Regional Coronary Venous Oxygen Saturation and Myocardial Oxygen Tension following Abrupt Changes in Ventricular Pressure in the Isolated Dog Heart

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ABSTRACT

Regional coronary venous oxygen saturation and myocardial oxygen tension (PO2) in isolated dog hearts were measured using two different methods—a new technique of microscopic oximetry and an improved oxygen electrode. Both methods yielded similar results. In studies of the isolated perfused dog heart developing a constant left ventricular systolic pressure, mean coronary venous oxygen saturation of blood in the subendocardial veins of the left ventricle was significantly lower than that in the subepicardial veins. Following abrupt increases in systolic pressure, subendocardial coronary venous saturations fell to even lower levels. Following abrupt decreases in systolic pressure, the mean subendocardial coronary venous oxygen saturation increased to levels above those in the blood of the subepicardial veins. These findings agree with the hypothesis that transient underperfusion and overperfusion of the subendocardial layers of the left ventricle occur with abrupt increases and decreases in ventricular pressure, respectively.

KEY WORDS: microscopic oximetry, Anrep effect, subendocardial venous O2 saturation, subepicardial venous O2 saturation, homeometric autoregulation, subendocardial ischemia, oxygen electrode, regional coronary vasoconstriction, regional coronary vasodilation.

By 1914, Starling and others in his laboratory had observed that a positive inotropic effect follows an abrupt increase in ventricular pressure (1–3). They attributed this effect to improved nourishment of the heart muscle, since it is accompanied by an increase in coronary blood flow (3). Later, Rosenblueth et al. and Sarnoff et al. (4, 5) found that the positive inotropic effect is independent of coronary blood flow; they thought it was a form of intrinsic adaptation or homeometric autoregulation; this view has subsequently been adopted by others (6–12).

More recently, evidence supporting the original Starling hypothesis has been reported (13). The study (13) suggests that the positive inotropic effect following an abrupt increase in ventricular pressure (termed the Anrep effect by Sarnoff [5]) is a manifestation of recovery from pressure-induced transient subendocardial ischemia. The ischemia is slowly corrected by vascular autoregulation with a redistribution of coronary blood flow to the ischemic areas. The study (13) presents several lines of evidence to support this hypothesis. (1) Contractility does not change following an abrupt increase in ventricular pressure when vasodilation of the coronary bed is nearly maximum. (2) The distribution of radioactive microspheres injected into the coronary bed indicates that relatively less blood supplies the subendocardial layers immediately after an abrupt increase in ventricular pressure. (3) The electrocardiographic changes following an abrupt increase in ventricular pressure are similar to those observed when coronary blood flow is compromised. Moreover, the new study (13) provides strong evidence that the coronary microvasculature is responsible for changes in contractility following both abrupt increases and abrupt decreases in ventricular pressure. However, techniques capable of confirming the precise effect of the changes in ventricular pressure on either transmyocardial tissue oxygen tension (PO2) or regional coronary venous oxygen saturation were not available at the time of this study (13).

Accordingly, we attempted to measure myocardial tissue PO2 with an improved technique using oxygen electrodes (International Bio-
physics Corporation). Considering the difficulties inherent in this technique, particularly those due to artifacts caused by the trauma of electrode insertion (14, 15), we also used a newly developed technique of microscopic oximetry. One primary purpose of this report was to describe the technique of microscopic oximetry in detail. A secondary purpose was to compare the findings from the polarographic and the oximetric techniques under controlled circumstances in the isolated heart. Finally, we attempted to determine whether either method could provide metabolic evidence that transient subendocardial ischemia accompanies an abrupt increase in ventricular pressure and, conversely, that a transient increase in subendocardial oxygenation accompanies an abrupt return of ventricular pressure to control levels.

**Methods**

**ISOLATED HEART PREPARATION**

Hearts from healthy mongrel dogs (10–20 kg) were excised under anesthesia and perfused with blood from an anesthetized donor. The isolated heart preparation has been previously described in detail (13, 16). In brief, arterial blood from an anesthetized donor dog was delivered to the aorta of the isolated heart so that the coronary perfusion pressure was maintained constant at 100 mm Hg. The left ventricle of the isolated heart contracted isovolumically; an arrangement of Starling resistances allowed it to develop any pressure below perfusion pressure. In addition, the pressure developed by the left ventricle of the isolated heart could be rapidly increased or decreased at will.

