Heart and Lungs as a Common Chamber during Extracorporeal Support of the Fibrillating Canine Heart

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Ventricular fibrillation was induced by electric shock in both closed- and open-chest animals. Total body perfusion was provided by extracorporeal circulation with a bubble oxygenator. The failure of left heart pressure to increase during prolonged observations indicates the probable competency of the aortic valve and retrograde flow of bronchial arterial blood to the right heart. Evidence for left-to-right flow is shown by reciprocal pressures in parallel chambers, by recovery of isotope in the pulmonary artery following left heart and aortic injection and by a cardiopneumonogram. The existence of a common mean pressure in the left heart, lungs and right heart during ventricular fibrillation confirms the hypothesis that the central circulation is in essence a system of tubes permitting the passive transfer of blood with the prevailing gradient.

SINCE ventricular fibrillation occurring spontaneously or experimentally is generally an acute phenomenon, few deliberate studies of the central circulation had been made prior to the availability of pump oxygenators. Under the circumstance of total body perfusion and induced ventricular fibrillation, systematic investigation of the hemodynamic adjustment of the cardiac and pulmonary circulation is possible.1,2 This investigation was undertaken to determine the blood flow pattern existing in the cardiopulmonary circuit when the propulsive action of both ventricles had ceased and extracorporeal function substituted. Because of the great change in pressure gradients at the onset of ventricular fibrillation, a complete reversal of normal antegrade flow is possible.

During studies with deliberate prolonged ventricular fibrillation supported with the pump oxygenator, it was noted that left heart dynamics closely paralleled induced changes in the right heart.4 Thus with a steady mean pressure in the right atrium the left atrial or ventricular pressures would immediately reflect changes in right heart pressure. In addition the failure of left heart pressures or volume to increase with time suggested the retrograde return of bronchial artery blood to the right heart. Evidence will be presented that during this experimental state all cardiac valves except the aortic are incompetent, permitting heart chambers and pulmonary vessels to function essentially as a common chamber.5 A common chamber is defined as a system of relatively nondeformable transfer tubes in which normal transvalve pressure gradients within the heart are absent and both retrograde and antegrade circulation may exist from right-to-left heart.

METHODS

Twelve healthy mongrel dogs of both sexes weighing from 15 to 25 Kg. were anesthetized with sodium pentobarbital (30 mg./Kg.) and artificially resired.

In closed chest experiments a No. 20 French Bardex catheter was introduced into the femoral vein and positioned in the inferior vena cava at the level of the diaphragm. In the open-chest animal the catheter was also introduced through the femoral vein and correct positioning in the cava was checked by palpation. The catheter was connected to half-inch Tygon tubing and led to the venous pump. The return of venous blood by gravity or syphonage was not as satisfactory in obtaining venous return as direct suction during the fi-
Fibrillating Heart and Lungs as Common Chamber

brilliating state. Following cannulation the animal was heparinized with 2 mg./Kgr.* The vertical stainless steel bubble oxygenator has been previously described.† Modified de Bakey roller type pumps‡ were used with a capacity of 4 L/min. flow. The arterial and venous pumps were occlusive and were calibrated for minute flow against a known resistance. The femoral artery was utilized for arterial return from the pump. The coronary sinus pump and reservoir were unnecessary for this preparation.

The arterial pump was not set to maintain a given arterial pressure as we preferred to regulate the system through control of the venous pump. Following the onset of ventricular fibrillation the venous pump was adjusted to withdraw the maximum available return. This speed was generally just under that necessary to produce venous flutter. Under these circumstances the mean arterial pressure appeared to be the resultant of the attainable perfusion rate and existing total peripheral resistance.

Arterial pressure was monitored in a femoral or carotid artery with a Statham P23D pressure transducer and recorded on a Sanborn Twin-Viso. The electrocardiogram was similarly recorded. Venous pressure was measured from the external jugular vein with a water manometer. Measurements of intracardiac pressures were made before, during and after the fibrillatory state. A zero reference point was adjusted to a midatrial level. In the closed-chest animal, a polyethylene catheter was introduced into the right ventricle from the external jugular vein and pressure recorded with the Statham transducer. After the induction of fibrillation the catheter was withdrawn across the tricuspid valve to determine the existing gradient. For left atrial pressure determinations, a bronchoscope was introduced through a tracheostomy and the atrial wall punctured with a transbronchial needle. Pressures in the left ventricle were measured by introducing a polyethylene catheter through a needle in turn introduced by direct anterior percutaneous puncture. In the open-chest animal, direct puncture of all cardiac chambers and large vessels was performed for determination of transvalve gradients.

