Regurgitation and vomiting with subsequent inhalation of gastric contents into the bronchial tree is a not uncommon occurrence and remains an important factor in the morbidity and mortality of anaesthesia. Hall (1940) reported the death of a normal, healthy primigravida which occurred as a result of aspiration of vomit during anaesthesia for a low forceps delivery. Many reports of similar incidents followed. In the U.S.A., Merrill and Hingson (1951) checked 42,439 deliveries by means of questionnaires to the major obstetric units and found that 59 maternal deaths were due to the inhalation of vomit, and they calculated that 2 per cent of all maternal deaths in the U.S.A. were due to this cause. Edwards et al. (1956), in a report on 1,000 deaths associated with anaesthesia, considered that 110 deaths were directly due to regurgitation or vomiting. This represents 18.6 per cent of a total of 589 in whom anaesthesia was thought to be responsible for death. Anaesthesia apart, this is a particular problem in debilitated and comatose patients whatever the cause. Fetterman and Moran (1942) studied the histology of the lungs in 469 consecutive autopsies from medical wards, and they were able to recognize food particles in association with inflammatory changes in the lungs in 27 cases. They concluded that many cases of bronchopneumonia in aged and feeble patients may be attributed to aspiration of food or vomit.

PREVENTION OF ASPIRATION OF VOMITUS

Those conditions in which it can be assumed that regurgitation or vomiting may occur under anaesthesia have been well described by Morton and Wylie (1951). However, it appears that this complication is particularly likely to occur in patients in whom the presence of gastric contents is unsuspected either through lack of the usual indications or by faulty assessment by the anaesthetist.

Emptying of the stomach.

In those patients in whom a “full stomach” is suspected, it has been assumed that the danger of aspiration could be avoided by emptying the stomach prior to the induction of anaesthesia. Edwards et al. (1956) recommend the use of a duodenal tube, catheter size 12, for this purpose. However, this is unreliable even with purely fluid stomach contents and large particles of food or blood clot are not necessarily removed even with the largest-bore stomach tube. The use of apomorphine to induce vomiting with consequent gastric emptying has been recommended by Holmes (1956) and White (1959). White, however, states that this method does not always produce active vomiting and, even when it does, it does not of necessity guarantee complete emptying. We believe that this practice should be avoided, since, quite apart from aesthetic objections to the use of apomorphine, cardiovascular collapse can be produced in ill and toxic patients and the possibility of active inhalation of vomitus by a conscious but debilitated patient cannot be disregarded.

Induction of anaesthesia.

In the anaesthetized patient, there can be no doubt that the use of a cuffed endotracheal tube affords the best protection against aspiration of re-
gurgitated gastric contents, and consideration should be given, therefore, to the possible ways of preventing such aspiration during the period of anaesthetic induction prior to the introduction of such a tube, and to the management of the extubation at the end of the operative procedure.

Induction of anaesthesia in a steep head-up or sitting position, using thiopentone-relaxant technique with rapid intubation has been recommended. (Hodges et al. (1959) used suxamethonium; Snow and Nunn (1959) used gallamine.) If regurgitation takes place in the paralyzed patient in the head-up position, aspiration is almost unavoidable. Two cases have been seen in which regurgitation has occurred when using this technique; in one there was a fatal outcome. Another disadvantage of this technique is that the combination of thiopentone and relaxant with a head-up tilt may result in cardiovascular collapse, particularly in ill patients. This is a potentially dangerous method and should not be used by the unskilled junior anaesthetist. A thiopentone-relaxant technique with a head-down tilt may be used on the assumption that gravity will prevent aspiration in an apnoeic patient. The danger of cardiovascular collapse is very much reduced in this position. When using thiopentone-relaxant techniques, we have noticed that, if regurgitation occurs, it tends to do so during inflation of the lungs, prior to intubation, or during laryngoscopy. This is believed to be due to incompetence of the cricopharyngeal sphincter, produced mechanically during laryngoscopy by stretching the sphincter as the larynx is drawn forward to expose the glottis and, during inflation, when the sphincter is opened by the pressure of the inflated gas. The patient is oxygenated before induction rather than prior to intubation and tracheal cartilage pressure is employed during laryngoscopy and intubation.

Induction using a volatile agent with spontaneous respiration and the patient in the lateral position with a slight head-down tilt may be employed, using combinations of oxygen and nitrous oxide supplemented by ether, cyclopropane, halothane, or vinesthene. With this type of induction, the laryngeal reflexes will be present to some degree until the patient is deep enough for intubation. Hyperventilation produced by carbon dioxide may be used to speed induction and to depress the vomiting mechanism. Regurgitation can still occur, particularly if there be any obstruction to the airway. Intubation and pharyngeal toilette are more difficult in these patients because of the position and the fact that the degree of relaxation is less than that obtained with the use of suxamethonium.

