The Effect on Blood Pressure in the Right Heart, Pulmonary Artery and Systemic Artery of Cardiac Standstill Produced by Carotid Sinus Stimulation


In two patients with hypersensitive carotid sinus syndrome, cardiac standstill was induced while pressures were recorded in the pulmonary and femoral arteries and right heart. With onset of ventricular standstill, pressures in the right atrium and right ventricle equalized and gradually increased, that in the femoral artery decreased, and that in the pulmonary artery fell until it became equal to right ventricular pressure, and then increased commensurately with it. All pressures then approached a uniform magnitude asymptotically; this uniform pressure is believed to correspond to the "static pressure." The manner in which the respective pressures returned to normal with resumption of normal cardiac rhythm is described, and certain unexpected findings are discussed.

Although the syndrome of carotid sinus sensitivity has been the object of considerable study,1-3 measurements of intracardiac and pulmonary arterial pressures during cardiac standstill in living man have not been reported. This presentation is the description of the measurements of these pressures during ventricular asystole induced in two patients by the stimulation of a hypersensitive carotid sinus in each.

Procedures and Methods

The patients were white men 44 and 67 years of age respectively. The former (G. S.) had been admitted to the hospital because of recurrent syncopal episodes which were considered to be due to hypersensitive carotid sinus syndrome. In the latter (K. H.), carotid sinus hypersensitivity was an incidental finding. This patient had diabetes mellitus, and moderate elevation of the systolic blood pressure, probably due to arteriosclerosis.

Cardiac catheterization was done with a double lumen catheter; pressures from the catheter lumens and from an indwelling brachial or femoral arterial needle were recorded by Hamilton manometers simultaneously with respiratory movements of the chest wall, recorded by means of a pneumograph, and one lead of the electrocardiogram. All measurements were made with the patient in the postabsorptive state and lying supine. The right carotid sinus was stimulated by externally applied digital pressure. The tracings presented in this report illustrate changes following stimulation of the right carotid sinus. In each patient asystole was induced during the simultaneous recording of pulmonary arterial and right ventricular pressures, and again while recording simultaneously right ventricular and right atrial pressures.

Results

The pressures in the right heart and pulmonary artery of each patient were normal during the control periods. In both patients ventricular asystole promptly followed stimulation of the carotid sinus, and lasted from 5 to 10 seconds, terminating in resumption of ventricular contractions despite continued pressure on the sinus.

In each instance, ventricular asystole occurred abruptly without any preliminary alteration of rhythmic cardiac contractions. With the onset of ventricular standstill, peripheral arterial pressures immediately diminished (fig. 1), rapidly at first and then more
slowly, falling to lowest values ranging from 14 to 22 mm. Hg. The right atrial and right ventricular pressures concurrently tended to equalize, and increased through an average of 6 mm. Hg. Pulmonary artery pressure at first fell until it was slightly less than right ventricular pressure, after which pulmonary artery and right ventricular pressures increased commensurately.

One or two atrial contractions occurred during ventricular standstill in two instances (figs. 1 and 2). The resulting pressure increment within the atrium is reflected by pressure increases of similar magnitude in the right ventricle and pulmonary artery. In certain tracings (fig. 2) there are visible momentary increases of the femoral arterial pressure coincident with atrial systole. Similar pulses were described by Wiggers and have been seen in many other tracings in this laboratory: they probably are caused by transmission of the pressure pulse from the left atrium through the left ventricle to the closed aortic valve.

The various pressures were influenced to a progressively increasing degree by the vigorous respiratory movements of the hyperpnea that developed after five to nine seconds of cardiac arrest. Expiration augmented, and inspiration reduced, all pressures. It is likely that the pneumograph, which merely indicates movements of the chest wall, is not, in point of time, a reliable index of intrathoracic pressure when respirations are abrupt. This deficiency may explain the apparent coincidence in some records (figs. 2 and 3) of a drop in all measured pressures with the attainment of maximal expiration.