The right ventricles of these hearts were decompressed. Since both the inferior and the superior venae cavae were ligated, all coronary venous blood was collected from the pulmonary artery under slight negative pressure and subsequently directed through a rotameter for the measurement of coronary blood flow before it was returned to the donor. Left ventricular pressure and coronary perfusion pressure were continuously monitored through catheters attached to pressure transducers (Sanborn 267B). The outputs of the pressure transducers and the oxygen electrode (described in the following section) were continuously recorded on a Sanborn 964 Polyviso recorder.

**MEASUREMENT OF MYOCARDIAL TISSUE PO2**

To determine myocardial tissue PO2, an oxygen analyzer (International Biophysics Corporation model 145-071) was used to measure the current flowing through a polarized, Hydron-covered gold electrode. Commercially available Hydron-covered probes (0.67 mm in diameter) with a time constant of approximately 20 seconds were used initially. These probes were calibrated at the end of the experiment with saline containing dissolved oxygen at various partial pressures. The PO2 of the saline, in turn, was established with a separate PO2 analyzer (Instrumentation Laboratories model 125A). Other probes with the same dimensions had a faster time constant (4–6 seconds); however, because calibration of these probes was unreliable, they only provided a relative indication of changes in PO2. Both types of probes were inserted into the left ventricular wall and secured with a single suture; the probes were dry when they were inserted, necessitating a 20-minute equilibration period prior to use.

**MISCRYOSCOPIC REFLECTION OXIMETRY**

**Technique and Optical System.**—A new technique of microscopic reflection oximetry was developed to determine the saturation of oxygen in the smaller veins of the ventricular myocardium.

For this analysis, the isolated heart was rapidly divided at its maximal diameter, and the apical portion was immediately quenched in a mixture of liquid propane and propane ice (−185°C). In several hearts, thermocouples were embedded at various depths in the wall of the right ventricle to record the speed of freezing. In these areas the temperature of the severed portion of the heart reached 0°C 10–30 seconds after division.

After the divided portion of the heart had been quenched in liquid propane, it was transferred to a liquid nitrogen bath and prepared for microscopic examination. First, all cracks in the specimen were filled with cold (−40°C) ethyl alcohol, and the specimen was resubmerged in liquid nitrogen. At that temperature, the alcohol acted as a glue which prevented further fragmentation of the frozen specimen. The cut surface was then filed flat with a cooled wood rasp and glued to an aluminum plate with frozen ethyl alcohol. Using a chilled hacksaw blade, a thin (3-mm) wafer of frozen tissue comprising the full thickness of the left ventricular wall was removed from the specimen. This wafer was further smoothed with a chilled rasp and later polished with a chilled double-cut bastard metal file until a smooth surface was obtained, and the larger, end-on arteries and veins could be clearly identified (Fig. 1). The wafer of tissue was then positioned in a bath of liquid ethyl alcohol and alcohol ice (−115°C) so that a thin layer of liquid alcohol covered the surface of the specimen. The alcohol immediately absorbed any water from the atmosphere adjacent to the specimen and thus prevented the deposition of a layer of frost. Furthermore, the film of alcohol provided a smooth optical surface.

The frozen specimen was next searched using a low-power objective lens (10x) on an American Optical Co. model 10 microscope fitted with a trinocular body (cat. no. 1043) and a modified vertical illuminator (cat. no. 3005). After selecting an appropriate small vein in the myocardium (50–150 μ in diameter), the high-dry objective lens was rotated into position. Coronary veins were identified by their

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1Kindly supplied by the International Biophysics Corporation.
FIGURE 1
Wafer of frozen tissue cut from the heart showing the left ventricle (left) and the right ventricle (right) in bath of alcohol and alcohol ice. The surface was polished as described in the text and covered with a layer of liquid alcohol. Arrow points to the location of a triad (artery flanked by 2 veins); the finding of triads facilitated identification of the vessels. An enlargement of the triad is shown in the bottom right of the figure.

wall thickness and branching: approximately 16% were in a triad consisting of an artery, readily identified by its thick wall, with two closely adjacent thin-walled veins (Fig. 1). No vessels were studied which could not be identified as veins. In any particular area, the \( \text{O}_2 \) saturation of the blood of veins identified by triads was never significantly less than that of non-triad veins. Both objective lenses were heated electrically (60°C) to avoid water vapor condensation on their frontal surfaces. A 35\( \mu \)m spot of white light was then directed onto the blood within the vessel: the intensity of reflected light was scanned continuously from 440 nm to 600 nm using a modified spectrofluorometer (Aminco-Bowman model 4-8208), and a continuous plot of intensity against wavelength was recorded on an Aminco X-Y recorder. A scanning rate of 100–200 nm min\(^{-1}\) was used.

