Invasive hemodynamic monitoring in obstetrics and gynecology

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Prior to the early 1970s, right heart catheterization was accomplished with semirigid catheters and fluoroscopy, thereby effectively limiting the procedure to cardiac catheterization laboratories (1). With the development of the flow-directed pulmonary artery catheter, however, the technology moved from the laboratory to intensive care units, operating rooms, and labor and delivery suites (2–7). Pulmonary artery catheterization provides information about left ventricular function that cannot be obtained with central venous pressure (CVP) monitoring. The combination of peripheral arterial and pulmonary arterial lines provides sufficient information to assess continuously both the cardiac and the pulmonary status of the patient. Use of pulse oximetry can provide continuous monitoring of arterial hemoglobin saturation (Sao₂). Clinical impressions can quickly be either reinforced or refuted with accurate hemodynamic measurements in critically ill patients, and therapeutic strategies can be calculated and their effects promptly evaluated.

Techniques

The decision to use invasive monitoring should include an assessment of the expertise of the physician to place the catheter and the expertise and availability of support staff to monitor the patient and maintain the equipment. Invasive monitoring should be carried out only in units with appropriate staffing ratios to allow continuous observation of the patient.

Cannulation of peripheral or central vessels for placement of intraarterial and pulmonary artery catheters should be accomplished by an individual skilled in these procedures and familiar with their associated complications. Detailed accounts of the technical aspects of line placement are available and will not be provided here. An increasing number of obstetrician–gynecologists are familiar not only with data collection and interpretation, but also with line placement (4). Additional assistance in line placement can usually be obtained from a trained intensive care specialist or anesthesiologist.

The standard flow-directed thermodilution pulmonary artery catheter (Fig. 1) includes a distal lumen at the catheter tip, a proximal lumen 30 cm from the catheter tip, a balloon lumen, and a thermistor. The distal lumen provides continuous measurement of the pulmonary artery pressure when the balloon is deflated and of the pulmonary capillary wedge pressure (PCWP) when the balloon is inflated. The proximal port can be used to monitor CVP or to administer fluids or drugs. Both the proximal and distal lumina of the catheter may be used to withdraw samples of venous blood for laboratory studies. Central core temperature can be measured and cardiac output calculated when the pulmonary artery catheter is used in conjunction with a thermodilution cardiac output computer. Additionally, fiberoptic pulmonary artery catheters are now being increasingly used, as they provide, in addition to all other parameters available from the standard pulmonary artery catheter, a continuous reading of the patient’s mixed venous oxygen saturation.

Data Collection

Continuous measurements of CVP and pulmonary artery pressure and intermittent measurements of PCWP are afforded directly by use of the pulmonary artery catheter (Fig. 2). Cardiac output can be measured intermittently by thermodilution. Heart rate and rhythm are observed through the use of continuous electrocardiographic monitoring. Systemic arterial pressure can be measured by sphygmomanometer or percutaneous arterial cannulation; the latter also provides easy access for

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arterial blood sampling. Mean pressure values can be determined for both the pulmonary arterial and the systemic circulation by electronic dampening of the respective tracing or by calculation with standard formulas. Additional hemodynamic values that reflect cardiopulmonary function and vascular resistance can be calculated as shown in the box. Stroke volume is a measure of the amount of blood pumped per contraction by the heart. Both cardiac output and stroke volume can be corrected for body size by dividing these values by body surface area to obtain the cardiac index and stroke index. Body surface area can be determined from standard nomograms (8); however, these data were collected in nonpregnant subjects, and comparable data are not available for pregnant women. Resistance to flow can be calculated from the right and left ventricles through determination of pulmonary and systemic vascular resistance, respectively. Pulmonary shunts and arterial–venous oxygen content differences are calculated by analysis of simultaneously obtained samples of mixed venous blood, drawn from the distal port of the pulmonary artery catheter, and arterial blood.

