THE EFFECT OF THE INHALATION OF VOMITUS ON THE LUNGS:
MORBID ANATOMY

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SUMMARY

The effect of the inhalation of vomitus will depend on whether the material is entirely liquid or contains large fragments. In the latter case, the result will be suffocation or, if the obstruction be less complete, massive collapse of a portion of lung accompanied by mediastinal shift. If the vomitus be fluid, there results the asthma-like syndrome named after Mendelson, who first drew attention to it. The irritative effect of the fluid vomitus is due to its hydrochloric acid content, and the acute inflammatory response is due to this chemical irritant. Pathogenic organisms are not usually present in this material, so that resolution occurs in a relatively short time, although artificial respiration may be required to tide the patient over the acute attack. In those few cases where pneumonia has resulted, contamination of the vomit as a result of oral sepsis has been suggested. Attention is also drawn to the importance of laryngeal malfunction in connection with the pulmonary syndrome in the newborn.

The effects of accidental inhalation of vomitus must have been known throughout history, but, following the introduction of anaesthesia with its resultant iatrogenic vomiting, morbid anatomists became more closely involved in the examination of such cases. Early reported cases dealt largely with the results of inhalation of large portions of recently ingested food material producing either suffocation, if large enough, or localized collapse followed by suppurative pneumonia with abscess formation, if a smaller bronchus were blocked. These cases, however, did not differ in their results from the accidental inhalation of teeth or foreign bodies which may have occurred while the laryngeal cough reflex was abolished during anaesthesia or from other causes such as over-indulgence in alcohol.

Mendelson (1946) distinguished between the clinical symptoms produced by the inhalation of liquid material such as fluid stomach contents and that produced by the aspiration of solid food. He described an asthma-like syndrome following the aspiration of liquid vomitus, frequently followed by cardiac embarrassment and pulmonary oedema. He also showed by animal experiment that hydrochloric acid is responsible for the changes which he described. These were bronchiolar spasm with an exudative reaction in the peribronchial tissues leading to disturbance of the pulmonary circulation severe enough to bring about cardiac failure in some cases. It was obvious from the large list of diagnoses that Mendelson cited, which had been made in cases where there had been aspiration of gastric contents, that the condition was not understood at that time and his elucidation of the problem fully deserves the honour that the syndrome should now bear his name.

As far as the morbid anatomist is concerned, the lungs are particularly difficult to examine at autopsy. Many changes occur as agonal phenomena and even more profound disturbances may result from the performance of the autopsy itself. Most of these changes are self-evident, but lead to considerable confusion if not taken into account by the pathologist. Rough handling of the cadaver may result in stomach contents reaching the trachea and main bronchi even before the autopsy is begun, and a well-known artefact is the post-mortem digestion which may occur as a result. When the chest is opened after death or, if there has been a tube in the thoracic cavity during life,
the lungs will partially collapse, giving a quite erroneous appearance in histological examination.

If these difficulties are to be avoided, it is best to close the trachea in the neck by forceps or by tying before the thorax is opened. If this be done, the lungs will remain distended and fixation can then be carried out by perfusion of the blood vessels, if it be desired not to disturb the content of the air passages. If dehydration and embedding be later carried out under reduced pressure, the final picture may approximate to that obtaining before death.

Before turning to a consideration of the abnormal findings following the inhalation of liquid vomitus, it may be as well to consider the normal lung appearances. By courtesy of Mr. G. A. Mason, Thoracic Surgeon of Shotley Bridge Hospital, I have been able to examine many hundreds of biopsies removed from the lingula following operations for mitral stenosis or patent ductus arteriosus. These biopsies were taken after reinflation of the lung at the end of the operation, the reinflation being controlled so that application of a clip, while pushing air into the portion of lung to be excised, did not over-distend it. These portions of lung, averaging 3 x 1 x 1 cm were then allowed to fix in formal saline without being opened. Penetration of the fixative in most cases was satisfactory and histological details were well preserved.

It soon became evident that the appearances differed very greatly from those usually seen in postmortem material and figure 1 illustrates this appearance, which is assumed to represent minimal departure from the normal. It will be noted that the normal lung is a delicate lacy network composed of alveolar walls and that the apparent rupture of these walls really represents the limit of the alveolar ducts which are so perforated with the openings of the alveoli that the spirally arranged, musculo-elastic wall only occasionally appears in a section cut in any one plane. Running diagonally across the field can be seen an interlobular septum with occasionally a small cleft representing a lymphatic channel. It will be noticed that the alveoli are empty and, in particular, are free from cells of any kind. This is the appearance of normal lung and it does not seem to show any abnormality following anaesthesia with controlled respiration for an operation involving opening of the thorax. It indicates that the delicate balance of the pulmonary circulation has been maintained so that the alveolar walls and bronchial passages have been kept moistened without excessive production of fluid leading to oedema. It also indicated that no irritant had acted on the alveolar epithelium to lead to its proliferation.

