Pulmonary Function during the Perioperative Period

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Postoperative pulmonary complications, a major cause of morbidity and mortality [1], lead to increased resource utilization, extended hospital stays and added costs. These complications are caused by successive changes in the structure and function of the respiratory system during the peri-operative period. Atelectasis forms upon the induction of general anesthesia and results in loss of lung compliance and a reduction in functional residual capacity. It may take weeks until pulmonary function is restored to pre-operative levels [2]. Although the natural history of postoperative atelectasis is usually one of spontaneous re-inflation, failure of these regions to re-inflate may result in pneumonia. The pulmonary consequences of thoracic and cardiac surgery have recently been reviewed [3]. This article reviews the etiology and consequences of pulmonary complications after abdominal surgery.

Peri-operative respiratory pathophysiology

The intraoperative period: general anesthesia

General anesthesia reduces functional residual capacity and predisposes to the development of atelectasis and hypoxemia. The multiple inert gas elimination technique to measure lung ventilation-perfusion relationships and computed tomography has been used extensively to examine patients during the induction and maintenance of anesthesia. CT revealed that densities form in the dependent portions of the lungs immediately upon induction of anesthesia with inhalation (e.g., isoflurane, halothane) and most intravenous (e.g., barbiturates) anesthetics [2]. Neuromuscular blocking agents (e.g., pancuronium) further contribute to density formation. The densities remained in the same dependent position when subjects were turned from the supine to the lateral decubitus position, suggesting that they were not pleural fluid but atelectasis [4]. Further evidence that the densities are atelectasis was their decreased size or total disappearance when 10 cm H_2O positive end-expiratory pressure was applied, and rapid return upon discontinuation of PEEP [4]. Histology of the atelectatic portions of anesthetized sheep lungs revealed completely collapsed parenchyma with only moderate vascular congestion and no interstitial edema, supporting the contention that the atelectasis is due to compression [5].

Atelectasis and decreased FRC are thought to be due to mechanical factors that reduce thoracic volume, leading to compression atelectasis. These factors include cephalad movement of the diaphragm, decreased cross-sectional area of the rib cage and increased intrathoracic blood volume. Studies during general anesthesia with neuromuscular blockade suggested cephalad diaphragmatic displacement at end-expiration that is probably due to loss of end-expiratory diaphragmatic tone [6]. This would permit transmission of abdominal pressure into the chest cavity, thereby increasing basal pleural pressure and causing alveolar collapse. Others found end-expiratory cephalad motion of the dependent portions of the diaphragm and caudad motion of the rest, but no consistent net shift in position. Stimulation of the phrenic nerve during anesthesia caused diaphragmatic motion and reduced the amount of dependent atelectasis [7]. This is cited as evidence that atelectasis formation is at least partially due to relaxation of the diaphragm. During general anesthesia with paralysis and in spontaneously breathing subjects anesthetized with halothane, CT scans revealed a reduced end-expiratory thoracic transverse cross-sectional area [8]. The decrease in total end-expiratory thoracic volume was greater than the reduction in gas volume measured by evaluating FRC. The thorax contains tissue and blood plus gas, thus the decreases in thoracic volume could be due to changes in blood volume as well as gas content. There are conflicting reports of whether anesthetized patients have increased [9] or decreased [8] blood volume. The contribution of each of the three factors to atelectasis formation under various anesthetic conditions needs further elucidation. Another

PEEP = positive end-expiratory pressure
FRC = functional residual capacity

factor contributing to intraoperative atelectasis is mechanical manipulation, i.e., retraction of abdominal contents against the diaphragm.

