

Noninvasive monitoring in the patient with heart disease

Joachim S. Gravenstein, M.D.

Gainesville, Florida

Nonpolluted or noninvasive are terms that were coined to describe things we had taken for granted, not requiring a defining term. For many decades it was unnecessary to describe monitoring as noninvasive. Then invasive methods were introduced and became commonplace. But now the terms invasive and noninvasive no longer suffice since we must deal with a spectrum ranging from noninvasive to highly invasive¹ with several intermediate gradations of invasiveness (*Table*). Only noninvasive techniques as defined here will be discussed.

Inspection

Inspection is probably the oldest and most respected of noninvasive monitoring techniques. Universally observed are the traditional eye signs, capillary filling after blanching the skin, the color of the conjunctival membrane, which we check for engorgement, blanching or a blue tint secondary to venous congestion, or arterial hypoxemia. The patient with heart disease might also be checked for distension of neck veins. The novice might be surprised to see distended neck veins after induction of anesthesia even in patients without heart disease. This change is similar to the general dilatation of the veins during sleep and anesthesia and a slightly elevated right atrial pressure commonly observed

Table. Invasive and noninvasive procedures for monitoring patients with heart disease

| | |
|--------------------|--|
| Noninvasive | |
| | Inspection; passive sensing of surface temperature, infrared emission, etc. |
| | Auscultation, external only |
| | Palpation, including arterial tonometry |
| | Passive and active electrical sensing with surface electrodes such as ECG, EEG, or impedance |
| | Externally applied and sampled low-level energy, such as ultrasound, light, and heat |
| | Gas sampling with skin surface probes |
| Minimally invasive | |
| | Cutaneous needle electrodes for EEG and ECG |
| | Cutaneous needle electrodes with stimulation for neuromuscular monitoring |
| | Cutaneous or muscular probes for tissue temperature and gases |
| | Abraded skin for gas sampling |
| | Intravenous injections and blood samples from capillaries or peripheral veins |
| Penetrating | |
| | Tympanic membrane probe for temperature |
| | Nasal septum probe for cardiovascular monitoring |
| | Pharyngeal or esophageal probe for temperature monitoring |
| | Esophageal probe for cardiovascular monitoring |
| | Stomach probe for pH |
| | Rectal probe for temperature |
| | Bladder catheter for renal function |
| | Uterine probe for fetal monitoring |
| Invasive | |
| | Arterial catheters and probes for various purposes |
| | Central venous probes |
| | Suprapubic catheters for urine samples |
| Highly invasive | |
| | Intracardiac probes for pressures and samples |
| | Transcardiac probes such as pulmonary artery catheters for pressures and flows |
| | Subarachnoid probes for pressure |
| | Intracranial probes for cerebrospinal fluid pressures or flows |

with the major inhalation anesthetics. Redistribution of blood within the body that occurs with sleep and anesthesia could be observed even more clearly with the help of infrared photography,

a gentle and noninvasive technique, but one not used for clinical monitoring.

One of the most important signs observed by inspection, one not easily recorded mechanically or electronically, is the pattern of respiration in a patient who is breathing spontaneously. Positive expiratory efforts with contraction of abdominal muscles during light anesthesia or in respiratory distress are important signs too often obscured by the use of muscle relaxants. The rocking chest with the upper thorax rising little or not at all during inspiration, whereas the abdomen and the lower thorax are pushed up are good indicators of muscle weakness or obstruction in the upper airways and are seen more often in patients with congestive failure who have little muscular reserve and who can become dyspneic when reclining.

Palpation

Another noninvasive method perhaps as old as inspection is palpation. We feel for equal excursion of the upper thorax after intubating a trachea, we test for changes in the skin temperature, we feel for thrills and, particularly late in anesthesia before we return the patient to spontaneous ventilation, we feel for laryngeal motion to make sure that no tug is noticeable. A tracheal tug that had not been present preoperatively indicates a degree of respiratory difficulty particularly unacceptable in patients with heart disease. These tugs must be palpated because they can be subtle and not visible in obese patients or in patients with short necks. A finger placed over the thyroid cartilage will detect even a slight laryngeal motion on inspiration.

Pask² may have overstated his case when he suggested that in British operating theaters only a finger on the pulse was necessary for monitoring. I agree

with him, however, that I would be willing to surrender electrocardiographic and blood pressure measurements before giving up inspection and palpation.

