Pre-Operative Respiratory Evaluation and Management of Patients for Upper Abdominal Surgery

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Patients presenting for surgery, be it on an elective or emergency basis, do so in the hope that the anesthetic will be without risk. Yet complications which arise are not always due to anesthesia. More often, the surgical process and factors intrinsic to the patient are major determinants of outcome. Pre-operative assessment allows review of the patient and the proposed surgery, and formation of a plan of management for the pre-, intra-, and post-operative anesthetic care. This paper provides an overview of the pre-operative assessment and management of patients who are to undergo upper abdominal surgery, with the aim of minimizing their risk of post-operative pulmonary complications. In particular, factors which contribute to the development of post-operative respiratory problems are described.

PURPOSE OF PRE-OPERATIVE ASSESSMENT AND MANAGEMENT

Pre-operative respiratory evaluation and management should be undertaken for two reasons. First, physicians should be able to identify patients with factors placing them at increased risk. Second, any reversible component should be treated or eliminated, thus potentially reducing the risk. Indeed, this is the aim of any pre-operative assessment or consultation. All patients undergoing upper abdominal surgery are at risk for post-operative respiratory complications, and those with diseases of, or affecting, the respiratory system are at increased risk. Specifically, the risk is of morbidity and mortality due to respiratory complications, or what Harrison describes as “failures in control of respiratory homeostasis” [1].

Since the first reports of fatal anesthetic complications were published, respiratory problems have been noted as occurring most frequently (although perioperative cardiac morbidity is the leading cause of death after anesthesia and surgery [2]). John Snow was the first epidemiologist anesthesiologist and made careful recordings of all his cases. His first death on the table was due to “the want of admission of sufficient air to the lungs” [3]. One hundred and fifty years later, Tiret and co-workers prospectively surveyed complications associated with anesthesia in France between 1978 and 1982. Almost 200,000 cases were studied, and causes of death (within 24 hours) and coma (persisting longer than 24 hours) were analyzed. More than half the deaths and comas attributable to anesthesia were due to respiratory problems [4]. It

Abbreviations: ACD: anesthetic contributory deaths ARDS: adult respiratory distress syndrome ASA: American Society of Anesthesiologists CCB: calcium channel blocker COPD: chronic obstructive pulmonary disease FET: forced expiratory technique FEV1,FVC: ratio of forced expiratory volume in one second FEV1,FVC: ratio of forced expiratory volume in one second to forced vital capacity FRC: functional residual capacity FVC: forced vital capacity PDPV: postural drainage, percussion, and vibration REM: rapid eye movement SaO2: saturation of arterial oxygen V/Q: ventilation/perfusion ratio

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must be remembered, however, that studies reporting anesthetic-related respiratory complications include all problems affecting the pulmonary system, from mishaps such as unrecognized esophageal intubation to post-operative respiratory failure on the ward.

Although all these problems are of importance, this paper will consider only post-operative complications originating "from the lungs." Thus, no attempt will be made to discuss such problems as hypoxia due to perioperative mechanical misadventure [5] or pulmonary thromboembolism [6,7]. These complications are not specific to upper abdominal surgery and are therefore beyond the scope of this review.

In general terms, pulmonary complications represent "an accentuation of normal post-operative pulmonary changes" [8]. The spectrum of complications is large and can range from subclinical atelectasis to difficulty weaning a patient from the ventilator to post-operative respiratory failure/arrest. All patients suffer some changes in respiratory function post-operatively, and the chance of altered outcome can be applied to this group as a whole. For example, 6–10 percent of all patients with normal lung function may develop post-operative respiratory complications [9]. Predictions can also be made for subsets of this population, as defined by the presence or absence of certain factors. Patients with pre-operative pulmonary disease have a statistically significant higher risk of mortality (15.6 percent) than those without pulmonary disease (1.6 percent) and may require post-operative mechanical ventilation 20 times more often than healthy patients [10]. The degree of risk for a single patient cannot, however, currently be defined, for two reasons. First, statistics apply to populations, not individuals. Second, lung function seems not to be as precisely measured and evaluated as that of, for example, the heart [2,11], perhaps reflecting the difference between "moving air" and "pumping blood." Thus, as stated by Goldstein and Keats, the estimate of anesthetic risk for individual patients remains "almost entirely intuitive" [12].

CONTRIBUTORY FACTORS

The factors which place a patient at increased risk may be classified as surgical, patient, and anesthetic (Table 1). Although all three are of importance when considering the problem of mortality, the interaction of surgical and patient factors is "responsible for approximately 90 percent of perioperative deaths" [13].

Surgical Factors

These are related primarily to the procedure site and duration. Complications such as post-operative pain, the presence of large amounts of traumatized tissue, and infection are also contributory.

Upper abdominal surgery encompasses procedures of the spleen, stomach, upper duodenum, pancreas, liver/biliary tree, and transverse colon. The term "upper abdominal surgery" can, however, be used to refer to any laparotomy which extends above the umbilicus and into the vicinity of the diaphragm. (Indeed, Beecher defined "laparotomy" to include "rest in bed, tight bandaging, post-operative medication" [14].) The importance of surgical factors is reflected in studies comparing outcome after various types of surgery. A review of about 100,000 anesthetics between 1972–1977 yielded a mortality rate of 6.6 percent for upper abdominal procedures in comparison to 2.8 percent for lower abdominal procedures [15]. Garibaldi et al. prospectively
studied 520 elective surgical patients to assess risk factors for post-operative pneumonia. The overall incidence was 17.5 percent, with those undergoing upper abdominal surgery affected three times more often [16]. Most recently, major upper abdominal surgery has been shown to be associated with a risk of 33 percent for the development of pulmonary complications in comparison to 16 percent after major lower abdominal surgery [17]. The same authors found an in-hospital mortality rate of 4.1 percent after gastrointestinal abdominal surgery, in comparison to the overall in-hospital mortality rate of 1.2 percent [18].