Ventricular fibrillation was induced by a single electric shock applied to the closed chest or exposed heart. The average strength of the stimulus was 150 V at 0.5 sec. Most of the open-chest animals were sacrificed at the completion of the experiment although initial defibrillation was uniformly successful. In the intact animal, defibrilla-

*Abbott Company, Chicago, Ill.
†Mark Company, Randolph, Mass.
‡Abbott Company, Chicago, Ill.

Table 1.—Average Levels of the Measured Pressures in mm. Hg in Three Open Chest Dogs

<table>
<thead>
<tr>
<th></th>
<th>Pre fibrillation</th>
<th>Ventricular fibrillation (mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>4/2</td>
<td>4</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>15/0-2</td>
<td>4</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>20/0-2</td>
<td>4</td>
</tr>
<tr>
<td>Left atrium</td>
<td>6/3</td>
<td>4</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>110/0-3</td>
<td>4</td>
</tr>
<tr>
<td>Aorta</td>
<td>110/90</td>
<td>92</td>
</tr>
<tr>
<td>Femoral artery</td>
<td>110/90</td>
<td>90</td>
</tr>
</tbody>
</table>

*Abbott Company, Chicago, Ill.
†Mark Company, Randolph, Mass.

RESULTS

Transvalve Gradients

In order to determine the status of cardiac valves during ventricular fibrillation and total body perfusion, pressures were registered directly from the chambers and large vessels in 3 open-chest animals (table 1).

Following the induction of fibrillation and establishment of pump oxygenator support, the pressures were again determined in the respective cardiac chambers and large vessels. It was evident that a mean pressure of approximately 4 mm. Hg existed throughout both sides of the heart and lungs. In the fibrillating left ventricle, mean pressures varied from 2 to 6 mm. Hg, while the left atrium with its independent beat registered pressures from 4 to 8 mm. Hg. The pulmonary artery pressure varied from 4 to 8 mm. Hg, while the right ventricle and right atrium varied from 2 to 6 mm. Hg. During the period of these measurements the femoral artery pressure was 90 mm. Hg, while the aortic root pressure was 92 mm. Hg.

Following the completion of pressure measurements, the heart was promptly defibrillated with a single shock of 220 V at 0.5 sec. and maintained without further support a peripheral arterial pressure of 130/80 mm. Hg.

The absence of any differential pressure on either side of the mitral, tricuspid or pulmonary valve indicates the functional incompetence of these valves during this experimental state. The failure of left ventricular
Effect of Changes in Right Heart Volume on Atrial Pressures

In 2 closed-chest animals a comparison was made of the effects of increasing right heart blood volume on right and left atrial pressure during ventricular fibrillation. Although right heart pressure could be raised by increasing the rate of aortic perfusion, this flow during steady state conditions was already near maximum. With slowing of the venous pump and maintenance of the arterial pumping rate, a prompt increase was noted in right atrial pressure (fig. 1). Under these conditions the lung in effect becomes an acute venous pool. Unless measures are undertaken to immediately reduce the right heart pressure, bloody, irreversible pulmonary edema will ensue. In our experience this has generally occurred at levels exceeding 30 mm. Hg. It is clear that during ventricular fibrillation 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. If this gradient is maintained, i.e., by continuing the arterial pump, there is no opportunity for the lungs or left heart to decompress. On the other hand, if the arterial pump is stopped simultaneously or just prior to stopping the venous pump, an initial rise of pressure will occur in the right heart, but eventually this gradient approaches zero and pressures through the vascular bed approach a uniform level-static pressure.

Prefibrillation pressure in the left atrium ranged normally from 1 to 3 mm. Hg more than the right. During ventricular fibrillation and retrograde aortic perfusion this difference was maintained. Pressure changes in the right atrium were immediately reflected in the left atrium. A spontaneous or deliberate increase of right heart volume was always paralleled by a subsequent rise in left atrial pressure. Again, as right atrial pressure decreased the left atrial pressure would fall, remaining 2 to 5 mm. Hg higher than the right.

