Pulmonary Artery Wedge Pressure Lability in the Bovine[1] (36875)

FRED L. ANDERSON, ROBERT B. WRAY, THEOFILOS J. TSAGARIS,
AND HIROSHI KUIDA
(Introduced by G. E. Cartwright)

Cardiovascular Division, Veterans Administration Hospital and University of Utah College of Medicine, Salt Lake City, Utah 84112

Pulmonary-artery wedge (PAW) pressure is generally accepted as a reliable reflection of pressure in the pulmonary capillary—pulmonary vein (PV)—left atrial (LA) compartment (1–4). Droszcz, *et al.*, however, recently presented data which suggest that in the bovine species, PAW pressure is quite labile and at times may not represent PV or LA pressure (5). During the course of studies on the bovine pulmonary circulation over the past 10 years, we occasionally have encountered some difficulty in obtaining valid and reproducible PAW pressures. We have also noticed that small volumes of heparinized physiological saline used to flush catheters occasionally elicited sustained elevation of this pressure. In the light of these observations, we undertook a systematic study of the bovine PAW pressure and the factors that influence it. An analysis of these data forms the basis for this report.

Methods. Fifteen studies were carried out in 10 normal hereford calves (average weight 92 kg) purchased randomly at auction. Seven additional calves with brisket disease (BD), obtained from high-altitude enzootic ranges and of similar age and weight, were each studied once. Each calf was studied lying on its side without premedication or general anesthesia. The external jugular vein, carotid artery, and a branch of the femoral artery (FA) and vein were surgically exposed under local lidocaine anesthesia. Two #8 Cournand catheters filled with heparinized saline were introduced into the external jugular vein; under fluoroscopic guidance, one was advanced to the pulmonary artery (PA) and the other to the PAW position. Another #8 Cournand catheter was introduced into the common carotid artery and advanced to the left ventricle (LV). Teflon cannulae (#18 gauge), 14 cm in length, were inserted into a branch of the FA and femoral vein, respectively, and advanced proximally. These catheters and cannulae were connected to P23Db pressure transducers. An electrocardiogram was used to determine heart rate. Pressures and electrocardiogram were recorded on an oscillographic recorder (Minneapolis Honeywell, Denver, Colorado) at a paper speed of 1 mm/sec.

Criteria for satisfactory catheter impaction and a valid PAW pressure included: (1) a pressure equal to the LV diastolic pressure with an atrial pulse contour and (2) an oxhemoglobin saturation in a blood sample obtained from the wedged catheter that was equal to or greater than that of a sample obtained simultaneously from the LV. Gentle sustained negative pressure with a 10 ml glass syringe was necessary to adequately sample the wedged catheter. A blood sample was obtained from the wedged catheter regularly before each series of injections and frequently before each single injection.

After recording pressures during a control period, 1–5 ml of either normal saline (NS), 5% dextrose in water (D5W), or blood (venous or arterial) was injected gently into the PAW catheter over a 5–10 sec period. When PAW pressure stabilized, another injection of the same solution with isoproterenol, 1–2 μg/ml added, was made into the same catheter. After PAW pressure reached a steady level a third injection of saline alone was made. This sequence of 3 injections constituted a series and was repeated several

[1] This study was supported by the U.S. Public Health Service (Grants number HE-07618 and HE-250) and the Utah Heart Association.

