THE ACID ASPIRATION SYNDROME

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DESCRIPTION OF THE SYNDROME

Clinical Features

A diagnosis of Acid Aspiration Syndrome was made when an "asthmatic" attack together with signs of pulmonary oedema occurred in patients with no previous asthmatic history, who had had a period of unconsciousness due to disease or drugs, and who were known or suspected to have vomited. Bronchospasm and cyanosis in these patients progressed within 30 minutes from onset to a severity that threatened life. There was a sinus tachycardia with rates in excess of 120 per minute, and clinical and electro-cardiographic evidence of right heart strain. Radiography of the chest provided further evidence in support of the diagnosis. (see section on radiology).

The disease was most frequently associated with obstetric anaesthesia (5 out of 7 cases described—see case reports; Mendelson, 1946; Hausmann and Lunt, 1955; Merrill and Hingson, 1951), but occurred also during anaesthesia for general surgery (Dines et al, 1961; Johnson, 1962; Case 4, page 9), coma from other causes (Irons and Apfelbach, 1940), and heavy sedation that depressed the laryngeal reflexes (Case 5). The episode of vomiting or regurgitation followed by aspiration of gastric contents into the lungs was not noticed in every instance (Parker, 1954) (Case 1 and 5). In these instances, the resulting disease had to be distinguished from bronchial asthma, acute cardiac failure, bronchopneumonia, pulmonary thrombo-embolism and amniotic fluid embolism, pneumothorax and atelectasis. The time interval between aspiration and the onset of symptoms varied from several minutes (Cases 1, 3, 4 and 5) to several hours (Cases 2, 6 and 7).

Pathology at Autopsy

The pleural and pericardial cavities contained sero-sanguinous fluid. The lungs were overdistended with air and weighed one and a half times normal. The lesions were extensive (Fig. 1) the lungs were oedematous and haemorrhagic and hyperaemic areas were unevenly distributed throughout the lung tissue (Fig. 2). In places, the tissue was stained with haemolysed and altered blood. Post mortem changes were more rapid and advanced than in death from other causes.

The tracheal and bronchial mucosa was hyperaemic and the air passages contained varying quantities of exudate (Fig. 3).

On microscopic examination there was extensive desquamation of the bronchial mucosa. The alveolar capillaries were intensely congested. The alveoli were filled with red blood cells, oedema fluid and desquamated epithelium, and in some sections, undigested food particles. The tissue did not stain well because of post mortem changes but the red blood cells in the sections looked dehaemoglobinised.

Autopsy should be performed within two hours of death as rapidly advancing post mortem changes obscured the characteristic features of the disease (Irons and Apfelbach, 1940).

Radiology

Radiographs of the lungs were taken in all instances and showed irregular bilateral shadows with no change in the position of the mediastinum. There were no areas of atelectasis. The radiographs resembled those found in viral pneumonias (Fig. 4). There were no pleural effusions. These radiological changes disappeared completely in patients who recovered Mendelson, 1946; Parker, 1954; Marshall and Gordon, 1958; (Figs. 5 and 6), and there were no pulmonary sequelae during short-term follow-up.

Treatment

Aspiration of regurgitated acid stomach contents during anaesthesia was treated by first inserting into the trachea, a cuffed endotracheal tube. As much as possible of the foreign material was removed by suction through this tube. Bronchial lavage was performed by pouring 10 ml. of normal saline at 37°C down the endotracheal tube every 5 minutes for 30 minutes. Bronchoscopy under general anaesthesia was performed if solid material was suspected of

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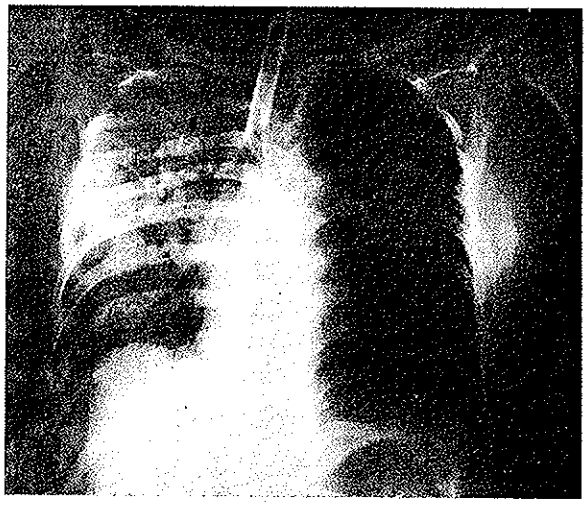

Fig. 4. Chest X-ray of patient who aspirated acid and was treated with I.P.P.V. via a tracheostomy. The shadowing resembled that seen in viral pneumonia; but in this particular instance was mainly unilateral.

