A Critique of Flow-directed Pulmonary Arterial Catheterization

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In 1953, Lategola and Rahn reported the use in dogs of a hand-fashioned, balloon-tipped, self-guiding catheter for cardiac and pulmonary arterial catheterization and occlusion.1 During the following 15 years, several self-guiding right-heart catheters were developed for human use, but none was balloon-guided, and none became well known.2-5 In 1970, Swan et al.6 reported their initial experience with a multilumen, balloon-tipped, radiopaque, extruded polyvinyl chloride catheter (with adequate frequency response) that met three criteria: 1) reliable, prompt passage to the pulmonary artery, 2) minimal arrhythmias, and 3) passage without fluoroscopy. The catheter may be introduced either by cutdown or percutaneously in one of several veins (femoral, antecubital, axillary, subclavian, external jugular, proximal basilic or internal jugular).6-10 Placement is optimally performed by continuous pressure monitoring with electronic transduction and oscillographic display.11-12 After the catheter tip is advanced into the thorax (detected by respiratory fluctuations in the pressure trace), the balloon is inflated and the catheter is further advanced, relying on blood flow to direct the catheter tip through the tricuspid and pulmonic valves into the pulmonary artery. Details of techniques of catheter passage and of care of the catheter are straightforward and widely reprinted.6,14-16 Since the initial report, a family of balloon-tipped catheters has been developed to allow: 1) in-vivo oximetry,19 2) pulmonary angiography,20 3) pediatric cardiac catheterization,21 4) transvenous cardiac pacing,22 5) His-bundle electrocardiography,23 6) thermodilution cardiac output determination,24 and 7) artifact-free cardiac monitoring.25

The catheter has been used in enormously diverse clinical situations.7,9,14,26-30 In general, the indications for its use appear to be: 1) continuous hemodynamic monitoring following complicated acute myocardial infarction, 2) management of fluid balance in non-cardiogenic pulmonary edema, in non-cardiogenic shock, and following cardiopulmonary bypass, and 3) evaluation of therapeutic interventions with mechanical ventilation, vasoactive drugs, hemodialysis and assisted circulation. Swan has recommended that a pulmonary-artery catheter should be used when one thinks that a central venous pressure (CVP) measurement is needed.37 Several review articles detailing Swan Ganz (S-G) catheter use are available.17,38-42 This article critically examines certain limitations inherent in pulmonary arterial monitoring and discusses its use in clinical anesthesia.

Pulmonary “Capillary” Pressure

Pulmonary “capillary” pressure refers to the pressure measured through a cardiac catheter impacted into a branch of the pulmonary artery in such a fashion that there is a free communication between the catheter tip and the capillary–venous compartment of the lung. This pressure has been called pulmonary wedge pressure (PWP), pulmonary-artery wedge pressure (PAWP), pulmonary capillary pressure (PCP), and more recently, pulmonary-artery occlusion pressure (PAOP). Three criteria have been thought necessary for a true wedge pressure:33 1) blood withdrawn from wedge position should be fully saturated with oxygen, 2) the pulmonary-artery phasic contour should change to a left atrial trace on wedging, and 3) mean wedge pressure should be less than mean pulmonary arterial pressure; it has been observed that a wedged catheter blood sample may or may not be 100 per cent oxygen-saturated in patients who have pulmonary shunts and during positive end-expiratory pressure (PEEP).34,43,48 Thus, only the latter two criteria now apply.34 Initial studies in both animal and man confirmed the
validity of the pulmonary-artery wedge pressure as an index of pulmonary venous pressure and left atrial pressure (LAP).43

