Prevention of respiratory complications after abdominal surgery

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Introductory article

Prevention of respiratory complications after abdominal surgery: a randomised clinical trial

JC Hall, RA Tarala, J Tapper, JL Hall

Objective. To evaluate the prevention of respiratory complications after abdominal surgery by a comparison of a global policy of incentive spirometry with a regimen consisting of deep breathing exercises for low risk patients and incentive spirometry plus physiotherapy for high risk patients. Design. Stratified randomised trial. Setting. General surgical service of an urban teaching hospital. Patients. 456 patients undergoing abdominal surgery. Patients less than 60 years of age with an American Society of Anesthesia classification of 1 were considered to be at low risk. Outcome measures. Respiratory complications were defined as clinical features consistent with collapse or consolidation, a temperature above 38°C, plus either confirmatory chest radiology or positive results on sputum microbiology. We also recorded the time that staff devoted to prophylactic respiratory therapy. Results. There was good baseline equivalence between the groups. The incidence of respiratory complications was 15% (35/231) for patients in the incentive spirometry group and 12% (28/225) for patients in the mixed therapy group (P = 0.40; confidence interval -3.6% to 9.0%). It required similar amounts of staff time to provide incentive spirometry and deep breathing exercises for low risk patients. The inclusion of physiotherapy for high risk patients, however, resulted in the utilisation of an extra 30 minutes of staff time per patient. Conclusions. When the use of resources is taken into account, the most efficient regimen of prophylaxis against respiratory complications after abdominal surgery is deep breathing exercises for low risk patients and incentive spirometry for high risk patients. (BMJ 1996;312:148-53)

Postoperative respiratory morbidity continues to be a major factor in the utilisation of resources and maintenance of hospitalisation after major surgery. The introductory article draws attention to this but, as the aetiology, prevention and management are multifactorial, the approach of this review to this subject has been broadened.

The incidence of pulmonary complications is higher after upper abdominal or chest surgery than operations on other parts of the body. These wounds produce a severe and prolonged alteration in pulmonary mechanics. Impaired ventilation and ineffective expectoration result in a postoperative failure of expansion or progression of collapse of lung segments, thereby encouraging infection. The ensuing shunt with venous admixture results in hypoaxaemia. Postoperative oxygen supply may therefore falter while oxygen demands are increased due to metabolic hypermetabolism and hypercatabolism of the neuroendocrine stress response to trauma. At the same time the work of breathing is increased due to the need for increased alveolar ventilation (because of shunt induced carbon dioxide retention), a stiffened abdominal wall and, possibly, diaphragmatic dysfunction. These pathophysiological changes underpin the events in the immediate postoperative period and morbidity and mortality depend upon their severity. The main factor behind all these events, and the one which is most amenable to modulation, is severe postoperative pain.

This discussion paper will review the effects of an abdominal incision, its analgesic management, and postoperative physiotherapy on the generation of postoperative respiratory complications.

Effects of anaesthesia and an abdominal incision on pulmonary physiology

Some great minds have pondered the problem of postoperative complications. Pasteur, Haldane and Beecher were all convinced of the importance of active collapse of the lung after abdominal operations with shallow breathing as the major cause of postoperative hypoxia and pulmonary complications.
Intraoperative and postoperative changes in lung volumes

Major alterations occur in respiratory volumes in all patients following abdominal surgery, involving a decrease in functional residual capacity (FRC) but with minimal change in the closing volume (CV). When CV exceeds FRC, atelectasis in the dependent lung regions becomes inevitable. This change is most exaggerated in the elderly, the obese, in smokers, and in those with pre-existing cardiopulmonary disease. General anaesthesia, irrespective of the anaesthetic agents used, causes a reduction in FRC of approximately 18% (the only possible exception being ketamine). Body posture affects lung volumes, with a change from supine to sitting increasing CV only slightly but increasing FRC significantly. Thus, in the immediate postoperative period the sitting position is preferred and early mobilisation is to be actively encouraged. Sufficiently effective analgesia must be established and maintained so that these activities are not impeded by pain.