The common chamber effect of the pulmonary bed and heart during supported ventricular fibrillation is seen during increases in right heart pressure. The gradient establishing retrograde blood flow from the left heart is reversed and with continued antegrade flow the lungs and left heart distend...
with blood. A recording catheter in the left ventricle reflects a steady, nonfibrillating left atrial wave increasing in amplitude with the corresponding increase in right heart pressures (fig. 2).

**Left-to-Right Heart Flow**

In order to demonstrate a blood flow pattern under the given experimental conditions from left-to-right heart the following radioisotope studies were carried out. The best results from a series of 6 open-chest animals are reported:

* **Aorta to Left Atrium and Pulmonary Artery.** Twenty-five µc. of I-131 (HSA)* was injected retrograde via the femoral artery catheter used for arterial input from the pump oxygenator. After injection, samples of blood were withdrawn simultaneously at 2 sec. intervals by means of a double automatic pipetting machine,‡ adjusted to deliver 1.0 ml. into a sample vial each 2 sec. The first sampling site was left atrium. Following equilibration a background sample was determined and an injection of 50 µc. made into the aorta from the femoral artery. Sampling was done from the main pulmonary artery.

**Results.** The early appearance of the tracer substance in the left atrium is anticipated from the known bronchial artery inflow to this chamber10 (fig. 3). The delay in appearance of the isotope in the pulmonary artery which reached a maximum concentration from three to four minutes after injection is consistent with a low volume of blood flow from the left-to-right heart.

* **Left Atrium to Pulmonary Artery and Right Ventricle.** Twenty-five µc. of I-131 (HSA) was injected into the left atrium. Simultaneous collections were made from the pulmonary artery and right ventricle.

**Results.** The maximum recovery of the isotope in the pulmonary artery was detected 5 min. after injection. Further delay in appearance time was seen in the right ventricle where the peak recovery was between 8 and 10 min. (fig. 4). Because of the very low volume and rate of flow from left-to-right heart, these data are not considered of quantitative value. The rate of retrograde flow in the pulmonary artery has been measured in

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*Human Serum Albumin, Abbott Laboratories.
†Filamatic Duplex Automatic Pipetter, Model DAB, National Instrument Company, Baltimore, Md.
separate experiments and found to vary from 8 to 10 ml./min.

**Aorta to Pulmonary Artery, Left Ventricle to Pulmonary Artery with Proximal Occlusion of Both Vessels.** In this experiment 25 μc. of I-131 (HSA) was injected directly into the femoral artery in a retrograde direction. To avoid any possibility of reflux blood flow from either the aorta to left ventricle or right ventricle to pulmonary artery, a Potts clamp was placed across the root of both vessels at the time of injection. Sampling was carried out from the pulmonary artery on the lung side of the occluding clamp. The clamp across the great vessels was released and following equilibration an injection of 50 μc. of I-131 (HSA) was made into the left ventricle. The occluding clamp had been reapplied to the aorta and pulmonary artery just prior to the second injection. Sampling was again taken from the pulmonary artery.

**Results.** The peak of the recovery curve following aortic injection and pulmonary artery sampling was seen in the first 1 and 2 min. (fig. 5). This suggested that the major portion of bronchial artery blood returned to the left atrium and was then redirected in a retrograde manner through the pulmonary capillary bed to appear in the pulmonary artery. On the other hand, it is known that not all bronchial artery blood is returned to the left atrium but may partition and flow via bronchial-pulmonary artery shunts (precapillary) directly into the pulmonary artery.

The absence of any isotope recovery in the pulmonary artery following left ventricular injection was not unexpected. With the pulmonary artery occluded, the left-to-right gradient is not transmitted to the left chambers. Bronchial artery blood coming from the high pressure aorta apparently flows to areas of low resistance which, in this instance, included is both left atrium and pulmonary artery. This would explain the presence of the isotope in the pulmonary artery in the absence of a negative gradient transmitted from the right heart. Blood in the left ventricle, on the other hand, coming possibly from Thebesian but largely from a left atrial source has no escape except to return to the left atrium, where the pressure is equal or greater. In the absence of any pressure head or transmitted gradient, blood in the left ventricle is essentially static.