Gunnarsson et al. [10] found that anesthesia with an enflurane/nitrous oxide/O_2 mixture increased intrapulmonary shunt and was paralleled by decreased perfusion of areas with low ventilation. This was not seen when nitrogen was substituted for nitrous oxide and was ascribed to absorption of nitrous oxide resulting in atelectasis. The investigators speculated that the initial compression atelectasis is followed by absorption atelectasis [10]. Further evidence of absorption atelectasis came from studies with 100% O_2. More dependent lung density formed with 100% than with 30% O_2 [11]. After re-expansion of atelectasis with an inflation to vital capacity, collapse recurred more readily with 100% than with 40% O_2. Anesthetized patients ventilated with 100% O_2 subjected to a vital capacity maneuver and then ventilated with 10 cm H_2O PEEP had less atelectasis than those ventilated without PEEP [12]. PEEP may thus be employed when 100% O_2 is needed to prevent intra-anesthesia hypoxemia. Besides PEEP, pressure control inverse-ratio ventilation also reduced the intraoperative A-aDO_2 gradient. However, both maneuvers did not influence the postoperative A-aDO_2 gradient [13].

Since absorption of soluble gases (O_2 and nitrous oxide) accentuate anesthesia-induced compression atelectasis, it would seem useful to use low O_2 concentrations during surgery to prevent both intra- and postoperative atelectasis. Yet there were no differences in postoperative lung volumes (VC and FEV_1) between 30% and 100% O_2 after hysterectomy, colon resection and upper abdominal surgery [14]. Intraoperative absorption atelectasis may not be a major contributor to reductions in postoperative lung volumes, so the practice of using elevated O_2 concentrations to prevent intraoperative hypoxemia may continue.

Intraoperative atelectasis is associated with a reduction in FRC greater than the 0.5 L decrease seen with a change from the upright to supine position. The latter is caused in part by cranial displacement of the diaphragm. Induction of general anesthesia with or without neuromuscular blockade decreases FRC by another 0.5 L. The intraoperative FRC decrease is associated with impaired oxygenation during both spontaneous and mechanical ventilation. Both increased ventilation/perfusion mismatch and shunting contribute to lower PaO_2. Young healthy anesthetized subjects increased V/Q mismatch with little increase in shunt, while older patients (37–64 years) had more shunting (8.6%). Elderly patients (>65 years) had greater V/Q mismatch and more shunting (15%). Other researchers did not find that shunt and atelectasis increased with age, although the elderly had more low V/Q areas. Intravenous barbiturates cause less shunting and V/Q mismatch than inhalation anesthetics, which is most likely due to preserved hypoxic pulmonary vasoconstriction . The amount of atelectasis on CT scan correlated well with the magnitude of the shunt, but not with V/Q inequality. Hypoxemia in smokers, the elderly and the obese tends to be more severe. Patients with chronic bronchitis had little shunting and little or no atelectasis on CT, possibly due to air trapping. They did have severe V/Q mismatch that was attributed to enhanced perfusion of areas with already low ventilation.

During laparoscopic abdominal surgery the abdomen is insufflated with CO_2. The increased abdominal pressure results in decreased total respiratory system, lung and chest wall compliance and increased pulmonary resistance. Some, but not all, studies have noted further accentuation with a move to Trendelenburg position. The effects of insufflation appear to reverse rapidly upon completion of the procedure with few reported long-term consequences.

The intraoperative period: spinal and epidural anesthesia

During spinal and epidural anesthesia with local anesthetics, patients breath spontaneously and often receive supplementary sedation and analgesia. Spinal and epidural anesthesia by themselves have no detrimental effects on ventilation, but supplemental sedatives and analgesics such as intravenous midazolam may depress resting minute ventilation. Spinal anesthesia to a T10-T11 level with bupivacaine or lidocaine did not change tidal volume, respiratory rate or minute ventilation, while end-tidal PCO_2 decreased. Lidocaine spinal anesthesia to a higher (T3-T8) level increased tidal volume, minute ventilation and mean inspiratory flow rate, but decreased respiratory rate. Lumbar epidural anesthesia (10 ml of 2% lidocaine) did not alter resting ventilation but increased the response to hypercapnia [15], while thoracic epidural anesthesia increased minute ventilation by 13% without changing the response to CO_2 [16]. Some of these effects could be due to serum concentrations of local anesthetics. The intravenous infusion of lidocaine, mepivacaine and bupivacaine in human volunteers did not affect resting minute ventilation nor the response to hypercapnia, but slightly increased the response to hypoxemia [17]. The administration of a local anesthetic into the thoracic epidural space has little effect on FRC despite an expected reduction in end-expiratory intrathoracic volume due to rib cage muscle paralysis. This lack of an affect on FRC may be due to the reduced intrathoracic volume being counterbalanced by decreased thoracic blood volume [18]. The latter is caused by reduced venous return secondary to local anesthetic-induced peripheral vasodilation.