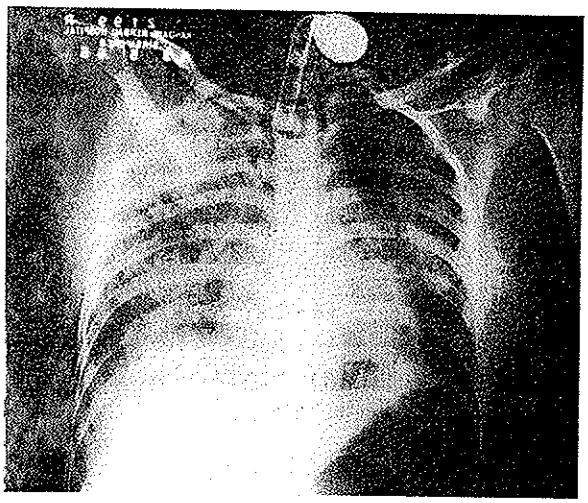

Fig. 5. Chest X-ray of patient with aspiration syndrome (as in Fig. 4.) taken one day later.

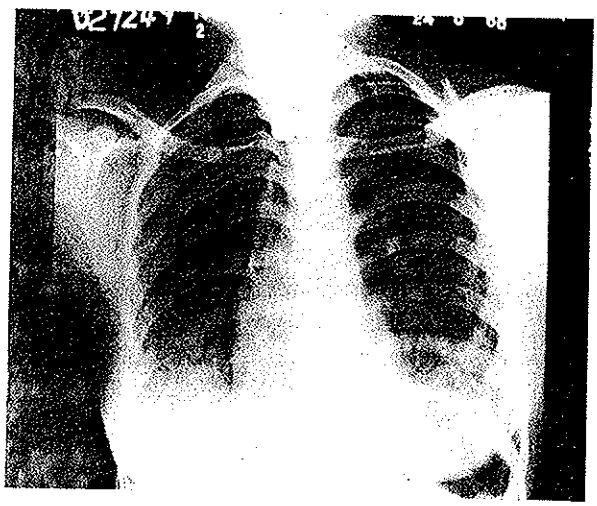

Fig. 6. Chest X-ray of same patient as in Figs. 4 & 5 taken 12 days later, showing that the lungs were radiologically normal.

having been aspirated. Intermittent positive pressure ventilation (I.P.P.V.) was carried out to maintain gaseous homeostasis between these manipulations.

Bronchospasm was treated with subcutaneous adrenaline (1 ml. of 1:1,000 administered over 60 seconds), intravenous aminophyline (0·25-0·5 gm. in 20 ml. water) and intravenous hydrocortisone. Prophylactic antibiotic cover was provided.

Three patients (Cases 2, 4 and 5) were treated by prolonged I.P.P.V. with oxygen in addition to the measures described above. But although gaseous homeostasis appeared to be maintained, clinical and electrocardiographic signs of acute right heart strain and failure were progressive. Death occurred from cardiovascular collapse that did not respond to treatment with steroids, transfusions, vasopressors or digitalis.

CASE REPORTS

Seven illustrative cases are recorded. Four patients died. Five were associated with general anaesthesia for obstetric procedures and one was in a general surgical patient (Case 4). One patient was not anaesthetised but received heavy sedation during labour (Case 5).