**Aorta to Pulmonary Artery, Left Ventricle to Pulmonary Artery with Aortic Occlusion Only.** This experiment was identical with the previous one with the exception that only the aorta was occluded by the Potts clamp. An injection of 25 μc. of I-131 (HSA) was made in a retrograde direction into the abdominal aorta from the femoral artery catheter. Sampling was done in the pulmonary artery. After equilibration an injection of 50 μc. of I-131 (HSA) was made in the left ventricle and sampling was again carried out from the pulmonary artery.

**Results.** The peak of pulmonary artery collection after retrograde aortic injection was seen at 2 min. (fig. 6). Thereafter there was a gradual decrease in radioactivity over the next 4 min.

The appearance of the isotope in the pulmonary artery demonstrates the left-to-right flow pattern. With the presence of the gradient from left-to-right heart, blood entering the left atrium is shunted to the pulmonary artery to mix with blood appearing from the bronchial-pulmonary artery (precapillary) anastomoses.

The recovery curve in the pulmonary artery following left ventricular injection showed a slow increase to a maximum value at 9 to 10 min. This was followed by a gradual fall off of radioactivity over the next 10 min.

Accumulation in and movement of blood from the left ventricle during fibrillation and extracorporeal circulation appears to be largely dependent on the pressures and volumes existing in the left heart. With blood moving from the left atrium to the pulmonary artery the isotope is slowly drawn in retrograde manner to appear in the pulmonary artery 8 to 10 min. after injection.

**Cardiopneumonogram.** For this experiment 2 dogs were utilized before satisfactory roentgenograms were obtained. Each was
FIBRILLATING HEART AND LUNGS AS COMMON CHAMBER

Fig. 7. A. Control film for catheter position in beating heart. Two milliliters of 70 per cent Urokon filling coronary arteries and descending thoracic aorta. B. Onset of ventricular fibrillation and extracorporeal perfusion. Five milliliters of dye injected. Exposure at 3 sec. Left heart filling principally atria, beginning visualization of pulmonary veins. C. Five seconds after injection. Pronounced left atrial filling with retrograde flow into pulmonary veins. Note absence of ascending or descending aortic filling. D. Thirty seconds after injection. Further filling of pulmonary veins and small venous radicles. E. One hundred and sixty-five seconds, capillary density decreasing, increasing pulmonary artery filling and beginning right heart contrast. F. Four hundred and five seconds, pulmonary artery filling more pronounced. Aortic valve well shown in relief from residual dye in left heart due to low dilution factor. G. After 2 hours of ventricular fibrillation. Dye cleared from heart and lungs. Four milliliters of Urokon, 70 per cent, injected into superior vena cava when venous pressure was 4 cm. saline. Dye being drawn in retrograde direction down inferior cava from negative gradient of pump. H. Effect of stopping both the arterial and venous pumps simultaneously (heart still fibrillating). With decreasing arterial pressure and rising venous pressure, antegrade flow begins. Five milliliters of Urokon injected into superior vena cava when central venous pressure was 12 cm. saline. Filling of both cavae and right heart.

anesthetized and transported to the room containing the Schönänder biplane rapid x-ray film changer. The necessary cannulation of the femoral vessels was performed and a polyethylene catheter was introduced into the left ventricle by anterior percutaneous needle puncture. The lungs were ventilated to aid filling of the vessels and avoid congestion. A scout film was made for a check of catheter location and showed coronary and ascending aorta filling (fig. 7A). Ventricular fibrillation was induced and pump oxygenator support established. When a steady state existed with stable femoral artery pressure of 90 mm. Hg and venous pressure of 3 to 5 mm. Hg, 15 ml. of 70 per cent Urokon was injected into the left ventricular catheter. A series of exposures were made with manual timing. The resulting left to right flow could be visualized as the pulmonary veins, arteries and, subsequently, the right heart and cavae filled (fig. 7B-F).

Discussion.