With the resumption of ventricular contractions the peripheral arterial systolic and diastolic pressures rose progressively with each beat. In no case was the peripheral arterial pulse pressure greater during recovery than in the control period. In contrast, in certain instances (fig. 2) some of the first cardiac contractions were accompanied by higher systolic, diastolic and pulse pressures in the pulmonary artery than during the control period at comparable phases of respiration. When such increases in pulmonary artery pressure occurred, they apparently developed with those systoles which accompanied the inspiratory phase of respiration.

With resumption of ventricular contractions, the pulmonary artery pressures in every instance returned to control values much more quickly than those in the systemic artery.

**Discussion**

The course of events following carotid sinus stimulation in these patients may be construed as follows: ventricular arrest occurs with the heart in diastole. The tricuspid and presumably the mitral valves open with diastole as usual, permitting equalization of the atrial and ventricular pressures on each side of the heart. Because the pulmonic and aortic valves are closed, the relatively high pressure in the pulmonary and aortic arteries causes blood to flow into their respective venous beds, distending and increasing the pressure within them. Thus, from the beginning of asystole there is a progressive increase of blood volume and pressure within the systemic venous tree and right heart on one hand, and the pulmonary veins and left heart on the other. The pressure within the pulmonary artery, however, falls for only one or two seconds when it becomes lower than the concurrently increasing right ventricular pressure. At this point the pulmonic valve opens and as pressure in the right heart continues to rise, blood flows into the pulmonary artery causing the pressure within it to rise also. Thus, from the time of opening of the pulmonary valve the combined volume of blood in the pulmonary vessels and left heart increases progressively (with certain interruptions as described below) above that present normally.

From the foregoing analysis, it is clear that during cardiac standstill blood will continue to flow as long as there is a gradient of pressure from the aorta through the greater and lesser circulations to the left ventricle. As asystole continues, this gradient approaches zero and pressures throughout the vascular bed approach a uniform level: static pressure.

The concept of static pressure, suggested by Starling and studied by Starr, refers to the residual pressure which would exist throughout the entire vascular tree if the
pumping action of the heart terminated and blood flow ceased. Starr measured intracardiac pressures immediately after death in humans who did not have congestive heart failure, and found static pressure to be 5 to 16 mm Hg. He recognized that change of vascular tone probably occurs at death, and while his values therefore may be subject to error, they nevertheless were consistent from patient to patient and probably serve as a good index of the actual magnitude of true static pressure.

In the tracings which form the basis for this report, all pressures during ventricular standstill approach a uniform pressure asymptotically. They indicate, therefore, that static pressure in our patients under these circumstances is between 9 and 18 mm Hg: the upper limit is the average lowest value reached by the peripheral arterial pressure, and the lower limit is the average highest pressure attained in the great veins and right heart. These values are subject to error owing to the influence of the diminution of vascular tonus which may be an element of the response to carotid sinus stimulation in most cases, and owing perhaps to vascular response to asphyxia. Furthermore, it has been shown that respiratory movements can produce circulation of blood through the vascular system in the absence of cardiac contraction. Therefore, even if asystole had been greatly prolonged in these cases, blood flow might never have stopped completely as long as respiratory movements persisted—and static pressure thus would not be reached. However, the nearness of our values to those found by Starr suggest that both approximate the true value for static pressure.

Toward the end of the period of ventricular asystole, all blood pressures were influenced to a greater degree by respirations than during the control period. It is probable that the increased forcefulness of the respiratory movements during cardiac arrest contributes to this alteration. Furthermore, it has been shown above that as asystole continues the volume of blood in the pulmonary vascular bed and left heart becomes greater than in the normal state. As a result the lungs become more turgid than normally and the alterations of thoracic volume due to respiratory movements may be expected to be accompanied by greater pressure variations within the thoracic cavity and the pulmonary vessels.

While it is hazardous to subject records of this type to minute analysis, it nevertheless appears that, of all the pressures, that in the pulmonary artery varies the most with respirations; and that on certain, if not all, occasions the pressure in the pulmonary artery exceeds that in the right ventricle briefly during expiration. Accordingly the pulmonary valve must close momentarily, and the flow of blood from the right ventricle into the pulmonary artery may then be interrupted briefly.