CASE 1

Caesarean section was performed on a healthy 41 year-old woman admitted in labour with a transverse lie and prolapsed cord. General anaesthesia was induced with a thiopentone, succinylcholine, nitrous oxide and oxygen sequence, and a cuffed endotracheal tube was inserted. Anaesthesia was maintained with nitrous oxide, oxygen, succinylcholine in intermittent doses and I. P.P.V. Shortly after the commencement of surgery, severe bronchospasm occurred. This was relieved by the intravenous injection of 0·5 gm. of Aminophylline. Surgery was completed successfully. A live foetus was delivered. The patient regained consciousness at the end of the anaesthetic and was returned to the ward 60 minutes after the induction of anaesthesia.

Thirty minutes later, the patient developed severe bronchospasm, respiratory distress, cyanosis and cardiovascular collapse. Death occurred within minutes.

At autopsy, the only abnormal findings were in the respiratory system: "trachea and bronchi contained a large amount of blood-stained frothy fluid. Lungs; gross oedema of

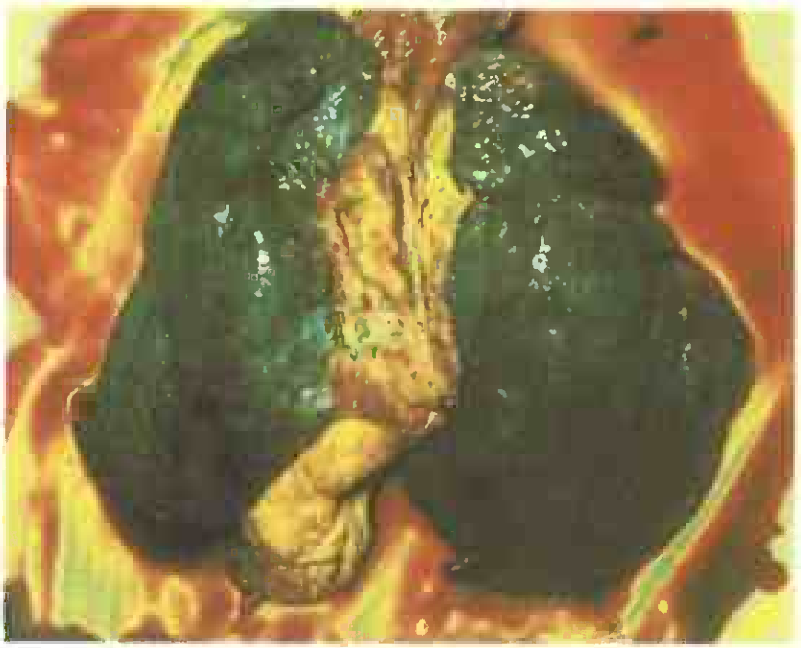

Fig. 1. Posterior aspect of lungs and mediastinum at autopsy showing extensive haemorrhagic lesions of overdistended, oedematous lungs.

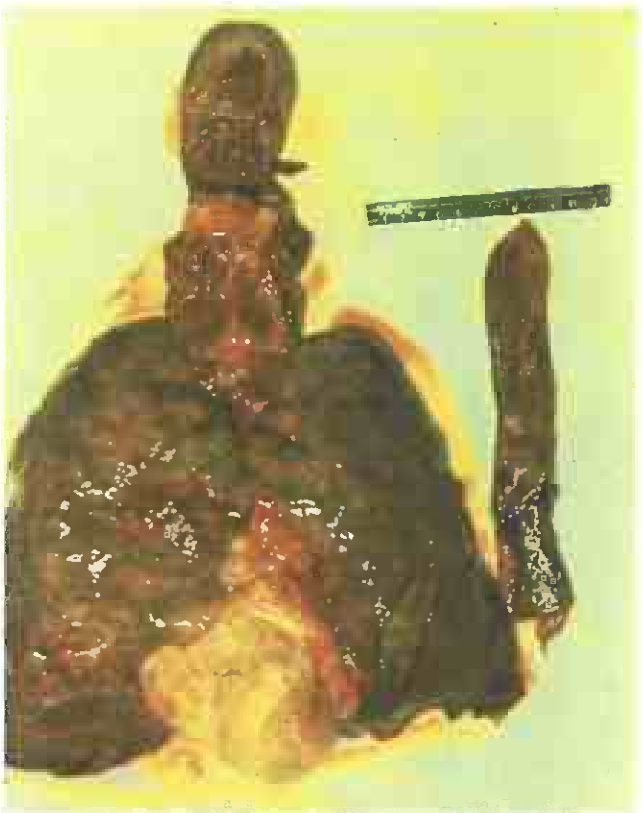

Fig. 2. Anterior aspect of lungs and mediastinum showing uneven distribution of haemorrhagic and hyperaemic areas and staining with haemolysed blood.

