply tangible proof for certain logical assumptions that Orias (1932) while working in this laboratory was obliged to make in order to explain the pressure changes immediately following coronary occlusion. To account for the immediate reduction in duration of ventricular systole, this investigator postulated a prompt reduction of contractile power in the ischemic area. In order to account further for the negligible decline of systolic pressure or in some instances its complete absence before compensatory reactions of other regions had time to develop required the further assumption that the contractions in the ischemic area were of shorter duration. Our observations have demonstrated the occurrence of both conditions. Myographic curves have shown that localized anoxemia produces the same abbreviation of contraction in the affected zone that generalized anoxemia does upon the whole heart (Sands and DeGraff, 1925). No evidence was obtained however that it exerts an initial increase in contraction as seemed to follow from studies of general anoxemia.

Our observations that a marked systolic expansion of the ischemic region replaces shortening makes it evident that a considerable fraction of the total pressure developed is lost in producing such distention. We would therefore supplement Orias' theoretical analysis of fundamental mechanisms by adding the suggestion that the hypodynamic levels which so often occur despite a rise of initial tension may not necessarily be due to a fatigue of the remaining contraction fractions, but can be accounted for by the loss of pressure in expanding the regions in which contractions are enfeebled or absent.

4. The myographic study of localized ventricular areas which we have
introduced and a knowledge of the sequential changes that follow deprivation of blood supply should prove useful in estimating the value of drugs in coronary occlusion, both those that might insure a better collateral supply and those that might act through a direct effect on the muscle. Unless it can be shown that some degree of immediate improvement in contraction occurs in the regions affected, no great practical value as regards maintenance of function and avoidance of subsequent pathological changes can be anticipated.

SUMMARY

1. An optical myograph suitable for recording localized contractions from a ventricular surface and a technique for its correct application are described.
2. Normal myograms recorded simultaneously with aortic or ventricular pressure curves, though slightly deformed by oscillations during the isometric contraction and relaxation phases clearly show the natural shortening which occurs during ventricular ejection and the lengthening which follows isometric relaxation.
3. Occlusion of a main coronary branch is followed by an evolving series of myographic changes which indicate progressive enfeeblement of contraction to the extent that approximately within a minute the area stretches during isometric contraction, remains stretched during systolic ejection and shortens quickly during isometric relaxation; in short, the myogram is completely inverted. Similar changes in contraction of the right ventricle occur following ligation of the right coronary artery. These observations demonstrate convincingly the functional inadequacy of described collateral circulation in normal hearts.
4. Reestablishment of the normal blood supply is followed by a reversed series of myographic changes with restoration of normal vigorous contractions provided the period of ischemia is not too long in duration.
5. Failure of shortening is due to enfeeblement or abrogation of contraction and not to failure of impulses to reach the areas involved, or to excite them.
6. The oxygen requirements for maintaining efficient contractions in the normally working heart are high as evidenced by our failure to maintain efficient contractions when an area is perfusing with highly oxygenated Locke's solution.
7. The observations supply tangible proof for the correctness of Orias' hypothesis that coronary occlusion produces an early abbreviation of total ventricular systole with little or no decline of systolic pressure through a progressive decrease in amplitude and duration of contraction in the ischemic area. Our results suggest further that the tendency for development of hypodynamic ventricular beats following coronary occlusion may
not necessarily be due to fatigue of the remaining contracting fibers, but can be explained by loss of pressure in expanding the regions in which contractions are enfeebled or absent.

8. Several clinical implications of our results are briefly discussed.

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