Hemodynamic values for healthy nonpregnant subjects (9) can be compared with those obtained in healthy

**Formulas for Deriving Various Hemodynamic Parameters***

| Parameter                                      | Formula                                                                 |
|------------------------------------------------|-------------------------------------------------------------------------|
| Mean arterial pressure                         | Mean pressure = (systolic pressure + 2 (diastolic pressure))/3           |
| Stroke volume (SV) (ml/beat)                   | SV = CO/HR                                                              |
| Stroke index (SI) (ml/beat/m²)                  | SI = SV/BSA                                                             |
| Cardiac index (CI) (L/min/m²)                   | CI = CO/BSA                                                             |
| Pulmonary vascular resistance (PVR) (dyne · cm · sec⁻³) | PVR = [(MPAP - PCWP)/CO] x 80¹ |
| Systemic vascular resistance (SVR) (dyne · cm · sec⁻³) | SVR = [(MAP - CVP)/CO] x 80¹                                           |

*Abbreviations: BSA = body surface area (m²); CO = cardiac output (L/min); CVP = central venous pressure (mm Hg); HR = heart rate (beats/min); MAP = mean systemic arterial pressure (mm Hg); MPAP = mean pulmonary artery pressure (mm Hg); PCWP = mean pulmonary capillary wedge pressure (mm Hg).

¹Conversion factor: 1 mm Hg/1 L/min = 80 dynes · cm · sec⁻³.
TABLE 1. HEMODYNAMIC VALUES IN HEALTHY NONPREGNANT, PREGNANT, AND POSTPARTUM SUBJECTS

| Parameter (Units)                        | Nonpregnant | 36–38 Weeks of Gestation† | Postpartum |
|-----------------------------------------|-------------|---------------------------|------------|
| Heart rate (beats/min)                  | 60–100      | 83 ± 10                   | 71 ± 10    |
| Central venous pressure (mm Hg)         | 5–10        | 3.6 ± 2.5                 | 3.7 ± 2.6  |
| Mean pulmonary artery pressure (mm Hg)  | 15–20       | —                         | —†         |
| Pulmonary capillary wedge pressure (mm Hg) | 6–12       | 7.5 ± 1.8                 | 6.3 ± 2.1  |
| Mean arterial pressure (mm Hg)          | 90–110      | 90.3 ± 5.8                | 86.4 ± 7.5 |
| Cardiac output (L/min)                  | 4.3–6.0     | 6.2 ± 1.0                 | 4.3 ± 0.9  |
| Stroke volume (ml/beat)                 | 57–71       | 74.7                      | 60.6       |
| Systemic vascular resistance (dyne cm sec^-5) | 900–1,400   | 1,210 ± 266               | 1,530 ± 520 |
| Pulmonary vascular resistance (dyne cm sec^-5) | <250        | 78 ± 22                   | 119 ± 47   |

* Where available, data are given as mean ± standard deviation.
† Values in pregnant patients were determined with patient in left lateral decubitis position.
† = Not reported.

Adapted from Rosenthal MH. Intrapartum intensive care management of the cardiac patient. Clin Obstet Gynecol 1981;24:789-807 and Clark SL, Cotton DB, Lee W, Bishop C, Hill T, Southwick J, et al. Central hemodynamic assessment of normal term pregnancy. Am J Obstet Gynecol 1989;161:1439-1442

primiparous women studied in the lateral recumbent position at 36–38 weeks of gestation and 11–13 weeks postpartum (10) (Table 1). Compared with a nonpregnant woman, a normal pregnant woman in the third trimester will have an increased cardiac output. The increased cardiac output is accounted for primarily by an increase in stroke volume, but also by increased heart rate. Mean arterial pressures were not different when measured in the late third trimester and at 3 months postpartum. In contrast, both the systemic and pulmonary vascular resistance, as determined by invasive monitoring, are significantly lower during the third trimester of pregnancy.

Data Interpretation

Assessment of cardiac function consists of evaluation of preload, afterload, heart rate, and myocardial contractility. If any of these four variables is abnormal, initial therapy should be targeted at correcting the specific dysfunction. Invasive monitoring allows for almost instantaneous assessment of therapeutic maneuvers. Additionally, significant pulmonary dysfunction in the face of normal cardiac function can quickly be determined to be due to a primary lung injury and therapy targeted for the specific pathophysiology involved. Assessment of cardiac function must include a systematic examination of each area that can lead to heart failure.

