

## VOMITING, REGURGITATION, AND ASPIRATION IN ANAESTHESIA, II

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IN A PREVIOUS COMMUNICATION (1) we discussed the incidence of and the clinical and pathological findings associated with aspiration of stomach contents. We shall now consider the treatment of this complication of anaesthesia. Earlier papers on the subject of aspiration which were referred to in our previous communication stressed prevention and made little mention of specific measures of treatment.

Morton and Wylie (2) suggested ways of preventing regurgitation and suggested procedures to be used if regurgitation or vomiting occurred. These authors believed that a relaxant should not be given since it would predispose to more regurgitation. They considered immediate bronchoscopy to be "lethal."

Merrill and Hingson (3) in reviewing mortality and morbidity from aspiration in maternal cases found that prompt bronchoscopy gave excellent results in decreasing the degree and duration of morbidity.

Housmann and Lunt (4) reported three cases of aspiration in obstetric patients and drew attention to the fact that pulmonary oedema rather than bronchospasm is present. They considered post-partum adrenal insufficiency to be the major factor in the pathogenesis of the condition. This they believed to be the result of loss of the placenta secretion of adrenocorticotrophic hormone and glucocorticoids. This leads to the theory that the body cannot respond adequately to stress in the immediate postpartum period, and cortisone is therefore recommended as essential therapy in the treatment of the aspiration syndrome.

The treatment presently used in the teaching hospitals of the University of Toronto is devised essentially to establish and clear an airway and as far as possible to prevent pulmonary complications by removal of the aspirated material and prevention of pulmonary reaction by steroid therapy. The pharynx, larynx, trachea and bronchi are cleared of all regurgitated material. This must be done quickly and often in stages, as the patient must be oxygenated at the same time. To facilitate this we have found it advisable to use a fast-acting relaxant. This not only gives the anaesthetist easy access to the larynx and trachea via endotracheal tube or bronchoscope, but also prevents the patient from aspirating material into the lung periphery with gasping respirations. Bronchoscopic examination of the larynx, trachea, and bronchial tree is usually carried out. It has been found beneficial to wash out any remaining fluid and food particles with saline. If applicable, general anaesthesia is discontinued.

The chief aim of therapy is to prevent pulmonary complications following aspiration of stomach contents. Pulmonary and bronchial oedema, consolidation and atelectasis may appear within a few minutes of the accident. Pneumonitis, lung abscess, or bronchopneumonia may follow in a few hours or days. Pulmonary fibrosis, localized or generalized, may follow in a few weeks. The serious nature of this complication is exemplified in case 7, reported below. Intravenous cortisone is given immediately and continued along with ACTH for a minimum

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of three days. Antibiotic therapy, antispasmodics, and oxygen with or without aerosol inhalation are used as deemed necessary. This method has proved effective in decreasing morbidity and preventing sequelae.

The case histories presented below were collected from several Toronto hospitals. The anaesthetists were able to recall vividly much information to supplement the details on the anaesthetic charts. This was an indication to us of the importance with which the problem was viewed by our colleagues.

#### CASE HISTORIES

##### *Case 1*

A middle-aged female had a general anaesthetic for dislocation of the right elbow. The injury occurred at 4.00 P.M., the patient ate a meal at 5. P.M. and the anaesthetic, thiopentone sodium, N<sub>2</sub>O and O<sub>2</sub>, was commenced at 10.15 P.M.. At 10.30 P.M. she regurgitated and aspirated while under general anaesthesia. The patient was intubated and clear fluid and mucus were aspirated from the trachea. At 10.50 P.M. the patient was bronchoscoped. No erythema or bile staining of the tracheal mucosa was noted. The airway was clear. Scattered rhonchi were noted in both upper lung fields. The patient was admitted to hospital and given therapy of nasal oxygen and aerosol inhalations. She was asymptomatic during the night. Physical examination twelve and thirty-six hours after the accident revealed no positive chest findings. The patient was discharged. A chest X-ray taken the day of discharge, but unfortunately not reported until later, noted parenchymal infiltration of the upper lobes of both lungs (Fig. 1). This is of interest in view of the negative findings on physical examination.