The postoperative period

There is a significant incidence of hypoxemia during transport from the operating to the recovery room and

VC = vital capacity

FEV_1 = forced expiratory volume in 1 sec

V/Q = ventilation perfusion

during recovery room stay. The incidence and severity is surgery site related, being greater for thoracoabdominal than abdominal surgery and least after peripheral surgery. The causes of hypoxmia during this period include atelectasis, residual neuromuscular blockade and residual general anesthesia. The latter may cause alveolar hypoventilation, reduced ability to detect inspiratory resistive loads, and depressed ventilatory responses to CO_2 and O_2. Prospectively collected data on 24,157 recovery room patients after general anesthesia revealed an incidence of critical respiratory events – unanticipated hypoxemia (SaO_2 <90%), hypoventilation (respiratory rate <8 beats/min or $PaCO_2$ >50 mmHg) or upper airway obstruction – requiring active intervention in 1.3%. The affected patients had longer stays, higher unanticipated intensive care unit admissions and were more likely to develop cardiac problems [19]. A multicenter study of 198,103 patients found a similar rate (1.9%) of adverse respiratory events [20]. A prospective study using continuous SaO_2 monitoring found a 17.8% incidence of hypoxemia (SaO_2 ≤90% for ≥1 min) and a 7.8% incidence of severe hypoxia (SaO_2 ≤85% for ≥1 min) [21]. Aggressive treatment of the hypoxemia reduced the incidences to 11.6% and 3.3%, respectively.

During the first few postoperative hours, there is little or no further change in the static pulmonary pressure-volume relationship or dynamic compliance [22]. Following superficial or extremity surgery, most of the changes in lung volume revert to preoperative levels within 24 hours [22]. In contrast, after upper abdominal surgery, respiratory abnormalities persist past the immediate postoperative period and are usually characterized by arterial hypoxemia without hypercarbia. FRC decreases after surgery, reaching its nadir after 16–24 hours and is associated with a further reduction in compliance, possibly due to collapse of air spaces [22]. After major upper abdominal surgery, FRC can decrease to 60–70% of pre-operative levels and may return to only 70% of normal by postoperative days 7 to 10 [22]. Immediately following upper abdominal surgery, VC drops to about 40% of pre-operative values and remains depressed for 10–14 days [28]. Maximal inspiratory and expiratory efforts are reduced after upper abdominal surgery and are associated with ineffective coughing. Lower abdominal surgery causes less reduction in FRC and VC. Factors other than operative site that influence the reduction in pulmonary volumes include incision type, age, duration of surgery, pain, and abdominal distension. Laparoscopic cholecystectomy also caused a restrictive impairment, but less depression of FVC, PImax, PEmax and PaO_2, than did open laparotomy.

A major contributor to the decreased lung volume after upper abdominal surgery is a breathing pattern characterized by decreased tidal volume, increased respiratory rate, and absence of sighing [23]. This pattern persisted for as long as 10 days and was unaffected by the epidural administration of the narcotic fentanyl [23], indicating that pain is not a major cause of these changes. Additionally, the ratio of abdominal to rib cage motion is reduced [23]. This decreased abdominal motion was ascribed to reduced diaphragmatic excursions. Fluroscopy on the day following surgery showed up to a 60% decrease in diaphragmatic excursions. The diaphragmatic dysfunction is not caused by altered contractile properties, since no loss of diaphragmatic strength was observed during direct phrenic stimulation [24]. Instead, it is thought to be due to reflex activation of peritoneal, diaphragmatic and esophageal receptors [24] that provoke activation of a central neural mechanism, resulting in inhibition of phrenic nerve output. Epidural blockade to T4 with the local anesthetic bupivacaine increases diaphragmatic activity and restores some abdominal motion probably by reducing afferent input and interrupting the inhibitory reflex [25]. This reflex can also be partially overcome by asking a patient to voluntarily switch from predominantly thoracic to abdominal motion.