Upper abdominal surgery results in abnormal lung function, which is not unlike that seen in obese patients in the supine position [19]. Although the respiratory changes are interrelated, they are best described as primary, secondary, and tertiary interacting factors, as defined by Craig [20]. The primary changes are those of the pulmonary mechanics. The secondary changes represent abnormalities in gas exchange. The tertiary changes are those of respiratory control (and are discussed below).

The pulmonary mechanical dysfunction, or what Beecher termed “crippling of the respiratory mechanics” [21], is best characterized as a restrictive pattern of ventilation. The small tidal volume requires an increased respiratory rate to maintain minute ventilation. Inspiratory capacity and vital capacity are reduced. The latter decreases to 40 percent of normal pre-operative values immediately after upper abdominal surgery, in contrast to a reduction to 60 percent after lower abdominal surgery. At one week post-operatively, vital capacity is still abnormal, at 70 percent
and 85 percent of pre-operative values, after upper and lower abdominal surgery, respectively. Full recovery requires at least three weeks. Functional residual capacity (FRC) is also reduced, but the decline occurs slightly later, at 12–16 hours post-operatively. The nadir to 70 percent after upper abdominal surgery is reached at one day post-operatively. FRC becomes normal gradually over seven to ten days [20]. Maximum inspiratory and expiratory flow rates are also decreased [22].

Abnormalities in gas exchange occur in two phases [20,22]. The first is in the immediate pre-operative period and lasts up to about two hours (see below). The second phase is in the later post-operative period, because of pulmonary mechanical dysfunction. The limitations in inspiratory capacity restrict the patient’s ability to cough or sigh effectively. The changes in FRC, although of smaller volume, are of more importance because of the greater effect on oxygenation. The reduced FRC impinges on the closing capacity, or the volume at which small airways close. When this occurs, atelectasis is produced. Microatelectasis is suggested by the presence of “cough, low-grade fever or a widened alveolar-arterial gradient” [23]. These changes may occur in 25–75 percent of patients after upper abdominal surgery [24]. Macroatelectasis is visible on chest roentgenogram, with loss of segmental, lobar, or lung volume [25]. Decrease in surfactant activity helps to maintain airway closure [22]. Ventilation/perfusion (V/Q) mismatch and right-to-left shunting of blood are seen. Arterial blood gas values usually show arterial hypoxemia with normocarbia, with the reduction in FRC correlating with the degree of hypoxemia [20], and the lowest values found some 24 hours post-operatively. If hypercarbia is found, then respiratory failure may be imminent [22].

Why does this happen? Ford and co-workers were able to demonstrate the pulmonary changes in a study of 15 patients undergoing elective cholecystectomy [26]. They found a significant decrease in tidal volume without a change in minute volume in the immediate post-operative period. One-second forced expiratory volume and vital capacity were also decreased. The alveolar-arterial gradient widened, and patchy atelectasis was noted in nine of ten patients for whom chest roentgenograms were obtained. The most important finding was that of diaphragmatic dysfunction, as assessed by: changes in swings in transdiaphragmatic pressure during quiet tidal breathing (significantly decreased); the ratio of changes in gastric to esophageal pressure swings (significantly decreased); and the ratio of changes in abdominal to rib-cage diameter (decreased). The reduction in diaphragmatic activity was accompanied by a shift in the pattern of breathing, from predominantly abdominal to predominantly rib-cage. These findings, which returned toward normal by 24 hours post-operatively, were postulated to cause the reduced vital capacity, atelectasis, and hypoxemia seen after cholecystectomy.

More recent investigators have suggested that the diaphragmatic dysfunction may be due to “inhibitory reflexes of phrenic activity arising from the abdominal compartment (abdominal wall and/or viscera)” [27]. The same authors also showed that a single injection of 0.5 percent bupivacaine producing thoracic epidural block partially reversed the diaphragmatic dysfunction. Blockade resulted in an increase in tidal volume, vital capacity, and abdominal contribution to respiration, as well as a decrease in respiratory rate. The changes in forced vital capacity (both before and after blockade) were “parallel to the changes in diaphragmatic function.” The investigators determined that the diaphragmatic dysfunction was not due to a
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considered to be a "key mechanism underlying respiratory dysfunction" after upper abdominal surgery [27].

All surgery produces tissue destruction, and the skill of the surgeon is inversely proportional to the degree of trauma. Whenever the barrier of the skin is breached, infection may follow. Wound infection is a complication of surgery occurring in about 5 percent of patients undergoing "clean" procedures (e.g., elective, non-gastrointestinal); 10 percent of "clean-contaminated" procedures (e.g., gastrointestinal); 20 percent of "contaminated" procedures (e.g., major spills from the gastrointestinal tract); and 30–40 percent of "dirty" procedures (e.g., established infection) [38]. Infection contributes to respiratory problems in three ways. First, intra-abdominal sepsis occurs in direct relation to the duration of operation [39] and contributes to abdominal distention. This augments the pathophysiologic changes in respiratory dynamics started by the surgical process, and the resultant atelectatic lung is at risk of infection. Second, pathogenic aerobic gram-negative bacteria normally found within the gut may overgrow. These bacteria, and the endotoxin which they produce, escape across the poorly functioning gut mucosa. Once in the blood stream, the endotoxin, acting on Kupffer liver cells, causes release of mediators. These cytokines, as well as inducing ischemic changes in the gut, may also trigger cellular and humoral cascades to produce oxygen-derived free radicals and the pathological changes of adult respiratory distress syndrome (ARDS) [40,41,42]. Third, despite no loss of muscle mass, sepsis is associated with respiratory muscle fatigue [43], particularly diaphragmatic weakness [44]. Respiratory muscle blood flow and diaphragmatic oxygen consumption are greatly increased, in disproportion to respiratory work load. This finding would suggest a metabolic problem, such as uncoupling of oxidation/phosphorylation [44].