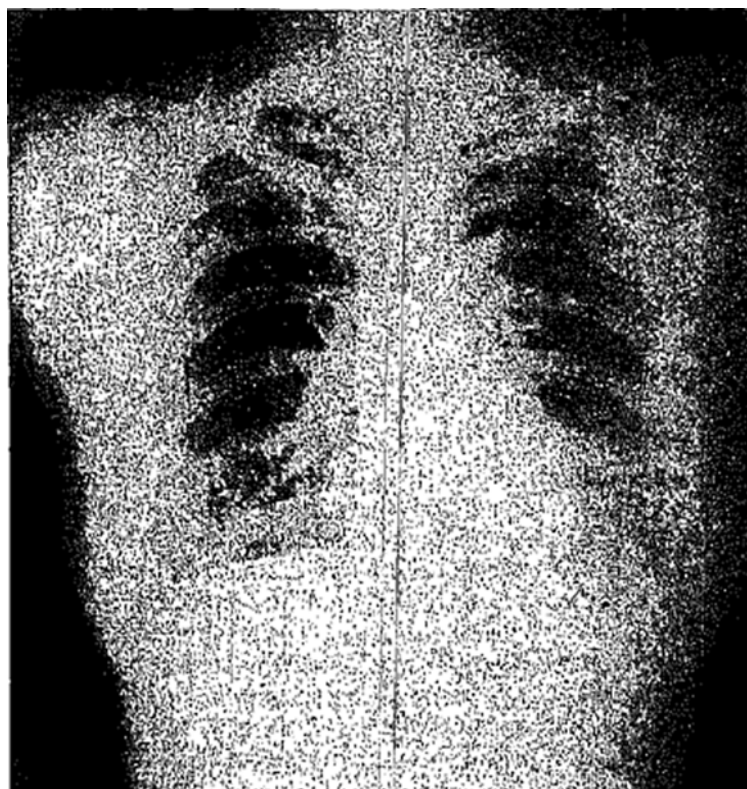


FIGURE 1

*Case 2*

A 40-year-old male was admitted in November, 1956, for drainage of an appendiceal abscess. Two weeks later a laparotomy was performed for small bowel obstruction. Two hours postoperatively he vomited and aspirated. The patient was immediately bronchoscoped and was given 100 mg of Solu-cortef® intravenously and antibiotic therapy was commenced. A portable chest X-ray taken the following day showed "an area of infiltration in the right lower lobe in keeping with pneumonitis" (Fig 2A). The patient was pyrexia for four days. A chest X-ray taken the day of discharge, sixteen days later, was reported to be clear (Fig 2B).