Intubation of the trachea under local anaesthesia prior to induction has also been proposed. It is conceivable that this would increase the likelihood of aspiration following extubation.

In patients who are extremely ill, toxic or shocked, intubation of the trachea before induction and without local anaesthesia may often be performed without undue distress to the patient.

**Management of recovery.**

The incidence of aspiration of stomach contents following extubation may be minimized by ensuring that protective laryngeal reflexes have returned before the endotracheal tube is removed. The patient should be on his side with a competent person in attendance until he is fully conscious.

**THE EFFECTS OF INHALATION OF VOMIT**

The first problem in any case of inhalation is that of acute respiratory obstruction, which, if unrelieved, may result in death of the patient from asphyxia. Reflex cardiac arrest can occur at this stage. Secondly, although adequate ventilation has been re-established, the patient may subsequently develop pulmonary complications. These include Mendelson's syndrome, absorption atelectasis and aspiration pneumonia. Lung abscess, empyema and bronchiectasis may occur as a late result.

**A. Acute respiratory obstruction.**

The inhalation of solid or fluid gastric contents leads to mechanical obstruction of the respiratory passages with resultant anoxia and hypercapnia. In the unparalyzed patient, there is at first moderate hyperpnoea followed by great respiratory effort, particularly in the expiratory phase, and apnoea then supervenes. During the initial stages, which are associated with increased sympathetic activity, there is a rise in blood pressure which later falls. The heart cannot incur an oxygen debt and soon weakens, the ventricles dilate and fibrillate; asystole follows.

The inhalation of even a small amount of gastric content may produce very severe bronchial spasm which results in asphyxia, if it be not relieved. Reflex vagal cardiac arrest can occur from stimulation of the larynx or bronchial tree by aspirated material.
THE EFFECTS OF THE INHALATION OF VOMITUS ON THE LUNGS

B. Mendelson's syndrome.

In 1946, Mendelson described a previously unrecognized pulmonary complication of general anaesthesia.

The actual aspiration often escaped recognition, but was followed by an "asthmatic-like" syndrome with distinct clinical, radiological and pathological features.

There was cyanosis, tachycardia, dyspnoea, and bronchospasm, but no mediastinal shift or massive atelectasis. Wheezes, rales and rhonchi were heard sometimes over the affected portions of the lungs. X-rays revealed irregular, soft, mottled densities. Progressive cardiac embarrassment and pulmonary oedema sometimes supervened.

Mendelson was able to reproduce this syndrome in rabbits using N/10 hydrochloric acid and unneutralized human fluid gastric contents. At autopsy, there was congestion and oedema throughout the lungs, a wavy bronchiolar pattern indicative of spasm, with peribronchiolar haemorrhage and exudate, usually with areas of secondary emphysema. The bronchial mucous membrane had sloughed in places.

When normal saline, distilled water or neutralized fluid gastric contents were injected into the rabbit's lung, a brief phase of obstructive respiration ensued, but breathing quickly returned to normal. The lungs showed only minute scattered areas of atelectasis. It was concluded that the features of Mendelson's syndrome could be attributed to the aspiration of gastric hydrochloric acid.

The cases described by Mendelson (1946) and Parker (1956) were confined to obstetrical anaesthesia, but there have been other cases reported associated with patients anaesthetized for other surgical procedures, and also in patients in coma.

In this department, five cases which had the features of Mendelson's syndrome have been seen. In two cases, the onset of signs occurred almost immediately following aspiration, and in these cases intense bronchospasm was the most prominent feature.

In three other cases, following treatment for aspiration, which was known to have taken place during anaesthesia, the patients appeared to be quite normal at the termination of anaesthesia, and the onset of a Mendelson type of syndrome was delayed for 2-4 hours. Pulmonary oedema was the most prominent feature of these cases.

C. Absorption atelectasis.

Blockage of a bronchus with subsequent absorption of the air contained in the alveoli beyond the obstruction allows that part of the lung to collapse. Lobular collapse is usually prevented by interalveolar gaseous exchange via the pores of Kohn (1893), but when respiration is depressed this is absent (Van Allen, Lindskog and Richter, 1931).

Bronchi may be blocked by the aspirated material itself or by secretions which are produced in response to stimulation of the bronchial mucosa by this material. Lobular collapse is often associated with small areas of compensatory emphysema.