Altered ventilatory pattern

Alterations in ventilatory mechanics occur both during surgery and for a long period of time afterwards. The characteristic postoperative mechanical abnormality in respiration is a restrictive pattern of ventilation with a significant reduction in vital capacity (VC), tidal volume (Vt), forced expiratory volume in one second (FEV), and FRC, and the principal cause of these abnormalities is pain. No other factor has greater importance.

To compensate for the reduced efficiency of breathing, carbon dioxide retention, and reduced Vt there is an increase in respiratory rate. Minute ventilation is maintained or increased at the expense of an increase in the work of breathing and therefore oxygen demand. The postoperative use of sedatives and opiates impairs the normal sigh mechanism which is responsible for maintaining small airways patency and FRC. Spontaneous deep breaths which help to restore FRC are abolished by a combination of pain and narcotic analgesics.

Alterations in gas exchange

Gas exchange is impaired intraoperatively due to a ventilation/perfusion mismatch which persists long into the postoperative period. It is the accepted view in the literature that this phenomenon of postoperative hypoxaemia in the absence of hypoventilation is inevitable. Physical therapy is of importance in that removal of secretions and re-expansion of collapsed basal lung segments will restore gas exchange.

Postoperative hypoxaemia resulting from the respiratory abnormalities discussed above is often compounded by systemic opiate use such as myocardial infarction and insulin lung, pulmonary complications, cerebrovascular accidents, thromboembolism, delayed wound healing, and prolonged convalescence with fatigue and inability to work (table 1).

Impairment of pulmonary defence mechanisms

Mucociliary clearance is adversely affected by anaesthesia and dry gas ventilation. Systemic opiates and sedatives suppress the cough reflex and inhibition of its expulsive force by pain renders it less effective. Gastric and oral aspiration are encouraged by the use of these same drugs. Sputum retention with bacterial overgrowth of the airways is therefore encouraged, while at the same time the stress response to surgery impairs the immune system, suppressing natural killer cell activity. Infectious complications have been reported in up to 50% of patients undergoing upper abdominal or thoracic operations, the incidence paralleling that of atelectasis.

Table 1 Some of the effects of hypoxaemia (compounded by the neuroendocrine stress response to trauma) in the postoperative period

<table>
<thead>
<tr>
<th>System</th>
<th>Stress-related complicating mechanisms</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Increase in cardiac work (1CO demands but (SVR), hypercoagulability</td>
<td>Myocardial insufficiency and infarction, thromboembolic phenomena</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Immune suppression, hypercoagulability</td>
<td>Infection, embolic phenomena</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>Multifactorial, hypercoagulability</td>
<td>Delirium, cerebral thromboembolism</td>
</tr>
<tr>
<td>Renal</td>
<td>Vasoconstriction</td>
<td>Oliguria, acute renal failure</td>
</tr>
<tr>
<td>Wound</td>
<td>Immune suppression, generalised vasoconstriction</td>
<td>Poor healing, dehiscence</td>
</tr>
<tr>
<td>Generalised</td>
<td>Hypertension, hypercoagulability</td>
<td>Chronic fatigue, prolonged convalescence</td>
</tr>
</tbody>
</table>

CO = cardiac output; SVR = systemic vascular resistance.
PAIN MANAGEMENT

As the respiratory abnormalities discussed above set the stage for postoperative respiratory complications, it is vitally important to attempt their alleviation, the outcome of the patient depending heavily on these factors. Inadequate analgesia prevents early ambulation and deep breathing and prolongs hospital stays. As pain is not only the patient's principal complaint, but is also the cause of many of these changes, one must be careful to ensure that it is not the main causative factor open to modulation. It is critically important that the analgesic method is chosen which can best improve pain and pulmonary function. Effective analgesia will improve and even reverse the effects of surgery on the pulmonary mechanics and prevent pulmonary complications.1 3 4 6 9

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Table 2: Generation and “balanced” prevention and management of postoperative pain

<table>
<thead>
<tr>
<th>Event</th>
<th>Effect</th>
<th>Modulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanical, thermal (diathermy) and chemical stimulation of nociceptors</td>
<td>Peripheral sensitisation</td>
<td>NSAID premedication and maintenance</td>
</tr>
<tr>
<td>Transformation of nociceptor information to CNS</td>
<td>C, A, sympathetic fibre activation</td>
<td>Opiates only useful for C-fibre afferent input, local anaesthesia required for others (regional anaesthesia)</td>
</tr>
<tr>
<td>Opiate sensitisation</td>
<td>Central sensitisation</td>
<td>Opiates and regional anaesthesia before surgical stimulus</td>
</tr>
</tbody>
</table>