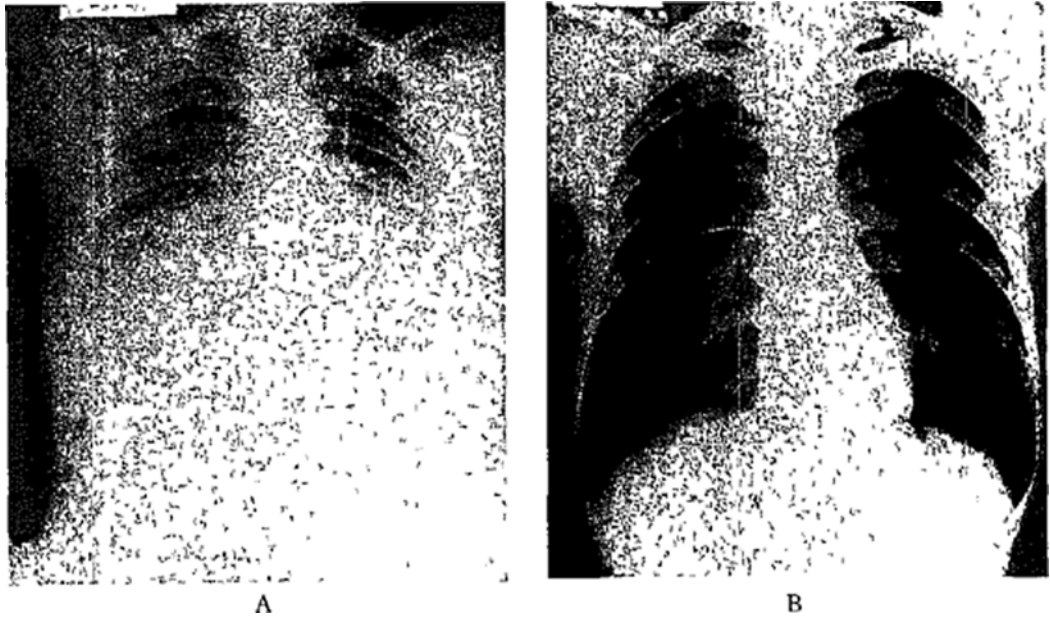


FIGURE 2

*Case 3*

A 25-year-old primiparous patient vomited during induction of general anaesthesia for delivery. The anaesthesia was discontinued, and the pharynx was cleared. The patient delivered spontaneously. Antibiotic therapy only was given during the postpartum period. Figures 3A, 3B, and 3C are X-rays taken one day, seven days, and seven weeks postpartum. These films show the slow resolution of the infiltrative process, which was still incomplete at seven weeks.

*Case 4*

A young primiparous patient was taken to the delivery room 22 hours after admission to hospital. During induction of general anaesthesia with  $N_2O$  and  $C_3H_6$  the patient vomited and aspirated. The pharynx was cleared and the patient was intubated with the aid of succinylcholine and oxygenated. A small amount of dark fluid was aspirated from the trachea through the endotracheal tube. The anaesthetic was discontinued and the baby was delivered. At this time the chest was reported clinically to be clear. Immediately postpartum the patient was given a broadspectrum antibiotic.

Twelve hours postpartum there were râles and coarse rhonchi in both lungs. A chest X-ray (Fig 4A) was reported as showing right upper lobe and left lower lobe bronchopneumonia. At this time she was given therapy of "Compound F" 50 mg stat and t i d, cortisone 25 mg stat and q i d, and Aminophylline Suppositories q i d. This medication was continued for three days.

Twenty-four hours postpartum the patient had no clinical signs in her chest X-rays taken seven days postpartum reported the chest to be clear (Fig 4B)