Balloon-tipped and ordinary pulmonary-artery catheters have been shown to give identical PAOP readings.6 Whereas pulmonary-artery catheters without a balloon tip must be manually advanced to measure PAOP and then withdrawn, the S-G catheter requires only intermittent inflation of the balloon to let blood flow pull the catheter tip out into a wedge position; thus, catheter manipulation is avoided and long-term monitoring becomes more feasible. Artifactual PAOP readings can be produced by an overinflated balloon occluding the catheter tip or an eccentrically inflated balloon not producing complete occlusion of the vessel lumen; inflating the balloon with the minimum volume of air sufficient to yield a PAOP trace and maintaining a central position of the catheter tip will avoid these problems.42,46-49 Analysis of PAOP (by S-G) as a measure of LAP has shown very good correlation in most studies.40-51 LAP has also been estimated from measurements of pulmonary arterial end-diastolic pressure (PAEDP). PAEDP is usually only 1-2 torr higher than PAOP and LAP; however, in critically ill patients, the correlation between PAEDP and PAOP is unreliable because of either pre-existing pulmonary hypertension or increased pulmonary vascular resistance secondary to acute cardiopulmonary failure.14,50-54 Normal hemodynamic values in the pulmonary artery are 20/12 (torr), with a mean of 13 torr and a mean PAOP of 6-12 torr. Caution must always be used in interpreting a single reading; sequential readings are thus necessary. The frequency of readings will, of course, depend on the instability of the patient, and might be as often as every 30 seconds, but certainly no less often than every 15 minutes.

The widespread availability of S-G catheters prompted a re-evaluation of central venous pressure measurements. Previously accepted as an adequate guideline for managing shock and hypovolemia,55-56 poor or absent correlation was found in comparing PAOP, PAEDP, or LAP with CVP in seriously ill patients.1,29-31,51,57-63 In the absence of cardiopulmonary dysfunction, CVP remains a reliable assessment of right and left heart filling.31,58 but in serious illness right ventricular function and left ventricular function are so disparate that not only the absolute CVP but even CVP changes are unreliable and misleading estimates of left heart filling.57,60

Some controversy does remain. First, a recent large series showed a good correlation of PAOP and LAP at pressures of 10 torr or less, but as PAOP increased the prediction of LAP from PAOP was subject to considerable error; at PAOP's greater than 15 torr the 95 per cent confidence interval for predicting LAP was at least ±5 torr. This was attributed to a change in the ratio of pulmonary arterial to pulmonary venous compliance. Second, evidence has been found for a pulmonary venous waterfall effect at the exit of the large pulmonary veins from the surface of the lung.65 A pulmonary vascular waterfall effect is said to occur in a collapsible vascular segment when the intravascular downstream pressure is less than the pressure surrounding the collapsible section.66 Under these conditions the vessels exposed to the surrounding pressure partially collapse and blood flow becomes independent of the more negative downstream pressure. Under no-flow conditions, a wedged upstream pressure will fall to the level of the pressure around the collapsible segment and not to the more negative downstream pressure. With a waterfall effect in the pulmonary veins and at very low LAP's, PAOP will be higher than LAP. This work66 was done in the dog with an open thorax; the conditions under which it might apply to man are unknown.

Whether PAOP reflects LAP during mechanical ventilation with PEEP is in doubt; PEEP raises alveolar pressure and creates a waterfall effect at the alveolar level, thus keeping PAOP higher than LAP.55,57,68 The presence or absence of a PAOP-to-LAP discrepancy during PEEP will depend on the complex relationship of: 1) the height of the S-G catheter tip above the left atrium during wedging; 2) the left atrial pressure; and 3) the level of PEEP: if the catheter tip wedges in a region of lung where alveolar pressure exceeds pulmonary venous pressure, PAOP will reflect alveolar pressure and not LAP.55,68 (The recent suggestion of Benumof et al.69 that a PAOP-to-LAP discrepancy will arise only when alveolar pressure exceeds pulmonary arterial pressure appears in error.69) Since the S-G catheter tip can distribute to peripheral regions of lung,69 it is easily conceivable that a change in patient position (supine to sitting or lateral decubitus) would produce a sudden, unexpected error in PAOP estimates of LAP. Equally, a change in the level of PEEP or LAP or a random migration of the S-G catheter tip during wedging would produce or eliminate an alveolar waterfall effect without a change in patient position. Unfortunately, the presence or absence of an alveolar waterfall effect during PEEP is not discernible with only an S-G catheter.