![FIG. 3. Ventricular function (Starling) curve for a heart with both normal function and during failure. Pulmonary capillary wedge pressure or central venous pressure represents fiber length (preload), and cardiac output (CO) represents fiber shortening. Note that in order to maintain cardiac output, the failing heart is required to function at higher preloads.](Int J Gynecol Obstet 42)
Preload
Preload is determined by end-diastolic intraventricular pressure and volume, thus setting the initial myocardial fiber length. Right and left ventricular end-diastolic filling pressures are clinically assessed by CVP and PCWP, respectively. Cardiac output plotted against PCWP gives a cardiac function curve for the left ventricle. The ventricular function curve shown in Fig. 3 demonstrates that a failing heart requires a higher preload (filling pressure) to achieve the same cardiac output as a normally functioning heart. Therapeutic manipulation of the ventricular filling pressures and simultaneous measurement of cardiac output allow calculation of the optimal preload (ie, the construction of a Starling ventricular function curve) at the patient’s bedside. Preload can be increased by the administration of crystalloid solution, colloid solution, or blood and can be decreased by the use of a diuretic, a vasodilator, or by phlebotomy.

Afterload
Afterload is the tension of the ventricular wall during systole and is dependent on ventricular end-diastolic radius, aortic or pulmonary arterial diastolic pressure, and ventricular wall thickness (11). The magnitude of the increase in right or left intraventricular pressure during systole depends primarily on pulmonary or systemic vascular resistance, respectively (Fig. 4). In the presence of heart failure, increases in afterload worsen the degree of failure by decreasing both the stroke volume and the cardiac output. Afterload, like preload, can be increased or decreased as mandated by clinical circumstances. Increases can be mediated via alpha-adrenergic agonists such as phenylephrine, while decreases can be achieved with a number of vasodilating agents. Sodium nitroprusside infusions are used most commonly to decrease afterload in medical–surgical intensive care units, while hydralazine is the agent most commonly used in obstetrics.

Myocardial Contractility
The inotropic state of the heart is defined as the force and velocity of ventricular contractions when preload and afterload are held constant. Although cardiac output can be measured directly, its adequacy is best assessed by clinical parameters, including the patient’s acid-base balance, mental status, urinary output, and, in pregnant patients, fetal heart rate tracing. In low-output cardiac failure, both preload and afterload should be optimized through therapeutic manipulation. If this fails to restore the cardiac output to an acceptable level, attention should be directed to improving myocardial contractility. Beta-sympathomimetic agents such as dopamine and isoproterenol are effective in improving cardiac output acutely. Depending on the cause of myocardial failure, either short- or long-term therapy with digitalis may be necessary.

Heart Rate
Although unusual in obstetric–gynecologic patients, heart block can compromise cardiac output. In this circumstance, treatment with either atropine or cardiac pacing is indicated. Similarly, sustained tachycardia can lead to heart failure due to shortened systolic ejection and diastolic filling times or myocardial ischemia, especially in the presence of stenotic cardiac valvular lesions. The pathophysiologic basis of tachycardia (eg, fever, hypovolemia, pain, hyperthyroidism) should be determined and corrected. Heart rate can also be controlled with propranolol or digoxin. Calcium channel blockers can also control heart rate, but the safety of these drugs for use in pregnant women is not known.

**Indications for Invasive Monitoring**
Clinical conditions for which invasive hemodynamic
monitoring may assist in the management of obstetric–gynecologic patients as follows:

- Sepsis with refractory hypotension or oliguria
- Unexplained or refractory pulmonary edema, heart failure, or oliguria
- Severe pregnancy-induced hypertension with pulmonary edema or persistent oliguria
- Intraoperative or intrapartum cardiovascular decompensation
- Massive blood and volume loss or replacement
- Adult respiratory distress syndrome
- Shock of undefined etiology
- Some chronic conditions, particularly when associated with labor or major surgery:
  - New York Heart Association class III (symptoms with normal activity) or IV (symptoms at bed rest) cardiac disease (structural or physiologic) (12)
  - Peripartum or perioperative coronary artery disease (ischemia, myocardial infarction)

Considerable controversy exists as to the need for routine use of a pulmonary artery catheter during the administration of either conduction or general anesthesia in women with severe pregnancy-induced hypertension. To date, no study has shown improved outcomes in such women.

Clearly, invasive monitoring will not be necessary in every patient with one of these conditions, nor is this an all-inclusive list of indications for monitoring. In most instances, the conditions and indications for invasive monitoring are identical to those found in other areas of medicine or surgery. Additionally, pregnancy is not a contraindication for invasive hemodynamic monitoring, and the same standards and criteria should be used in the selection of pregnant women for invasive monitoring as for nonpregnant patients.

