Effect of cardiac rhythm on vena caval blood flows

ALAN L. PINKERSON, MYRON H. LURIA, AND EDWARD D. FREIS
Veterans Administration Hospital and Department of Physiology, Georgetown University School of Medicine, Washington, D. C.

Effect of cardiac rhythm on vena caval blood flows. Am. J. Physiol. 210(3): 505-508. 1966.—Cannulating electromagnetic flow probes were inserted into superior and inferior venae cavae of anesthetized mongrel dogs. The relationship of instantaneous vena caval blood flows to intracardiac pressures and the surface electrocardiogram was observed during normal and various abnormal heart rhythms, including isolated atrial beats, and ventricular systoles not immediately associated with atrial activity.

METHODS

Twenty-five mongrel dogs were anesthetized with 25 mg/kg sodium pentobarbital intravenously. The chest was opened in the right fourth intercostal space and respirations maintained with intratracheal positive pressure. After the administration of 5 mg/kg heparin sodium, the intrathoracic portion of the inferior vena cava was dissected free from surrounding tissue and occluded at the diaphragm and entrance to the right atrium withatraumatic clamps. The occluded segment of vena cava was divided and the ends of a Statham cannulating electromagnetic flow probe (Q series, i.d. 7 mm) were inserted and tied into the cut ends of the vein. The superior vena cava was then dissected free, occluded, divided, and a similar flow probe was tied in place. A Dallons-Telco catheter-tip manometer was passed through the ligated azygous vein at the cardiac end of the superior vena cava flow probe and advanced into the right atrium. Right ventricular and aortic pressures were recorded through standard cardiac catheters. The surface electrocardiogram was monitored throughout each study. Blood flow in the ascending aorta or main pulmonary artery, when obtained, was detected with a Statham cannulating electromagnetic flow probe (Q series). All physiological parameters were recorded on a Sanborn multichannel direct-writing oscillograph.

After initial observations during normal sinus rhythm, various arrhythmias were induced by direct stimulation of the atria or ventricles, electrical stimulation of a vagus nerve, or by the administration of digitalis, acetylcholine, or norepinephrine. Changes which occurred during spontaneous arrhythmias also were recorded. In five
animals the thoracotomy incision was closed. With unassisted respiration and negative intrathoracic pressure breathing, similar parameters were recorded during normal and abnormal heart rhythms.

RESULTS

During normal sinus rhythms blood flow decreased in both venae cavae at the time of the atrial pressure rise associated with atrial contraction. In early ventricular systole atrial pressure declined and blood flow increased in the venae cavae (Fig. 1). Peak atrial pressure either coincided with or preceded the maximum decrease in flow by up to 40 msec. The ratio of decreased flow associated with atrial contraction to the subsequent increase was variable. Later in ventricular systole and in early ventricular diastole blood flow in the venae cavae decreased.

Figure 2 illustrates similar parameters recorded during atrial activity at the time of ventricular standstill produced by electrical stimulation of the left cervical vagus nerve. The timing, magnitude, and configuration of the flow profiles in the venae cavae, which were associated with the isolated atrial beats, closely approximated those seen during normal sinus rhythm. Following a decrease in flow associated with atrial contraction, flow increased at the time of atrial relaxation. The flow increase was not significantly influenced by the absence of an associated ventricular systole.

During ventricular systoles not preceded by atrial contractions, the normal phase of rapid atrial inflow in early ventricular systole was absent. In Fig. 3, ventricular escape beats occurred during electrical stimulation of the right cervical vagus nerve. With the isolated ventricular beats, right atrial pressure initially declined slightly and then increased throughout the remainder of ventricular systole. The venae cavae blood flow changes were opposite in direction to the changes in atrial pressure. Blood flow now decreased during ventricular systole as...
opposed to the normal increase in forward flow. In early ventricular diastole, at the time of opening of the tricuspid valves, flow began to increase instead of the continued decline recorded during normal sinus rhythms. When a ventricular beat was accompanied by an atrial systole rather than the atrial relaxation which normally occurs at this time, marked reversal of flow was recorded in the venae cavae (Fig. 4). This period of regurgitant blood flow from the atrium into the venae cavae was associated with a large increase in intra-atrial pressure. The subsequent phase of rapid atrial inflow, which was also markedly accentuated, occurred during the falling limb of the atrial pressure wave in late ventricular systole or early ventricular diastole.

