The Derivation of Coronary Sinus Flow During Elevation of Right Ventricular Pressure

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The derivation of coronary sinus flow during increased pulmonary and right ventricular pressures was resubmitted to experimental study. The primary reduction and subsequent augmentation in coronary flow following pulmonary artery constriction and the persistence of a greater flow after release preclude the concept of a variable contribution via thebesian channels from the right ventricular cavity. When the magnitude of the increase is also considered, it is difficult to escape the conclusion that drainage from the right ventricle is also involved. The use of coronary sinus blood in studies of metabolism of the left ventricle under various dynamic conditions seems precarious.

WHILE it is generally agreed that elevation of right ventricular pressure by pulmonary artery constriction causes an increased coronary sinus flow, the mechanism for this increase is still controversial. Johnson and Wiggers, who first made this observation, suggested that the increased resistance against which the thebesians drain venous blood into the right ventricle causes the diversion of blood from the thebesians into the coronary sinus, or even produces a reversal of flow from the right ventricle through the thebesians into the coronary sinus. They therefore concluded that measurement of coronary sinus outflow could not be used as an index of coronary artery inflow.

Since flow in the right and left coronary arteries first decreased and then increased after pulmonary artery compression, Gregg and his associates concluded that the greater left and right coronary inflows could account for the augmented coronary sinus flow. Additional experiments led Gregg to the affirmation that coronary sinus outflow may reasonably be used to estimate directional changes in flow through the left coronary arteries. This statement has been misinterpreted by some investigators to the effect that blood drained by the coronary sinus necessarily represents that which has passed largely through the left ventricle and can therefore be employed in metabolic studies of this chamber. Since coronary sinus catheterization is accomplished relatively easily, both in animals and man, the question as to the derivation of coronary sinus blood during pulmonary artery constriction was subjected to further experimentation.

METHODS

Dogs weighing 8 to 15 Kg. were anesthetized with sodium pentobarbital and morphine sulfate. The chest was opened, artificial respiration started, and the heart suspended in a pericardial cradle. Coagulation was prevented by an initial intravenous injection of heparin (4 mg. per kilogram) supplemented by a 5 mg. dose every half hour.

A large cannula was placed in the left jugular vein and attached by a short rubber tube to a metal T-tube. This was in turn connected by another short length of rubber to an appropriately curved metal cannula which was inserted via the right auricular appendage into the coronary sinus and tied there by a previously placed loop of thread. The
circuit from the auricle to the jugular vein was immediately established so that coronary sinus flow was not obstructed at any time. A damped saline manometer in the circuit between the T-tube and the coronary sinus approximately indicated changes in the coronary sinus pressure. At the conclusion of each experiment fluid was injected into the cannula to check absence of leakage around the cannula; the sinus and surrounding area were also dissected and examined for evidence of obstruction of side branches. In several experiments a small branch leading to the posterior left ventricular wall and entering near the mouth of the sinus had been blocked by the cannula tip, but these experiments are included since each animal served as its own control and the directional changes could be determined.

Coronary sinus flow was recorded optically by shunting the blood through the T-cannula into a venous flow meter. It consisted of a glass burette, firmly clamped into a vertical position. A narrow metal tube extending from the bottom of the burette to several inches above it was connected to a sensitive Gregg manometer by lead tubing. The whole system was filled with saline. Blood from the coronary sinus was diverted through the T-tube into the burette; as the blood level in the burette rose, the resulting change in hydrostatic pressure within the metal tube was transmitted to the Gregg manometer. The flow meter was standardized under static conditions by adding increments of the animal's own blood and recording the deviation of the manometer beam. In short, the pressure changes could thus be expressed in terms of volume flow. Calibrations of the manometer were in all cases linear. After each record the blood was drained from the burette and reinfused into the animal. Flow was calculated from the records for successive 10 second intervals and plotted as in the accompanying figures. Aortic and right ventricular pressures were also recorded with calibrated Gregg optical manometers by methods that need not be again described. Right ventricular pressure was elevated by drawing up an adjustable loop of cord placed around the pulmonary artery just distal to the conus. An effort was made to produce sufficient constriction to elevate right ventricular pressure without inducing significant changes in aortic pressure.

Results

Forty-three sets of observations were made on 12 dogs whose mean arterial pressure was around 100 mm. Hg or above. In these, mean coronary flow was continuously recorded with aortic and right ventricular pressures during a control period, during pulmonary artery constriction, and after release.

Effects of Elevating Right Ventricular Pressure. In 8 out of 43 observations coronary sinus flow fell immediately after pulmonary artery constriction and remained low during several minutes of compression. Three of these could be accounted for by the marked decline of aortic pressure to less than 60 mm. Hg, a result of too great pulmonary artery constriction. Two other experiments were complicated by ventricular alternation. The three other observations were not the initial ones in the animals. Data from one of these eight experiments are shown in figure 1.
in the majority of our experiments, it is regarded as the typical one for a heart in prime condition.

A representative observation from this group is illustrated in figure 2. No significant change in aortic pressure occurred but, after a control period, abrupt elevation of right ventricular pressure at C diminished the flow from 42 to 20 cc. per minute. During this same period coronary sinus flow dropped from 30 cc. to 23 cc. per minute. However, although aortic pressure, 20 seconds after pulmonary artery constriction, was only 92/64 mm. Hg, coronary sinus flow had risen to the control level (30 cc. per minute), and at the end of five minutes of elevated right ventricular pressure, when aortic pressure was the same as control, it had risen to 42 cc. per minute. Evidently the greater coronary sinus flow could not have been due to changes in aortic pressure. Finally, the data from 20 observations in which aortic pressure changes were slight following pulmonary artery constriction were averaged for successive 10 second periods. The average pattern was a 4 per cent fall of coronary sinus flow within 10 seconds after constriction and a secondary 15 per cent rise with respect to the control flow at the end of five minutes.