The optical system is diagrammed in Figure 2. Light from a high-intensity xenon arc source (A) was focused on a fiberoptic bundle\(^*\) containing 19 closely packed 50\( \mu \)m fibers and was delivered to the vertical illuminator (B) where it passed through a polarizing filter. The polarized light was partially reflected by a half-silvered mirror (C) to the 40x objective lens of the microscope. This reflection formed a small (35\( \mu \))

\*Gift from the American Optical Company.
image of the fiberoptic bundle on the frozen specimen (D). The light reflected from the 35µ area was collected by the same objective lens, passed through the half-silvered mirror with some attenuation, and focused by a lens to form an image of the 35µ area on the end of another fiberoptic bundle (E). The field subtended by the fiberoptic bundle limited the collection of light to the 35µ area so that back-scattered or diffused light returning from the specimen outside the illuminated circle was not picked up by the sensing fiberoptic bundle. A polarizing filter (E) excluded approximately 99% of the directly reflected light and minimized such reflections from the lens surfaces, the alcohol surface, and the ice crystals in the blood under study. The light received by the fiberoptic bundle (E) was delivered to the scanning spectrophotometer represented by the grating (F) and the photomultiplier tube (G). The amplified photomultiplier output (Y-axis) and the output from a wavelength potentiometer linked to the grating (F) (X-axis) were fed into the X-Y plotter (H) to record the light intensity over the wavelength span from 440 nm to 600 nm.

Calibration and Measurement.—For calibration, ten samples of dog blood were obtained with oxygen saturations ranging from 0% to 100%. After mixing, a portion of each sample was analyzed for oxygen saturation with a spectrophotometer (Beckman model C). The remainder of each sample was placed in a plastic tuberculin syringe and rapidly frozen in liquid nitrogen. Disks of frozen blood surrounded by a plastic ring were obtained from the syringes with a chilled saw, filed smooth, transferred to the bath of liquid alcohol and alcohol ice, and scanned as described in the preceding section. The ten curves obtained were then measured every 2 nm, corrected for photomultiplier sensitivity, and replotted using a Hewlett-Packard calculator (model 9810A) and plotter (model 9862A).

As in transmission oximetry, the percent of oxygen saturation can be calculated from the ratio of two absorption coefficients: one at an isobestic wavelength and the other at a wavelength at which the absorption coefficients of reduced and oxygenated hemoglobin differ widely. (Isobestic wavelength is defined as the wavelength at which the absorption coefficient is the same for oxyhemoglobin and reduced hemoglobin.) Because of the addition of white light and the slight motion of the specimen in the alcohol bath, it was necessary to modify this method slightly. First, the isobestic wavelengths of frozen dog blood were identified. From data for human blood, it was assumed that four isobestic points would be present over the range of wavelengths observed. Next, the amplitudes of replotted curves were initially adjusted so that all ten calibration curves had the same height at the first wavelength suspected to be isobestic. The dispersion at other isobestic wavelengths was noted. The wavelength at which the first suspected isobestic point was plotted was varied slightly to produce minimal dispersion at other possible isobestic wavelengths. Finally, since white light reflected from some crystals, it was assumed that a small but variable amount of white light was added to the back-diffused red light from the blood. Since the ratio of light at two isobestic wavelengths should be constant, it was assumed that any variation in the ratio of intensities of two isobestic wavelengths was a result of the addition of white light. Accordingly, replotting was undertaken after the appropriate amount of white light had been subtracted from all wavelengths, thus making the ratio of intensities at two isobestic wavelengths constant. Minor changes in these two wavelengths were then made to minimize the dispersion at the other two remaining isobestic wavelengths. The best fit was found with isobestic wavelengths of 497, 523, 544, and 566 nm.