Although the absence of any transvalve gradient during supported ventricular fibrillation suggests the mitral, tricuspid and pulmonary valves are incompetent, it may also
be a reflection of the minimal flow across the valve. This is particularly true with the mitral valve as seen by the slow movement of isotope from the left ventricle to the pulmonary artery. As the superior vena cava has not been separately cumulated in the present study, blood returning from the head and upper extremity will enter the right atrium. In addition, the normal return of coronary sinus blood is to this chamber. During the cardiopneumonogram it was noted that dye injected into the superior vena cava was drawn into the inferior vena cava (fig. 7G).

Because of the negative gradient existing in the inferior vena cava (transmitted from the venous pump), it is believed that the majority of blood entering the right atrium from whatever source is subsequently drawn into the inferior vena cava to then pass into the extracorporeal circulation. However, a decrease in this gradient with resultant increase in right heart volume will cause right atrial blood to flow to the right ventricle and reflux into the pulmonary artery. Such a possibility was obviated first by attempting to maintain steady state conditions during the injection with a venous pressure of 2 to 4 mm. Hg in the right atrium. Second, the aortic injection of the isotope this material could, under these conditions, enter the right atrium via the coronary circulation and reflux into the pulmonary artery. Such a possibility was obviated first by attempting to maintain steady state conditions during the injection with a venous pressure of 2 to 4 mm. Hg in the right atrium. Second, the aortic injection of the isotope was made while the pulmonary artery was occluded at its root, thus mechanically avoiding reflux from the right heart. The subsequent appearance time in the pulmonary artery was similar, i.e., maximum isotope recovery from 2 to 4 min., whether the pulmonary artery was unoccluded or occluded.

It is recognized that considerable increase in left intraventricular blood volume may occur as from aortic regurgitation prior to elevations in pressure. Thus, pressure determinations may not be a reliable indication of the existence of aortic regurgitation. Several methods were chosen to demonstrate that the aortic valve remained wholly competent during this experimental state, i.e., supported ventricular fibrillation.

1. Intraventricular pressures were always similar to those existing in the left atrium. Increases in right heart volume transmitted through the pulmonary capillary bed were reflected by atrialization of the left ventricle. The pulsatile wave of the pump characteristically seen in the peripheral femoral artery was never seen in the ventricular tracing.

2. Because no apparent increase was seen in left ventricular mean pressure during periods of 1 to 3 hours of supported ventricular fibrillation it was believed that all blood entering this chamber must necessarily reflux into the left atrium and retrograde through the lung. Since no possibility existed of left ventricular pressure increasing to the extent of exceeding aortic root pressure (established by the pump oxygenator), increases in left ventricular volume must decompress by left-to-right heart pathways. In 1 experiment with a polyethylene catheter in both atrium and ventricle for simultaneous pressure recording, increments of 50 ml. of blood up to 500 ml. were injected into the left ventricle with no resultant change in pressure in either chamber.

3. During the isotope studies, injection of I-125 (HSA.) into the left ventricle revealed no reflux of material into the pulmonary artery when it was occluded (fig. 5), whereas with the artery unoccluded an appreciable recovery curve was found (fig. 6). This would indicate the effect of a gradient in the right heart on the movement of blood from the left.

4. To further eliminate the possibility of aortic regurgitation possibly not reflected by pressure measurements, the isotope studies were carried out with clamping of the aortic root, which also occluded coronary flow. In these experiments (fig. 5 and 6), the isotope was detected in the pulmonary artery following aortic injection, regardless of the presence of a right heart gradient, i.e., with the pulmonary artery both occluded and nonoccluded.

Consideration of simultaneous pressure
recordings from both atria during ventricular fibrillation indicates the pendulum effect of blood moving in the lesser circulation. Spontaneous or deliberate increase of pressure in the right heart is nearly instantly paralleled by a corresponding increase in left heart pressure. The gradient for antegrade flow is quite low. In dogs, Rodbard and Wagner performed an anastomosis between the right auricular appendage and the main pulmonary artery, thus by-passing the pumping action of the right ventricle. They were able to demonstrate a flow of venous blood from the auricle to the distal pulmonary artery when the venous pressure in the right atrium was 9 to 14 cm. of saline. In the human, Dexter and associates noted that a gradient of only 6 mm. Hg was needed to propel blood from the pulmonary artery to the pulmonary capillary bed.