In addition, PEEP increases intrathoracic pressure and thus all intravascular and intracardiac pressures. The true distending pressure of a vessel or of a
cardiac chamber under these conditions is the intraluminal pressure minus the surrounding pressure. Calculation of transmural pressures (PAOP – intrapleural or esophageal pressure) has been recommended to preserve the accuracy of PAOP in estimating distending pressures during PEEP. Surrounding pressures have been taken with an esophageal balloon and with a fluid-filled intrapleural catheter. The latter technique has been criticized by Craig as measuring pleural liquid pressure, which can be quite different from pleural surface pressure. It is pleural surface pressure that determines expansion of the lung and is transmitted to intrathoracic extrapulmonary structures. Use of an esophageal balloon pressure must be taken with reservation owing to the possible artifacts introduced by the esophagus and surrounding structures. Even if pleural surface pressure is measured accurately, calculation of transmural filling pressures is potentially in error, as the pressure around the heart is more negative than pleural surface pressure and varies with lung volume.

Some have objected that in patients with chronic obstructive pulmonary disease (COPD) an elevated PAOP may be an artifact of the pulmonary abnormality and does not necessarily indicate left ventricular failure (LVF). In general, however, elevated PAOPs in COPD have been shown to reflect true LVF. In patients who have severe obstructive pulmonary disease, the wide swings in intrathoracic pressure during "active expiration" can influence PAOP interpretation; ideally, in this circumstance transmural PAOP should again be used; however, this technique is not routinely employed as it requires an esophageal balloon.

How, then, should PAOP be measured during elevated or rapidly changing intrathoracic pressure? The following suggestions might be useful. Pulmonary arterial pressures should be taken as the average over several heart beats at end-expiration with the transducer at the mid-axillary line and referenced to atmospheric pressure. For the mechanically ventilated patient who has a respiratory rate so rapid that a stable end-expiratory trace cannot be seen, the ventilator should be momentarily disconnected during the measurement. During vigorous spontaneous respiration, the patient with COPD or severe respiratory distress should be instructed to hold his breath at end-expiration; if this is not possible, transmural pressures must be taken or apnea induced.

Positive end-expiratory pressure of modest amounts (perhaps to as high as 10–12 cm H2O) appears to induce clinically unimportant errors in end-expiratory PAOP estimates of LAP. Temporary withdrawal of PEEP during wedging would also appear valid. During higher levels of PEEP (>10–12 cm H2O) no consistently reliable measurement protocol exists; it is possible that further experience comparing PAOP with LAP or LVEDP in patients with severe acute respiratory failure will confirm one report showing no PAOP to LVEDP discrepancy during PEEP to as high as 90 cm H2O.

**Left Ventricular Filling**

It is widely accepted that the Frank-Starling law applies to the intact human heart; i.e., at any given functional state the force of ventricular contraction is dependent upon its end-diastolic volume or end-diastolic wall tension. For want of a technique for routine ventricular volume determination, investigators and clinicians have relied on ventricular end-diastolic pressure as an index of end-diastolic volume and tension. Two questions arise: 1) how reliable is left ventricular end-diastolic pressure (LVEDP) in assessing left ventricular end-diastolic volume (LVEDV), and 2) how reliable is PAWP in estimating LVEDP.

During diastole the left ventricle fills passively and the pressure developed is exponentially related to the volume. An elevated LVEDP is commonly taken to signify the presence of LVF: a normal LVEDP is assumed to be evidence against its presence. In fact, apparent left ventricular (LV) function as reflected by LVEDP can change in many ways. Among them are a true change in the LV contractile state, a change in the diastolic properties of the LV, and hypervolemia. Particularly following acute myocardial infarction (AMI) or acute hemodynamic inter-