Animal studies have demonstrated that isolated gallbladder bed stimulation without cholecystectomy increased the rib cage:abdominal motion ratio to the same degree as gallbladder removal. Traction or compression of the gallbladder for 30 seconds in anesthetized spontaneously breathing dogs decreased tidal volume, diaphragmatic electromyography output and transdiaphragmatic pressures [26]. EMG electrodes and sonomicrometry crystals placed on the diaphragm during abdominal surgery demonstrated impaired diaphragmatic shortening and reduced EMG activity following recovery from anesthesia. Laparoscopic surgery provides an opportunity to examine what occurs in humans when the right upper quadrant is stimulated without a large skin incision [27]. Diaphragmatic function and breathing pattern were significantly impaired after laparoscopic cholecystectomy, but not after laparoscopic hernia repair, presumably because of the lack of right upper quadrant stimulation. These observations help explain why on the first day after both laparoscopic cholecystectomy and open laparotomy similar degrees of dependent atelectasis were seen on CT scans. It appears that stimulation of the upper abdomen even without surgery alters respiratory patterns.

The reduced abdominal movement following surgery may be due not only to decreased diaphragmatic excursions, but also to increased activity of the abdominal external oblique and lower intercostal muscles during expiration, followed by an abrupt decrease in tone with the onset of inspiration [28]. These findings were correlated with simultaneous recordings of intragastric pressure, and it was proposed that decreased abdominal motion is not caused by diaphragmatic dysfunction but by loss of abdominal tone on inspiration, making the mechanical

Plmax = maximal inspiratory effort
Pemax = maximal expiratory effort

EMG = electromyography

coupling of diaphragmatic contraction to lower rib cage movement less efficient. Others have proposed that pain-induced tonic contraction of the abdominal musculature may cause cephalad diaphragmatic displacement, compressing the lower lobes and further reducing lung volume. Not all investigators concluded that abdominal muscle activity contributes to the postoperative respiratory patterns. Dureuil et al. [29] failed to observe abdominal muscle activity on the day after upper abdominal surgery. Raimbult et al. [30] found increased abdominal tone 2, but not 4 hours following surgery. This increased tone was ascribed to the residual effects of fentanyl that was no longer present 4 hours postoperatively. They attributed the reduced abdominal compartmental motion to diaphragmatic, not abdominal muscle dysfunction.

Diaphragmatic dysfunction, small tidal volumes and reduced coughing due to pain are the probable cause of atelectasis formation in dependent areas of the lung. This may explain why VC and FRC continue to fall during the first 24 hours after upper abdominal surgery. Generalized fatigue contributes to pulmonary dysfunction by impairing the ability to cough and breathe deeply and by reducing muscular and respiratory efficiency as noted during graded exercise. Surgical division of the abdominal muscles and resultant pain may impair proper muscle function, reducing abdominal motion and contributing to reduced lung volumes.

Studies indicate that FRC may fall below the closing capacity, a measure of small airway closure. The closing capacity is felt to increase after surgery because of decreased FRC, abdominal distension, surgical dressings and interstitial edema. The supine position further reduces FRC and increases closing capacity. If closing capacity increases after surgery, it may be one of the mechanisms of atelectasis formation, i.e., tidal breathing is unable to open closed small airways. There are conflicting opinions as to the relationship between the degree of airway closure and the degree of hypoxemia. In general, postoperative hypoxemia parallels changes in FRC.