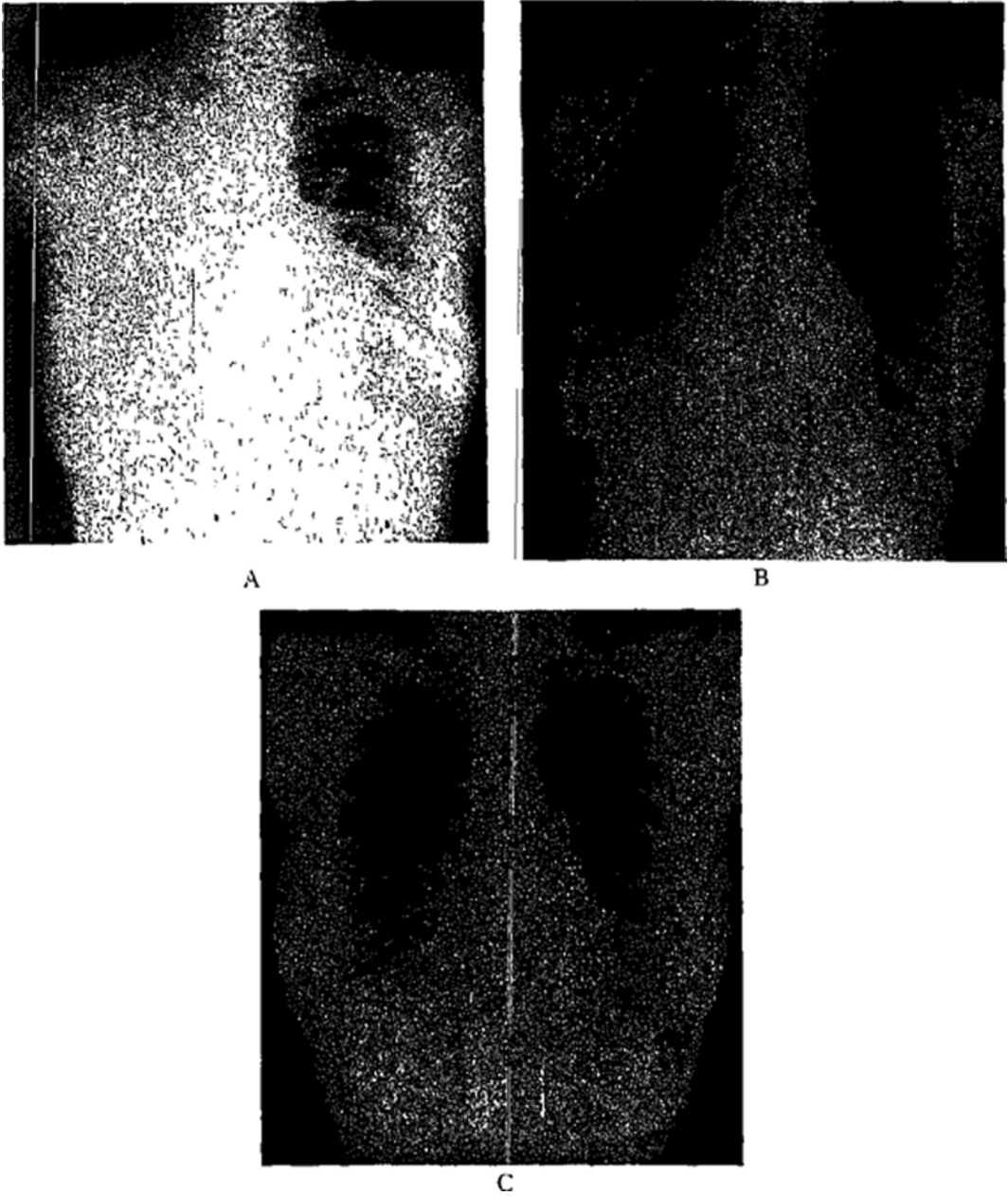


FIGURE 3

#### Case 5

A 21-year-old primiparous patient was presented for Caesarean section after forty-two hours of labour. Thirty minutes preoperatively the patient vomited a small amount of brownish liquid. The stomach was not emptied. General anaesthesia was induced with thiopentone. A cuffed endotracheal tube was introduced with the aid of succinylcholine. At this time a small amount of brownish fluid was noted in the pharynx, but none could be aspirated from the bronchial tree. Anaesthesia was maintained with  $N_2O$  and trichlorethylene and oxygen. The conduct of the anaesthesia was uneventful.

A few minutes after extubation the patient was noted to be slightly cyanotic. This was readily reversed with oxygen. A coarse rhonchus was present in the right chest. On the ward her condition deteriorated. Thirty minutes postoperatively râles were present in the right chest. Oxygen inhalations failed to eliminate the cyanosis. Pulse rate was 200 per minute. Moist bubbling râles were noted over the entire chest. A portable chest X-ray (Fig 5A) showed generalized "snow storm" appearance. Hydrocortisone 100 mg was given immediately intravenously and a further 100 mg was given slowly during the next two hours. The patient began to improve after one hour of therapy. Her respiratory rate fell from 30 to 24 and her pulse rate from 200 to 120. The cyanosis disappeared. Râles were heard only in the right upper chest. A further 100 mg of hydrocortisone was given during the next six hours. Metacortin was given for three days to a total dosage of 105 mg. A broad spectrum antibiotic was given for 48 hours. The patient steadily improved and was discharged on the tenth day postpartum. A chest X-ray taken on that day was reported to be normal (Fig 5B).