NSAID = non-steroidal anti-inflammatory drug.

shown to be prevented, and the subsequent development of chronic pain has been prevented.26

Intercostal nerve blocks are logical as most postoperative pain arises from the intercostal spaces and not the abdominal wall itself. The procedure is straightforward, the skills are easily acquired and taught and useful analgesia has been demonstrated in many studies, pulmonary function has been improved, and pulmonary complications have been reduced.28 29 Reduced postoperative pulmonary complications have been found in some studies30 31 while others dispute this.32 33 There are, however, an array of drawbacks. The site of the epidural catheter is contraindicated in patients who are anticoagulated or have a bleeding tendency. (Injection of non-steroidal anti-inflammatory drugs is controversial.34) Technical failures form a significant proportion of patients in most studies.35- 37 Hypotension due to high bilateral sympathetic blockade may not be a problem intraoperatively as the event is anticipated, but postoperatively drops in blood pressure (which can be unpredictable) have been reported with thoracic catheterisation in greater than 60% of cases38 and for this reason patient mobilisation can be severely restricted. Urinary retention is also to be expected,39 with or without a degree of motor weakness of the lower extremities, which further limits postoperative mobilisation. Neuropathological complications are fortunately rare,40 as are infections in the epidural space, even though all the natural barriers are breached. Non-steroidal anti-inflammatory drugs are useful adjuncts in the treatment of moderate or severe pain, they are morphine sparing and have useful properties as far as a reduction in inflammation and stress inhibition are concerned.41- 43

The generation and prevention and management of postoperative pain is summarised in table 2.

Minimally invasive surgery

The surgeon’s response to all these sequelae is to minimise the atteri on input to the CNS through the use of laparoscopic surgery in preference to laparotomy. This has led to a revolution in surgical practice. However, clinical outcome data supporting this approach are poor. Most prospective studies have not been randomised and results are simply audits of results. Two prospective randomised studies have been per-
formed in patients undergoing appendicectomy which showed no difference. A further study has shown a reduction in the length of hospital stay, but with no difference in complications. A physician controlled discharge times are a poor indicator of superiority of one treatment over another.

There are insufficient data to assess open versus laparoscopic pyloromyotomy as only one study has been performed of which the results were almost equivocal. Four prospective randomised studies of inguinal hernia repair have been undertaken. One showed less pain and reduced postoperative complications in the laparoscopic group, but the recurrence rate will not be known for a number of years. Two others concluded that a laparoscopic approach was as good as an open approach although the operative costs were higher. The third was difficult to interpret as the open group had local anaesthesia compared with general anaesthesia for the laparoscopic group, although the results were strongly and significantly in favour of the open approach.

Laparoscopic cholecystectomy seems to be the indication for this approach which has been most studied. In one randomised study of open cholecystectomy versus endoscopic sphincterotomy no differences were found in mortality and morbidity, but there was a higher recurrence of symptoms with the endoscopic approach, leading to the conclusion that open surgery was preferable. In a prospective randomised comparison of laparoscopic versus small incision cholecystectomy involving 200 patients Majeed et al found that the laparoscopic approach took longer to do and had no significant advantages in terms of hospital stay or postoperative recovery. MCM ahon found that laparoscopic cholecystectomy led to shorter hospital stays and a quicker return to normal activities than an open approach, but there was no difference in the incidence of complications and operative costs were higher. Pulmonary function in terms of spirometric values and oxygenation has been shown to be better in laparoscopic than in laparotomy cholecystectomy in a number of studies. Very little attempt was made in these studies to utilise regional analgesia which, in our view, is difficult to justify when all the inadequacies of systemic opiates are taken into account. Rademaker et al did use epidural analgesia in a comparison of pulmonary function and stress responses in laparoscopic versus subcostal incision cholecystectomy. Oddly perhaps, epidural analgesia was used in one of the laparoscopic groups rather than the subcostal incision group. The laparoscopic groups did better in terms of pulmonary function even though the endocrine stress response was similar.