Postoperative respiratory complications

Based on many studies performed over a 40 year period the reported incidence of postoperative pulmonary complications varies from 6% to 76%. When reviewing reports of postoperative complications it is important to note the type of patient population, assess the definitions and objective diagnostic evidence used and examine the study end point. For example, Williams-Russo and colleagues [31] reviewed a study of abdominal surgery patients among whom 15% had major and 14% had minor abnormalities on chest X-ray, while 2% had clinical signs without radiographic abnormalities. They questioned whether the complication rate was 15%, 29% or 31% (i.e., does atelectasis on chest X-ray in an afebrile asymptomatic patient who does not require any additional intervention and whose hospital stay is not prolonged, constitute a "complication"). Studies should be evaluated as to whether they assess pathophysiological abnormalities (e.g., hypoxemia, chest X-ray findings of atelectasis) or actual clinical problems (e.g., pneumonia). Reported complication rates vary with the study design. Higher incidences are reported when complications are specifically sought by investigators than when only those identified by the clinical staff are included. Higher incidences emerge from high risk populations (e.g., age and chronic obstructive pulmonary disease).

Atelectasis seen on chest X-ray is frequently used as evidence of a pulmonary complication. Yet, compared to CT scans, portable chest X-rays have poor sensitivity but good specificity for diagnosing lung consolidation. Fever, often used as a criterion for postoperative pulmonary complications, occurs for a variety of reasons and it is unclear whether atelectasis is one of them. In 92% of postoperative fevers no infection or pathological process was found [32]. Therefore, fever alone is not used as evidence of a pulmonary complication. The definition of pneumonia varies; most define it as a new infiltrate on chest X-ray, but some also include indirect evidence such as the presence of one, some or all of the following: fever, discolored sputum, elevated white blood cell count, and administration of antibiotics.

Postoperative pulmonary complications occur with some frequency. Postoperative pneumonia is associated with considerable morbidity and mortality (30–46%). A Danish prospective study of over 7,000 abdominal surgical, urological, gynecological and orthopedic operations found that 4.1% suffered postoperative complications (respiratory failure requiring >24 hours of mechanical ventilation, X-ray proven atelectasis or pneumonia, isolation of a pathogen from the lung, or respiratory insufficiency requiring therapeutic intervention) [33]. The incidence of pulmonary complications after upper abdominal surgery with general anesthesia was 18% and lower following major orthopedic (6.8%) and lower abdominal (8.5%) procedures. Atelectasis and pneumonia were more frequent following general (4.5%) than regional (1.9%) anesthesia. Emergency surgery and operating time of more than 180 minutes were associated with a greater incidence of complications. Others found that pulmonary complications requiring therapeutic interventions ranged from 1–3% after elective to 10% after emergency surgery [34]. Respiratory insufficiency (PaO_2 <60 mmHg, tracheostomy or endotracheal intubation) was found in 3% of 1,332 adults after abdominal surgery [35]. A retrospective review of 181 consecutive elective abdominal aortic aneurysm repairs showed major complications (pneumonia, re-intubation for pulmonary insufficiency and postoperative intubation for >24 hours solely for respiratory problems) in 26 (16%), including 17 (9%) who developed pneumonia [36]. Age greater than 70 years, body weight >150% of ideal, American Society of Anesthesiologists class IV, pre-operative FVC ≤80% of

predicted, forced expiratory flow rate ≥6 0% or less of predicted, crystalloid replacement of >6 L and operative time >5 hours were associated with increased risk for pulmonary complications [36]. Other authors have found that impaired pre-operative cognitive function, smoking within 8 weeks of surgery, pre-operative sputum production, postoperative nasogastric intubation, history of cancer, increased body mass index, and upper abdominal incision are risk factors for postoperative complications. Stremple and coworkers [37] used discharge diagnostic codes from two large (>4 million patients/year) databases and found a 9% complication rate after major abdominal surgery (partial gastrectomy and other gastroenterostomies, n= 3,399) in males >18 years. The rate was 6% for colonic surgery (n=16,954), but only 2% for total knee replacements (n=9,249).

Each of the above studies have limitations, especially the latter, which relies on the diagnostic coding performed by medical records personnel following discharge. Yet studies published during the 1990s, as well as older reports, demonstrate that postoperative respiratory problems are common and occur at rates equal to or higher than cardiac and other complications. The incidence of pulmonary complications is lower following laparoscopic abdominal surgery, most likely because of better preservation of ventilatory function. In a series of 75 laparoscopic adrenalectomies only one case of atelectasis was reported.

