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# Monitoring in Anesthesia

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"The point requiring most skill and care in the administration of the vapour of ether is, undoubtedly, to determine when it has been carried far enough." So wrote John Snow in 1847,(16) implying as the era of surgical anesthesia began that the anesthetic state was precarious, and that the risk to the patient made monitoring mandatory. According to Webster a monitor is one who teaches, admonishes and warns. In medicine, we use the term to describe the watching of vital functions by periodic or continuous measurement or assessment. The need to monitor has increased with time, particularly during the most recent decades, in response to the public's demand for safety. These rising expectations for safe anesthesia may be the ultimate motive force behind the monograph on "Monitoring in Anesthesia." We now live in an age which is in transition from regarding good health as God's gift to viewing it as a constitutional right, guaranteed every citizen.(12) A mishap is no longer accepted as an "Act of God," but found to be a fault for which someone must be held liable. Today's citizens insist on living no shorter time than their predicted life span and favor that research which is most likely to lengthen their lives. Only the natural disaster is beyond reprisal, and only because no effective reprisal has been found. The meaning of all this for anesthesiologists is that they are no longer protected by the axiom that "accidents will happen." They soon will be expected to perform with near-zero mortality and morbidity.

The basic task of our profession has not changed since John Snow became the first physician to devote full time to Anesthesia. We are expected to produce freedom from pain for the patient undergoing surgery—and to do so effectively and efficiently. Effectively means consistently producing good operating conditions combined with freedom from pain. Efficiently means doing this with a minimum of complications or loss of life. From the early days, the anesthesiologist has needed a conceptual understanding of the physiological effects of anesthesia, good tools and techniques of administration, and means of monitoring and evaluation. With time, other tasks have been added, which we shall discuss later.

During the entire first century of surgical anesthesia the emphasis with respect to

monitoring was on assessment of the depth of anesthesia, starting with John Snow's description of the five degrees of etherization (16) and on to Guedel's famous stages and planes. (10) Throughout the first century of anesthesia, most anesthesiologists confined themselves to the task of producing anesthesia, while evaluating the depth of anesthesia, as observers of respiration, circulation and other signs. At the turn of the century, record keeping and measurement of blood pressure were added to the anesthesiologist's basic task. Record keeping during anesthesia was suggested by F.B. Harrington and the first records were kept in 1894 by his "house pupils," E.A. Codman and H. Cushing, who at first charted pulse and respirations. The following is an excerpt from a letter from Cushing to F.A. Washburn, dated February 10, 1920: (2)

"I think we both became very much more skillful in our jobs than we otherwise would have become, owing to this competition, but it was particularly due, I think, to the detailed attention which we had to put upon the patient by the careful recording of the pulse rate throughout the operation.

"Subsequently, on going abroad and getting interested in blood pressure, I discovered in use in Padua a simple recording instrument in Riva-Rocci's clinic. On returning home I came to utilize this always during the course of my neurological operations so that the procedure might be as comparable as possible to the records taken upon a kymograph during an experiment in the laboratory. A much more elaborate ether chart was thereupon prepared, on which not only pulse rate and respiration but the systolic blood pressure was recorded.

"On Dr. Councilman's instigation a paper was read here in Boston, January 19th, 1903, on the subject of The Routine Determination of Arterial Tension in Operating Room and Clinic. This was the beginning, I think, of the general use of a blood pressure apparatus in hospital wards, whether medical or surgical, for though the principle was not new the old Gartner tonometer was most unsatisfactory because in cases of low blood pressure, the most important ones, it was utterly unreliable.