Fig. 3. Grossly overdistended lungs. The trachea and bronchi have been opened to show intense hyperaemia of tracheo-bronchial mucosa.

PLATE I

both lungs. Large red areas on posterior aspects of both lungs". (Fig. 1) An autopsy diagnosis of bronchopneumonia was registered.

CASE 2

Lower segment caesarean section was performed on a 22 year-old primigravida with cephalo-pelvic disproportion to terminate a 30 hour trial of labour. During induction of anaesthesia the patient regurgitated stomach contents into her lungs. A cuffed endotracheal tube was inserted and tracheobronchial toilet was carried out. Surgery was completed, a live baby was delivered, the patient recovered from anaesthetic and was returned to the ward.

Two hours later, the patient was found to have severe bronchospasm. She was cyanosed and gasping, with blood-stained froth issuing from her mouth and nose. 100 mg. Hydrocortisone and 0·5 gm. Aminophylline were given intravenously. A cuffed endotracheal tube was inserted, large quantities of pink froth were aspirated from the tube and 100% oxygen was administered via the endotracheal tube. Cyanosis was still present but the colour improved. There was no cardiovascular collapse. Thirty minutes later cardiac arrest occurred. External cardiac massage and I.P.P.V. were instituted. About 5 minutes after commencing cardiac massage the heart started to beat again. The pulse rate was 120 per minute and the systolic blood pressure 90 mm. Hg. The patient was digitalised. Spontaneous respiration began at a rate of 60 per minute but the pupils were dilated and did not respond to light. The patient did not regain consciousness and 15 minutes later started generalised convulsions that were controlled by intravenous thiopentone and intramuscular phenobarbitone. A diagnosis of anoxic cerebral damage was made and 60 minutes after cardiac arrest an unsuccessful attempt was made to induce hypothermia. The patient died hours later with a rectal temperature of 104°F.

The only abnormalities were in the respiratory system. "Pleural cavities contained a small amount of blood-stained fluid. Lungs slaty blue and heavy with multiple subpleural petechiae. Pleura tense. Cut surface firm, dark blue and friable. On squeezing abundant frothy blood-stained fluid exudes."

Diagnosis: Pulmonary oedema following caesarean section.

CASE 3

Lower segment caesarean section was carried out on a 23 year-old primigravida with cephalo-pelvic disproportion to terminate a failed 18 hour trial of labour. Passive regurgitation of fluid stomach contents into the pharynx and aspiration into the lungs occurred during induction of anaesthesia with cyclopropane and oxygen. The patient was paralysed with succinylcholine, a cuffed endotracheal tube was inserted, tracheo-bronchial toilet was performed and anaesthesia was maintained with nitrous oxide and oxygen, intermittent succinylcholine and I.P.P.V. The patient was bronchoscoped and 3 ml. of black fluid were sucked out of the right main bronchus. No solid foreign material was found in the bronchus. Surgery was successfully completed and a live baby was delivered. The patient regained consciousness and was returned to the ward with no apparent ill-effects from the episode of aspiration of stomach contents into the lungs.

Thirty minutes later, the patient had severe bronchospasm and tachycardia. Blood pressure was 120/80 mg. Hg. There was no cyanosis, no mediastinal shift and no lung consolidation.

The patient was treated with intravenous aminophylline, inhalation of 100% oxygen, and pencillin and streptomycin. The attack responded to this treatment and the patient was discharged well on the seventh day.

CASE 4

A 55 year-old man was operated on for intestinal obstruction due to a strangulated left inguinal hernia. There was no pathology in the respiratory system. He was also suffering from neurosyphilis, had aortic incompetence, was grossly dehydrated and severely jaundiced. The abdomen was very distended. A Ryle's tube was passed into the stomach and 2½ pints of black fluid were removed. Anaesthesia was then induced with ether, nitrous oxide and oxygen from a Boyle's machine.