Single or multiple areas of collapse may occur affecting a whole lung, lobe or segment. The site of the collapsed area largely depends on the position of the patient at the time of aspiration. When the patient is supine, the apical segment of the right lower lobe is most commonly affected.

D. Aspiration pneumonia.

Acute infection is likely to occur in the collapsed areas, if the obstruction be not relieved. In patients who have recently undergone general anaesthesia, the cough reflex and ciliary activity may be depressed, and thus the removal of bronchial obstruction is delayed. The pneumonic process is usually mild, but if a virulent organism gains entry, then the condition may go on to suppuration. Aspiration of infected material may lead to a pneumonic process without previous collapse.

TREATMENT

The management of cases of aspirated gastric contents to be described is based upon experience gained from working in a busy casualty department where many patients are admitted in coma due to drug overdosage, trauma and diabetes, together with seven cases in which inhalation was associated with anaesthesia.

The regime of treatment used is set out below. Cases in which inhalation was associated with anaesthesia are tabulated (table I).

Re-establishment of the airway.

Rapid establishment of adequate pulmonary ventilation and oxygenation is essential.

If the patient be on an operating table when regurgitation or vomiting takes place, then the table is tipped head down at least 15 degrees, and the patient is rolled on to his right side to aid the drainage of vomit from the mouth and to try to
### TABLE I

<table>
<thead>
<tr>
<th>Surgical condition</th>
<th>Age</th>
<th>Stomach tube</th>
<th>Method of anaesthesia</th>
<th>Time, type, amount of aspiration</th>
<th>Lavage</th>
<th>Hydrocortisone</th>
<th>Bronchodilator</th>
<th>Tracheostomy</th>
<th>Mendelson's</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestinal Obstruction. Emergency. Carcinoma Colon.</td>
<td>74</td>
<td>Yes</td>
<td>Thiopentone-suxamethonium, Head-up tilt</td>
<td>When intubated, Small, fluid</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
<td>Yes</td>
<td>—</td>
<td>Recovery uneventful.</td>
</tr>
<tr>
<td>Forceps Delivery. Emergency. Previous L.S.C.S.</td>
<td>44</td>
<td>No</td>
<td>O₂-cyclopropane. Flat</td>
<td>Following induction. Very small fluid</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
<td>Died 14 hrs. postop. At autopsy, the bronchial tube was clear, lungs congested. Large parts of intestinal tract and posterior abdominal wall were necrotic.</td>
</tr>
<tr>
<td>Intestinal Obstruction. Emergency following Acute Pancreatitis.</td>
<td>57</td>
<td>Yes</td>
<td>N₂O, O₂ Halothane. Head-down tilt</td>
<td>When intubated. Fluid</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
<td>Yes</td>
<td>—</td>
<td>Died 1 week postop. At autopsy, peritonitis, basal pneumonia.</td>
</tr>
<tr>
<td>Laparotomy. Peptic Ulcer. 'Cold' Case</td>
<td>68</td>
<td>No</td>
<td>Thiopentone D.T.C. Flat</td>
<td>On inflation with O₂</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
<td>Recovered from pulmonary complications but died 2 weeks postop. from biliary peritonitis.</td>
</tr>
<tr>
<td>Resuture of part of an abdominal wound 3 weeks after gastrectomy. 'Cold' Case</td>
<td>58</td>
<td>No</td>
<td>Thiopentone-suxamethonium. Flat</td>
<td>Small fluid on intubation</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
<td>Yes</td>
<td>—</td>
<td>Recovery uneventful.</td>
</tr>
<tr>
<td>Posterior fossa craniotomy for Acoustic Neuroma</td>
<td>50</td>
<td>No</td>
<td>Thiopentone-suxamethonium. Intubation. N₂O. O₂. Halothane</td>
<td>On the ward 4-hour post-extubation</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
<td>Yes</td>
<td>—</td>
<td>Not a true Mendelson's syndrome but developed bronchopneumonia; after two weeks is making satisfactory progress.</td>
</tr>
</tbody>
</table>
confine any aspiration to the dependent right lung. Often the patient is in bed or on a trolley, in which case the head-down tilt can be obtained by placing the foot of the bed or trolley on the seat of a chair.

If possible, a laryngoscope is inserted into the mouth and the pharynx is cleared by suction under direct vision. If solid material is vomited, it is scooped out of the mouth using a finger wrapped in gauze (Bannister and Sattilaro, 1962), and any bolus seen to be occluding the laryngeal inlet is removed with Magill forceps. Oxygen is given as soon as an airway is established.

Some patients admitted in coma had regurgitated and presented with airway obstruction and tightly clenched jaws. If the airway be not completely obstructed, then oxygen is given using a face mask, and the pharynx may be cleared using suction with a nasal catheter.