"I mention this because it is not uninteresting, in view of the universal adoption, subsequently, of instruments to measure blood pressure, to recall that the Division of Surgery appointed a committee to report on the subject. This report appeared March, 1904, Bulletin No. 2 of the Division of Surgery, and the final conclusion of this committee was as printed: 'The adoption of blood pressure operations in surgical patients does not at present appear to be necessary as a routine measure." I find I have written on my reprint the verse from Dr. Holmes' Stethoscope Song:

Now such as hate new fangled toys Began to look extremely glum; They said that rattles were made for boys And vowed that this buzzing was all a hum'

Another addition to the basic task was the concern with outcome. Measuring outcome by studies of morbidity and mortality gradually assumed importance, although never the prominence deserved. Again, Snow was leading with his analysis in 1858 of fifty cases of death from chloroform.(17) Except for sporadic reviews by individuals and commissions, the next major steps came in 1934 with Rovenstine promoting systematic data collection,(14) and in 1935 when Ruth and co-workers began the work of The Anesthesia Study Commission of the Philadelphia County Medical Society in an attempt to learn why, when and how anesthesia fatalities occurred.(15) In Great Britain and Australia similar efforts followed. The largest data bases available to us

came decades later with the Beecher-Todd report (3) and with the National Halothane Study.(4)

One signal event in our history gave us a new task, namely the control of vital functions, what John Gillies referred to as "Physiologic trespass in Anesthesia." (6) Perhaps taking liberties with his fine phrase, I refer to such interventions as paralyzing and controlling respiration, instead of merely observing and recording it. And controlling blood pressure and temperature, instead of just observing and recording them. As single events or spectacular trials, we may trace control of respiration and circulation far back in history. But as a sustained clinical application, the control of ventilation came with the great surge in Thoracic Surgery, which started in the nineteen thirties, only to be partially suppressed by World War II, and then took off in earnest after the war. Control of circulation and temperature was prompted by advances in cardiovascular and neurological surgery around 1950. The change in anesthetic approach was spurred on by the need for surgical advances, and in turn the new anesthetic techniques made these surgical advances possible.

Control of respiration and circulation precluded measuring the depth of anesthesia according to Guedel's stages and planes. Because the safe use of one-agent deep general anesthesia with diethyl ether or cyclopropane depended on knowing the stage and plane; and because respiratory and circulatory signs were at the core of the Guedel scheme, it is not surprising that deep general anesthesia was found "unsuitable" when "physiologic trespass" was perpetrated. There simply was no way of measuring or assessing the depth of anesthesia under those circumstances. It is no coincidence that barbiturates, narcotics and muscle relaxants were discovered or rediscovered in time to introduce the balanced anesthesia technique needed for physiological trespass. (6,8) This was also the time, when the search was on for new methods of quantifying anesthetic action. Bickford and Faulconer's work on the electroencephalogram was done at this time. (5) During the last decade, we have made it common practice to omit the assessment of anesthetic depth. Instead we rely on an approach which uses general anesthesia so light that any danger of overdose is improbable. And we rely on muscle relaxants and narcotics for completion of the requirements we must meet. With respect to the depth or lack of depth of anesthesia, we may have come to rely excessively on probability. With a patient breathing 60% nitrous oxide, and fully curarized, after premedication and a thiopental sodium induction, it is probable that this patient is unconscious and will have no memory of the surgery. But it is not certain! On the contrary, it is certain that some patients will not be made fully unconscious by 60% nitrous oxide. The common use of narcotics as adjuvants in anesthesia can be rationalized in several ways. The most important is that the narcotic makes certain that, if the patient remembers, the memory is not of pain and suffering. For all the usefulness of probabilities, here is room for improvement.

A second addition of an important task also took place gradually. The medical management of the patient's overall functions and problems has become the responsibility of the anesthesiologist. Resuscitation, transfusion and fluid therapy came first, as soon as it became common practice to have an intravenous line open during anesthesia and surgery; and this has been the case for less than twenty-five years. Now we have made it routine to cannulate peripheral and central veins as well as arteries, and we obtain measurements which are continuously recorded as well as those which are intermittently sampled. Just a few decades ago one anesthesiologist gave anesthesia

and measured and recorded blood pressure, pulse and respiration every five or ten minutes. Now in complex cases it takes several people to anesthetize, monitor, measure and manage vital functions. It is amusing to recall what one Wm. Hooper Attree wrote to the editor of the Lancet in 1858:(1)

—In all cases where the inhalation of this great boon is advised by the faculty, or voluntarily desired by the patient, how much safer life would be if there were an act enforcing the employment of three persons—one to administer chloroform by Snow's apparatus, a second to watch the heart's action and the pulse, whilst a third be solely occupied as the operator. Each would have work enough to perform.