Postoperative management

Mechanical maneuvers

Following major surgery, maneuvers designed to induce a maximal inspiratory effort, mobilize secretions and increase FRC are used to prevent and treat atelectasis. These include early mobilization, chest physical therapy, deep breathing exercises, coached coughing, intermittent positive pressure breathing and incentive spirometry. Often, at least one modality is used after abdominal surgery [1]. Abdominal surgery patients receiving either intermittent positive pressure breathing, incentive spirometry or deep breathing exercises had significantly lower complication rates (21–27%) than control patients (48–60%). The treatments were equally effective [1], while deep breathing exercises and incentive spirometry were equally cost effective. It has been recommended that the former be used in low risk patients and the latter in high risk ones (ASA class >1 and age ≥60 years). Despite the effectiveness of these therapies, early mobilization is still the most cost-effective therapy. The superiority of physical therapies, such as percussion, positive expiratory pressure and inspiratory resistance, has not been demonstrated. Intermittent positive pressure breathing is costly, offers no advantage over other therapies and may be detrimental because it causes gastric distension. Most interesting was that even under study conditions these therapies were unable to reduce the complication rate to below 15–20% after abdominal surgery (except one study with a reported 6% complication rate). This may be due to the inability of intermittent maximal inspiratory efforts to completely overcome the atelectasis-forming effects of a resting respiratory pattern of small tidal volumes, narcotic-induced respiratory depression especially during sleep, and pain-limited coughing and deep breathing. Additionally, the effectiveness of incentive spirometry may be compromised because it increases mainly chest wall and not abdominal motion, thereby failing to effectively expand the dependent portions of the lungs where atelectasis develops most often [38].

Pain management

Patients after abdominal surgery have difficulty coughing, moving about and taking deep breaths because of pain. Many modalities provide effective pain relief after surgery, including parenteral, intrathecal and epidural narcotics and regional nerve blocks. Analgesic therapy is ostensibly used to make patients more willing to move about and participate in treatments designed to prevent and reduce atelectasis, such as incentive spirometry. Analgesia has long been considered part of the defense against the development of atelectasis and pneumonia. Yet evidence is sparse that current therapies actually reduce pulmonary complications, since most studies involve small numbers of patients and focus on analgesia scores and immediate side effects. Respiratory measurements are usually limited to traditional pulmonary function tests and blood gas analysis. Only a few investigations have examined respiratory mechanics, muscle function and breathing patterns. The impression based on available evidence is that opioid analgesia, even when administered in novel ways, has only a limited ability to reduce or attenuate postoperative alterations in pulmonary function and may even contribute to the pulmonary complication rate by causing respiratory depression. Also lacking is information whether analgesia can improve coughing effectiveness and facilitate the treatment and prophylaxis of atelectasis and pneumonia with incentive spirometry and deep breathing exercises. There is limited knowledge as to whether the newer approaches to pain management, especially epidural analgesia, are truly cost effective and improve outcome. The cost-benefit (reduced length of hospital stay, earlier recovery of gastrointestinal function) of superior pain relief has been demonstrated in colonic surgery, but more extensive study is needed with other types of major abdominal surgery. A meta-analysis of 48 small-randomized trials examined these issues and concluded that compared to systemic opioids, epidural opioids decreased the incidence of atelectasis and had a weak tendency to decrease pulmonary infections and overall pulmonary complications. Epidural local anesthetics when compared to systemic opioids decrease pulmonary in-

ASA = American Society of Anesthesiologists

fections and overall pulmonary complications [39].

The most commonly used analgesics, narcotics, are themselves respiratory depressants [Table 1]. The route of administration influences the effects of narcotics. Lumbar epidural morphine (3.5 and 7.0 mg), but not equal doses injected subcutaneously, increased end tidal PCO_2 and decreased the response to CO_2 – the latter persisting for as long as 24 hours. Intrathecal morphine (0.2, 0.4 and 0.6 mg) produced hypoxemic episodes and dose-related increases in $PaCO_2$ that were most pronounced after 6.5–7.5 hours and 17–20 hours. The respiratory changes are due to depression of central respiratory integrating centers and chemoreceptors as well as to depression of peripheral chemoreceptors.