If respiration be completely obstructed, then a mouth gag and the laryngoscope blade are used to force open the clenched jaws so that the airway may be cleared. Force should be used in preference to a short-acting relaxant for two reasons. Firstly, vital time may be wasted in drawing up, injecting, and waiting for the muscle relaxant to act. Secondly, the short-acting muscle relaxant will abolish the laryngeal reflexes and consequently greatly increase the danger of a massive aspiration. It is suggested that a short-acting relaxant be used only as a last resort.

A large-bore, cuffed endotracheal tube is passed into the trachea at the earliest opportunity and the cuff is inflated. This is then followed by inflation with 100 per cent oxygen, and endotracheal and endobronchial suction. A Pinkerton (1955) endobronchial catheter is used and has been found to be satisfactory. Great care is taken when sucking out the bronchial tree not to traumatize the bronchial mucosa.

**Bronchial lavage.**

In 1956 Scurr, in a personal communication to Gardner (1958), stated that repeated lavage of the bronchial tree with up to 200 ml of normal saline was thought to reduce the degree of pulmonary oedema following aspiration. This technique of lavage was based on the fact that the lungs of horses and dogs are very tolerant to instillation of water. We have used a technique of bronchial lavage in forty cases, six of which were associated with anaesthesia, with apparent clinical success.

**Technique.** When the patient is adequately oxygenated and no further aspirate can be removed by endobronchial suction, then the bronchial tree is “washed out” with normal saline, which is readily available in most hospital departments. Five to 10 ml of sterile normal saline are injected by syringe into the endotracheal tube, and this is followed by endotracheal and endobronchial suction and oxygenation. This sequence is repeated until the fluid aspirated is clear. A total of from 300 to 400 ml may be needed.

If the patient’s level of consciousness be such that the cough reflex is present, the injection of normal saline into the trachea will be followed by coughing, thus bringing the more deeply placed particles within reach of the sucker.

Mendelson (1946) and Bannister et al. (1961) found that normal saline per se caused little or no pathological change when instilled into the bronchial tree in experimental animals. Teabeaut (1952) reported that the presence of partly digested food particles in the lungs caused a reaction regardless of the pH of the foreign body. It is well known that particulate matter may cause bronchial obstruction with subsequent absorption collapse of the lung. In this series, most of the patients who had inhaled particulate matter, and who were treated by lavage, were found on subsequent bronchoscopy to have a clear bronchial tree. Thus it is felt that the use of normal saline as a vehicle for the removal of aspirated material is justified.

By utilizing the findings of Mendelson (1946) and Teabeaut (1952), Bannister et al. (1961) found that it was possible to produce the lesion of aspiration pneumonitis in experimental animals. Rabbits were anaesthetized with nitrous oxide, oxygen and ether; they were intubated and a light plane of anaesthesia was maintained so that breathing and the cough reflex were present, as in the induction stage of anaesthesia. Following intubation hydrochloric acid with pH range from 1.50 to 1.82 was instilled into the trachea using 4 ml of acid per kg body weight. The rabbits were turned from side to side to ensure dispersion of the acid to both lungs. All the rabbits were sacrificed at the end of 48 hours and gross and microscopic examination of the lungs was carried out. Typical lesions of aspiration pneumonitis were produced in a control group of eight rabbits treated with acid only.

In a group of six rabbits, the effects of instilla-
tion of sodium bicarbonate 3.75 and 1.87 per cent, sodium lactate N/10, calcium gluconate 5 per cent, normal saline and sodium hydroxide N/10 in a volume of 4 ml/kg were studied. All these solutions, except sodium hydroxide N/10, produced either no damage or slight damage. Sodium hydroxide N/10 produced lesions of similar severity to N/10 hydrochloric acid.

A further group of nineteen rabbits had hydrochloric acid instilled into the lungs, as above, followed by one of the above six “treatment fluids”. It was found that the lesions were aggravated in all cases. Bannister et al. suggested that this may be due to the fact that:

1) the large volume of fluid serves to push the hydrochloric acid deeper into the lungs;
2) mixing of the acid and the treatment solution was impossible because of the minute size of the interface;
3) the hydrochloric acid probably caused damage within a very short time.

They concluded that a large volume of fluid used as a diluent or as a neutralizing agent was dangerous, but that it was not necessary to infer that the use of a small volume of normal saline for loosening secretions in the trachea was contraindicated, provided that it was rapidly aspirated.

Whilst we must accept the experimental findings, we cannot entirely agree with the conclusions drawn, for the following reason:

The use of normal saline as a pure diluent, as in the experiment referred to above, could on theoretical grounds be expected to worsen the position.