What will be expected of the anesthesiologist in the future? Prediction is a risky game, but it would appear a safe bet that the anesthesiologist will be expected to provide anesthesia for good risk patients undergoing routine procedures at a zero or nearzero mortality and morbidity. This makes it a major objective to identify and eliminate those errors and accidents, in part randomly occurring, which contribute to the disturbing fact that roughly one third of all anesthesia deaths strike ASA class I and II patients.(3,7) With respect to anesthetic mortality in general, I shall venture a prediction. I think the public will view anesthesia-related mortality and morbidity in much the same way as drug induced mortality and morbidity.(11) As with drugs, anesthesia should be part of the cure, not part of the problem. Anesthetic mortalityshould be "undiscoverable" against baseline mortality. In good risk patients, having simple procedures, there is little or no baseline mortality, and anesthesia related mortality, therefore, is easily "discoverable." In fact, it stands out for everyone to see. This mortality must disappear. If there is any "acceptable" mortality of anesthesia, it is that which is "undiscoverable" against a high mortality baseline in high-risk patients. The unavoidable major effort to reduce "discoverable" mortality will have to rely heavily on monitoring, measurements, safety engineering procedures and modern decision making processes.

One other easy prediction is that even in the face of fiscal restraints, the demand for more complex surgery for older and more debilitated patients will increase. For one thing, the drive to vastly increase coronary artery surgery seems unstoppable. We will be expected to do anesthesia for this type of surgery without adding a "discoverable" "excess mortality" from anesthesia.(4,11) And, in general, we may expect successful medical treatment to result in the survivors being candidates for further medical treatment, a multiplier effect which Enoch Powell described as "the appetite for medical treatment vient en mangeant."(13) (Freely translated: as they eat they get hungrier!) Older patients may be a greater risk inherently; that will not stop them from expecting the utmost in anesthetic safety.

The exchange between operating room anesthesia and the intensive care unit is likely to go on. In the nineteen fifties we applied to intensive care, what we had learned in anesthesia. By the mid-nineteen sixties, the traffic had reversed and we returned critical care medicine to the operating room. The exchange continues, to the benefit of both environments. We train our residents in intensive care so that they can learn to take care of the critically ill patient under anesthesia in the operating room. A few will take a long-term interest in intensive care. But, that is a fringe benefit, not the principal aim.

There is little doubt that these high expectations can be met only by relying on measurements and monitoring to an increasing extent. Many of the necessary tech-

niques are already available or becoming available. Most are of the invasive kind, because the invasive measurement must precede the noninvasive, which, needless to say, is the ultimate goal. On the frontiers of the profession, where progress is being made, the invasive approach is likely to be unavoidable. In addition to monitoring and measurements, we need to introduce quality control, safety procedures, risk recognition and decision making procedures. Most importantly, we must abandon our complacent acceptance of human errors as unavoidable. The public will expect an "undiscoverable" or zero-excess mortality and morbidity in anesthesia. This book takes an important step towards that goal.

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# **Preface**

As quantitative truth is of all forms of truth the most absolute and satisfying, so quantitative error is of all forms of error the most complete and illusory.

George M. Beard, 1879

There are two reasons for monitoring patient responses during anesthesia and surgery. The first is to preserve a record of events so that future care of patients might benefit from what occurs in the present. The second and more important reason is to enhance the quality of care at the moment. In the latter case, for example, the electroencephalogram is observed during surgery to allow us to detect and hopefully correct inadequate cerebral perfusion. Similarly, fetal heart rate is continuously recorded to alert the obstetrician if fetal well-being is threatened.

Since the reasons for monitoring in anesthesia are primarily concerned with clinical care, we have oriented this text toward the clinician rather than the basic scientist. Thus, we emphasize the practical use of equipment on patients rather than emphasizing electronics and physics. We used several approaches to accomplish our objective. First, we asked our contributors to establish, when appropriate, a three-level hierarchy of monitoring. The first level includes those instruments and devices that should be utilized on all patients undergoing anesthesia and surgery—what could be called routine or essential monitoring. The second level includes those techniques that might be used for patients whose systemic disease or surgical procedure poses a threat in the absence of such a monitor. Measurement of cardiac output in patients with crippling heart disease would fit into this category. The third monitoring level encompasses techniques performed by specially trained individuals or in institutions with special interests. An example from this category might be measurement of cerebral blood flow during neurosurgical procedures. This last level, though having utility in terms of patient care, might be considered to border on research.