During induction, the patient regurgitated large quantities of black fluid which was aspirated into the lungs. A cuffed endotracheal tube was passed into the trachea, and tracheo-bronchial toilet was carried out. Anaesthesia was then maintained uneventfully with nitrous oxide, oxygen and ether via the endotracheal tube, for over three hours. At the end of the operation, severe bronchospasm developed and the patient had a tachycardia. Intravenous Hydrocortisone

was administered. The patient was left breathing 100% oxygen through the endotracheal tube. Respiratory distress increased during the next 15 hours together with the pulse rate. The patient was digitalised and I.P.P.V. was commenced. Cardiovascular collapse ensued resulting in death.

CASE 5

Death resulted from aspiration of stomach contents into the lungs while this patient was conscious. The patient was a 35 year-old gravida 10 admitted in her 35th week of gestation for treatment of severe pre-eclamptic toxaemia. She was treated for 4 days with rest and sedation, and the drugs administered were:-

Ist 24 hours: Sparine 150 mg.; Luminal 1 gr.; Serpasil 0·25 mg.; Chlortride 1 tablet

2nd 24 hours: Sparine 100 mg.; Luminal 6 gr.; Serpasil 1·0 mg.

3rd 24 hours: Sparine 50 mg.; Luminal 6 gr.; Sodium Amytal 6 gr.; Serpasil 0·5 mg.

4th 24 hours: Sodium amytal 18 gr.

7 hours prior to aspiration: Sodium Amytal 9 grains.

The patient was very drowsy but could be roused sufficiently to have a rice meal. The toxaemia was not improving and surgical induction of labour was attempted. The obstetrician noticed at the end of the procedure that the patient was in a severe "asthmatic" attack and was deeply cyanosed. She was given intravenous Aminophylline and placed in an oxygen tent, but her colour did not improve. The pulse rate rose to 180 per minute and the breathing became more laboured. There was no previous history of asthma. The symptoms developed rapidly to a severity that threatened life. Unlike true asthma which developes slowly, and because the patient had been heavily sedated, an alternative diagnosis of acid aspiration syndrome was proposed. The patient was bronchoscoped and rice grains were removed from the tracheo-bronchial tree. The patient was then intubated, tracheo-bronchial toilet was performed and 100% oxygen was administered. Intravenous Hydrocortisone and digitalis were given.

During the first 30 minutes, cyanosis became less but large quantities of pink froth were being removed from the trachea. I.P.P.V. was commenced and during the following 18 hours, the bronchospasm became less severe, the signs of pulmonary oedema disappeared and the patient delivered a premature foetus. The pulse rate, however, remained high. The patient presented the clinical picture of acute cor pulmonale.

After 18 hours I.P.P.V. was discontinued and the endotracheal tube removed. The respiratory symptoms were mild now but the cardiovascular signs became worse. The blood pressure which was 180/130 mm. Hg. fell to 70/40 mm. Hg. and the patient died 6 hours later.

CASE 6

A healthy 21 year-old gravida 3 was admitted in labour with triplets. General anaesthesia was necessary for forceps delivery and was attempted in the ward. During induction with cyclopropane and oxygen the patient regurgitated large quantities of fluid from the stomach, and some of it was aspirated into the lungs. An endotracheal tube was inserted and tracheo-bronchial toilet performed. On auscultation of the lungs coarse crepitations were audible. The operation was completed and the patient regained consciousness. There were no abnormal symptoms or clinical signs.

Three hours later, the patient developed an "asthmatic" attack, the pulse rate rose to 140 per minute, and the blood pressure fell to 86/70 mm. Hg. Treatment was commenced with intravenous hydrocortisone and penicillin and streptomycin. The patient improved rapidly and was sympton free six hours later. She was discharged five days later, with lungs clinically and radiologically normal.

CASE 7

A 29 year-old gravida 9 had a lower segment caesarean section performed under general anaesthesia. The anaesthetic was uneventful and no regurgitation or vomiting was noticed. She was returned to the ward and remained well on the first post-operative day. Thirty hours after induction of anaesthesia, the patient developed a severe "asthmatic" attack with cyanosis and pink froth emerging from the nose and mouth. A chest X-ray was taken and a diagnosis of acid aspiration syndrome was made. The patient was bronchoscoped, bronchial lavage carried out, and treatment commenced with intravenous hydrocortisone, penicillin and streptomycin. The patient improved rapidly and recovered completely. Irregular mottled shadows seen in the acute phase in X-rays were diminishing by the fifth day and resolved completely after a week. The patient was afebrile throughout.