Measurements were then made at wavelengths of 509, 536, and 557 nm, points between the isobestic points at which the variation in oxygen saturation gave the greatest dispersion. Regression equations were established between the oxygen saturation and the deviation of these three points from a straight line joining two adjacent isobestic points. Finally, a similar regression equation was established between oxygen saturation and the ratio of intensities was recorded at 481 and 497 nm. (The sensitivity of the photomultiplier tube was insufficient to establish the next isobestic wavelength below 497 nm; therefore, deviation from a straight line connecting isobestic points could not be used in this paper.) Therefore, four independent estimates were made of oxygen saturation. These estimates were then combined by weighting each proportionally to the inverse of its standard error of estimate to yield a final regression equation with a standard error of estimate of 5%. The Hewlett-Packard calculator was then programmed to make all the calculations when the raw intensities were supplied. Any scan was discarded if the intensity of light at 492 nm varied by more than 20% during the scan: this variation was attributed to motion of the specimen. All samples which had more than 30% white light or which had a standard deviation of the individual interim saturations of more than 25% were discarded (approximately 10%).

Both the calibration samples and the sections of ventricular tissue were stored for 4 days prior to study at −90°C. Although we cannot definitively prove that such storage did not alter the absolute values of oxygen saturation, the low metabolic activity at such temperatures makes this possibility very unlikely. Furthermore, hearts stored for as long as 4 weeks showed no significant difference from those stored for 4 days in the distribution of coronary venous saturations.

Results

MYOCARDIAL TISSUE P02 IN SUBEPICARDIAL AND SUBENDOCARDIAL LAYERS

In six isolated hearts, oxygen electrodes were inserted as described in both the subendocardial and the subepicardial portions of the left ventricular wall. The tips of the superficial electrodes were approximately 2 mm below the epicardial surface and the tips of the deep electrodes were inserted to 10 mm. While coronary
perfusion pressure was maintained at 100 mm Hg, left ventricular systolic pressure was abruptly lowered from 100 mm Hg to 45 ± 5 mm Hg, maintained at that level for 3 minutes, and abruptly returned to the control level of 100 mm Hg. Examples of the changes in myocardial tissue PO\(_2\) in both the subepicardial and the subendocardial layers are seen in Figure 3. Little change was observed in the PO\(_2\) recorded by the subepicardial electrode. The subendocardial electrode, on the other hand, showed a pronounced increase in PO\(_2\) when the ventricular pressure was lowered and an equally pronounced decrease in PO\(_2\) when the left ventricular systolic pressure was returned to control levels. Figure 3 illustrates that most recordings from electrodes with a slow time constant, i.e., from electrodes placed in the subendocardial layers of the ventricular wall demonstrated a slight but distinct overshoot in tissue PO\(_2\) when the ventricular pressure was abruptly lowered and an undershoot in tissue PO\(_2\) when the ventricular pressure was returned to control levels. These undershoots and overshoots in tissue PO\(_2\) that accompanied changes in ventricular pressure were more pronounced when the electrodes with fast time constants were used (Fig. 4). Calibration of the fast electrodes, however, was unreliable: therefore only relative changes in PO\(_2\) could be observed.

MICROSCOPIC OXIMETRY OF CORONARY VENOUS BLOOD

Four isolated hearts were allowed to contract isovolumically while the systolic pressure remained constant at 100 mm Hg. After a 3-minute period, during which the hearts were free of arrhythmias, they were severed at their maximum diameter, and the apical halves were rapidly frozen in a mixture of liquid propane and propane ice. In these hearts, determinations of the oxygen saturation of blood in the coronary veins of the left ventricular wall were made using the microscopic oximetric technique. These determinations were made on 42 vessels from the inner third and 46 vessels from the outer third of the left ventricular walls of all four hearts.
The findings in these hearts are graphically combined in Figure 5 (top); the cumulative percent of both subepicardial and subendocardial veins is plotted against the percent of saturation of blood in those vessels. The mean saturation of blood in the subendocardial veins averaged 33.8 ± 3.0% SE and that of blood in the subepicardial vessels averaged 51.7 ± 3.5%. Therefore, the oxygen saturation of blood in the subendocardial veins was significantly lower than that in the subepicardial vessels (P < 0.001).

MICROSCOPIC OXIMETRY OF CORONARY VENOUS BLOOD AFTER AN ABRUPT INCREASE IN SYSTOLIC PRESSURE

In four additional hearts the same determinations of coronary venous saturations were made immediately after an abrupt increase in left ventricular systolic pressure from 10 mm Hg to 100 mm Hg. Coronary perfusion pressure was maintained at 100 mm Hg. In these studies, the hearts were severed at approximately the time when the rise in end-diastolic pressure accompanying the abrupt increase in systolic pressure was maximum. The Anrep effect (13) was clearly shown in these hearts by a rise in end-diastolic pressure to an average of 10.1 mm Hg and a subsequent correction to an average of 2.3 mm Hg. Oximetric determinations were made on blood in 46 subepicardial veins and 36 subendocardial vessels of the left ventricular walls of these hearts.