Monitoring may be instituted prophylactically in the seriously ill or compromised obstetric–gynecologic patient preparatory to the stress of labor and delivery or a major surgical procedure. In the patient with acute and unexpected pulmonary edema, a primary lung injury (normal or low PCWP) can quickly be differentiated from heart failure (high PCWP) and specific targeted therapy administered. Volume status of the patient can be assessed by ventricular preload or filling pressures, which can be adjusted to optimize cardiac output. Assessment of volume status is often critical in patients with massive volume loss and replacement, sepsis, or oliguria that fails to respond to initial therapy. Although initial management of the majority of these acute events may not warrant invasive monitoring, failure to achieve the predicted clinical response to initial empirical therapy warrants consideration of invasive monitoring to collect additional information to guide and evaluate further therapeutic manipulations.

Central Venous Pressure

Right ventricular function and systemic vascular compliance can be assessed by CVP monitoring, which has been widely employed to establish the relationship of blood volume to vascular capacity. The primary disadvantages of this method relate to the fact that CVP levels may not necessarily be elevated in the presence of left ventricular failure and pulmonary congestion and, conversely, may be elevated in patients without evidence of pulmonary edema (13). Pulmonary capillary wedge pressure can be reliably assessed by CVP monitoring only in the absence of cardiopulmonary disease. It has been noted that in the presence of dissociated right and left heart function, absolute CVP values correlate poorly with left-sided filling pressures, and even changes in this modality may be misleading (13).

Complications

The complications of invasive hemodynamic monitoring can be subdivided into those occurring in conjunction with gaining vascular access and those related to the duration of monitoring. Peripheral arterial cannulation can be associated with hematoma formation, infection, and vessel thrombosis. Serious complications from insertion, such as gangrene and loss of a digit or extremity, occur in less than 1% of cases (Table 2) (14, 15). Gaining central venous access for either a pulmonary artery catheter or a CVP catheter can also be associated with vessel wall damage and hematoma formation, pneumothorax, inadvertent arterial puncture, and persistent bleeding. The pulmonary artery catheter, unlike CVP lines, frequently will cause transient arrhythmias as it is passed through the heart, but only 1% of patients will require pharmacologic treatment. A significant complication is hemorrhage as a result of disconnection of the catheter from the intravenous line. Rarer but reported complications include
TABLE 2. COMPLICATIONS OF PULMONARY-ARTERY CATHETERIZATION

| Complication                     | Incidence (%)|
|----------------------------------|--------------|
| Premature ventricular contractions | 15–27        |
| Arterial puncture                | 8            |
| Superficial cellulitis           | 3            |
| Thromboembolism                  | ?            |
| Pneumothorax                     | 1–2          |
| Balloon rupture                  | <1           |
| Pulmonary infarction/ischemia    | 1–7          |
| Pulmonary artery rupture         | <1           |
| Catheter knotting                | <1           |
| Catheter-related sepsis         | 1            |

Hankins GDV, Cunningham FG. Severe preeclampsia and eclampsia: controversies in management. Williams obstetrics, supplement 1991;18:11. Reprinted by permission of Appleton and Lange, Inc.

Knotting of the catheter, rupture of the pulmonary artery, and thromboembolism. The risks of both sepsis and thromboembolism increase with duration of catheter placement, and all line complications are proportional to the experience of the operator. Given a broad range of medical and surgical patients with conditions necessitating invasive monitoring, 3% will sustain a major complication, including death (4, 14, 15). In assessing the need for monitoring, potential benefits must be weighed against the reported risks, taking into consideration that many complications, such as arrhythmias and pneumothoraces, are more likely to occur when dealing with a patient in extremis.

Summary

Indications for invasive hemodynamic monitoring in obstetrics and gynecology are much the same as in any other area of medicine and surgery. The clinical decision of whether to employ monitoring cannot be made according to absolute criteria but must be made on an individual basis. Obviously, not every patient will benefit from or need invasive hemodynamic monitoring; proper patient selection is important, as is the availability of skilled physicians, nurses, and ancillary support personnel. The information collected at the patient's bedside can be used to both guide and evaluate therapeutic maneuvers. Timely adjustment and titration of therapeutic maneuvers and treatments would logically have a significant impact on the outcome of a critical illness or on patients on the verge of cardiac or respiratory decompensation. Nonetheless, a randomized prospective study of the efficacy of the pulmonary artery catheter in obstetrics and gynecology does not currently exist, nor is such a study likely with the already widely accepted use of these technologies.

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