**Patient Factors**

As even 6–10 percent of patients with normal lung function develop major post-operative respiratory complications after abdominal surgery [9], the presence of abnormalities increases risk. For example, Pedersen et al. found that patients with pulmonary disease required mechanical ventilation 20 times more often than those without pulmonary disease [10]. Abnormalities may be defined as intrinsic or extrinsic. The former include obstructive airway disease, obesity, and other diseases. Of extrinsic factors, infection and smoking are the major examples.

Obstructive airway disease encompasses those conditions with reversible (asthma [9]) and irreversible (chronic bronchitis [45,46], emphysema [46]) components. Patients with asthma are more likely to be young, those with emphysema old. Some 4–5 percent of the British population have asthma, in comparison to 8 percent (female) and 17 percent (male) with chronic bronchitis [46]. The incidence of post-operative respiratory complications in these patients is twice that of "normal" patients [36]. The combination of pre-existing obstructive lung disease with the restrictive defect acquired as a result of surgery may invoke fatal pulmonary compromise.

Massive obesity is a risk factor because of its effect on increasing respiratory work. Alone, obesity rarely results in respiratory failure, but when combined with other factors, such as prolonged upper abdominal surgery, the need for mechanical ventilation may become obvious [47]. Thus although most physicians would intrinsically believe that massive obesity was a risk factor for post-operative pulmonary
dysfunction, certain studies do not demonstrate this premise. Garibaldi et al. showed that massive obesity was associated with an increased incidence of pneumonia, but not to a level of statistical significance when this factor was controlled for site or duration of surgery [16]; however, obesity may influence outcome indirectly. Many obese patients may actually be poorly nourished. These individuals and others with more obvious malnutrition may therefore be at significantly increased risk for the development of post-operative pneumonia, especially if the serum albumin concentration is low [16,48].

Just as it is difficult to delineate the degree of contribution of anesthetic and surgical factors to the development of post-operative complications, so it may be to define the importance of other non-pulmonary problems. For example, patients with chronic obstructive pulmonary disease often have associated cardiovascular complications. These conditions range from pulmonary artery hypertension, to right ventricular hypertrophy and right congestive heart failure (cor pulmonale) [49]. The presence of other conditions may precipitate the development of complications due to those conditions, e.g., pulmonary dysfunction causing hypoxia resulting in myocardial dysfunction. Or, those conditions may exacerbate pulmonary dysfunction, e.g., left ventricular failure causing pulmonary edema. Thus, in a recent prospective study of 7,306 anesthetics, the researchers considered cardiac and pulmonary complications together [50].

Not only may other medical conditions contribute to lung problems, but the drugs used to treat them may produce pulmonary dysfunction. For example, non-selective beta-adrenoreceptor antagonists used in the treatment of hypertension can induce severe attacks of asthma. Selective beta, adrenoreceptor antagonists are less problematic but should probably be avoided. Angiotensin converting enzyme inhibitors can produce bronchial reactivity and cough because of decreased metabolism of inflammatory substances in the bronchial mucosa such as bradykinins. Calcium channel blockers (CCB), which are used extensively to treat coronary spasm and hypertension, do not have a major effect on resting bronchial tone and may reduce bronchospasm induced by exercise, cold air, histamine, and cholinergic agonists [51]. Some effect is also seen with allergen-induced bronchospasm, but dilation of pulmonary arteries may be produced, especially in the presence of hypoxic pulmonary vasoconstriction [52]. Furthermore, because excitation-contraction coupling appears to be dependent on extracellular calcium, the CCB may have deleterious effects on diaphragmatic function [53].

The patient with a respiratory tract infection presents a clinical dilemma: to anesthetize or not. If the patient is a child, many anesthesiologists would postpone surgery. Although post-operative symptoms may not be increased in those anesthetized with "uncomplicated" upper respiratory tract infections, intra-operative complications may be more frequent, as may be the incidence of transient post-operative hypoxemia in the recovery room. For adult patients, the evidence is unfortunately not quite so clear [54], although the accompanying malaise may provoke some patient-postponed procedures.

The patient who smokes is at risk of respiratory and other complications in the immediate perioperative period. The inhalation of cigarette smoke acutely increases tidal volume by stimulating epipharyngeal mucosal receptors and triggering the "aspiration reflex" [55]; however, this reaction seems to be the only "beneficial" result. Smoking has been described as having the same effect on closing volume as
that of aging ten years [56]. When closing volume exceeds FRC, the resultant airway closure produces arterial hypoxemia. Patients with lung disease from cigarette smoking also have increased mucous secretion, but decreased mucociliary transport [57], thus predisposing them to the development of atelectasis and its sequelae [36]. Although Strandberg and colleagues could not show a significant difference in transverse area of regions of atelectasis (using chest tomography), only pre- and intra-operative measurements were made [58]. Surprisingly, those who smoke have no greater a decrease in cough threshold (or cough sensitivity) than those who do not. Dilworth and colleagues tested 12 smokers and 14 non-smokers with aerosols of capsaicin and citric acid before and after upper abdominal surgery under general anesthesia. Most patients had a substantial decrease in cough threshold on day one, with a trend toward normal by day four [59].