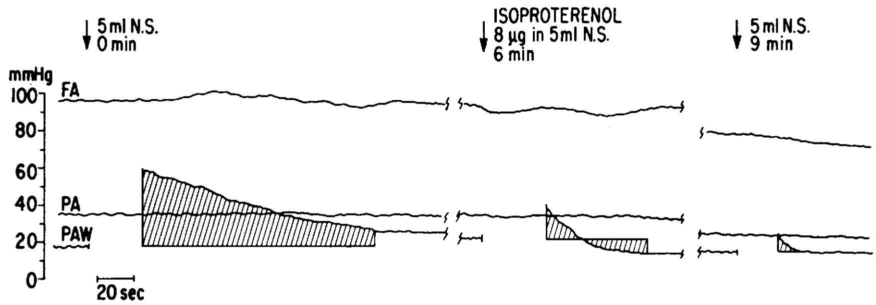

FIG. 1. Results of a typical experiment showing sequential changes in femoral artery (FA), pulmonary artery (PA), and pulmonary-artery wedge (PAW) pressures to sequential injections of: (1) Normal saline (NS), (2) NS with isoproterenol, and (3) NS alone over a period of 9 min. Hatched portions indicate areas determined by planimetric integration above and/or below baseline pressure level to determine net change in pressure.

times in each experiment. The volume and injection rate in each series was constant.

The response to a solution injected into the wedged catheter was analyzed according to the change in PAW pressure (mm Hg) $\times$ the time (sec). Using time as the ordinate and pressure as the abscissa the area of pressure change was determined by planimetry. The resultant area was expressed in cm^2. In those instances in which the pressure rose above the control level initially and then fell below that level before stabilization, the fall in pressure below the baseline was subtracted from the rise above to yield the difference. Positive values indicate a net pressure rise above the control level whereas negative values indicate a pressure fall below control level.

Results. The method of analysis and the results of a typical experiment are shown in Fig. 1. The usual response to the initial injection of 5 ml NS into the wedged catheter was a rise in PAW pressure, occasionally above PA pressure, followed by a slow return to control level. Occasionally a sustained pressure rise lasting 10–20 min was observed. In 5 series no significant change in PAW pressure occurred. The occurrence and degree of response elicited by NS were independent of the volume tested. In the control state, before injection of any solution, the PAW and LV diastolic pressures were identical. The injection of isoproterenol usually elicited a fall in pressure, although a few exceptions were observed. The third injection (5 ml NS without isoproterenol) usually failed to elicit any pressure rise. The average response of the PAW pressure of this series of 3 injections performed 19 times in 10 normal calves was 16.8, —3.6, and 1.1 cm^2, respectively. The accumulated data for these series are shown in Fig. 2.

An analysis of all test injections of either D5W or arterial or venous blood that were made under conditions in which no isoproterenol had been given for the preceding 20 min is shown in Fig. 3. In 27 experiments on 5

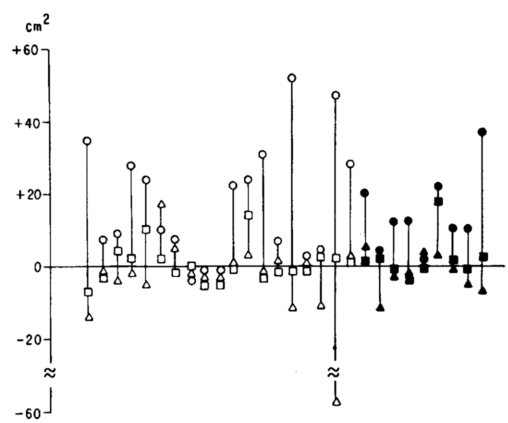

FIG. 2. Results of 19 experiments in 10 normal calves (open symbols) and 9 experiments in 7 brisket calves (closed symbols) showing net changes in area due to sequential injections into the wedged pulmonary catheter of: (1) 5 ml normal saline (circles), (2) 5 ml saline containing isoproterenol (triangles), and (3) 5 ml normal saline without isoproterenol (squares).

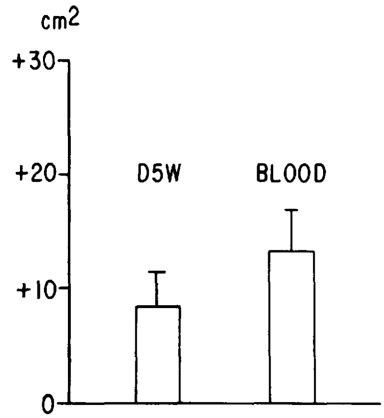

FIG. 3. Comparison of 11 test injections into the wedged pulmonary artery catheter of 5% dextrose in water (D5W) in 3 normal calves with 16 test injections of either venous or arterial blood in 3 other normal calves. Mean change in area above baseline pulmonary-artery wedge pressure ± standard error is shown.

calves (2 normal and 3 brisket), average increase in PAW pressure using D5W was 8.5 cm^2 (11 experiments) and 13.5 cm^2 using either arterial or venous blood (16 experiments). The difference was not statistically significant.