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<td>AMI = acute myocardial infarction</td>
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<td>C(a–v)O2 = arterial–venous oxygen content difference</td>
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<td>COPD = chronic obstructive pulmonary disease</td>
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<td>CVP = central venous pressure</td>
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<td>LAP = left atrial pressure</td>
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<td>LV = left ventricle</td>
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<td>LVEDP = left ventricular end-diastolic pressure</td>
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<td>LVEDV = left ventricular end-diastolic volume</td>
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<td>LVF = left ventricular failure</td>
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<td>LVSWI = left ventricular stroke work index</td>
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<td>PAEDP = pulmonary arterial end-diastolic pressure</td>
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<td>PAOP = pulmonary-artery occlusion pressure</td>
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<td>PAWP = pulmonary-artery wedge pressure</td>
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<td>PCP = pulmonary capillary pressure</td>
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<td>PEEP = positive end-expiratory pressure</td>
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<td>PVAO = mixed venous oxygen tension</td>
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<td>PWP = pulmonary wedge pressure</td>
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<td>S-G = Swan-Ganz</td>
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<td>SVO2 = mixed venous oxygen saturation</td>
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ventions (vaspressor and vasodilator infusions), impaired ventricular relaxation and decreased diastolic ventricular compliance may account in part for an elevation in LVEDP without any change in LVEDV. Under these conditions it may be incorrect to assume that an elevated LVEDP represents an impairment of myocardial contractility. A systematic study of the relationships of LVEDV, LVEDP, ventricular relaxation and ventricular diastolic compliance in many forms of cardiovascular dysfunction (anesthesia, trauma, sepsis, burns) is not available.

Second, the relationship between PAOP and LVEDP is complex. In subjects who have normal cardiovascular systems, PAOP, LAP and LVEDP are essentially interchangeable. Following myocardial infarction, atrial contraction makes a greater contribution to left ventricular filling (probably due to changes in ventricular compliance) and raises LVEDP much higher than mean PAOP. LVEDP is often 10 torr or more greater than LAP or PAOP. One study of anesthetized patients with coronary-artery disease showed LVEDP greater than PAOP. PAOP now reflects the left ventricular diastolic pressure prior to atrial contraction and is a poor estimate of left ventricular filling, but still provides reliable information about pulmonary venous hypertension and pulmonary edema. The lower the cardiac output, the more important is atrial contraction for ventricular filling and the less reliable is mean PAOP for estimating LVEDP. During severe respiratory failure, PAOP has been shown in a few patients both to equal LVEDP and to underestimate LVEDP.

But the relationship of PAOP and LVEDP during cardiopulmonary failure has not been systematically investigated.

Attempts to get a better estimate of LVEDP than that given by PAOP have been made and the pulmonary-artery occlusion "a" wave appears most valid. The wedge pressure trace is a reflection of the left atrial phasic events and can show prominent "a" and "c" waves. Measurement of the "a" wave pressure of the wedged pressure trace (rather than mean PAOP) seems to correlate very well with LVEDP. Unfortunately, the "a" wave is rarely distinctly seen in the wedge trace, and the "a" wave pressure has not been widely used.

**Ventricular Function**

An early modification of the S-G catheter was a multiple-lumen, thermistor-tipped S-G unit that permitted thermodilution determination of cardiac output. The principles of measurement are as follows: If a known quantity of cold solution is introduced into the circulation and adequately mixed, recording of the resulting cooling curve at a downstream site allows calculation of net blood flow. In practice 10 ml of 5 per cent dextrose at 0°C or room temperature are injected into the superior vena cava or right atrium via the proximal lumen of the S-G. The thermistor allows recording of the baseline pulmonary-artery blood temperature and the subsequent temperature change. The resulting curve may be analyzed manually by simple planimetric methods or by computer.

Validation studies of the thermodilution technique using S-G catheters show both good reproducibility and good correlation with the dye dilution methods of measuring cardiac output. However, large variations in baseline pulmonary-artery temperature related to cardiac and respiratory cycling may occur, especially in mechanically ventilated critically ill patients, which lessens precision and accuracy, such errors may be minimized by repeating the cold injections during the same point in the respiratory cycle. Only a modest correlation between thermodilution and Fick oxygen methods has been found in one study.