Continuous postoperative monitoring of respiratory rate and O_2 saturation has demonstrated untoward effects of narcotic analgesia. Following cholecystectomy, total hip replacement and upper abdominal surgery, continuous intravenous morphine infusions caused more episodes of desaturation (SaO_2 <80%), more tachyarrhythmias, and more ventilatory disturbances such as central and obstructive apnea, paradoxical breathing, slow respiratory rates and small tidal volumes, than epidural analgesia with bupivacaine, a local anesthetic. Obstructive apnea and desaturations occurred during sleep and continued for 5–6 nights following surgery. Respiratory disturbances occurred more frequently in older (66–75 years) patients. Postoperative sleep disturbances with suppression of rapid eye movement sleep are exacerbated by opioids and may contribute to episodic hypoxemia. The number and duration of pre-operative overnight desaturations failed to predict postoperative respiratory problems nor did pre-operative snoring. The contribution of apneic episodes to the postoperative decreases in lung volumes and atelectasis needs clarification. Gill et al. [40] observed a close relationship between the duration of hypoxemic episodes and myocardial ischemia. The effect of these hypoxic episodes on cerebral function, especially in the elderly, is of concern.

Table 1. Ventilatory effects of intravenous morphine

- Decreased minute ventilation
- Slower respiratory rate
- Reduced sighing
- Depressed ventilatory response to hypoxia
- Reduced ventilatory response to hypercapnia, accentuated by sleep
- Decreased rib cage, but not abdominal, motion

The ventilatory effects of narcotics led to the development of agonist/antagonists such as nalbuphine and pentazocine. Unlike pure agonists, these drugs do not depress ventilation in a dose-related fashion, but produce maximal respiratory depression at low clinical doses ("ceiling effect"). However, these drugs are not used much since they offer no advantages over pure agonists. There has also been interest in analgesics that have a lower propensity for respiratory depression, e.g., the non-steroidal anti-inflammatory drugs such as ketorolac.

Conclusions

The peri-operative period is marked by many changes in the structure and function of the respiratory system, some of which contribute to postoperative complications. This article reviews these issues, but leaves many observations unexplained and many questions unanswered. Most lacking are standardized definitions for clinically significant postoperative pulmonary complications which are needed to facilitate inter-study comparisons. Since pulmonary function is altered both during surgery and the immediate postoperative period, the interrelationship of these two periods needs to be better defined, e.g., are there associations between the degree of intraoperative atelectasis and the severity of recovery room complications? In addition, the effects of analgesic regimens on pulmonary function need further study.

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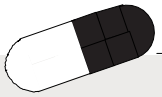
I think this is the most extraordinary collection of talent, of human knowledge, that has ever been gathered together at the White House – with the possible exception of when Thomas Jefferson dined alone.

John F. Kennedy, 35th American President (1917–63), addressing Nobel prize winners in 1962

Capsule



Walking and golfing

Parkkari et al. studied the effects of regular walking during a golf game on various health and fitness indicators in middle-aged men.

The study subjects were 55 healthy male golfers aged 48 to 64 years who had been sedentary during the 7 months before the study, and 55 age-matched, similarly sedentary controls. During the 20 week study, those in the intervention group were encouraged to play golf two to three times a week; the controls were not. Measurements of body composition, cardiorespiratory performance, motor and musculoskeletal fitness, blood pressure, and serum lipid, glucose, and insulin levels were obtained at baseline and after the 20 week study. The results showed reductions in weight of 1.4 kg, in waist circumference of 2.2 cm, and in abdominal skinfold thickness of 2.2 cm. Golfers also had significantly greater increases in serum high density lipoprotein (HDL) cholesterol levels and in the ratio of HDL cholesterol to total cholesterol. The authors conclude that regular walking has many positive effects on the health and fitness of sedentary middle-aged men. Walking during a golf game is characterized by high adherence and low risk of injury and is therefore a good form of health-enhancing physical activity.

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