As it is known that bronchial arteries provide a continuous flow of arterialized blood both at a pre- and postcapillary level, both the left and right sides of the heart receive this flow during asystole or ventricular fibrillation. The steady low volume of bronchial arterial blood into the left atrium would correspondingly expect in time to result in some increase of left heart pressure if retrograde flow were not possible. As long as the pulmonic valve remains incompetent, the 2 sides of the heart are in free communication through the pulmonary vascular bed. Ross and co-workers found that right atriotomy would thus prevent elevation of left atrial pressure during cardiac arrest and total body perfusion.

**SUMMARY**

It is believed that left-to-right blood flow is a consequence of establishing a gradient in the right heart during ventricular fibrillation and extracorporeal perfusion when a common mean pressure exists in the cardiac chambers and pulmonary vessels. The existence of a common chamber throughout the heart and lungs during this experimental state is shown by the absence of differential pressure gradients across all valves except the aortic. The reflection of pressure changes in the right heart by the left atrium illustrates the reciprocal relationship that exists in a common chamber system through the intermediary of the pulmonary capillary.

In addition to the transmission of pressure changes between left and right heart, both the partition and retrograde pattern of bronchial arterial flow was demonstrated by the detection of radioactive material in the left atrium and pulmonary artery following aortic injection. Following injection of the isotope into either left atrium or ventricle, it was recovered at various intervals in the pulmonary artery and right ventricle. The possibility of aortic regurgitation, although not reflected by pressure studies, was further eliminated by an occlusive clamp.

The movement of blood from the fibrillating left heart to right is largely dependent upon a transmitted gradient from the right. Labeled material in the left ventricle was static unless the pulmonary artery was unoccluded. However blood entering the pulmonary artery at the precapillary level from a bronchial source may be detected in the absence of such a gradient. Finally, visual demonstration of retrograde flow was accomplished by a cardiopneumonogram. Radiopaque material injected into the left ventricle was followed in transit through the pulmonary capillary bed to the right heart and cavae.

**ACKNOWLEDGMENTS**

We are indebted to Dan Hayden, Ph.D. and Mr. William Korcski for technical assistance, and Dr. Harold Dodge, Cardiologist, Veterans Administration Hospital, for review of the manuscript.

**SUMMARIO IN INTERLINGUA**

Es opinante que le fluxo sinistro-dextere de sanguine es un consequentia del estabilimento de un gradienti in le corde dextere durante fibrillation ventricular e perfusion extra-corporee quando un commun pression medie existe in le cameras cardiae e le vasos pulmonar. Le existentia de un camera commun que include le corde e le pulmones in ille stato experimental es demonstrate per le absentia de omne gradiente de pression dif-

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ferential trans omne le valvulas, excepte le valvula aortic. Le reflexion de alterationes del pression in le corde dextere per le atrio sinistre illustra le relation reciproque que existe in un systema a camera commun per le intermediario del capillares pulmonar.

A parte le transmission de alterationes de pression inter le corde sinistre e le corde dextere, tanto le partition como etiam le configuration retrograde del fluxo bronchoarterial esseva demonstrate per le detection de material radioactive in le atrio sinistre e le arteria pulmonar post injectiones in le aorta. Post le injection del isoto in (1) le atrio sinistre o (2) le ventriculo sinistre, illo esseva retrovate post varie intervallos in le arteria pulmonar e le ventriculo dextere. Le possibilitate de regurgitation aortic, ben que non reflectite in studios de pression, esseva eliminate additioualmente per le uso de un crampa de occlusion.

Le movimento de sanguine ab le fibrillante corde sinistre verso le latere dextere depende in grande mesura de un gradiente transmitite ab le latere doxtere. Materiales marcate in le ventriculo sinistre remaneva static, excepte post disocclusion del arteria pulmonar. Tamen, sanguine que entrava in le arteria pulmonar al nivello precapillar ab un fonte bronchial pote esser detegite in le absentia de un tal gradiente. Finalmente, le demonstration visual de fluxo retrograde esseva effectuate per medio de un cardiopneumogramma. Material radio-opac in le ventriculo sinistre esseva observate in transitato a transverso le vasculatura pulmonocapillar verso le corde e le venas cave dextere.

REFERENCES


**Errata**


page 539: figure 1 and figure 2 have been reversed.
legend for figure 2, last word of fourth line should be “decades.”

page 541: column 3, table 4 should be headed “f R(m).”
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