Perhaps this is not too surprising, as patients in the operating room or intensive care unit are rarely in a steady hemodynamic state and the thermodilution method is an average over 4–10 heart beats, while the Fick method averages over 2–3 minutes. In addition, there are other potential errors in both methods. Ultimately, there is no standard by which to compare accuracy. Each method must be considered as an estimate, but the thermodilution methods appear particularly simple and efficient.

With the easy availability of frequent cardiac output determinations in critically ill patients, construction of ventricular function curves has been recommended. Left ventricular stroke work index is plotted versus left ventricular filling pressure. Filling pressure is altered by such maneuvers as administering fluid challenges, diuretics and vasopressors, or by varying mechanical ventilation, PEEP and assisted circulation; Starling curves may then be constructed. Because of the previously mentioned disparity between PAOP and LVEDP, Starling curves so constructed from PAOP must be considered critically. It is easily seen that since true left ventricular filling pressure may be 8–10 torr greater than

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\text{cardiac output in } \frac{\text{ml}}{\text{min}} \times (\text{mean arterial pressure in torr}) = \frac{\text{LVSWI in gram-meters/m}^2}{\text{occlusion pressure in torr}} \times \frac{\text{heart rate in beats/min} \times \text{body surface area in m}^2}{13.6}
\]

† LVSWI (in gram-meters/m²) = heart rate in beats/min x body surface area in m² x 13.6.
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PAOP in certain patient groups, all points derived using PAOP may be erroneously plotted leftward and upward, thus creating a left-shifted Starling curve and suggesting left ventricular function better than in fact.

Crexells et al. constructed Starling curves for the left ventricle with volume challenges in patients following AMI and found maximum LVSWI at PAOP 14–18 torr; this, then, was considered to be the optimal filling pressure for adequate cardiac performance without risk of pulmonary congestion. However, since it is well demonstrated that pulmonary edema can develop in association with low colloid oncotic pressure or increased pulmonary capillary permeability at normal PAOP’s, PAOP’s of 14–18 torr may not be tolerable for adequate gas exchange in all patients. Whether survival following AMI is enhanced by manipulating PAOP to 14–18 torr is not proven. Others have not found an optimal left ventricular filling pressure following AMI.

Swan and co-workers, using hemodynamic data obtained by S-G catheter, divided patients with AMI into subsets and suggested specific therapies. The therapies are dependent on continual reassessment of hemodynamics with pulmonary-artery catheters. Although classification of patients within these groups is of prognostic value, little evidence that overall morbidity or mortality may be changed by the suggested manipulations is yet available.

Rapid diagnosis of certain complications of AMI and open-heart surgery (ruptured interventricular septum, acute mitral insufficiency, and cardiac tamponade) and serial hemodynamics during intra-aortic balloon pumping have been facilitated by pulmonary-artery monitoring. However, it was recently found that cardiac tamponade is probably better detected by CVP monitoring.

Analysis of Mixed Venous Blood

As a simpler method of assessing effective tissue perfusion or oxygenation in seriously ill patients, serial sampling for determination of blood oxygen saturation in the right atrium, right ventricle, or pulmonary artery has been used, since mixed venous blood oxygen saturation is directly proportional to cardiac output when arterial oxygen content and oxygen consumption remain constant. Normal mixed venous blood O₂ saturation is 70–75 percent; values less than 60 percent have been associated with heart failure and values less than 40 percent with shock. Only true mixed venous blood from the right ventricle or pulmonary artery can be used in seriously ill patients, as right atrial samples are poorly mixed and may give falsely elevated whole-body venous oxygen saturations. Fiberoptic S-G catheter oximeters have been developed to monitor mixed venous blood oxygen saturation (SVO₂) continuously. Also, serial measurements of mixed venous blood oxygen tension (PvO₂) have been used to assess circulation and oxygen therapy (normal 38–42 torr) and the arterial–venous oxygen content difference (Ca–V O₂) (normal 3.5–5 vol per cent) has been used to detect and differentiate co-existent cardiac failure and respiratory failure. Clinical decisions based on Ca–V O₂, PvO₂, and SVO₂ must be made cautiously. It is important to remember that a single C(a–v)O₂, SVO₂, or PvO₂ is, strictly speaking, uninterpretable without a simultaneous cardiac output determination, but a very low PvO₂ (<20 torr) or SVO₂ (<40 per cent) or a very high C(a–v)O₂ (>9 vol per cent) by itself does confirm a very severe derangement of oxygen transport. There is another possible error. As contamination of desaturated mixed venous blood by saturated pulmonary capillary blood is possible during aspiration through the non-wedged S-G, withdrawal of blood should be slow and abnormally high values suspect.

