Further Experimental Evidence that Pulmonary Capillary Pressures Do Not Reflect Cyclic Changes in Left Atrial Pressure (Mitral Lesions and Pulmonary Embolism)

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Wedged catheter pressures (PC) were compared with those in the aorta, left atrium, and pulmonary artery before and after production of mitral stenosis, insufficiency, and pulmonary embolism. In general it was found that "PC" pressures follow closely changes in left atrial pressures even when pulmonary artery pressure changes in an opposite direction (for example, pulmonary embolism). However, properly recorded "PC" pressure pulses did not display the large cyclic pressure variations recorded from the left atrium during mitral stenosis and insufficiency. It is concluded that pressures recorded through a properly wedged catheter represent the constant pulmonary capillary pressure but do not reflect cyclic pressure variations in the left atrium.

In a previous communication1 experimental evidence was presented that a catheter properly wedged into a peripheral pulmonary artery records a rather constant pressure, generally a little higher than that in the left atrium. Therefore, such pressures can be fairly taken as an index of pulmonary capillary pressure (PCP). However, it was questioned whether phasic left atrial pressures can be transmitted through capillaries to such a catheter, as others have believed.2-5 Evidence was presented that when phasic systolic variations are recorded through a wedged catheter they are caused by transmission of pulmonary arterial pressure to the catheter tip. Instead of giving information regarding left atrial pressures they must be regarded as artefacts from improper lodgment of a catheter. These studies did not exclude the possibility that large pre-systolic and systolic pressure elevations, characteristic respectively of mitral stenosis and regurgitation, might be transmitted backward through dilated venules and capillaries. Since such waves have been reported chiefly in patients with such lesions it seemed important to determine whether they can develop during experimental mitral lesions and, if not, how pulmonary capillary pressures are affected by such lesions.

METHODS

Dogs were anesthetized with morphine and sodium barbital. The chest was opened in the midline under artificial respiration and the heart and lungs were exposed. Mitral stenosis was created by passing a suture by means of a large curved needle through the base of the left atrium parallel to the interatrial septum. The suture was started posterosilaterally to the pulmonary conus and emerged posteriorly at the interatrial groove. An additional fixation stitch was taken on the left lateral aspect of the AV ring. By firmly tying down the suture thus placed, the mitral orifice could be reduced to a narrow opening near the interatrial septum. Mitral insufficiency was produced by thrusting a trocar described by Wiggers6 through the left ventricular wall and passing it between the mitral cusps so that its tip lay in the left atrium. The valve leaflets folded around this round instrument so that its tip lay in the left atrium. Usually the valve leaflets folded around this round instrument so that no or only a slight degree of incompetence resulted. When the obturator was withdrawn a large channel for regurgitation immediately became avail-
able. As in a previous investigation,1 pressures were recorded by Gregg type manometers from the aorta, pulmonary artery, left atrium, and from a catheter introduced through a pulmonary vein and wedged into a peripheral arterial branch. The latter yielded “PC” pressures.

For comparisons all pressures except aortic were measured in mm. H2O. The surface of the animal board was used as an arbitrary zero level for all pressures.

RESULTS

Figure 1 illustrates typical effects on the aforementioned pressures before (A) and after experimental production of mitral stenosis (B). The first set of records (A) was obtained after the mitral orifice had been pulled together by the ligature. The aortic pressure pulse indicates that the effects of the lesion had been overcompensated as far as arterial pressures were concerned. But they show the peaked appearance and abridgement of systolic ejection reported as characteristic effects of reduced left ventricular action reported by other experimenters in this lesion (Katz and Siegel7, Opdyke and Brecher8). The atrial contraction wave in the left atrial pressure pulse is definitely increased and the pressure during ventricular systole is also elevated. The pressure values at the summit and trough of the a wave and at the end of systole are indicated on the record. The pulmonary capillary pressure is also raised considerably (250 mm. H2O), but displays no evidence of the great cyclic pressure variation in the left atrium.

Figure 2 illustrates typical changes following production of a large mitral leak. Segment A shows records taken with the atroventricular sound in place. The atrial pressure pulse again shows a slight degree of regurgitation. The catheter curve is essentially a straight line, indicating a pressure of 130 mm. H2O. This is slightly higher than the left atrial pressures shown directly on the curve. On withdrawal of the plunger, creation of a marked mitral insufficiency caused typical pressure changes.
in the left atrium and a slight elevation of PC pressure at a constant level. In order to create a greater distention of the smaller pulmonary vessels and to intensify the volume of regurgitation an infusion of 50 cc. saline solution was given rapidly. At the end of the infusion the records shown in segment B were obtained.