Auscultation

Auscultation is relatively new to inspection and palpation not only historically in physical diagnosis but also in anesthesia. The precordial or esophageal stethoscope is now used routinely. No anesthetic should be given without that simple instrument. After tracheal intubation we listen for breath sounds over both lungs. In the patient with vascular disease a bruit may be heard over a narrowed carotid artery. Any change in sounds of the bruit or cessation of the bruit on one or the other side occurring with hypotension signals a change in blood flow that may become insufficient to perfuse the brain during such episodes under anesthesia.

Blood pressure

Anesthesiologists use changes in arterial pressure as signals for a change in anesthetic management more often than any other routinely monitored variable.³ This emphasizes that cardiovascular changes brought about by anesthetics have great importance during anesthesia, and that as yet there is no better way of monitoring these changes than by following the blood pressure, even if with noninvasive techniques we can do this only every few minutes.

Electrocardiogram

The electrocardiogram (ECG) represents a noninvasive method now so firmly established that few anesthesiologists would be willing to give anesthesia without an ECG tracing on an oscilloscope. That is true even for short procedures in healthy patients and even though changes in the ECG trigger

changes in anesthesia management.^{3,4} The ECG in most uncomplicated anesthetic and operative procedures is simply a security blanket and potentially a disaster alert. In patients with heart disease the ECG has a different role.

Systolic time intervals

Even though much has been written about systolic time intervals (STI), it might be useful to review the definition of these intervals. STI refer to the brief and definable episodes during left ventricular systole, i.e., from the beginning of electrical activity in the ventricle to the closing of the aortic valve. This period is called the total electromechanical systole (QS₂) lasting from the Q wave of the ECG to the second heart sound (S₂). The pre-ejection period (PEP) stretches from the Q wave to the beginning of blood flow with opening of the aortic valve; the left ventricular ejection time (LVET) occupies the rest of the electromechanical systole. Although these measures are straightforward they are not always easily obtained. Difficulties begin with the Q wave of the ECG, which is frequently not recordable and forces us to use the beginning of the R wave, which may not reflect the initial activation of the septum. With bundle branch block and other ventricular conduction disturbances the interpretation of the ECG may be even more difficult.

The PEP itself can be divided into an electromechanical interval (the time from the beginning of the Q wave to the beginning of the rise in ventricular pressure) and the isovolumic contraction time (the time during which left ventricular pressure rises until the aortic valve opens and blood begins to flow across the valve). Since the electromechanical interval is brief, quite stable and not easily measured, the electromechanical interval and the isovolumic contraction time are combined into the

PEP, which reflects primarily events in the isovolumic contraction time.

A microphone over the precordium helps us to detect the first high-frequency component of the second heart sound and thus the closing of the aortic valve, i.e., the end of the electromechanical systole. Also to be defined is the beginning of blood flow across the aortic valve, which will signal the end of the PEP and the beginning of LVET. This measurement of opening of the aortic valve is difficult to obtain noninvasively. A number of methods have been described. Most frequently investigators try to detect the upswing (the foot) of the pulse pressure wave in a carotid artery. This can be relatively easily accomplished with a suitably shaped transducer applied to the skin of the neck. However, the pulse wave arrives in the carotid artery a little after it rises in the root of the aorta due to the pulse wave transmission time. To measure the transmission time, the pulse wave is recorded from its beginning to its dicotic notch. Provided the shape of the pulse wave is not distorted as it travels from

the root of the aorta peripherally, the time from the beginning of the pulse to the dicotic notch is equivalent to LVET. Since the duration of QS_2 is not known, LVET can be subtracted from it to obtain PEP. The *Figure* summarizes these relationships.

Of all the noninvasive techniques that have been used in the assessment of cardiac function, STI are particularly helpful because they permit continuous preoperative, intraoperative, and postoperative measurements of the cardiovascular system. They are also easily applied and comfortable to the patient. Other methods that do not involve the application of energy (such as echo) or of invasive techniques include the ballistocardiogram (which cannot be employed in the operating room) and phonocardiography.