Several recent studies have examined the role of smoking in the development of perioperative hypoxemia. Riley and colleagues monitored the decrease in saturation of arterial oxygen (SaO₂) during transfer from the induction room to the operating room. Using multiple regression analysis, they showed that transfer time, smoking history, and ventilatory status during transfer “accounted for 64 percent of the variation in the fall in SaO₂” [60]. Recovery room oxygenation on arrival and then 15 minutes later was evaluated by Hudes et al. No statistically significant effect of a history of smoking was noted, although “obesity and age” were significant factors in patients who were hypoxemic on arrival [61]. Brown and co-workers determined the incidence of hypoxemia in two groups of patients, in the pre-operative period and in the first hour of post-operative recovery. In the pre-operative group, 21 percent had episodes of hypoxemia (some of whom smoked). In the post-operative group, 80 percent of patients showed saturations below 90 percent. Increased susceptibility to hypoxemia was found in patients who were either male, smoked more than ten cigarettes a day, or received opioids intra-operatively [62]. The incidence and duration of hypoxemia (SaO₂ < 90 percent) in the recovery room was also studied by Moller et al. Cigarette smoking was identified as a significant risk factor in comparison to lung disease alone, which was not [63]. The most specific study of the effect on changes in post-operative arterial oxygen saturation after elective intra-abdominal surgery was carried out by Tait and colleagues. Although the non-smoking group of patients was significantly older than the smokers, the severity of hypoxemia seen on arrival in the recovery room was significantly greater in the smokers [64].

Non-respiratory effects of smoking include activation of the sympathetic nervous system, with increases in heart rate, blood pressure, and plasma concentrations of noradrenaline and neuropeptide Y. These two substances coexist in sympathetic postganglionic perivascular nerves in both the myocardium and “most vascular beds” [65]. Their release may contribute to the pressor response seen after smoking, and a relationship with coronary spasm has been postulated [65]. One problem which has not been shown to be related to cigarette smoking is that of increased risk of pulmonary aspiration of gastric contents. Adelhoj and co-workers failed to demonstrate a significant difference in gastric volume and pH between smokers and non-smokers (after overnight abstention from smoking) [66]. More recently, Hardy and colleagues found similar results. In addition, no gastroesophageal reflux was detected, although those who smoke should have diminished lower esophageal tone, thus contributing to regurgitation [67].
Anesthetic Factors

The general effect of anesthesia is one of depression, albeit for the most part controlled, of the body’s vital functions. Anesthetic factors which contribute to the development of post-operative respiratory problems do so through the presence or continued activity of drugs which alter or depress the respiratory system. The anatomical changes in the respiratory system are reflected mainly in the development of hypoxemia in the early post-operative period. Depression of ventilation occurs either centrally (opioids, sedatives) or peripherally (neuromuscular blockade). Defense mechanisms may also suffer: residual incompetence of pharyngeal reflexes predisposes to aspiration; impaired host defenses may contribute to the development of pneumonia. Thus, the choice of anesthetic may influence post-operative respiratory function.

Anatomic changes induced by anesthetic agents often persist into the post-operative period. Although they are not specific to anesthesia for upper abdominal surgery, they are mentioned here because they complement or magnify changes induced by these operations. Thiopentone tends to constrict airways, whereas ketamine has a bronchodilator effect, even in patients with severe reactive airways disease such as status asthmaticus. Inhalation agents all prevent or reverse bronchospasm [68].

Partial occlusion of the oropharynx and total occlusion of the nasopharynx results from posterior movement of the tongue, soft palate, and epiglottis, toward the posterior pharyngeal wall [69]. This area may be the major site of interference with the pharyngeal airway [70].

As well as the decrease in FRC attributed to the surgeon’s hand near the diaphragm, anesthesia induces changes in FRC. This process is considered as “compression atelectasis in dependent regions of the lungs” [71]. A decrement is seen immediately after induction, virtually no matter which drugs (including neuromuscular blocking agents) are used [72]. The major contributors to the anesthetic-induced decrement are thought to be movement of blood (in or out of the thorax), cephalad shift of the diaphragm [73], and decrease in volume of the rib cage. Interestingly, there is apparent disagreement, as to the magnitude of the contributing changes, between investigators in Sweden and the United States [69,72].

All these changes impair ideal gas exchange. Arterial hypoxemia may be due to residual anesthetic agents causing hypoventilation, diffusion hypoxia, shivering-induced increased consumption of oxygen [69], and residual abolition of hypoxic pulmonary vasoconstriction [69,74]. Redistribution of ventilation, to the “top” or poorly perfused regions of the lungs, as well as hemodynamic factors such as changes in cardiac output, also contribute. Pulmonary edema in the post-operative period may be associated with severe hypoxemia. The condition usually occurs with relief of (although occasionally during development of) acute severe upper airway obstruction [75].