Local anaesthetics with laparoscopic cholecystectomy have been used intraoperatively with two opposing views of their efficacy. It seems that, with fairly substantial doses given immediately after induction of the pneumoperitoneum and repeated at the end of surgery, pain can be reduced. A fully comprehensive review of studies undertaken with laparoscopic cholecystectomies has been made by Downs et al. Laparoscopic-assisted ileal pouch anal anastomosis compared with standard laparotomy failed to reduce the duration of postoperative ileus or length of hospital stay. We are forced by these data to question the philosophy of the seemingly headlong rush into minimally invasive surgery.

**Modification of perioperative risk factors**

Studies on the effectiveness of modification of risk factors on outcomes are difficult to interpret due to the defining criteria used. The incidence of pulmonary complications varies enormously after abdominal and chest surgery. Important diagnostic criteria should include sputum changes, abnormalities in auscultation, radiological changes, fever, leucocytosis, and hypoxaemia. Smoking has the effect on CV of adding 10 years to one's age so that the functional consequences of airways closure during tidal breathing will occur earlier and to a greater extent in smokers than in non-smokers. Chronic obstructive pulmonary disease is associated with copious production of viscid sputum which leads to obstruction of airways with distal collapse and exacerbated shunting. Cessation of smoking improves lung function by approximately one month and improvement continues for up to 18 months. Even a few days abstinence will improve mucociliary transport.

Obesity causes a restrictive defect in pulmonary function because of a reduction in chest wall compliance, all lung volumes, including FRC, being affected. If surgery is elective then obese patients should be encouraged to lose weight. Muscular strength should be maintained as far as possible in the malnourished or hypercatabolic patient through adequate nutrition.

**Predictors of pulmonary complications**

A number of studies have tried to identify predictors of pulmonary complications. In a study of 278 patients pre-existing respiratory morbidity and poor exercise tolerance were found to be predictors of mortality whereas pulmonary function test results in this respect were unhelpful. However, in a study of patients undergoing thoracotomy for oesophagectomy VC was found to correlate positively with the risk of complications. Smokers with a history of respiratory illness have a significantly longer hospital stay. Studies on the effect of smoking on postoperative respiratory complications have been mixed. In a prospective randomised study of smoking versus non-smoking patients undergoing cholecystectomy, smoking did not affect surgical outcome but poor smoking history was associated with a higher incidence of postoperative complications. Smoking has the effect of reducing the patient's ability to utilise residual volume, increasing the chance of residual air.

**Physical therapy**

Physical therapy has a valuable role to play in the prevention of complications as well as their treatment, although the type of therapy which should be used is not entirely clear. Various methods of physical therapy have been shown to improve measured pulmonary function – for example, VC and FRC – and a meta-analysis showed a significantly beneficial effect on the prevention of complications. However, single treatment modalities – for example, incentive spirometry, coughing and breathing exercises and intermittent positive pressure breathing – have yet to have their individual roles defined.

In their study of 456 patients undergoing abdominal surgery Hall et al found a small and non-significant reduction in complications from 15% for high and low risk patients given incentive spirometry versus 12% in low risk patients given breathing exercises and incentive spirometry along with conventional physiotherapy in high risk patients. This small improvement was thought to be worth the investment in terms of utilisation of manpower resources, although the addition of conventional physiotherapy added significantly to staff time. In conclusion, major alterations in pulmonary mechanics and ventilation/perfusion relationships result from anaesthesia and abdominal surgery and the principal inhibitor of chest cage motion is severe postoperative pain. Atelectasis, hypoxaemia, infection, and respiratory...
distress occur in some normal and many high risk patients. The development of postoperative pulmonary complications ought to be favourably influenced by effective regional anesthetics. Results in thoracic surgery are unequivocal, although the situation in abdominal surgery is less clear cut. The use of minimally invasive surgery has not been conclusively shown to confer any benefit in terms of improved pulmonary function and reduced complications compared with standard open surgery when systemic opiates are the mainstay of analgesic management. The use of physical therapy has a valuable role to play in the prevention of complications as well as their treatment, although the relative values and indications for different therapies have still to be conclusively determined.

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