Structure of alveolar wall.

It is important to understand the structure of the delicate alveolar wall, and a fortunate chance has enabled me to demonstrate this structure. A biopsy specimen from a case of mitral valvular disease was inadvertently placed in normal saline instead of in fixative and, as a result of autolysis, the tissues dissociated, but the material was processed before all structural detail was lost. Figure 2 shows the appearance obtained. It will be noticed that the epithelium of the alveoli has been loosened from the underlying tissue and is represented in profile by a very thin strand of cytoplasm with the nucleus bulging out in the middle.

The other noticeable finding is the presence of cells lying apparently free in the lumen of the alveoli and alveolar ducts. Figure 3 shows at a greater magnification several of the endothelial cells which appear to form a continuous covering to these alveolar walls except where there is an alveolar pore. Between the two layers of epithelium, like the filling in a sandwich, lies the ground substance in which course the alveolar capillaries and elastic fibres. In the normal lung the alveolar epithelium appears thin with few nuclei, and is so inconspicuous as to go unnoticed in ordinary preparations although it has, of course, been demonstrated by the electron microscope.

With regard to the cells apparently lying free in the lumen, a little consideration will show that their position is literally insupportable and that, in the absence of oedema fluid, they must have been detached to float in fixing fluid and that this appearance must be an artefact.

Results of irritation.

Mendelson (1946) showed experimentally that it was the acid content of the vomitus which caused the lungs to be irritated and to react with bronchospasm and patchy areas of haemorrhage and oedema, which is marked around the blood vessels. This experimental finding in rabbits was
FIG. 1
Biopsy from lingula at end of operation for patent ductus arteriosus in a female aged 15. Appearances normal apart for slight overdistension. Note delicacy of structure and absence of desquamated cells.

(H. & E. ×100)

FIG. 2
Biopsy from lingula from a woman aged 29 after operation for mitral stenosis. The specimen was not fixed and the alveolar epithelium has been dislodged.

(H. & E. ×92)
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FIG. 3
Higher power view fig. 2 to show structures of alveolar wall. (H. & E. x410)

associated with cyanosis and laboured breathing accompanied by the appearance of a pink froth at the mouth.

It is evident, then, that the aspiration of vomitus containing hydrochloric acid has a violent irritative action on the bronchial tree, producing bronchial spasm, and that some of the fluid must reach the capillary bronchioles and even the alveoli with the production of an acute inflammatory reaction. The patchy areas of collapse may be due to absorption of air due to the obstruction by spasm of certain of the bronchi, but it must be borne in mind that, as Hoffstaedt (1953) points out in a review of the condition of pulmonary atelectasis, there is a possibility of an endobronchial reflex being the cause of the collapse.

Pulmonary oedema.

Whatever the cause of the areas of atelectasis, there is no doubt that pulmonary oedema is produced. The control of the pulmonary vascular circulation is not well understood and the finding of marked oedema of the lungs at postmortem in cases of sudden death from coronary thrombosis seems to indicate that the condition can occur with great suddenness. It is also found in experimental animals which have been killed by a blow on the head, and the picture is that of filling up of the alveoli while the alveolar ducts remain free. Figure 4 shows the appearance in the lungs of a rabbit killed by an overdose of ether in the course of a few minutes. The peripheral parts of the lung are particularly well supplied by lymphatics and any interference with access to these by thickening of the alveolar walls or septal tissues usually leads to a proneness to attacks of pulmonary oedema. This can be demonstrated in cases of mitral stenosis where the lungs become indurated and stiffened due to increase in the tissues and, although the lymphatics appear to undergo considerable compensatory increase, oedema often supervenes. Figure 5 shows an interlobular septum (compare figure 1) showing slight thickening and prominent lymphatic channels in a biopsy from a case of mitral stenosis.

Another factor to be considered is the great distensibility of the pulmonary vascular bed, especially of the precapillary arterioles which, in expiration, fill with blood and may occupy an amount of space almost as big as an alveolus. Figure 6 shows how greatly distensible these vessels are when filled with an injection of starch granules. These vessels could well be the source of the oedema fluid which appears to accumulate so rapidly if they were to become dilated either as a result of nervous reflex stimulation or in response to an inflammatory irritant.