Other Uses

Measurement of PAOP has proved essential to discriminate LVF in the presence of acute respiratory failure as traditional signs of LVF (tachycardia, engorged jugular veins, hepatomegaly, gallop rhythm) become nonspecific and insensitive. The use of S-G catheters in severe acute respiratory failure has also revealed the almost universal presence of pulmonary hypertension and elevated pulmonary vascular resistance. Calculation of right ventricular stroke work index shows a threefold elevation over normal values; but afterload reduction to prevent right heart failure does not appear possible. Pulmonary angiography has been facilitated in critically ill patients by use of an indwelling S-G catheter.

Complications

Initial reports of S-G catheter use mentioned frequent minor problems, these included; transient arrhythmias during passage, balloon rupture, catheter thrombus, catheter coiling in the right ventricle, and local infection at the cutaneous insertion site. More serious complications have also been reported (table 1); some of these are preventable or treatable. Rhythm disturbances may usually be terminated by withdrawal of the catheter, but deaths have resulted. Pulmonary infarction can probably
be averted if the pulmonary artery trace is continuously monitored so as to permit immediate recognition and withdrawal of the catheter should inadvertent wedging occur.\textsuperscript{37} Fatal pulmonary arterial rupture has occurred; keeping the S-G catheter as close as possible to the pulmonic valve (while still able to obtain PAOP) and slow balloon inflation have been recommended to avoid this complication.\textsuperscript{10}

Other complications, such as aseptic thrombotic endocardial vegetations, subacut e bacterial endocarditis, and endocardial mural thrombosis, appear unavoidable if a catheter must remain in the central circulation for prolonged periods.\textsuperscript{138-141}

Considering the wide use, remarkably little serious morbidity or mortality has been reported. Perhaps this technique is inherently safe; however, without a large-scale, multi-center, prospective study (such as the Cooperative Study on Cardiac Catheterization\textsuperscript{142}), under-reporting of the true complication rate must be suspected.

### Use during Anesthesia

The availability of pulmonary-artery catheterization has been a boon to the study of pulmonary and systemic hemodynamics during diverse anesthetic circumstances; for example, halothane and enflurane anesthesia,\textsuperscript{138} pentolinium hypotension,\textsuperscript{139} large-dose morphine,\textsuperscript{130} nitrous oxide with morphine,\textsuperscript{87} and induction of anesthesia.\textsuperscript{140} Some of the limitations in using PAOP to evaluate left ventricular function, previously described, must be kept in mind when interpreting such studies. Sørensen and Jacobsen\textsuperscript{141} found large transient increases in PAOP for a few minutes immediately following endotracheal intubation with a barbiturate and succinylcholine induction; this was associated with large increases in pulmonary arterial and systemic blood pressures. While these increases in PAOP might reflect left ventricular failure, changes in left ventricular relaxation or compliance might also be involved.

Another question remains. What is the proper clinical use of pulmonary-artery catheterization in the operating room? To visitors of large hospital centers (especially those doing open-heart surgery), there appears to be no widely accepted common criteria for S-G use.\textsuperscript{‡} In some centers very few patients are catheterized; in others, the anesthesiologists seem to follow the dictum of Swan\textsuperscript{37} that if a CVP line is considered, a S-G should be used. Yet pulmonary-artery catheterization is expensive and has definite risks.