A second direction we have taken is to include chapters which address the monitoring needs of a clinical subject or subspecialty of anesthesia, such as obstetrical or neurosurgical anesthesia. The purpose of these chapters is to enable the clinician to use a monitoring scheme appropriate to a particular clinical problem; for example, high-risk pregnancy. Although this latter approach has resulted in some repetition and overlap (the use of a doppler ultrasonic flow probe has been covered under monitoring of the cardiovascular system as well as under monitoring during neurosurgical anesthesia), this overlap was considered desirable as well as unavoidable.

Finally, we have tried to help the clinician to be better able to evaluate hardware from the variety of manufacturers that offer such systems. One additional goal in this

regard is to make physicians skeptical of instruments they use, as well as to make them aware of what needs to be done to enhance the functioning of and safety of electrical equipment. We recognize the limitations imposed on such a text by the extraordinary rate of development of new machines and systems that must confound and confuse the practitioner, as well as render obsolete some of the techniques discussed. We know of no way to avoid this problem, which is common to most technically oriented texts.

Our sincere thanks to those who helped with this project and who, after all, made it possible—the contributing authors, the typists, our editor and, of course, our colleagues at UCSD whose support gave us the time and inspiration to see it through.

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### 1

# Monitoring the Depth of Anesthesia

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#### INTRODUCTION

The early use of anesthetic agents brought the realization that anesthesia also carried the risk of death. The margin between clinically useful doses of ether or chloroform and doses producing unwanted depression was not great. Our predecessors dealt with this problem by assessing the depth of anesthesia. This assessment was an important part of our training and practice up to 20 to 30 years ago. The development of halothane and enflurane and the increasing use of balanced anesthesia lessened the need to determine depth. Recent discoveries (which will be discussed later) may have restored that need.

The assessment of depth resulted from the desire to limit anesthetic administration to clinically useful dose ranges. The relationship between anesthetic dose and effect has three parts: the anesthetic dose, the sites affected, and the manifestations of the effect at those sites (Fig. 1.1). There is also a fourth dimension: factors other than anesthesia may act on the same sites. These factors include drugs, disease, surgery, and physiological stresses such as hypoxia or hypercapnia. Their effect may be to modify the response to anesthesia and hence the signs of depth.

#### MAC

One part of our scheme presents a relationship which is remarkably constant. The alveolar anesthetic concentration required to eliminate movement in 50% of patients in response to surgery (MAC) is unaffected by duration of anesthesia (1), by respiratory or metabolic acid-base changes (1,2) (except that MAC decreases as PaCO<sub>2</sub> exceeds 80 to 90 torr), by PaO<sub>2</sub> changes (until PaO<sub>2</sub> falls below 40 torr) (3); by anemia (4), by hypertension (1,5), by hypotension to a mean arterial pressure of 50 torr (6), or by sex (7). Two stresses that do decrease MAC are aging (8-10) (Fig. 1.2) and Supported in part by USPHS GM15571

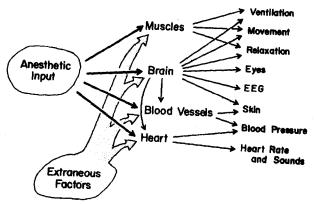


Figure 1.1. The effect of anesthetic input to muscles, brain, blood vessels, and heart may be manifested by alterations in the elements listed on the right of the figure (e.g., ventilation and movement). It is these manifestations that we use to define "depth" of anesthesia. The picture is complex: anesthesia not only affects muscle, brain, blood vessel, and heart, but such tissues additionally may interact to alter the picture we see. For example, an anesthetic may directly depress the heart, but the brain (via the autonomic nervous system) may compensate for this depression. Finally, extraneous factors such as age, surgery, disease, hypovolemia, hypoxia, hypercapnia, and medications (e.g., reserpine, alphamethyldopa, vasopressors, atropine, narcotics, and ganglionic blocking agents) may alter the tissue response to anesthesia and thereby