Although all of the illustrations shown were recorded in animals with an intact pericardial sac, its removal in no instance altered the records obtained. In animals with the thoracotomy incision closed and with normal spontaneous negative-pressure respirations, pressure and flow patterns were superimposed on respiratory variations. The pressure and flow changes during normal and abnormal cardiac cycles, however, were qualitatively similar to those recorded in the open-chest animal.

**DISCUSSION**

Although the use of electromagnetic flow probes for the measurement of instantaneous vena caval blood flows has been suggested (2), they have not, to our knowledge, been previously used in the venae cavae. The records so obtained successfully demonstrated the time relationships between instantaneous blood flow into the right atrium as correlated with the various electrical and pressure events of the normal and abnormal cardiac cycles. The electromagnetic flow probes were of high sensitivity with a low signal-to-noise ratio, frequency response ±10% at 100 cycles/sec, and linearity within the flow range studied. The cannulating type of flow probe prevented vessel constriction and base-line shifts even when the probe was subjected to cardiac and respiratory motions.

The instantaneous blood flow patterns observed in the venae cavae during normal cardiac cycles agree with those reported by previous investigators (3, 4). As in the
present experiments, these earlier studies in man and laboratory animals without tricuspid insufficiency have demonstrated decreased flow with atrial systole and increased flow with atrial relaxation and ventricular systole. Since the tricuspid valve is closed during ventricular systole, the rapid increase in blood flow must result in accumulation of blood in the right atrial cavity. The subsequent decreased vena caval blood flow which continued beyond the time of opening of the tricuspid valve suggests that the blood available for rapid filling of the ventricle in early diastole had been obtained primarily from the right atrium rather than from the venous reservoirs.

Intra-atrial pressure contours and vena caval blood flows during isolated atrial activity closely resembled the pressure and flow waves recorded during the immediately preceding sinus beats. This similarity indicated that atrial rather than ventricular activity is the main determinant of the fluctuations in vena caval blood flow recorded during normal cardiac beats. The slight decrease in peak flow velocity during isolated atrial relaxation, however, may be attributed both to the absence of downward motion of the atrioventricular ring described during normal ventricular contractions (2) or to a gradual increase in right heart pressures, since the right ventricle is not emptying.

When ventricular systole is not accompanied by an atrial relaxation wave, the normal phase of rapid atrial inflow during early ventricular systole is not seen. Atrial inflow now declines during ventricular contraction and increases when the tricuspid valve opens in early diastole. This early diastolic increase in vena caval flow shows that ventricular filling at this time is now associated with and may be more dependent on increased blood return from the veins.

Although the loss of rapid atrial inflow during early ventricular systole can be attributed entirely to the absence of a simultaneous atrial relaxation, other possible contributing factors should also be mentioned. In the absence of a preceding atrial contraction, prelosure of the tricuspid valve does not occur and the atrium may be partially filled by regurgitant blood from the right ventricle (6). Also the normal downward motion of the atrioventricular ring may not be as effective in the isolated ventricular beat as in normal sinus rhythm.

When atrial and ventricular systoles occur simultaneously, marked alterations are produced in the normal pattern of right atrial inflow. During such an arrhythmia the atrium contracts against a closed tricuspid valve and produces an augmentation of intra-atrial pressure. Since intra-atrial blood cannot move into the ventricle, it is driven back into the venous system causing reversal of flow. With atrial relaxation and consequent fall in intra-atrial pressure, blood again flows rapidly back into the right atrium.

The present studies demonstrate that a disturbance in the normal sequence of atrial and ventricular contractions has considerable influence on instantaneous venous return to the heart. During normal rhythms, atrial relaxation at the time of ventricular systole induces a rapid flow of blood from the venous system into the atrium. This provides a reservoir of atrial blood which is available for rapid ventricular filling in early diastole. When the normal relationship between atrial relaxation and ventricular contraction is disturbed, early ventricular filling may be more dependent on blood drawn from the venous reservoirs.

REFERENCES