Effects of Reducing Right Ventricular Pressure by Releasing Pulmonary Artery Constriction. In only 6 of the 36 observations under consideration did an immediate drop in coronary flow follow pulmonary artery decompression. Three of these observations were final ones in animals and in one case cardiac alternans was present. Since the heart may have been impaired the reactions might not represent normal ones. However, in no case did the coronary sinus flow decline to previous control levels. Figure 3 illustrates one of the other experiments in which the heart was judged to be in good condition. Coronary sinus flow decreased immediately from 42 to 38 cc. per minute as right ventricular pressure returned essentially to control levels; but it may be noted that the return of coronary sinus flow only gradually returned to control levels, 40 seconds after decompression.

In 10 observations a slight increase of coronary sinus flow 5 to 10 seconds following release of pulmonary artery constriction was noted. In six of these, however, there had previously been a depression of aortic systolic pressure because of excessive pulmonary artery constriction. However, increase of flow from 45 cc. per minute to 50 cc. per minute following abrupt lowering of right ventricular pressure is demon-
strated in the experiment plotted in figure 1, in which aortic pressures showed no significant changes. However, in this experiment coronary sinus flow had fallen from 54 cc. per minute to 34 cc. per minute in the first 10 seconds following pulmonary artery constriction, and had risen to 45 cc. per minute in the subsequent 20 seconds of increased right ventricular pressure.

In 20 observations coronary sinus flow remained unchanged for the first 5 to 20 seconds after release of pulmonary artery constriction. After this interval it always decreased. The sudden marked reduction of right ventricular pressure did not affect coronary sinus flow. This reaction occurred in 11 first trials on different dogs and is considered the most characteristic reaction. These experiments are illustrated by the graph of figure 2. With release of the pulmonary artery constriction at R the coronary sinus flow remained at the same level (49 cc. per minute) for the next 10 seconds, although right ventricular pressure suddenly fell from 39/5 mm. Hg to 29/3 mm. Hg. In the next 30 seconds coronary sinus flow fell progressively to 41 cc. per minute, although aortic pressure showed no significant changes.

**Discussion**

The observations presented show that in good preparations augmentation of coronary sinus flow following elevation of right ventricular pressure by pulmonary compression is a delayed, not an immediate effect; indeed, the greater flow is preceded by a primary reduction. Further, it was found that coronary sinus flow remains augmented and only gradually returns to control values after release of pulmonary artery constriction and restoration of natural right ventricular pressures.

These observations preclude the conception that the changes in coronary flow are mechanical effects caused by elevation of right ventricular pressure. The hypothesis of Wiggers and Johnson that the augmentation of coronary flow is due to diversion of blood from the right ventricle via thebesians to the coronary sinus is therefore not supported.

Since the coronary sinus flow increases when aortic pressure remains the same or falls slightly, some peripheral vascular mechanism must be postulated. Gregg and his associates suggested that metabolites released through increased cardiac work might exert a dilator action on coronary vessels. However, since the greater work is limited to the right ventricle, while the bulk of blood draining into the coronary sinus is derived from the left coronary arteries, it seems improbable that metabolites originating in the right ventricle could dilate arterioles in the left. Accordingly one of two possible explanations needs to be adopted: (1) the suggestion of Gregg and his associates that the augmented coronary sinus flow is due to metabolic dilation of left coronary branches which supply the right ventricle, or (2) that the right coronary artery contributes relatively more blood to the coronary sinus outlet than is postulated by these investigators. Our experiments do not allow a final decision between these two alternatives. However, it may be noted in the graphs of figures 2 and 3 that the stabilized coronary sinus flow during pulmonary artery compression augmented 18 and 25 per cent respectively. Furthermore, 20 experiments in which aortic pressures remained unchanged were averaged for successive 10 second periods and the average increase in flow at the end of five minutes amounted to 15 per cent. It has certainly not been demonstrated that the left coronary twigs overlapping the right ventricle are sufficient in number and size so that their dilation could increase coronary sinus flow by 15 per cent or more. Also, the initial reduction of coronary sinus flow following pulmonary compression must involve right ventricular tributaries regardless of whether they are derived from the right or left coronary arteries. The conclusion therefore seems warranted that quantitative or even directional changes in coronary sinus outflow cannot safely be used as a criterion of left ventricular blood flow under altered dynamic conditions. The importance of this conclusion in the study of metabolic changes in the left ventricle based on studies involving coronary sinus blood is apparent.

**Summary**

Coronary sinus flow, aortic pressure and right ventricular pressure were simultaneously and
continuously recorded during abrupt constriction and release of the pulmonary artery. The sudden elevation of right ventricular pressure resulted in an initial fall of coronary sinus flow in the first 10 seconds and then a secondary rise. This occurred whether aortic pressure was constant or depressed. Following release there was, most often, either a rise or no change of flow for 5 to 20 seconds, gradually followed by a decline of flow.

The time, course and magnitude of these changes do not support the concept that changes in right ventricular pressure are capable of affecting coronary sinus flow by altering thebesian flow into or from the right ventricle. On the other hand, they strongly suggest that a larger and more variable aliquot of the total coronary sinus flow is derived from branches supplying the right ventricle than is presently believed. The importance of this deduction in studies of left ventricular metabolism based on studies on coronary sinus blood should be apparent.

REFERENCES

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