Despite the fact that during cardiac standstill each ventricle probably accumulates a larger volume of blood than in normal diastole, the pulse pressure in the systemic arteries is not increased above control magnitudes in any of the recovery beats. This may be due to two

Fig. 1. Patient K. H. From above downward: femoral artery, right ventricle, right atrium. The pneumogram is superimposed on the intracardiac pressure tracings. In all of the tracings, downward movement of the pneumograph represents inspiration. Ventricular asystole lasted nine seconds. Two atrial contractions occurred during this period. The second of these may have come from an ectopic focus within the atrium, for no typical P is identifiable in the electrocardiogram. Despite the relatively long duration of ventricular asystole, there is no increase in right ventricular pulse pressure during recovery.

Fig. 2. Patient K. H. From above downward: femoral artery, pulmonary artery and superimposed pneumogram, right ventricle. Ventricular standstill lasted 10 seconds. Two atrial contractions occurred during ventricular asystole. Small pulses are identifiable in the femoral artery tracing which coincide with atrial contractions. The pulses in the right ventricle and pulmonary artery are increased in the first, second and third recovery beats.

Fig. 3. Patient G. S. From above downward: brachial artery, pulmonary artery, right ventricle, pneumogram. Ventricular standstill lasted 6.5 seconds. The apparent coincidence of a drop in all pressures with attainment of maximal expiration, during asystole, is probably artefactual.

Fig. 4. Patient G. S. From above downward: brachial artery, right ventricle, right atrium, pneumogram. Ventricular standstill lasted eight seconds. The pulse pressure in the third recovery beat of the right ventricle is greater than in the control.
factors. Peripheral arteriolar relaxation may occur during activation of the hypersensitive carotid sinus reflex. This event in itself would permit an increase in stroke volume to be introduced with relatively small pulse pressure. Furthermore, the process of “run-off” of blood from the arterial to the venous side during ventricular asystole depletes the arterial blood volume and, as a result, the systemic arteries are less distended with blood. This development, too, will permit the introduction of an increased stroke volume with a relatively small increment of pressure.

In every instance the blood pressure in the pulmonary artery returned to normal during recovery much more quickly than that in the systemic artery. In certain instances, the pulmonary artery systolic, diastolic and pulse pressures exceeded those of the control period, whereas this never was the case in the systemic artery. One possible reason for these differences in the behavior of pressure between the greater and lesser circulations is that by the end of asystole the pulmonary vascular bed and left heart contain more blood than normally, while the systemic circulation is correspondingly depleted of blood. Systolic discharges into the somewhat distended pulmonary vessels will, therefore, be attended by relatively higher pressures than in the case of the aortic artery. Further, it is possible that the right ventricle, which is probably more distensible than the left, accumulates a greater presystolic volume than the latter, and its first systolic ejections may be greater than those of the left ventricle. The minor augmentation of blood flow that occurs during inspiration may account for the coincidence of the increase of pulse pressure with this phase of respiration. Why this increase is present in some instances and not in others, despite the coincidence of systole with inspiration, we are unable to explain.

**Summary**

In two patients ventricular standstill was induced by stimulation of a hypersensitive carotid sinus, while respiratory movements and pressures in the pulmonary artery, right heart, and a peripheral artery were recorded.

In every instance of ventricular asystole, the systemic arterial pressure fell, and right heart pressure rose. The pulmonary artery pressure at first fell until it equalled right ventricular pressure; then it rose commensurately with that in the right ventricle. All pressures then asymptotically approached a uniform magnitude which is thought to approximate “static pressure.”

The hyperpnea which developed during asystole exerted an exaggerated effect upon the central blood pressures.

With resumption of ventricular contractions, all pressures returned to the control values. The pressures in the pulmonary artery recovered more quickly than those in the systemic arteries; occasionally the former exceeded the control values during some of the recovery systoles.

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**References**


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