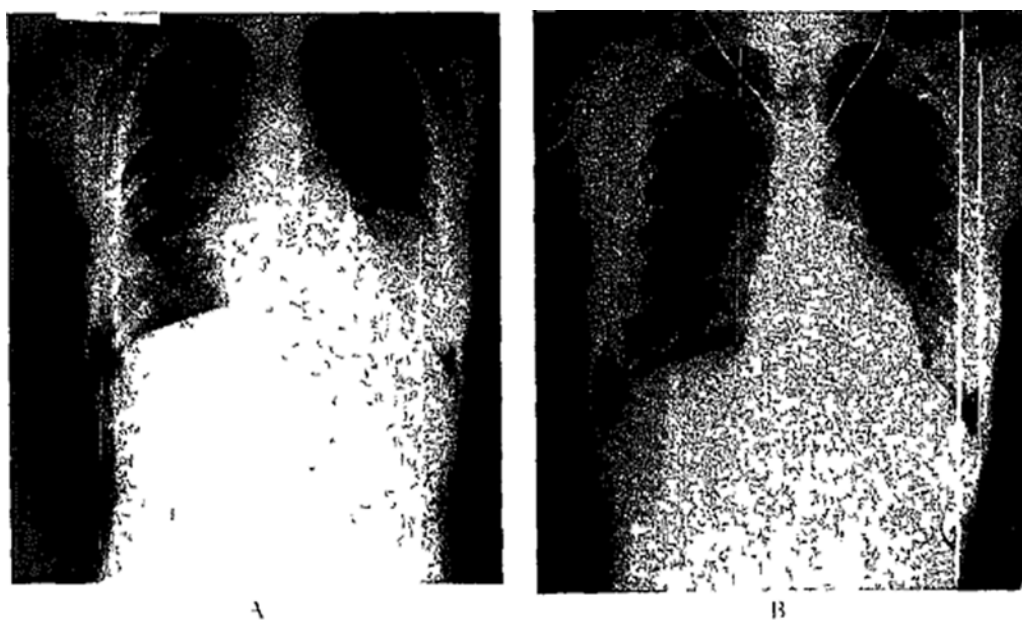


FIGURE 4

#### Case 6

A 21-year-old primiparous patient, in labour for sixteen hours, was delivered under general anaesthesia with  $N_2O$ , trichlorethylene, oxygen, and curare. During anaesthesia the patient vomited brownish fluid. Aspiration was suspected and she was intubated. Brownish fluid was aspirated from the trachea and bronchi through the endotracheal tube. The upper respiratory tract was lavaged with 200 cc of saline. On physical examination the patient was dyspnoeic and had râles in both lungs. The dyspnoea and râles continued for about 48 hours. A chest X-ray taken the evening of delivery showed mottled densities in both lung fields, mainly in the lower segments (Fig 6A). Solúcortef 100 mg was given immediately and repeated in 8 hours. One dose of aminophylline was given. Antibiotic therapy was given for three days and metacortin 10 mg q 8 h for one day. Although she was in an oxygen tent, the patient was dyspnoeic during the first day except when she was maintained in Fowler's position. There was steady clinical improvement, and the patient was discharged seven days postpartum. A chest X-ray taken on the day of discharge showed very little residual infiltration (Fig 6B).