The findings in all hearts in which the apical halves were rapidly frozen after an abrupt increase in systolic pressure are graphically shown in Figure 5 (middle) as a plot of the cumulative percent of veins against the percent of oxygen saturation. After an abrupt increase in systolic pressure, mean coronary venous oxygen saturation averaged 28.2 ± 4.0% SE in the subendocardial veins and 50.3 ± 3.9% in the subepicardial vessels. According to these studies, after an abrupt increase in systolic pressure the saturation of blood in the subendocardial veins was again significantly different from that in the subepicardial veins (P < 0.001). Furthermore, subendocardial coronary venous saturations fell lower after an abrupt increase in systolic pressure compared with the saturations found after the ventricle developed a constant systolic pressure.

FIGURE 4

Recording from subendocardial PO₂ electrode (fast time constant), left ventricular pressure transducer at low gain, and left ventricular pressure transducer at high gain when systolic pressure was successively abruptly lowered and increased in an isolated heart preparation. Lack of scale (top) indicates that only relative recordings of PO₂ could be obtained with fast electrodes. Note the pronounced overshoot in PO₂ when systolic pressure was lowered and, conversely, the undershoot in PO₂ when systolic pressure was increased.
Although the difference in these averages was not statistically significant. Subepicardial coronary venous saturations were roughly similar in both circumstances.

**Microscopic Oximetry of Coronary Venous Blood After an Abrupt Decrease in Systolic Pressure**

In four additional isolated hearts, determinations of left ventricular coronary venous saturations were made immediately after an abrupt decrease in left ventricular systolic pressure from 100 mm Hg to 20 mmHg. The hearts were severed at a point roughly coinciding with the maximum reverse Anrep effect (13). In these four hearts, oximetric determinations were made on blood in 38 subepicardial veins and 42 subendocardial veins.

The findings in all hearts in which the apical halves were rapidly frozen after an abrupt decrease in systolic pressure are shown in Figure 5 (bottom). After an abrupt decrease in systolic pressure, mean coronary venous oxygen saturations averaged 65.4 ± 2.8% SE in the subendocardial veins; these saturation levels were significantly higher than the control levels and exceeded the average of 56.5 ± 3.6% found in the subepicardial vessels.

Figure 6 is a summary of all of the studies involving microscopic oximetry of coronary venous blood. The plots of the cumulative percent of veins against the percent of oxygen saturation were roughly similar when the findings in subepicardial vessels were compared regardless of whether the specimens were obtained while the ventricle was developing a constant pressure or immediately after either an abrupt increase or an abrupt decrease in that pressure (Fig. 6, right). However, when the findings in subendocardial vessels were compared, three distinct patterns emerged (Fig. 6, left). Following an abrupt increase in systolic pressure subendocardial venous saturation levels fell even lower than the control levels, but following an abrupt decrease

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**FIGURE 5**

Plots of the cumulative percent of left ventricular coronary veins against the percent oxygen saturation of venous blood of both subepicardial and subendocardial veins in isolated hearts. Top: Data were obtained while the left ventricle contracted at a constant systolic pressure and determinations of O₂ saturation were made by microscopic oximetry. Mean oxygen saturation of blood in subepicardial veins was 51.7 ± 3.5% SE and that in subendocardial veins was 33.8 ± 3.0% SE (P < 0.001). Middle: Determinations of venous oxygen saturation made on specimens obtained immediately after an abrupt increase in ventricular pressure. Mean oxygen saturation of blood in subepicardial veins was 50.3 ± 3.9% SE and that in subendocardial vessels was 29.2 ± 4.0% (P < 0.001). Compared with Figure 5 (top) mean coronary venous saturation of blood in the subendocardial veins fell to a lower level immediately following an abrupt increase in systolic pressure. Bottom: Determinations of venous oxygen saturation made on specimens obtained immediately after an abrupt decrease in ventricular pressure. Mean oxygen saturation of blood in subepicardial veins was 56.5 ± 3.6% SE and that in subendocardial veins was 65.4 ± 2.8% (NS). Compared with Figure 3 (top) mean coronary venous saturation of blood in subendocardial veins rose to a high level following an abrupt decrease in systolic pressure; it even exceeded that in the subepicardial veins.
in systolic pressure the saturation levels were significantly higher ($P < 0.05$) than the control levels.

