sharp bend over the angular apex of the petrous portion of the temporal bone, enters the cavernous sinus through Dorelli's canal, and reaches the orbit through the superior orbital fissure.

Because of its long and angular course at the base of the brain, it is vulnerable to mechanical damage after intercranial pressure changes caused by spinal puncture and/or subarachnoid block.[1] Under these circumstances the onset of abducens nerve palsy is gradual. Diplopia develops in 3 to 21 days after prodromal symptoms of headache, dizziness, nausea, and stiff neck.

In our two cases diplopia appeared 15 to 20 minutes after nerve block was completed. It is conceivable that in the first case some of the 2-chloroprocaine injected into the sphenopalatine fossa could have reached the abducens nerve through the inferior orbital fissure.

In the second case the most probable sequence of events was diffusion of 2-chloroprocaine through the dural sleeve of the second cervical nerve into the spinal subarachnoid space. The ability of 2-chloroprocaine to diffuse through the dura has been demonstrated.[2] From the spinal subarachnoid space the local anesthetic may have diffused to the basal cisterns and from there to the vicinity of the abducens nerve on its way from the brain to the point where it pierces the dura. A second possibility that cannot be excluded is that 2-chloroprocaine was injected directly through the dural sleeve of the second cervical nerve into the spinal subarachnoid space and that the time lag between the block and the onset of abducens paralysis resulted from the time necessary for diffusion to the spinal fluid of the posterior fossa.

The two case histories illustrate that when local anesthetic agents are injected at sites from which they may diffuse to the vicinity of the relatively thin abducens nerve, transient abducens paralysis may develop. In the cases described, the paralysis subsided within three hours, with no after-effect.

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Indirect Measurement of Left-atrial Pressure in Surgical Patients—Pulmonary-capillary Wedge and Pulmonary-artery Diastolic Pressures Compared with Left-atrial Pressure

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Recently reported data have demonstrated the disparity between right ventricular and left ventricular function in seriously ill patients with or without obvious heart disease.[1-4] The use of right-heart filling pressure (right atrial or central venous) of such patients to estimate left-heart filling pressure may be misleading. As left-atrial pressure can seldom be measured in patients not undergoing cardiac surgery, indirect estimation of left-heart filling pressure may offer valuable clinical information. The introduction of a flow-directed balloon-tipped pulmonary-artery catheter * (Swan-Ganz) has made possible the widespread clinical measurement of both pulmonary-artery diastolic (PAD)

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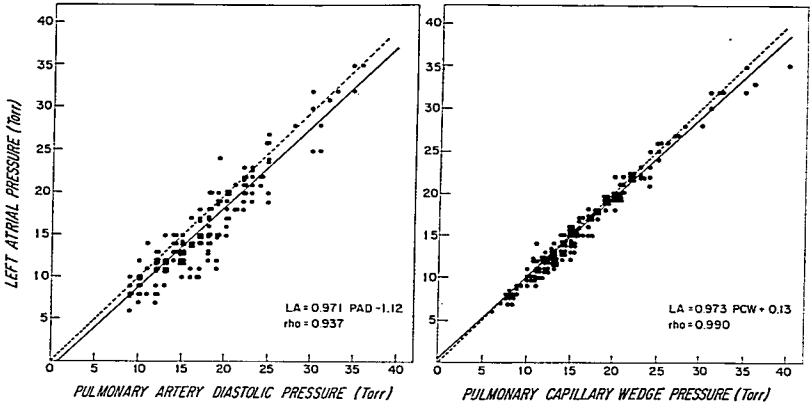

FIG. 1. Scattergram of the relationship of pulmonary-artery diastolic and pulmonary-capillary wedge pressures to left atrial pressure. Each dot represents one measurement. Dashed line is line of identity; solid line is calculated regression line.

pressure and pulmonary-capillary wedge pressure (PCW). The present communication compares PCW pressure and PAD pressure, as determined with a balloon-tipped catheter, with directly recorded left-atrial pressure (LAP) in a group of cardiac surgical patients.

METHODS

Eighteen cardiac-surgical patients for whose clinical management left-atrial-pressure measurements were needed were the subjects of this study. No attempt was made to exclude patients with altered pulmonary vascular resistance.

A Swan-Ganz pulmonary-artery catheter was inserted percutaneously via either an external or an internal jugular vein through a 12-gauge Argyle Medicut intravenous cannula † and guided into the pulmonary-capillary wedge position using constant-pressure monitoring.[5] The criteria used to verify a true wedge position were: 1) a characteristic waveform; 2) a mean pressure lower than the mean pulmonary arterial pressure; 3) the presence of respiratory variation; 4) the ability to withdraw arterialized blood (*i.e.*, a sample in which the oxygen tension was at least as high as the arterial oxygen tension).[6]

A left-atrial catheter was inserted prior to left-heart bypass in five patients, each of whom underwent surgical repair of a thoracic aortic aneurysm, and following total cardiopulmonary bypass in the remaining 13 patients. Measurements of left-atrial, pulmonary-artery diastolic and pulmonary-capillary wedge pressures were recorded from the time of insertion to 24 hours postoperatively.

Pressures were measured by Sanborn transducers (model no. 267BC) calibrated by mercury manometer and recorded on a direct writer. In most instances, left-atrial, pulmonary-artery diastolic, and pulmonary-capillary wedge pressures were measured serially by the same transducer. In some instances pressures were measured on equisensitive transducers. Mean pressures were derived by electronic integration.

Repeated measurements of values of individual patients were made in order to evaluate the responses of PCW and PAD pressures to changes in LAP. A total of 161 sets of measurements was made in these 18 patients. The number per patient ranged from four to 17.

† Sherwood Medical Industries, Inc., St. Louis, Mo. 63103.