When an equal amount of normal saline is added to a volume of hydrochloric acid of pH 1.6, and if the solution be completely mixed, it will have a pH of approximately 1.8. Thus there is twice the volume of fluid with a pH well within the irritant range. Teabeaut (1952) found that all solutions with a pH of less than 2.5 produced the lesions of aspiration pneumonitis when instilled into the lungs.

The technique of lavage described, with repeated injections of small quantities of saline and immediate suction, does not increase the volume of fluid in the lungs, and in theory it could materially alter the pH, if the total amount of fluid aspirated was small.

The instillation of 4 ml/kg of hydrochloric acid of pH 1.6 is equivalent to an aspiration of 280 ml of highly acid gastric juice in a 70 kg man. An aspiration of this type and quantity must be rare. Most of the aspirations seen in this department have been small in amount.

It is felt that there is a good case for using lavage on clinical grounds, and in that the experimental work does not deal with the problem of lavage, it is not applicable when using the above technique.

If aspiration occur during induction of anaesthesia, then the operation should, if at all possible, be postponed to a later date.

Supportive therapy.

During the initial stages in the treatment of aspiration, intravenous aminophylline 250 mg is given in an effort to reduce or prevent the element of bronchial spasm which is frequently present. If bronchospasm persist or become evident again later, further doses of aminophylline may be given or another bronchodilator agent may be used. Resistant bronchospasm severely reduces the pulmonary ventilation in these patients; and it is important that every effort should be made to overcome it. Intravenous hydrocortisone 100 mg is given as a routine and repeated if necessary. Bannister et al. (1961), in their studies into the general forms of treatment for aspiration pneumonitis, found that intravenous hydrocortisone 100 mg greatly reduced the severity of the pulmonary lesions in aspiration pneumonitis. This is supported clinically by Dines and his colleagues (1961) who described a case of severe aspiration pneumonitis in which there was a dramatic improvement in the patient following the intravenous administration of hydrocortisone.

Following pregnancy and labour, the adrenals may become exhausted and the patient may become unable to cope with the additional stress created by aspiration pneumonitis. Parker (1952) recommended the administration of hydrocortisone to obstetric cases who had aspirated gastric contents. Hydrocortisone is also of value in the treatment of bronchospasm. As a large number of the population receive systemic corticosteroids for their non-specific effects in the treatment of many diseases, it is quite possible that in an emergency a patient may present at operation or in a casualty department and no history of corticosteroid therapy be available. The administration of hydrocortisone in these cases may be lifesaving.

Hypotension, which may be a feature of the initial acute asphyxial episode, but which in our experience is more likely to be associated with the
THE EFFECT OF THE INHALATION OF VOMITUS ON THE LUNGS

asthmatic type of reaction occurring within 6 hours of the aspiration, is treated with vasopressor agents. Ephedrine, methyl amphetamine, methoxamine, and metaraminol have been used in the standard recommended dosage. Hypovolaemia, whenever it occurs, is treated with intravenous replacement therapy. A routine course of a broad spectrum antibiotic is started following aspiration.

Bronchoscopy.

When the initial danger of asphyxia is overcome, and the patient is well oxygenated, bronchoscopy is undertaken. This ensures that the segmental bronchi are patent.

In many cases, spontaneous respiration with adequate pulmonary ventilation is soon established. If, at this stage, the patient is conscious with an active cough reflex, and the danger of further regurgitation has been minimized by the passage of a stomach tube, the endotracheal tube may be removed.

All comatose patients remain intubated and, if the coma is expected to be prolonged (more than 24 hours), tracheostomy is performed.

During the first 24 hours after the aspiration, a careful watch must be kept on the patient. The increase in deadspace air as a result of extubation may make respiration inadequate with a resultant hypoxia and hypercarbia. Thus a vicious circle of respiratory depression is set up. A similar picture may be seen with the asthmatic type of reaction.

Should respiration become inadequate at any time, then the patient is re-intubated and inflated with oxygen, and the plasma carbon dioxide level is returned to normal limits by inflating the patient through a soda lime system.

The patient may require assisted respiration. At first this is done by hand, then later the patient is transferred to a patient-triggered respirator, if one be available. The use of intermittent positive pressure respiration may help to relieve pulmonary oedema by raising the intra-alveolar pressure. The patient is taken off the respirator at intervals and the adequacy of the spontaneous respiration is assessed. When this becomes adequate, the endotracheal tube is removed.

All patients receive intensive physiotherapy if their general condition permits.

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REFERENCES