DISCUSSION

Aspiration of regurgitated gastric contents into the lungs was recognised as the cause of death in 169 out of 1,074 necropsies described by Irons and Apfelbach (1940). They pointed out several important differences between the pathological features in death after aspiration of stomach contents and those following death from bacterial pneumonias. They showed that to establish diagnosis, autopsy should be performed within two hours of death as post-mortem changes progressed unusually rapidly in these cases and obscured the diagnostic features. They also showed that the organisms responsible for death from bacterial pneumonias could be obtained in pure culture from lung tissue and heart blood whereas in deaths from acid aspiration, heart blood was sterile and organisms cultured from lung tissue were identical with organisms cultured from the gastro-intestinal tract of the subject.

Gastric acid instilled experimentally into the trachea of dogs caused a non-fatal hyperaemia, but death occurred if the gastric contents were contaminated with bacteria (Apfelbach and Christianson, 1937). These experiments, however, were incompletely reported. An experimental study on rabbits led Mendelson (1946) to the conclusion that the acidity of the aspirated material caused a fatal disease closely resembling that seen in human beings. This has subsequently been confirmed by Lewinski (1965). Further experimental studies showed that inhalation of semi-digested food particles led to a foreign body reaction in the lungs (Teabeaut, 1952). Lesions due to both causes present a recognisable radiological picture. Lesions due to acid alone resolve in 7-10 days, but lesions from the latter cause persist longer.

This distinct and fatal clinical entity was identified over 26 years ago, but deaths from acid aspiration continue to be labelled as bronchial asthma, suffocation, massive atelectasis, partial atelectasis, disc atelectasis, pulmonary tuberculosis, fungus infection, pulmonary metastasis, drowned lungs, cardiac failure, pulmonary oedema and paroxysmal tachycardia (Mendelson, 1946; present case reports). For this reason, acid aspiration as a cause of death is not recognised as frequently as it occurs and thousands of cases probably go unrecorded, (Bannister and Sattilaro, 1962). Attempts to assay the incidence and prognosis of the disease are therefore inadequate and conclusions drawn from available figures are likely to be misleading unless interpreted with caution.

Culver, Makel and Beecher (1951) showed that aspiration of gastric contents into the lungs occurred in 16·3% of 300 general anaesthesia conducted without an endotracheal tube. No clinical disease was reported in this series. The result of retrospective studies that relate deaths from aspiration of gastric contents to the total number of anaesthestics given are presented in Table I.

The commonest method of describing the nature of the problem is by relating deaths from acid aspiration to total anaesthetic deaths (Table II). Weaver (1964) on the basis of a study in Connecticut (Greene, 1959), estimated that 238 avoidable deaths would occur when 9.5 million anaesthesias were administered. All these methods underestimate the size of the problem and each provides only a limited perspective. Further, the total incidence of the syndrome is unknown and prognosis as determined from case reports in the literature varies greatly (Table III). Irons and Apfelbach (1940) diagnosed aspiration of gastric contents as a cause of death in 169 out of 1,074 autopsies. The Department of Pathology of the Chicago Lying-in Hospital named this as a cause of death in 20% of all autopsies. Four out of seven patients we have treated have died. We therefore, conclude that aspiration of acid stomach contents into the lungs is common and carries a grave prognosis.

It has been shown in experimental animals that instillation of concentrated solutions of Hydrocortisone into the trachea, together with parenteral administration of normal doses of this drug, reduced the severity of the pulmonary lesions produced by entry of hydrochloric acid into the tracheobronchial tree (Lewinski, 1965). However, the immediate cause of death is cardiovascular collapse, and during the development of the disease, there is clinical and electrocardiographic evidence of right heart strain leading to failure. This is probably due to pulmonary hypertension which developes despite maintenance of adequate gas exchange by I.P.P.V. which has been shown to relieve pulmonary hypertension in acute-on-chronic bronchitis (Jones, 1966) and status asthmaticus that threatens life (Ambiavagar and Riding, 1966).