Results

Figure 1 shows the data from the 161 measurements in the 18 patients. The overall correlation coefficients were 0.990 for pulmonary-capillary wedge versus left-atrial pressure and 0.937 for pulmonary-artery diastolic versus left-atrial pressure. The slopes of the regression lines were similar (0.97), although examination of the y intercepts revealed a one-torr difference.

Figure 2 shows the frequency distribution of variation of individual measurements from the line of identity. The standard deviations of the measurements from the line of identity were 0.95 torr for PCW–LAP and 2.68 torr for PAD–LAP. The standard deviations of these measurements from the calculated regression lines were 0.87 and 2.14 torr, respectively.

The correlation coefficients, y intercepts, and slopes were derived from the values for each patient. The correlation coefficients for pulmonary-capillary wedge pressure versus left-atrial pressure ranged from 0.81 to 1.0, with a mean of 0.95 ± 0.05 (SD), and all achieved statistical significance. In contrast, the correlation coefficients of pulmonary-artery diastolic pressure versus left-atrial pressure ranged from 0.07 to 1.0, with a mean of 0.80 ± 0.25, and three were not significant. Likwise, the y intercepts and slopes were consistently closer to ideal when PCW was compared with LAP than when PAD pressure and LAP were compared.

Discussion

This study documents the clinical reliability of measuring LAP by means of the Swan-Ganz catheter in the pulmonary-capillary wedge position. The standard deviation of the difference between pulmonary-capillary wedge pressure and left-atrial pressure was less than one torr, a difference beyond the limits of clinical detection. In contrast, although overall correlation was good, the pulmonary-artery diastolic pressure was a less reliable indicator of changes in left-atrial pressure in individual patients.

Left ventricular cannulation would certainly be the best available method to monitor filling pressure if it could be accomplished safely. Indeed, two groups have demonstrated that left-ventricular catheterization without use of fluoroscopy is possible in critically ill patients.[7, 8] However, the inherent dangers of left-ventricular catheterization (systemic embolization; ventricular arrhythmias) militate for use of an indirect method when one is available. Estimation of left-atrial pressure offers a suitable alternative, although it does not always reflect LVEDP in a number of circumstances. Prominent among these is left-ventricular disease when sinus rhythm is present,

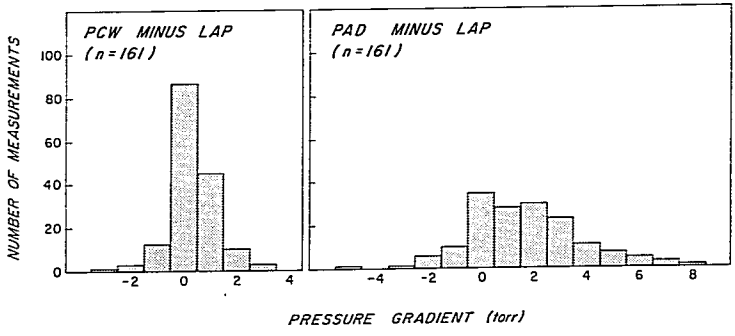

Fig. 2. Frequency distribution of the pressure gradients. Only 17 of 161 LAP–PCW pressure gradients were greater than ±1 torr.

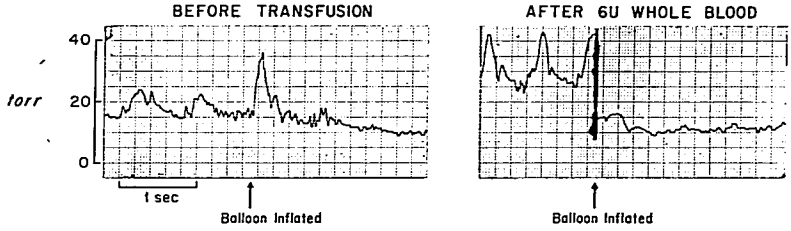

FIG. 3. Increase of pulmonary-artery diastolic pressure without change of pulmonary-capillary wedge pressure. The difference between PAD and PCW pressures increased from 5 to 15 torr after rapid infusion of 6 units of whole blood during resection of an abdominal aortic aneurysm. Acute changes in pulmonary vascular resistance may lead to erroneous conclusions about the level of left-atrial pressure when PAD pressure is used as an index. ("Balloon-inflated" signifies PCW pressure.)

where the average LVEDP–LAP gradient has been shown to increase from 0.2 to 9 torr.[9]

The lack of agreement between PAD and PCW pressures (or LAP) is explained primarily by a lack of time for pressure equalization across the pulmonary vascular bed during tachycardia (heart rate faster than 115/min) [10] or by an abnormally increased pulmonary vascular resistance. The latter may be permanent or temporary. We have frequently found increases in PAD pressure without changes in PCW pressure during rapid infusion of banked blood (fig. 3). Obviously, reliance upon PAD pressure for left-heart filling pressure under these circumstances would lead to errors in therapy.

We conclude that PCW pressure is a reliable index for clinical estimation of left-atrial pressure. When it is not possible to obtain PCW pressure, PAD pressure usually approximates it. Interventions causing changes in the relationship between PCW and PAD pressures need to be defined.

ADDENDUM

Since submission and acceptance of this manuscript, Rahimtoola and associates [11] have reported an average gradient of 6.7 torr between pulmonary-artery end-diastolic pressure and mean PCW pressure in patients with acute myocardial infarction who had slightly to moderately increased pulmonary vascular resistance (PVR greater than 2 units). Twenty-one of 48 patients with acute myocardial infarction in their series had such elevations of pulmonary vascular resistance. Myocardial infarction may thus be another situation in which acute changes in PVR cause varying gradients between PAD and PCW pressures.

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