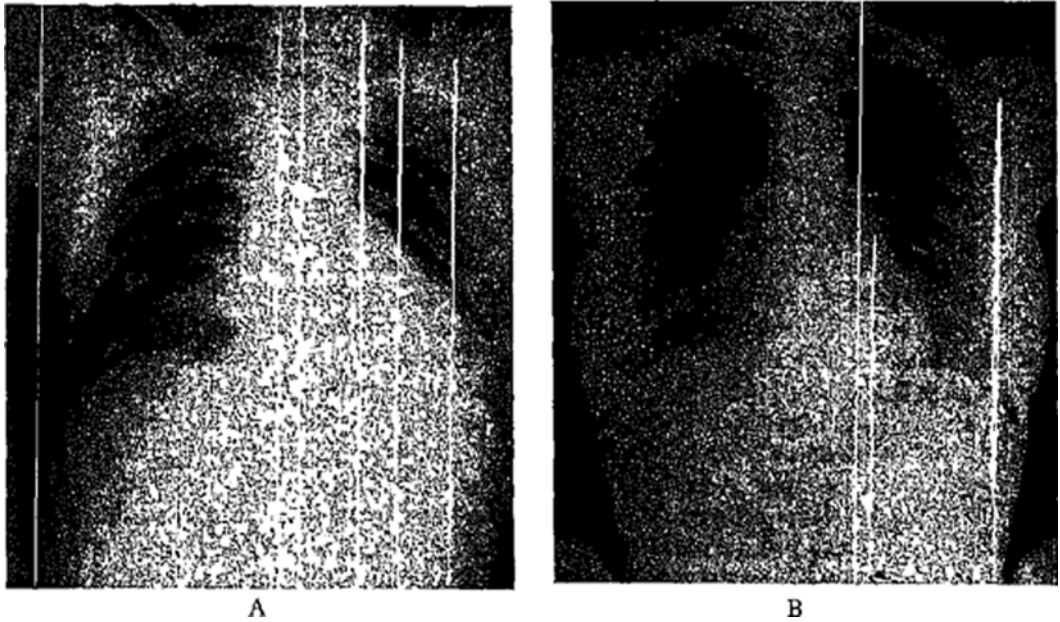


FIGURE 5

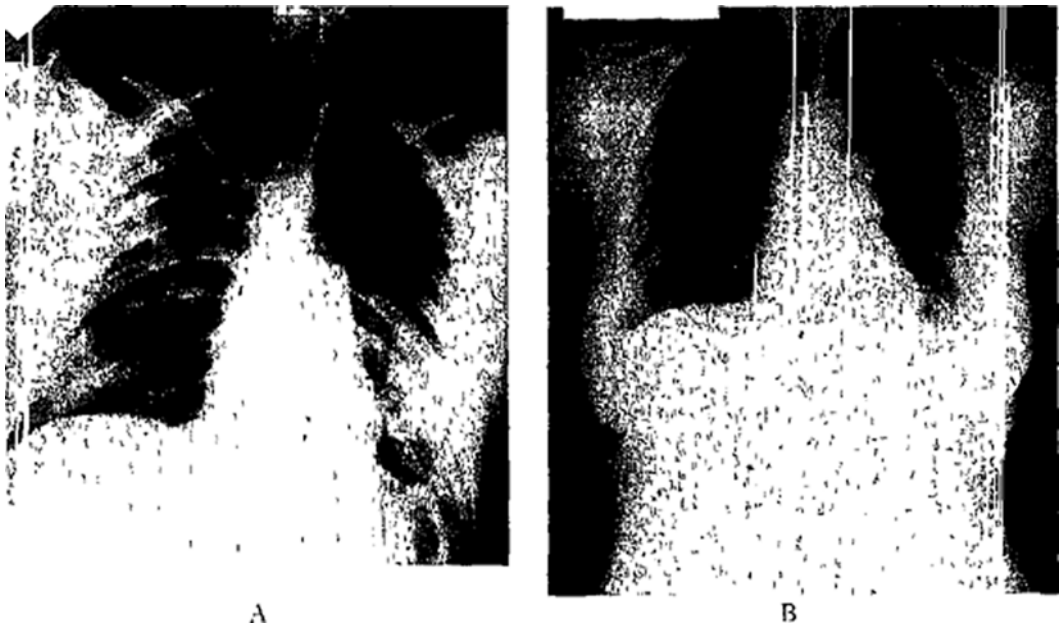


FIGURE 6

*Case 7*

A young primiparous patient had an episode of cyanosis and tachycardia during general anaesthesia for delivery. Immediately postpartum the patient was cyanotic and on physical examination was found to have a blowing apical systolic murmur, gallop rhythm, bilateral basal râles, and pitting oedema of both ankles. Her heart rate was 160 per minute and blood pressure 100/80. The heart was found to be enlarged. The patient was considered to have rheumatic heart disease with failure and was treated with quinidine, digoxin, and aminophylline. Her breathing remained laboured and difficult. Nine days postpartum she was given Cortone 25 mg b.i.d. for six doses. A

chest X-ray taken at the end of this therapy (12 days postpartum) showed pulmonary congestion and cardiac enlargement. The patient at this time was transferred to another hospital.

Physical examination at the time of transfer showed her temperature to be 100 F., pulse rate 120 per minute, and blood pressure 90/60. She had gallop rhythm, a systolic heart murmur, râles in the left chest, and an enlarged liver. The patient responded well to digitalis therapy until four weeks postpartum when she complained of a dry cough and shortness of breath. She had one episode of haemoptysis. Following this a physical examination of the chest revealed bilateral consolidation. The patient expired one week later (five weeks postpartum). During the final stages of this illness, an ECG was reported normal, and blood and sputum cultures were non-contributory. The chest X-ray showed increased mottling throughout both lung fields.

At autopsy the lungs were heavy, oedematous, firm, and airless, with patchy consolidation and haemorrhage. There was a slight pleural effusion. The heart weighed 400 gm., and showed no evidence of valvular disease. The spleen showed infarction and there was a small infarct in the lower lobe of the left lung. No evidence of amniotic debris was found in the pulmonary arterioles. The lung tissue showed marked desquamation of alveolar living cells, patchy deposits of haemosiderin, and alveolar haemorrhages. The pathologist ended his report on the case as follows, "The cause of death in this case is obscure. It may have been a result of amniotic fluid embolism at delivery, or possibly an aspiration of gastric contents during anaesthesia." The anatomical diagnosis was: (i) diffuse haemorrhages of lungs with fibrosis; (ii) infarct of lung; (iii) cloudy swelling of myocardium.