In 1908 and 1910, Pasteur published descriptions of post-operative respiratory complications which, as well as interesting from a historical perspective, also provided a strong hypothesis for their development [28,76]. He ascribed massive collapse of the lung as being due to deficiency of “inspiratory power” [76]. Although his theory related to diaphragmatic dysfunction, the term may well be applied today. In his 30-year analysis of the “clinical failures” responsible for anesthetic-contributory deaths (ACD), Harrison classified the mechanisms/causes of the 145
deaths in this period. Failure "to ensure adequate pulmonary ventilation" was the second most common cause of ACD (20 percent). Most of these deaths occurred immediately post-operatively because of hypoventilation due to peripheral depression from residual neuromuscular blockade or central depression by opioids [1]. Other workers have shown similar problems. Pedersen et al. noted respiratory insufficiency due to overdose of narcotics or residual neuromuscular blockade in almost 2 percent of cases [10]. Tiret et al. reported respiratory depression to be the cause in 50 percent of deaths in the post-operative period attributable to anesthesia. Unlike Harrison's study, this research showed that narcotics were the major cause of respiratory depression [4].

The effect of narcotics on post-operative ventilatory function was clearly shown by Catley and co-workers, who compared the effects of morphine infusion with regional anesthesia after cholecystectomy or total hip replacement in otherwise healthy patients. The 16 patients who underwent upper abdominal surgery and then received opioids had 220 episodes of hypoxemia (SaO₂ less than 80 percent) in the first 16 hours after surgery. In comparison, the 16 patients who received regional anesthesia did not have any episodes of hypoxemia while breathing room air. Moreover, there was a significant difference in the two groups in their minimum oxygen saturations achieved. Five types of respiratory dysfunction were seen: decreased tidal volume, decreased ventilatory rate, obstructive apnea, central apnea, and paradoxical breathing. These conditions occurred only during sleep. Although sleep normally produces some mild changes in ventilatory pattern and arterial blood gas results, usually only males are affected, and changes in oxygen saturation are not significant unless the patients are obese. These authors clearly showed, however, that sleep was associated with marked changes in arterial blood gas values in these patients of both sexes. Furthermore, despite the anticipated changes in diaphragmatic function after cholecystectomy, the incidence of paradoxical breathing was similar in patients receiving morphine, after either cholecystectomy or total hip replacement [77].

Yeager et al. compared epidural anesthesia and post-operative analgesia with general anesthesia and parenteral narcotic administration. Patients underwent a variety of procedures which included intra-abdominal surgery. Morbidity, defined as organ failure, infection, or re-operation, was found in more than twice as many patients who underwent general anesthesia than who received epidural anesthesia/analgesia. Similarly, more than twice as many patients in the former group suffered respiratory failure (the need for mechanical ventilation for greater than 24 hours post-operatively, or for reintubation and mechanical ventilation after extubation) [78]. Although this study has been criticized because of its small size [79], the results are striking.

Pain and the opioids used to treat it are considered principal causes of sleep disruption after surgery. The importance of severe disruption of nocturnal sleep lies in the occurrence of "highly intense rapid eye movement (REM) sleep about the middle of the first post-operative week." This period coincides with the time when cardiac, neurologic, and psychiatric complications occur [80]. During normal rapid eye movement (REM) sleep, there is "relative loss of hypercapnic ventilatory response and preservation of hypoxic responses" [81]. Thus, administration of opioids, which also decrease CO₂ responsiveness and eliminate sighing [20], will compound this effect [81]. Furthermore, the drugs are often administered as a bolus according to schedule, rather than need, and usually without any form of monitoring
apart from intermittent observation and measurement of vital signs. The occurrence of opioid-induced respiratory depression is therefore easily explained.

Normal lung defenses are found at all levels of the respiratory tract. In the nose, filtration and gravity (mucosal impaction) are important in removing larger particles. At the level of the larynx, swallow and cough reflexes prevent aspiration. The mucociliary apparatus in the bronchi sweeps potentially dangerous debris from the airway. In the alveoli, macrophages, complement, lymphatic drainage, and surfactant help to repel bacteria and viruses [25,82]. Each of these defenses may be altered or depressed by anesthetics agents or equipment.

Drug-induced laryngeal incompetence results from such agents as lidocaine, either applied topically to the pharyngeal tissues or given intravenously. (Almost all anesthetics, except ketamine, when given in normal doses, produce this effect.) The presence of an endotracheal tube produces laryngeal incompetence, despite a functioning high volume-low pressure cuff. Unfortunately, the cuff may then act as a barrier to the upward movement of particles carried by the mucosal ciliary apparatus [83]. Furthermore, movement of the tube in relation to the trachea during each ventilatory cycle may allow migration of bacteria down the trachea [83]. Thus, upper respiratory pathogens can gain access to the lower regions of the lung. Traumatized mucosa may then allow bacteria to adhere to exposed basement membrane and permit colonization [83]. Contaminated equipment may also introduce bacteria directly into the respiratory tract [23,83].

Even without pre-existing tracheal intubation, patients are at increased risk of aspiration and aerosolization of pharyngeal contents during sleep. The incidence of discovery of these secretions was 1.5 times higher in those with “altered consciousness” in comparison to normal patients [82]. This process may be an important mechanism contributing to the development of post-operative pneumonia. Aspiration of as small a volume as 0.01 ml could inoculate up to $10^5$ bacteria, including Viridans streptococci (Streptococcus salivarius and Streptococcus sangius), Branhamella catarrhalis, and non-typable Haemophilus influenzae [82]. These bacteria represent some of those cultured from the mouths of non-hospitalized patients. Should a patient develop a respiratory tract infection while in hospital, however, the bacterial species are likely to be quite different and may include “endogenous” Escherichia coli and “exogenous” Pseudomonas aeruginosa [83].

Certain anesthetic techniques are more likely to induce problems than others. For example, the use of high frequency jet ventilation presents the difficulty of adequate humidification of entrained air, with resultant deleterious effects on respiratory mucosal function [84]. Compounding the mechanical effect is that of immunosuppression. Anesthetic agents, in particular the halogenated vapors and nitrous oxide, depress lymphocyte function (perhaps synergistically with the immunosuppression induced by the stress response from the trauma of surgery).