What do changes in PEP and LVET signify? The *Figure* shows that PEP will be longer when the steep slope of the PEP becomes less steep, i.e., when at early systole the pressure rises more slowly in the ventricle. As one would anticipate there is good correlation be-

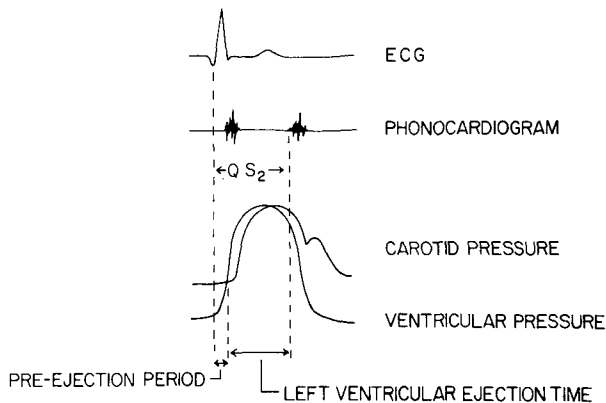


Figure. Systolic time intervals. The relationship between carotid pulse, ECG, and phonocardiogram. From Q wave of the ECG to 2nd heart sound (QS_2) is the total electromechanical systole. This time can be divided into the pre-ejection period (PEP = from Q wave to the beginning of blood flow across the aortic valve) and the left ventricular ejection time (LVET = from opening to closing of the aortic valve). Because the ventricular pressure cannot be sensed noninvasively, the carotid pressure curve is recorded. It is delayed, but from upswing of this curve to dicotic notch equals LVET. Consequently $QS_2 - LVET = PEP$.

tween PEP and the index of contraction, V_{\max} .⁵

As is true for other variables used to assess the heart, a number of factors can affect PEP, which lengthens not only with a weakening of contractility but also with a decrease in preload and an increase in afterload.

LVET occupies the time of block flow across the aortic valve. Intuitively, a short LVET should be expected with a small stroke volume. Again, interdependence exists and LVET is shortened also when heart rate increases or afterload falls.

Many investigators have combined PEP and LVET. We would anticipate a short PEP and long LVET to reflect good contractility and a large stroke volume, i.e., conditions likely to be found in a healthy heart; the opposite, a long PEP and short LVET, would reflect a weak myocardium ejecting little blood. Indeed the ratio PEP/LVET has been found to be greater than 0.4 in patients with heart disease or depressed from anesthesia, whereas small PEP/LVET ratios less than 0.4 are found in healthy or hyperdynamic states with sympathetic stimulation of the heart.⁶

Similar considerations lead to another useful value, that of LVET—PEP, which correlates well with stroke volume.⁷ Monitoring STI during anesthesia might therefore offer much useful information if arterial blood pressure and heart rate, all measured noninvasively, are available at the same time.

STI may be an attractive tool in the preoperative assessment of patients who have a history of myocardial infarction but who were not thought to be candidates for coronary angiography or another extensive cardiac workup. Weisler et al⁶ suggest that the PEP/LVET ratio and ejection fraction demonstrate concordant changes; both show little

difference between normal and patients with coronary artery disease, but both change significantly in patients with coronary artery disease who also had suffered a myocardial infarction.

In anesthesiology, a number of investigators have studied STI. Dauchot et al⁸ reported on three groups of 24 patients; one included patients with severe heart disease, another with moderate, and a third without heart disease. The patients were given either halothane-nitrous oxide-oxygen anesthesia or morphine-*d*-tubocurarine-nitrous oxide-oxygen anesthesia. Preoperatively, the patients differed only in that those with heart disease took many drugs and their systolic blood pressures were more than 150 torr as compared to 127 torr in patients without heart disease. The product of systolic blood pressure times heart rate times LVET was also higher in the two groups of patients with heart disease than in the healthy patients. As expected, preoperative systolic time intervals in these three groups were not significantly different.⁶ The patients with heart disease had been treated and were in optimal condition before coming to the operating room. However, with the administration of halothane the patient with heart disease showed much greater myocardial depression as reflected in pronounced changes in PEP/LVET than was true for the patients without heart disease. Indeed, the response of the patients with heart disease to halothane as assessed by STI could be used to predict the presence of heart disease. Thiopental-opiate-relaxant anesthesia techniques did not separate the patient in a similar manner.⁸

In an extension of this work Dauchot et al⁹ studied patients undergoing aortic reconstructive operations. All of these patients had vascular disease and many had a history of myocardial infarction.