Bedside S-G catheterization for preoperative evaluation of the elective surgical patient does not seem to be generally indicated. Although this has been recommended prior to vascular surgery,\textsuperscript{162} the history, physical examination and laboratory data are usually adequate to reveal evidence of myocardial ischemia, myocardial failure, and pulmonary edema, and to allow selection of therapy. This, of course, excludes patients for open-heart surgery, who usually require formal cardiac catheterization.

Routine S-G monitoring for open-heart surgery is

\textsuperscript{‡} Wong KC, Stanley TH: Personal communication.
probably an unnecessary expense, hazard, and inconvenience for most patients, with the exception of those with very severely impaired left atrial or left ventricular function. Anesthesia may be induced and maintained with arterial and central venous pressure monitoring; after cardiopulmonary bypass, direct IAP is easily and frequently used to adjust left heart filling. It is possible that certain patients who have coronary-artery disease will benefit by pharmacologic unloading of the left ventricle during hypertensive episodes prior to cardiopulmonary bypass to alleviate myocardial ischemia, and that this will be facilitated by S-G monitoring. This approach needs validation.

One situation where S-G monitoring should be routinely employed has been identified. Infrahepatic aortic cross-clamping during repair of abdominal aortic aneurysms or during aortofemoral bypass surgery in patients with severe coronary artery disease frequently produces severe hemodynamic disturbances and myocardial ischemia. Observation of PAOP and ECG seems necessary to detect and ameliorate these changes.

Early detection of venous air embolism during sitting intracranial surgery was reported; however, treatment of such embolism by withdrawal of air through the S-G does not appear superior to use of conventional right atrial lines. Since other sensitive methods for detecting air embolism exist (precordial Doppler monitoring and end-tidal CO₂ sampling), the routine use of pulmonary-artery catheterization for sitting craniotomies does not appear warranted.

Certainly, the critically ill patient (in particular, those with cardiogenic and septic shock and acute respiratory failure) whose care is dependent on pulmonary-artery catheterization will also benefit by its use during emergency surgical procedures. The anesthesiologist should be prepared to initiate S-G catheterization in these patients (by himself or by others) if the need has not been perceived.

Swan-Ganz catheters have been placed during many other types of surgical procedures but except for the operations mentioned above, it is the cardiopulmonary status of the patient that should determine catheterization. Overall, pulmonary-artery catheterization should never become a routine technique for most anesthesia care.

Conclusion

Pulmonary arterial monitoring with Swan-Ganz catheters must be considered one of the most important advances in the care of the critically ill. As I have detailed, a more critical attitude should be adopted in evaluating data derived from the catheter because of the inherent limitations in pulmonary arterial monitoring and unresolved details of cardiovascular dysfunction. Faute de mieux, its use should be encouraged in appropriate patient groups.

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Obstetric Anesthesia

CLOSING VOLUME AND PREGNANCY New pulmonary function tests (e.g., flow–volume loops and closing volume), as well as standard pulmonary function tests, were performed on 19 healthy pregnant patients in the third trimester and after delivery. It was found that the flow characteristics manifested in the flow–volume loops and the closing volume remained unaltered during pregnancy. With regard to the standard pulmonary function tests, no statistically significant change was found except for a decrease in the expiratory reserve volume and in the functional residual capacity during pregnancy. (Baldwin CR, and others: New lung functions and pregnancy, Am J Obstet Gynecol 127:235–239, 1977.)

ROLL-OVER TEST The supine pressor test was performed on 207 nulliparous young women between the twenty-eight and thirty-second weeks of gestation. The supine pressor test predicted pregnancy-induced hypertension in 78 per cent of those women in whom the condition subsequently developed. Ninety-six per cent of the women who failed to demonstrate a rise in diastolic pressure on position change remained normotensive throughout the remainder of the pregnancy. (Phelan JP, and others: Is the supine pressor test an adequate means of predicting acute hypertension in pregnancy? Am J Obstet Gynecol 128:173–176, 1977.)