**EVALUATION**

Respiratory “success” is governed by the balance between respiratory drive and load, which in turn depends on respiratory muscle endurance [43,47]. The aim of pre-operative evaluation is to determine if any of the influencing components is likely to change, such that respiratory “success” becomes “respiratory failure.” As for any patient, a careful history and physical are of great importance in detecting problems.
A thorough clinical examination has been shown to be effective in identifying patients at risk of post-operative surgical complications [85, 86].

**History Taking**

Once a patient has been assigned to have pre-operative assessment, this procedure should be done systematically. One way of doing so is to define a list of problems, which may then be organized according to degree of threat to life. For example, unstable angina would rank before non-insulin-dependent diabetes mellitus and long-standing hypothyroidism requiring thyroid hormone replacement. The physician should also attempt to determine if a link exists between problems. For example, patients with hiatus hernia often present with a chronic cough as their major symptom. A past history of certain conditions may also be of significance. For example, testicular carcinoma treated with bleomycin represents a risk factor for post-anesthetic respiratory dysfunction [87].

Certain specific symptoms should be sought. The patient with orthopnea may have respiratory or cardiac disease. If the former, then symptoms will be reported to develop rapidly, as chest wall and diaphragmatic mechanics are altered by a change from the vertical to the horizontal position. If the latter, then symptoms will be delayed some minutes to hours [23]. Dyspnea is a more general term meaning the sensation of some difficulty in breathing (remembering that most people are unaware of the “task” of breathing). The cause may be psychogenic or physical. A description of exercise tolerance, and especially a change, is of great importance. For example, the housewife may report giving up vacuuming, and the gardener raking, as these represent the most difficult daily tasks.

A cough, like sniffing, may be nothing more than a nervous habit. Unfortunately cough most often reflects respiratory disease, of the upper airway with chronic rhinitis, or of the lower tract, with asthma, bronchitis, bronchiectasis, or bronchogenic carcinoma [23]. Other infectious processes such as tuberculosis (in immigrants) and *Pneumocystis carinii* (in HIV-positive patients) must also be considered. Thus, not only the nature, timing, and length of the cough, but the color, consistency, and volume of sputum should be elicited. Yellow, red (or brown), and green are colors to be noted! White sputum suggests the absence of significant numbers of leucocytes present. Fresh, purulent sputum is usually yellow, but red or brownish if red blood cells are present. As secretions stagnate in the lungs, oxidation by leucocyte myeloperoxidase (or verdoperoxidase) causes sputum to become greenish. Although yellowish or greenish sputum suggests infection, asthmatic secretions may be yellow because of eosinophils or Dietrich’s plugs [88].

Wheeze suggests hyper-reactivity of the airways and may reflect severe asthma. Or, the patient may have simple exercise-induced wheezing related to water loss from the airways. In either case, the presence of pre-operative wheezing should alert the physician, as more severe bronchospasm may occur perioperatively.

The lifetime history of cigarette consumption should be calculated. The simplest value is that of the “pack-year,” equivalent to one pack of cigarettes smoked each day for one year. (Partial inhalation of the cigarette does not warrant a reduction in pack-years!) Many patients in their middle years who have smoked since their teens are astounded when told that they have a history of 70 to 80 pack-years. These patients, unfortunately, are at increased risk of both cardiorespiratory disease and
malignancies. The threshold for chronic bronchitis is apparently eight pack-years [25].

Physical Examination

Anesthesiologists, perhaps more than any other specialists, are trained to detect the presence of respiratory distress. The slight frown, flared nostrils, and firmly held shoulders are but three of the subtle signs of respiratory problems. Physical examination may also provide clues to a propensity for post-operative respiratory failure after upper abdominal surgery. For example, the presence of glossitis or cheilosis may indicate malnutrition [89], and "clubbing" may indicate severe cardiorespiratory disease. Examination should also encompass the "traditional" areas of interest of the anesthesiologist, such as the airway [90] and the cervical spine [91]. In addition, "morbidity and mortality in surgical patients usually derives from the failure of a vital bodily system." Therefore, "a proper clinical examination (i.e., something more than that usually undertaken by the operating surgeon) . . . is required to pick out those patients particularly at risk" [85].

Laboratory Examination

History taking and physical examination will identify which tests should be chosen to define numerically the patient's conditions. Although the "ideal test" should differentiate those at, or not at, risk, unfortunately such a test does not exist [23]. Furthermore, use of laboratory examination as a "screening device" for disease is not as successful as a careful history and physical examination [85], especially in the asymptomatic patient [86]. The patient will also be subjected to unnecessary discomfort and money wasted. As well stated by Roizen, the "15-minute history and physical examination are probably the best screening tests preparatory to anesthesia" [86]. Some suggested tests are listed in Table 2. Each of these can yield specific information, also listed in Table 2. In addition, some tests will provide extra data, of use in perioperative management. For example, the arterial blood sample can also be tested for carboxyhemoglobin concentration. Abnormal results (3–15 percent) sug-
gest excessive cigarette consumption or increased environmental exposure (home-, work-, or travel-related) [57].