During cross-clamping of the aorta LVET increased particularly in patients with ECG evidence of old myocardial infarction. The authors reported that observing the changes in PEP/LVET and LVET—PEP was helpful in conducting the anesthetic. PEP/LVET was taken to reflect “cardiac function,” whereas LVET—PEP to mirror stroke volume.

Smith et al¹⁰ compared halothane, enflurane, and isoflurane anesthesia in healthy men using STI to assess cardiac function. They found that the marked cardiac depression produced by enflurane was much ameliorated by hypercarbia. The authors postulate that the hypercarbia, so commonly seen in spontaneously breathing patients under enflurane, causes sympathetic stimulation, which in turn counteracts the myocardial depression produced by enflurane. This raises the question of enflurane anesthesia in patients with congestive failure who are known to respond to inhalation anesthetics with greater myocardial depression⁸ and who often show decreased sympathetic control of their hearts.

As mentioned, STI are not always easily measured. They may be spawning a derivative less precise but far more easily obtained, the QF interval¹¹ or, as it is sometimes called, the “dirty PEP”. QF simply measures the interval from Q wave (or upswing of R wave) to the time of the arrival of the pulse wave (F for foot of pulse wave) in the periphery. The QF includes the electromechanical interval, isovolumic contraction time (the sum of which equals PEP) and the transmission time of the pulse wave from aorta to wherever the wave is sensed. This simple measure may prove useful clinically, not because it will always faithfully parallel PEP, but because the anesthesiologist may find this

measure as helpful as blood pressure. The latter is also a variable influenced by a host of factors yet of proved utility.

Many new developments in monitoring patients will appear in the next few years. Anesthesiologists await with particular interest the demonstration of noninvasive methods that are meaningful, easily applied, and inexpensive. STI or QF hold promise.

References

1. Gravenstein JS, Newbower RS, Ream AK, Smith NT, eds. *Essential Monitoring: Current and Future Noninvasive Techniques*. New York: Grune & Stratton, 1980.
2. Pask EA. Hunt the signal. *Proc R Soc Med* 1965; **58**: 757–66.
3. Amaranath L, Burke P, Kreul J, Kiriluk V, Gravenstein JS. Why monitor? In: Gravenstein JS, Newbower RS, Ream AK, Smith NT, eds. *Monitoring Surgical Patients in the Operating Room*. Springfield, Ill: Charles C Thomas, 1978: 19–30.
4. Hur D, Gravenstein JS. Is ECG monitoring in the operating room cost effective? *Biotelemetry* 1979; **6**: 200–6.
5. Zanella J, Steinberg R, Katona P, Dauchot PJ, Gravenstein JS. Correlation of invasive measures of cardiac function with expressions derived from systolic time intervals in the anesthetized dog. In: List WF, Gravenstein JS, Spodick DH, eds. *Systolic Time Intervals*. Berlin: Springer-Verlag, 1980: 82–7.
6. Weissler AM, Stack RS, Sohn YH. The accuracy of the systolic time intervals as a measure of left ventricular function. In: List WF, Gravenstein JS, Spodick DH, eds. *Systolic Time Intervals*. Berlin: Springer-Verlag, 1980: 1–13.
7. Grum DF, Dauchot PJ. Correlation of systolic time intervals with stroke volume in man. In: Gravenstein JS, Spodick DH, eds. *Systolic Time Intervals*. Berlin: Springer-Verlag, 1980: 218–22.
8. Dauchot PJ, Rasmussen JP, Nicholson DH, et al. On-line systolic time intervals during anesthesia in patients with and without heart disease. *Anesthesiology* 1976; **44**: 472–80.
9. Dauchot PJ, DePalma R, Grum D, Zanella J. Detection and prevention of cardiac dysfunction during aortic surgery. *J Surg Res* 1979; **26**: 574–80.
10. Smith NT, Calverley RK, Eger EI, Quinn M,

Prys-Roberts C. Changes in systolic time intervals during halothane, enflurane or isoflurane anesthesia in healthy man. In: List WF, Gravenstein JS, Spodick DH, eds. *Systolic Time Intervals*. Berlin: Springer-Verlag, 1980: 265–72.

11. Reitan JA, Levine NA. A comparison of the pre-ejection period and the QF interval as in monitoring variable. In: List WF, Gravenstein JS, Spodick DH, eds. *Systolic Time Intervals*. Berlin: Springer-Verlag, 1980: 273–80.