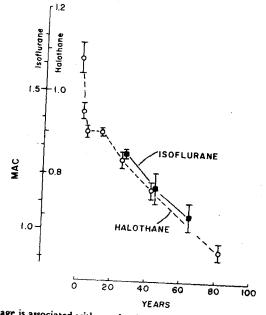


Figure 1.2. Facreased age is associated with a reduction in MAC for either halothane or isoflurane. The reduction is proportionally the same for both agents. (Reproduced with permission from Stevens WC et al: Minimum alveolar concentrations (MAC) of isoflurane with and without nitrous oxide in patients of various 2

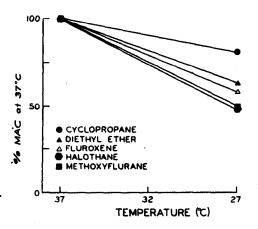


Figure 1.3. A decrease in body temperature reduces MAC for cyclopropane, fluroxene, ether, halothane, and methoxyflurane. Isoflurane is affected as much as halothane (14). Not all anesthetics are affected in the same manner: the reduction in MAC for a 10°C decrease in temperature is 50% for halothane but only 20% with cyclopropane. (Reproduced with permission from Regan MJ, Eger EI II: Effect of hypothermia in dogs on anesthetizing and apneic doses of inhalation agents. Anesthesiology 28:689-699, 1967.)

hypothermia (11-14) (Fig. 1.3). MAC also is decreased by the addition of narcotics (15-17) or diazepam (18) or by the presence or availability of central nervous system catecholamines (19-21). The effect of inhaled agents given concomitantly appears to be additive (22-24). That is, half a MAC of nitrous oxide plus half a MAC of enflurane produces absence of movement in response to incision in 50% of patients (24). To eliminate movement in 95% of patients requires an alveolar anesthetic concentration which exceeds MAC by 10% to 40% (7).

#### **Clinical Signs of Anesthetic Depth**

Many effects, other than movement in response to incision, vary with anesthetic dose and often differ from one anesthetic to another. Some of these effects are useful guides to the conduct of anesthesia (i.e., they are signs of depth); some are sought because they lessen the trauma or difficulty of surgery; some are avoided because they impose an unnecessary risk or compromise anesthetic or surgical care.

Some signs of anesthetic depth may be simply manifestations of increased sympathetic activity. One example is the progressive pupillary dilatation and diaphoresis seen with ether anesthesia. Similarly, with other anesthetics the pupillary dilatation, increased blood pressure and heart rate, and diaphoresis that appear subsequent to the start of surgery are similar examples of an incompletely attenuated sympathetic response to a noxious stimulus.

Increasing the depth of anesthesia does not necessarily abolish this response, although it alters the baseline value from which the change occurs. For example, respiration increases after surgical incision at all levels of isoflurane anesthesia (25) (Fig. 1.4).

Although several signs of depth are used, apart from movement in response to surgery, no single sign is applicable to all agents (Table 1.1). The use of a given sign depends on the agent or combination of agents used.

Eye signs. Increasing pupil size indicates increasing depth of ether, fluroxene, or cyclopropane anesthesia (26). It is of little value as a sign with halothane, enflurane, or methoxyflurane (26, 27), particularly if narcotic premedication has been given. It is of no value with nitrous-narcotic or ketamine anesthesia (27). Pupillary response to light

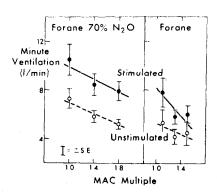


Figure 1.4. Isoflurane, either alone or with nitrous oxide, causes a dose-related depression of ventilation (above) and an associated increase in arterial carbon dioxide (PaCO<sub>2</sub>) levels. Ongoing surgery stimulates ventilation at any given dose and reduces PaCO<sub>2</sub> (not shown). The reduction in PaCO<sub>2</sub> is equivalent to the reduction which would be achieved in the unstimulated state by decreasing the anesthetic dose by 0.5 MAC. (Reproduced with permission from Eger EI II, Dolan EM, Stevens WC, Miller RD, Way WL: Surgical stimulation antagonizes the respiratory depression produced by Forane. Anesthesiology 36:544-549, 1972.)