Unlike cardiac disease, pulmonary disease offers no multifactorial risk scale combining history and physical examination and laboratory results [11,92]. There is no simple pre-operative exercise stress test of the lungs which can provide an objective means of functional assessment before upper abdominal surgery. In particular, no single or group of pulmonary function studies can identify patients at risk for respiratory complications after upper abdominal surgery [93]. Abnormal values which may suggest increased risk include values less than 50 percent for forced vital capacity (FVC), forced expiratory volume in one second (FEV₁₀), and the ratio of these two measurements (FEV₁₀/FVC). A recent comprehensive review, however, analyzed all articles published in English from 1966–1987 (Medline search). The reviewers were able only to recommend spirometry before abdominal surgery in patients who are “cigarette smokers or have respiratory complaints that have not been previously evaluated.” Spirometric assessment was considered most helpful in “raising the level of vigilance in the perioperative period and by suggesting helpful therapies” [94]. In addition, the values of pre-operative pulmonary function testing, when considered as a baseline, suggest a patient’s “total respiratory reserve” [95].

MANAGEMENT

The initial part of management is the determination of the optimal timing of pre-operative evaluation. This assessment must be done in concert with considering the length of time required to treat/reverse certain conditions. For example, the patient presenting for an emergency laparotomy for gangrenous cholecystitis needs immediate, swift attention. “Stat” laboratory examination should include a complete blood count, serum electrolytes, hepatic enzymes, prothrombin and partial thromboplastin times, arterial blood gases, electrocardiogram, and chest roentgenogram (possibly portable). The obese, middle-aged (but otherwise healthy) patient with asymptomatic cholelithiasis can be seen weeks before operation. Laboratory examination may be limited to a hemoglobin and electrocardiogram. In general, however, patients should be seen within one to two weeks of surgery. From this precept follows the decision as to where evaluation and management are best carried out. Evaluation carried out immediately pre-operatively can be done in the operating room, with blood for laboratory testing sent as intravenous and intra-arterial lines are also inserted. A more leisurely approach requires a defined area for assessment and potential treatment.

Surgical Factors

Once those factors which define risks are identified, they may be used to organize a plan of management. The first step is to ask if the surgery is necessary. Unfortunately, this question is intrinsically difficult for anesthesiologists (as they lack surgical training). At times, however, the most important role of the anesthesiologist is to act as the patient’s advocate and to question the need for, and/or extent of, surgery. For example, especially in the elderly patient, it is important to discourage the performance of “en passant” procedures [39]. Sometimes delay of even an hour or two will allow more precise pre-operative assessment and management [96]. One factor which should contribute to a decrease in respiratory complications is the declining incidence of certain types of operations. For example, H₂ receptor antagonists have
made dramatic changes in the treatment of ulcerative gastric lesions [97]. Even cholecystectomy for cholelithiasis may become an “endangered operation” with the introduction of topical and oral dissolution of gallstones, biliary lithotripsy, endoscopic sphincterotomy, and percutaneous extraction [98]. The development of “keyhole” surgery promises to produce a further decline in post-operative respiratory complications.

In the post-operative phase, techniques to maintain or restore FRC and thus increase lung expansion are helpful, but only if applied conscientiously to those predicated to be at increased risk of pulmonary complications [8]. These techniques include incentive spirometers, chest physiotherapy, and early mobilization [96]. Incentive spirometry should not be confused with the deflationary “blow bottle” [20]. Incentive spirometry is based on the theory of sustained maximum inflation opening closed airways and alveoli and preventing further atelectasis [22]. In 1982, Bartlett, the “father” of the “incentive spirometer” or “inspiratory breathing exerciser,” stated that post-operative pulmonary complications could be prevented “by frequent maximal lung inflation (i.e., a normal pattern of breathing) . . . when properly used, incentive spirometer breathing is an effective and inexpensive method to accomplish the objective” [99]. Two years later and citing “controversy” regarding “the routine use of aids to lung expansion in the prevention of pulmonary complications after abdominal surgery,” Celli and co-workers carried out a prospective study. They randomly assigned 172 patients undergoing abdominal surgery to one of four treatment groups and compared the incidence of pulmonary complications and length of hospital stay. Those receiving incentive spirometry, intermittent positive-pressure breathing, and deep breathing exercises had an incidence of 22 percent of complications versus 48 percent in the control group. Patients who had undergone upper abdominal surgery and then received incentive spirometry also had a significantly decreased length of hospital stay than did the control group. In addition, no complications of incentive spirometry treatment were seen, in comparison to an incidence of 18 percent with the intermittent positive-pressure breathing therapy [100]. In 1985, an American survey of “lung expansion maneuvers in the prevention and management of post-operative atelectasis” after abdominal and thoracic surgery found that incentive spirometry was used in more than 95 percent of hospitals [101].

Since these reports were published, two recent studies have carefully examined the effect of incentive spirometry [102] and diaphragmatic breathing maneuvers [103] after cholecystectomy. Incentive spirometry was found to increase “tidal excursion of the chest compartment . . . without any increase in abdominal tidal volume,” thus failing “to increase diaphragmatic movement” [102]. In contrast, coached diaphragmatic breathing increased “tidal excursion of the diaphragm,” but only when the patient was supine, rather than semi-recumbent. The authors stated that movement of the diaphragm is important in lower lung field ventilation “and that most atelectasis and infection occurs in the lower lung field” [103]. Thus, use of this technique could decrease the pulmonary complications of upper abdominal surgery.

Chest physiotherapy encompasses the forced expiratory technique (FET) and the triad, postural drainage, percussion, and vibration (PDPV). Both FET and PDPV are of benefit in patients with copious amounts of sputum in central airways. In those with atelectasis from sputum blockage of a major airway, PDPV is helpful but may produce bronchospasm and hypoxemia [104]. Monitoring of oxygenation is therefore suggested [104,105,106].
Patient Factors

Patient factors are potentially susceptible to the most manipulation. Intrinsic factors such as airway obstruction can be treated with bronchodilators, occasionally steroids (especially by inhalation therapy), and chest physiotherapy. Because of pre-existing respiratory defects, post-operative deterioration may be difficult to detect in the early stages of compromise [46]. Thus, a raised index of suspicion (from history, physical examination, and laboratory results) is particularly important.