Response of the alveolar epithelium.

The alveolar epithelium responds to the irritant by becoming thicker and at once more easily visible; indeed, it is often referred to as cubical when seen on section, and this change, which is most marked on the alveolar walls which abut on to the more sturdy tissues of the lung such as the blood vessels, bronchioles, and interlobular septa, is accompanied by an apparent proliferation of nuclei.

It would be unwise here to enter into a discussion as to the origin of the phagocytic cells of the alveolar walls, but there is no doubt that many of the epithelial cells do ingest particulate material
FIG. 4
Showing oedema largely confined to alveoli while the ducts remain free.
(H. & E. X 90)

FIG. 5
Slightly indurated lung from a case of mitral stenosis in a female age 36. Note the prominent septal lymphatics.
(H. & E. X 100)
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FIG. 6
Cat lung photographed through partially crossed Nicols prisms to show distended pre-capillary arterioles following experimental embolism with starch granules. Note relative size of dilated vessels to the alveoli.
(H. & E. ×220)

FIG. 7
Note thickening of alveolar epithelium with increase in nuclei in oedematous lung. Post-mortem specimen from a male aged 40.
(H. & E. ×550)
when in their more active plump state. Figure 7 shows the appearance of alveolar epithelium in this state; it has been slightly dislocated from its underlying alveolar septum.

The histopathology of the pulmonary lesion in Mendelson's syndrome suggests an inflammatory reaction to a chemical irritant acting mainly on the bronchi and bronchioles, with rather less direct action on the alveoli. This inflammatory reaction is evidently reversible and, only in those cases where there is bacterial contamination, will prolonged inflammation ensue. This is probably unlikely considering the nature of the stomach contents, as is borne out by the findings in Mendelson's original series of forty cases which nearly all recovered after an afebrile illness, and of which only four developed pneumonia, one developing a lung abscess.

Aspiration of solid gastric contents.

This complication of anaesthesia leads to complete or partial obstruction of the air passages and, if not cleared, will result in death from suffocation or, if a smaller bronchus be involved, will lead to massive atelectasis and mediastinal shift. The possibility of septic pneumonia is considerable and lung abscess is not uncommon. The possibility of contamination of aspirated material with organisms from the mouth must be borne in mind, as shown by Smith (1927) when he demonstrated the presence of fusiform bacilli in pulmonary abscesses. More recently Schweppe, Knowles and Kane (1961), in a study of lung abscess, showed that severe gingivo-dental disease with advanced oral sepsis was the single most commonly associated disease, being present in almost half of their cases. But apart from possible contamination of the vomitus in this way, it would appear that septic pneumonia is not such a frequent complication as it is in other cases of inhalation of foreign material.

Hiatus hernia.

A recent investigation by Carré (1960) into an unusual susceptibility to pulmonary infection in children with a partial thoracic stomach showed that this was associated with those cases in which night vomiting was a feature. On the other hand, no relationship was shown where the cases vomited during the day. The author puts forward the idea that vomiting at night when still asleep resulted in inhalation of vomitus, and thus in increase in pulmonary lesions which were found radiographically to be most frequent in the right middle lobe. Although this report might indicate that the lesions were infective in origin, the author gives only radiological findings in support and speaks of them as segmental. He does not record whether the episodes were accompanied by fever. It seems likely, however, that patients, especially very young children, with a partial thoracic stomach who vomit when semi-conscious, will aspirate vomitus producing a chemical inflammatory lesion. It would be of interest to know whether these episodes resolve each time or that if eventually a fibrosis of the lung would result.

Aspiration in the newborn.

Osborn and Flett (1962) have again emphasized the importance of laryngeal malfunction as a cause of the pulmonary syndrome of the newborn, and have produced convincing evidence that laryngeal spasm is an all-important factor. This is evidenced by the finding of ulceration of a pressure type on the "true" and "false" cords.

They also point out that partial asphyxiation of the foetus before birth will allow both inhalation and swallowing of the content of the upper air passages, but that the infant will still receive an oxygen supply via the umbilical cord so that it will not so readily drown in its own secretions. However, the accumulation of aspirated material will interfere with the establishment of normal respiration. They conclude that assessment of laryngeal function is of prime importance in all these cases and indicate a line of treatment with an "artificial larynx" after clearance of the airways.

REFERENCES