is brisk with small amounts of all the inhaled agents, becomes sluggish with increasing depth, and soon is exhausted. Unfortunately, the response is difficult to see with halothane, enflurane, or methoxyflurane because the pupils are small at all depths. Eye movement indicates, but is not necessarily a companion of, a low level of anesthesia. It is particularly useful on induction, disappears when anesthesia is achieved, and may not return when anesthesia is decreased except that nystagmus often reappears at inadequate levels of ketamine anesthesia. Eyelash and corneal reflexes disappear once anesthesia is achieved and normally do not return at useful surgical levels of anesthesia.

Blood Pressure. Decreasing arterial blood pressure is the most commonly used sign of increasing depth of halothane (28) or enflurane (29) anesthesia. The imposition of surgery increases the blood pressure by a variable amount (30). Both the depression due to anesthesia and the increase due to surgery (or other stimuli) are related to cardiovascular status (blood volume, myocardial contractility, blood vessel reactivity, catecholamine stores) as well as to factors which influence sympathetic sensitivity (acid-base or electrolyte status, age, hormonal alterations). The result is that although we know the qualitative change in pressure with change in anesthetic dose, the quantitative relationship must be assessed individually for each patient. The relationship of pressure and dose also may be used to estimate depth of methoxyflurane anesthesia (27). Ether and fluroxene do not produce a consistent change in pressure except that deeper levels of fluroxene cause an increase. Cyclopropane causes an increase which is sustained over the range of clinically useful doses (28). Blood pressure increase is used as a sign of inadequate depth with nitrous-narcotic, nitrous-Innovar or nitrousketamine anesthesia (27). However, these increases in pressure may occur in the presence of an anesthesia which is satisfactory in all other respects. Furthermore, the addition of substantial amounts of fixed agent does not always lower the pressure.

Pulse Rate and Heart Rhythm. Pulse rate reflects so many influences that it is a relatively poor sign of anesthetic dose. It varies inversely with arterial pressure as dictated by the action of the baroreceptors. Thus it increases with halothane or enflurane and decreases with cyclopropane and often with balanced anesthesia. Sympathetic output also may be increased by anesthetics at a normal pressure. Thus ether and fluroxene anesthesia are associated with an increased pulse rate.

Ventricular arrhythmias may appear with any anesthetic. They are more likely to

Table 1.1. Usefulness of various signs or measures in the clinical assessment of depth.\*

| 140k iii Ostuniss         | 0             |            |       | Pulse                 |   |                      |             |
|---------------------------|---------------|------------|-------|-----------------------|---|----------------------|-------------|
| Anceshoein                | Pupil<br>size | Eye        | Blood | rate and/or<br>rhythm | Respira-<br>tion                        | Muscle<br>relaxation | Sweating    |
| Allesment                 | 2010          |            | 7     | +                     | +++++++++++++++++++++++++++++++++++++++ | +++                  | +           |
| Ether                     | +++           | +          | н     | -                     |   | -                    | +           |
| Cyclobrobane              | ++            | +          | +     | +                     | +                                       | +                    | ٠,          |
| Halothane                 | +             | #1         | +++   | +                     | ++                                      | +                    | <b>&gt;</b> |
| Enfluence                 | +             | +1         | +++   | ++                    | +++                                     | ++                   | 0           |
| Killiulalic               |               | ۱ +        | +     | ++                    | ++                                      | ++                   | 0           |
| Metnoxymurane             | + 4           | 4 <b>C</b> |       | +                     | +                                       | 0                    | ++          |
| N <sub>2</sub> 0-narcotic | <b>&gt;</b> ' | > <        | - 1   | - 4                   | +                                       | c                    | +           |
| N <sub>2</sub> 0-innovar  | 0             | •          | +-    | <b>-</b> 1            |   |                      | 0           |
| N.0-ketamine              | 0             | +          | ++    | H                     | +                                       | >                    | >           |

\*Usefulness is indicated on a scale of 0 (not useful) to + + + + (very useful).