This case almost certainly is one of regurgitation and aspiration.

#### Case 8

A 19-year-old primiparous patient was admitted at 8 P.M. after five hours' labour, and delivered at 3.45 A.M. the following morning. At 8.00 A.M. she was seen by the anaesthetist because of dyspnoea and a drop in blood pressure. The patient was cyanotic and dyspnoeic. She had tachycardia, hypotension, and bilateral pulmonary consolidation. She was given hydrocortisone, antibiotics, digoxin, and aminophylline, but continued to deteriorate. The patient died two days following admission, thirty-six hours after delivery.

At autopsy the right lung weighed 840 gm. The cut surface showed areas of necrosis up to 1 cm. around the moderate-sized bronchi. These areas exuded pus. The left lung weighed 490 gm. and had a similar appearance.

On microscopic examination very little aerated lung tissue was seen. The alveoli were filled with oedema fluid. There was necrosis and destruction of the alveolar walls. The oedema fluid was haemorrhagic and diffusely infiltrated with polymorphonuclear leukocytes. There were many small abscesses. Anatomical diagnosis: (i) haemorrhagic pulmonary oedema; (ii) acute aspiration pneumonia with suppuration; (iii) bilateral pleural effusion; (iv) right-sided cardiac failure.

#### Case 9

A primiparous patient was admitted in labour at 11 P.M. and was taken to the delivery room two hours later. The patient vomited solid food on induction of general anaesthesia. There was complete obstruction of the airway. The pharynx was cleared manually and then aspirated. The larynx was aspirated under direct vision and an endotracheal tube passed, through which the patient was alternately and gently oxygenated. Twenty minutes later the patient was bronchoscoped without the need of relaxant or anaesthetic. The bronchial mucosa was quite red, and bled when touched by instruments.

Forty-five minutes after the accident the patient was conscious. An area of atelectasis in the right lower lobe was cleared by voluntary coughing. Solucortef® 100 mg. was given intravenously over a period of 6 hours and repeated q. 12 h. ACTH 20 units was given immediately and b.i.d., and Dicrysticin 2 cc. was given immediately.

The following morning the patient was afebrile, but had an elevated pulse rate, elevated respiratory rate, and an area of atelectasis in the right lower lobe. This quickly cleared when the patient was given oxygen and aerosol (Alevaire®) inhalations. The pulse and respiratory rates returned to normal. Cortisone was discontinued the third day, and ACTH on the fifth day. The patient was discharged on the tenth day and remained well.

#### Case 10

A 4-year-old child came to operation with a stomach full of fruit juice and ginger ale. Actually the stomach was distended although this was not noted until the patient regurgitated and aspirated following induction of anaesthesia. Fluid was aspirated from the pharynx and the patient was intubated. Endobronchial aspiration was carried out via the endotracheal tube. The child was allowed to wake up enough to cough vigorously, but the anaesthetic was continued and the operation completed.

Four hours later the patient was bronchoscoped. There was much secretion in the bronchi. The vocal cords were red, but there were no haemorrhagic areas and the bronchial mucosa did not bleed when touched with the instrument.

At this time the child was given 100 mg. of Solucortef, followed by 50 mg. in the next 8 hours. Metacortin 30 mg. (total dose) was given over the next three days. Chloromycetin was given for three days.

At the time of bronchoscopy there were rhonchi and scattered râles in both lungs. The following day the chest was clear on auscultation, but a small area of collapse was noted in the posterior segment of the lower lobe of the right lung. The third day there was an occasional rhonchus in the right lung. On the fifth day there were no abnormal chest findings.

#### DISCUSSION

The ten cases of aspiration of vomitus here described illustrate the value of the specific therapy recommended. The serious sequelae if the condition is not recognized and adequately treated may also be noted. The contrast between the treatment required for and the sequelae of aspiration of solid and liquid gastric contents is evident in comparison of Cases 6 and 9. Cases 7 and 8 show many important details. Aspiration was not recognized in either case. The rapid, fatal course in Case 8, in spite of vigorous therapy is remarkable. We feel this may be due to the fact that the aspiration was not recognized, and the anaesthetic was allowed to continue without removal of the aspirated material (cf. Case 4).