The aortic pressure pulse indicates that effective compensation for the valvular lesion had taken place as a result of the infusion. The left atrial pressure curves show a larger atrial wave and a pronounced systolic regurgitation wave with murmur vibrations. Pressures are indicated on the record. The pulmonary capillary pressure is elevated to a level of 190 mm. H2O but remains constant except for slight systolic oscillations. Certainly the pronounced systolic elevation of left atrial pressure is not transmitted to the wedged catheter.

In experiments reported in this and a previous publication it has been shown satisfactorily that elevations of phasic left atrial pressures are transmitted as a rise of mean pressure to a catheter properly impacted in a peripheral pulmonary artery. It remains to be shown that a general decline of left atrial pressure leads to the expected drop of PC pressure, even when pulmonary arterial pressure is elevated considerably. This was demonstrated by producing pulmonary embolism by injecting a blood clot into a femoral vein.

Figure 3 is an example of the results obtained from such experiments. Segment A shows that, except for a systolic impact, catheter pressure is recorded as a straight line under normal conditions. At the V point a normal pressure gradient of 22 mm. H2O exists between the left atrium and the catheter. Segment B was taken three minutes after injection of an embolus. Pulmonary arterial pressure had risen considerably, while left atrial pressure had declined. Actual pressure values are indicated directly on the curves. Pulmonary capillary pressure retains its even character throughout the heart cycle at a reduced pressure level of about 115 mm. Hg. At the V point the pressure relationships of left atrial and catheter pressures were practically the same as under normal conditions shown in curves of segment A. At autopsy a small embolus was located in the right lower lobe; not in the same vessel which contained the catheter.

**DISCUSSION**

In anesthetized dogs with open chests small catheters can be impacted in peripheral pulmonary vessels under better control than is possible in animals and humans via the right ventricle. Using this technic, it was again found that PC pressures recorded from properly impacted catheters show a nearly constant pressure throughout the cardiac cycle. Normally this pressure exceeds left atrial pressure at the end of ventricular systole and probably represents fairly well the constant pressure which exists in pulmonary capillaries. Under abnormal conditions it rises and falls with the general trend of left atrial pressure, even when pulmonary arterial pressure changes in an opposite direction. This was again shown to be true during experimental pulmonary embolism.

No evidence was found that the exaggerated pressure variations in the left atrium consequent to mitral lesions can be transmitted through the pulmonary capillary resistance to an arterial branch into which a catheter has been wedged. Such results reaffirm a previous suggestion that presystolic or systolic waves recorded from pulmonary catheters should be suspected as due to artefacts. Systolic pressure variations were recorded only from improperly placed catheters which allow pulmonary ar-
arterial pressure to creep around the catheter to its orifice. Such waves show pressure variations greatly in excess of left atrial pressure and can therefore not represent a transmitted pressure from this chamber.

The question has quite properly been raised whether in chronic human mitral disease accompanied by pulmonary congestion the pulmonary capillaries may not be dilated sufficiently to allow the passage of left atrial pulses. These experiments cannot supply an answer to this query. But such an assumption cannot be made a priori; it must be supported by hemodynamic evidence that the recorded PC variations correlate both as to time and pressure values with pressure pulses in the left atrium. Possibly this may soon prove feasible in humans as a prelude to operative procedures for the relief of mitral stenosis.

**SUMMARY**

1. Small catheters were impacted into peripheral pulmonary arteries in anesthetized dogs with open thorax. Pressure variations recorded through them (PC pressure) were compared with those in the aorta, left atrium, and pulmonary artery before and after production of mitral stenosis and insufficiency.

2. Records from properly wedged catheters showed no pressure fluctuations comparable to those of the left atrium. Since the constant PC pressure exceeds left atrial pressure slightly at the end of ventricular systole and since it rises and falls with the general level of left atrial pressure, even when pulmonary arterial pressure changes in opposite directions (for example, during pulmonary embolism), these pressures probably represent reasonably well the constant pulmonary capillary pressure.

3. Experimental mitral stenosis is characterized chiefly by a pronounced presystolic wave in addition to a general rise of pressure in the left atrium. A catheter record follows this general trend of pressure, and probably corresponds closely to a constant elevation of pulmonary capillary pressure. But no evidence has been found that a corresponding presystolic wave of pressure is transmitted.

4. Experimental mitral insufficiency is characterized in addition by a prominent systolic regurgitant wave as the general pressure level increases in the left atrium. Again, pressures recorded from an impacted arterial catheter respond by a constant pressure elevation, but neither the large presystolic nor systolic wave in the left atrium is transmitted.

5. While the transmission of left atrial pulsations through dilated pulmonary capillaries is a priori conceivable in persons with chronic mitral disease, direct hemodynamic evidence must be supplied before it can be accepted as a fact.

**REFERENCES**


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