Obesity, especially in the short term, is not susceptible to treatment, although the advent of very low-calorie diets, with synthetic amino acid supplementation and close medical supervision, may offer hope, even in as short a time as six weeks; however, the obese patient who suffers from sleep apnea may benefit from nasal continuous positive airway pressure, especially in the perioperative period [107]. The patient with weight loss and wasting may benefit from "nutritional repletion" before major surgery, provided that sepsis is not present [43, 48, 85]. Aminophylline and theophylline may improve respiratory muscle strength, particularly of the diaphragm [108]. This improvement may, however, be at the cost of increased oxygen consumption and potentiation of respiratory muscle fatigue [43]. Therapy may also be instituted for certain other diseases. The progression of emphysema in alpha-1-anti-trypsin deficiency may be "halted" through the use of "augmentation therapy" with purified alpha-1-anti-trypsin [109].

Extrinsic factors such as smoking or occupational exposure to carbon monoxide should be reduced, not only because of the dangers of hypoxia but because of the interference with pulse oximetry monitoring [105]. Patients should be encouraged to stop smoking in the pre-operative period. After 12 to 24 hours, concentrations of carbon monoxide and nicotine are reduced, and after 48 hours, the carboxyhemoglobin of smokers should fall to that of non-smokers [57]. Ciliary beating is improved after two to three days. After one to two weeks, sputum volume is significantly reduced (although most ex-smokers would attest to the seeming rebound increase in sputum production on first quitting). Post-operative respiratory morbidity is influenced "greatly" after four to six weeks, when some improvement in the degree of small airways disease is seen on pulmonary function testing. A minimum of six to eight weeks is required to allow recovery of the immune system and to effect changes in drug metabolism [57]. Not until the patient has stopped smoking for some eight to 12 weeks is there "an improvement in post-operative respiratory morbidity" [46]. Infection requires antibiotic therapy, with repeated clinical examination to assess efficacy. Pulse oximetry and chest roentgenography may also be helpful in monitoring resolution of the infection.

Anesthetic Factors

The patient with airway disease from cigarette smoking will have an increase in alveolar dead space; this condition becomes worse with increasing age. Use of a nomogram (to determine ventilatory requirements) and end-tidal pCO₂ (to determine adequacy of ventilation) may lead to hypoventilation. This effect can be minimized by ventilation with large volumes (greater than 800 ml) at slow rates (8–12 breaths/minute), facilitating gas exchange in areas of low V/Q with long time constants [110]. The ventilatory requirements may be 25 percent greater than expected [111].
Other anesthetic factors may be reduced by the use of short-acting drugs (possibly by infusion), thus ensuring the minimum blood concentrations of respiratory depressants. Unfortunately, despite seemingly advantageous pharmacokinetics, such short-acting drugs as alfentanil have been associated with profound respiratory depression in patients who were apparently awake and talking [112]. In addition, the use of benzodiazepines (specifically midazolam) in conjunction with fentanyl or other opioids may result in an increased incidence of hypoxemia and apnea. The drugs act synergistically on the ventilatory center, decreasing hypoxic ventilatory drive [113]. Such reports would suggest the need for careful titration of drugs, the administration of oxygen in the recovery room, and peripheral arterial oxygen saturation monitoring. A raised index of suspicion for the possibility of respiratory depression in patients, especially those who have received naloxone, may help to prevent tragedies. Furthermore, an aggressive approach to the problem of post-operative hypventilation (i.e., continuing or instituting intermittent positive-pressure ventilation) is usually safer than allowing the patient to breathe spontaneously and waiting for improvement or deterioration. The latter often leads to disaster, as well shown by Harrison [1]. Should ventilatory support be necessary, use of techniques such as airway pressure release ventilation may yield an increase in CO₂ elimination and avoid conventional mechanical ventilation [114].

A careful plan for provision of analgesia, with appropriate monitoring (i.e., pulse oximetry), may avoid problems with hypoventilation and possibly respiratory/cardiac arrest in the recovery room and on the ward. In addition to standard analgesic regimes using intramuscularly administered opioids, the use of transcutaneous nerve stimulation [20], intravenous drug administration, transdermal drug administration, patient-controlled analgesia, nerve blocks, and intraspinal/epidural administration of opioids and/or local anesthetics may be of benefit in selected patients. Even psychological preparation may be helpful [20].

CONCLUSIONS

Patients undergoing upper abdominal surgery will suffer a decrease in ventilatory drive, a restrictive respiratory defect, and general depression of immune function. These changes will be magnified in those patients with underlying respiratory disease and compounded in those with other problems such as cardiovascular disease. Pre-operative evaluation allows definition and possible treatment or correction of underlying problems. Unfortunately, no single test or combination of tests will allow prediction of the risk of complications; however, simply identifying and defining the problems can be of benefit. Over the past decade, mortality from post-operative respiratory failure has not increased, despite the increasing age and American Society of Anesthesiologists' (ASA) classification of patients undergoing major abdominal and other types of surgery [22]. One of the contributing factors to this improvement in outcome is careful perioperative management. Although best results will be achieved if the patient can be seen some weeks in advance, even those patients who present for emergency surgery can benefit from a coherent plan of management, designed to minimize the contribution of surgical and anesthetic factors and thus to improve outcome.

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