Discussion. The criteria for a valid PAW pressure alluded to earlier were adhered to strictly. Pressures at sites from which fully oxygenated blood (greater than 90% saturation as determined with an oximeter or comparable to simultaneously obtained LV blood) could not be obtained were discarded. Solutions were injected gently to avoid dislodging the catheter tip by recoil. Generally, but not always, sampling of blood from the catheter for determination of oxyhemoglobin saturation was performed to verify position following the injection of a solution. It was consistently observed that blood could be withdrawn more freely from the wedged catheter following an injection of solution containing isoproterenol.

The fact that the PAW pressure response was similar following the injection of varying volumes (1–5 ml) of either NS, D5W, or blood suggests that neither the type of injectate nor its volume within the limits tested is critical. The reproducible sequence of changes in PAW pressure, *i.e.*, rise following initial injection of NS, fall following isoproterenol and failure to rise following the injection of NS after isoproterenol, provide strong evidence that changes observed are not artifacts.

The complexity of the bovine pulmonary microcirculation makes any attempt to determine whether the present findings are a result of pre- or post-capillary vasoconstriction or relaxation formidable. Our interpretation of the changes observed in the present study is that the injection of the test solution is made into a vascular segment characterized by high vascular resistance downstream from the tip of the wedged catheter which limits egress of the injectate. Whether or not this is elicited by the injectate (*i.e.*, a myogenic response) or pre-existed we cannot say. The fall in PAW pressure following the injection of isoproterenol is interpreted to be due to relief of this obstruction by the well known vasodilatory property on pulmonary vascular smooth muscle of this agent. Failure of the PAW pressure to rise after the 3rd injection of NS can be explained by persistence of the vasodilatation produced by isoproterenol.

In a number of experiments the PAW pressure reached levels equal to or greater than simultaneously recorded PA pressure. Such an effect most simply is explained by injection into a segment of limited distensibility or vascular capacity with high outflow resistance. Another explanation that could account for this observation is the presence of bronchial artery collateral vessels communicating with the vessel in which the catheter tip is wedged and from which egress is obstructed. These communications have been thought to be pre-capillary.

These observations serve to reemphasize the rather considerable and perhaps exceptional pulmonary vasomotor reactivity that may occasionally occur in the bovine and to encourage caution in accepting PAW pressure as a reflection of PV, LA or LVed pressure in hemodynamic studies in this species.

Summary. In 10 normal calves and 7 calves with brisket disease, injections of small volumes of normal saline into the wedged pulmonary artery catheter were shown to

produce transient but significant increases in pulmonary-artery wedge pressure that could be reversed with isoproterenol. Whether this occurred at a pre- or post-capillary site or both could not be determined.

The technical assistance of Mr. Don Anton and Filimon Ukradyha, Ph.D. is greatly appreciated.

1. Connolly, D.C., Kirklin, J. W., and Wood, E. H., Circ. Res. **2**, 434 (1954).

2. Hellems, H. K., Haynes, F. W., Gowdey, J. F., and Dexter, L., J. Clin. Invest. **27**, 540 (1948).

3. Hellems, H. K., Haynes, F. W., and Dexter, L., J. Appl. Physiol. **2**, 24 (1949).

4. Werko, L., Varnauskas, E., Eliasch, H., Lagerlof, H., Senning, A., and Thomasson, B., Circ. Res. **1**, 337 (1953).

5. Drosozcz, W. A., Latorre, F., and Reeves, J. T., J. Appl. Physiol. **24**, 640 (1968).

Received June 16, 1972. P.S.E.B.M., 1972, Vol. 141.