There is no proved cause of vomiting during anaesthesia and it may be associated with the use of any anaesthetic agent or technique. Safety is associated with the skill of the administrator rather than with a particular agent or technique (Report of Anaesthesia Study Committee, New York, 1964).

TABLE I

INCIDENCE OF DEATH FROM ASPIRATION OF STOMACH CONTENTS DURING ANAESTHESIA

Author		Total no. of Anaesth.	Deaths
Dieckmann	1945	46,000	2
Jeffcoate	1953	3,522	3
Hingson	1963	26,810	3

TABLE II

ASPIRATION OF STOMACH CONTENTS AS A CAUSE OF ANAESTHETIC DEATH

Author		Total no. of Deaths	No. due to Aspiration
Gordon	1947	51	18
Merrill & Hingson	1951	309	59
Lock et al	1955	45	13
Edwards et al	1956	598	110
Clifton & Hotten	1963	52	8
Dinnick	1964	400	48
Graff et al	1964	58	15
Hingson	1964	4	3

TABLE III

PROGNOSIS FOLLOWING ACID ASPIRATION

Author	Year	No. of Cases	No. of Deaths
Hall	1940	15	5
Dieckmann	1945	45	2
Hartzell & Minninger	1946	20	0
Mendelson	1946	66	0
Parker	1954	7	5
Hausmann & Lunt	1955	3	0
Marshall & Gordon	1958	10	2
Dines et al	1961	1	0
Johnson	1962	1	0
Berris & Kasler	1965	3	0

Vomiting and passive regurgitation from the stomach have been shown to occur when the stomach is full, the intra-abdominal pressure raised, and the intergrity of the cardio-oesophageal sphincters is disturbed (O'Mullane, 1954). Many methods have been described to reduce the incidence of regurgitation and aspiration during induction of anaesthesia, such as induction with thiopentone in a head-up position, followed by a muscle relaxant and rapid intubation of the trachea (Snow and Nunn, 1959); the use of stomach washouts or apomorphine to empty the stomach prior to induction of anaesthesia (White, 1959); crico-thyroid compression during induction (Sellick, 1961); occlusion of the oesophago-gastric orifice with balloons (Macintosh, 1951); intubation of the conscious subject under local anaesthesia (Wycoff, 1959); and surgery under regional anaesthesia. None of these procedures are foolproof. Further, the acid aspiration syndrome occurs in the absence of general anaesthesia (Waters and Harris, 1940; Clark, 1963). It appears impossible to eliminate aspiration of stomach contents into the lungs as a cause of morbidity and mortality. The methods of treatment available now are inadequate. This disease often occurs in otherwise fit and healthy subjects and as it is usually associated with the induction of general anaesthesia, it is a truly iatrogenic disease. Further, under law, anaesthetists are responsible to their patients and not to surgeons (Wasmuth, 1961). It is therefore imperative that the patho-physiology resulting from aspiration of acid into the lungs should be more closely investigated in the expectation that a more effective therapeutic regime may evolve.

SUMMARY

Seven patients who aspirated acid stomach contents into their lungs are described. The disease resulting from this cause is described as a recognisable clinical syndrome. The features by which this disease may be diagnosed are described, the literature is reviewed and the problems related to prevention and treatment are discussed.

Aspiration of regurgitated stomach contents into the lungs results in mechanical airway obstruction if the regurgitated material is solid, and a recognisable clinical syndrome if the inhaled material is acid gastric secretion (Irons and Apfelbach, 1940; Mendelson, 1946).

This syndrome is a clinical entity resembling bronchial asthma. It differs from asthma in the rapidity with which it progresses to a fatal outcome, the circumstances surrounding its occurrence, and its radiological and pathological peculiarities.

The clinical features, pathology found at autopsy and the treatment used are described in seven patients. The relevant literature is reviewed and the methods of prevention and treatment are discussed.

ACKNOWLEDGEMENT

We are grateful to the Consultant obstetricians and surgeons who allowed us to describe their cases and the Department of Pathology, University of Singapore for performing the autopsies and preparing the illustrations.

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