**Discussion**

A primary goal of this paper was to describe a new physiological technique for the regional determination of venous oxygen saturations that can be applied to virtually any organ; the advantage of this technique is the relative simplicity of its application. In this regard, we are not certain that the superior coolant properties of liquid propane are necessary, because once the heart is severed there is no coronary blood flow to the severed portion and the coronary venous blood should presumably remain stationary in the veins where very little metabolic activity occurs. However, this coolant was used in place of liquid nitrogen to freeze the tissue as quickly as possible.

Disadvantages that should be mentioned include the fire hazard of the propane, the high cost of the spectrophotofluorometer used to measure the intensity of reflected light, and the time-consuming analysis of data from each point. This latter disadvantage will probably be overcome by computer analysis.

Other disadvantages include the standard error of the method which is moderate at 5%. So far, attempts to reduce this value by using additional isobestic points have been unsuccessful. An additional problem lies in the identification of veins; 16% of the veins were found in the presence of an artery, clearly identified by its thick walls (triad). A final disadvantage is that the organ involved had to be destroyed to obtain the data.

Two findings emerged from the studies in which electrodes with both fast and slow time constants were used to measure $P_{O_2}$ within the ventricular wall. First, the deeper electrodes consistently recorded a lower $P_{O_2}$ than did the superficial ones; this finding has been reported elsewhere (14, 17, 18). Second, following an abrupt increase in systolic pressure, there was a transient decrease in the subendocardial $P_{O_2}$ and, conversely, following an abrupt decrease in systolic pressure, there was a transient increase in $P_{O_2}$. This latter finding agrees with the hypothesis that the Anrep effect and the reverse Anrep effect are representative of the recovery from transient subendocardial ischemia and a manifestation of transient overperfusion of subendocardial regions, respectively (13).

There are shortcomings of polarographic estimates of myocardial tissue $P_{O_2}$. The negatively polarized electrode itself consumes oxygen; also motion of the electrode can increase the availability of oxygen and increase current flow, thereby indicating an overestima-
tion of tissue Po2. Denaturation of protein on the electrode can poison it and, conversely, cause an underestimation of Po2. Most important, any damage to local tissue attending the insertion of the electrode can cause serious artifactual changes, if the electrode itself records the Po2 in a damaged area. All of these shortcomings have been discussed in previous reports (14, 15). Therefore, measurement of myocardial tissue Po2 with polarographic techniques has been viewed with substantial skepticism.

Nevertheless, the results obtained with the microscopic oximetric technique were consistent with the findings obtained with the polarographic technique in the isolated heart. The saturation of blood in the subendocardial veins was significantly lower than that found in the subepicardial layers when the heart was developing a constant systolic pressure; a lower tissue Po2 is expected to attend any significantly lower venous saturation. Also, with an abrupt increase in systolic pressure, the oxygen saturation of blood in the subendocardial coronary veins fell to even lower levels. Although the average decrease was not statistically significant (from 33.8% to 28.2%), it should be remembered that there is a finite limit to which any organ can reduce the saturation of oxygenated blood. Only 18% of the subendocardial veins showed oxygen saturations of 20% or less while the ventricle was developing a constant systolic pressure (Fig. 6, left). On the other hand, 45% of the subendocardial veins showed saturations of 20% or less after an abrupt increase in systolic pressure.

With an abrupt decrease in systolic ventricular pressure, the average oxygen saturation of blood in the subendocardial veins was higher than that in the subepicardial veins. The average subendocardial venous oxygen saturation following an abrupt decrease in systolic pressure was significantly higher than that found when the ventricle was developing a constant pressure. This finding was consistent with the transient elevation in Po2 recorded by subendocardial electrodes when systolic pressure was abruptly decreased.

Perhaps the most interesting finding obtained with the microscopic oximetric technique was the range of coronary venous oxygen saturations in both the subepicardial and the subendocardial layers when the heart was developing a constant pressure or when this pressure was abruptly altered. Although this range could be a characteristic of isolated hearts, the inhomogeneity implies that, in adjacent areas within the heart, there is likewise an inhomogeneity of oxygen consumption, coronary blood flow, or both. This finding suggests the possibility that, within the heart, some areas are at rest when adjacent areas are intensely active.

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