Cases 1 to 6 illustrate the difference in the pulmonary sequelae between those cases which receive inadequate or delayed treatment and those which received adequate and immediate treatment. In Case 1, although the condition was recognized, it was not considered that there was sufficient pulmonary pathology present at the time of bronchoscopy to require more specific therapy. More serious complications may have been prevented by the fact that the aspirated gastric secretions were partially neutralized by ingested food. This difference was discussed in our previous communication (1).

#### CONCLUSIONS

1. Aspiration of gastric contents is a serious complication of general anaesthesia and a major cause of morbidity and mortality.
2. Immediate recognition and immediate treatment are necessary to prevent prolonged morbidity or mortality.



3. Successful therapy must include prompt complete removal of any irritant from the respiratory tract and prevention of inflammatory reaction by specific drug therapy.

#### OUTLINE OF RECOMMENDED TREATMENT

##### *Immediate*

1. Stop the general anaesthetic immediately and continually oxygenate the patient while carrying out the following steps.

2. Clear the pharynx and larynx with suction and manually if necessary.

3. Prevent further aspiration by lowering the head of the table and giving a fast, short-acting relaxing agent.

4. Clear the larynx and bronchial tree by direct laryngoscopy and bronchoscopy.

5. Lavage the bronchial tree with soda bicarbonate solution or saline.

##### *Specific Drug Therapy*

1. Cortisone 100 mg. should be given intravenously immediately and 200 mg. during the next 24 hours, followed by 50 mg. twice a day for two days, or until the chest is clear.

2. ACTH 20 units twice a day for three days.

3. Antibiotics: a broad spectrum antibiotic or combination of antibiotics should be given as a preventative measure.

4. Oxygen by mask, tent, or nasal catheter should be given, sufficient to maintain adequate oxygenation.

5. Aminophylline intravenously or in suppositories may be given.

6. Expectorant cough mixtures may aid in the production of thin easily removed bronchial secretions.

7. Inhalation therapy with a detergent aerosol may be beneficial in the prevention or treatment of atelectasis.

#### ACKNOWLEDGMENTS

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#### RÉSUMÉ

Au cours d'un exposé antérieur, les auteurs ont parlé de la fréquence et des séquelles cliniques pathologiques de l'aspiration de liquide gastrique. Dans cette communication-ci, il sera question du traitement de cette complication de l'anesthésie avec l'anamnèse des histoires de cas de dix malades.

L'aspiration de liquide gastrique est une complication sérieuse de l'anesthésie générale et une cause fréquente de morbidité et de mortalité. Pour prévenir la morbidité et la mortalité, il s'impose de reconnaître le fait immédiatement et de pratiquer le traitement aussitôt. Dans un traitement efficace, il faut inclure

l'ablation précoce et complète de toute substance irritante dans les voies respiratoires et faire la prévention d'une réaction inflammatoire par les médicaments appropriés.

#### TRAITEMENT SUGGÉRÉ

##### *Immédiatement*

1. Suspendre immédiatement l'anesthésie générale et continuer à oxygéner le malade tout en faisant ce qui suit.
2. Nettoyer le pharynx et le larynx avec un aspirateur et avec les mains si nécessaire.
3. Prévenir une nouvelle aspiration en abaissant la tête du malade et en administrant un myorésolutif à action rapide et courte.
4. Nettoyer le larynx et l'arête bronchique en faisant une laryngoscopie directe et une bronchoscopie.
5. Faire un lavage de l'arête bronchique avec une solution physiologique ou bicarbonatée.

##### *Médication spécifique*

1. Administrer immédiatement par voie endoveineuse de la cortisone 100 mg. et, durant les 24 heures suivantes, 200 mg.; ensuite 50 mg. b.i.d. durant deux jours ou jusqu'à ce que le poumon soit clair.
2. ACTH 20 unités b.i.d. durant trois jours.
3. Des antibiotiques. Comme mesure préventive, administrer un antibiotique à grand spectre ou encore une association d'antibiotiques.
4. Donner, soit par masque, tente ou cathéters nasaux, de l'oxygène en quantité suffisante pour maintenir une bonne oxygénation.
5. On peut aussi donner de l'aminophylline par voie endoveineuse ou en suppositoires.
6. Des mixtures expectorantes peuvent aider à éliminer des sécrétions fluides et faciles à expectorer.
7. On peut aussi, en prévention ou comme traitement d'atélectasie, faire inhaler un détergent par aérosol.

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