air-raid warden, who told me that several of the dead found by his rescue party had been suffocated by dust—the mouth, nose, and throat being completely blocked, while no other injuries were visible.

E. Oil.—The last physical agent we encountered was oil, and the only problem was to clean the victim up in the midst of a heavy rush of casualties. The gas-decontamination chamber provided a satisfactory answer.

TABLE VI.—Non-surgical (132 Cases)

Description	No.	Remarks	Deaths
Chronic sick transferred from other hospitals	32	4 T	
Crippled and infirm	15		
Psychological shock	39		
Surgical shock	28		
Dust:			
Corneal abrasions	17	Eyes affected as well	1
Respiratory tract	4		
Oil	1		

Conclusion

An attempt is made to review 259 air-raid casualties admitted to an E.M.S. hospital in the recent flying-bomb attacks and to analyse the types of injuries and the agents which caused them. I include three further tables to aid the analysis.

TABLE VII.—Eye Injuries (52 Cases)

Agent	No. and Description	Deaths
Glass	20 eyes with perforating wounds 1 conjunctival abrasion. (2 T)	1
Dust*	17 corneal abrasions and conjunctivitis	
Blast	1 traumatic iritis 1 ruptured iris 2 contusions 3 subconjunctival haemorrhage 4 hyphaemia	
Bomb splinter	1 perforating wound of globe	
Fear	2 cases of acute dilatation of the pupils, which did not contract to light	

* It is probable that many of these lesions were due to blast and that the presence of dust was coincidental. In several cases the corneal and scleral epithelium had been wiped off, leaving a denuded circular area of varying diameter, the largest about half an inch.

TABLE VIII.—Fractures (39 Cases)

Agent	No. and Description	Deaths
Masonry	23 (mostly severe fractures), 1 T	3
Glass	4 minor fractures	1
Bomb fragments	5 severe 3 minor	
Cause unknown	4	

TABLE IX.—Head Injuries (23 Cases)

Agent	No. and Description	Deaths
Masonry	7 cranial fractures 1 cerebral irritation (blood in C.S.F.) 1 unconscious	6
Blast	1 paralysis of left arm 1 partial paralysis of left arm 1 mental confusion 3 contusions 4 concussions	
Cause unknown	1 unconscious	
Bomb splinter	1 contusion 1 concussed (4 hours' retrograde amnesia) 1 lodged in frontal lobe (now convalescent)	1

Guidance to local authorities in England and Wales as to the lines on which proposals to provide temporary housing should be framed is given in a memorandum entitled *Temporary Accommodation* by the Ministry of Health and the Ministry of Works (H.M. Stationery Office; 6d.). This matter, it says, should not be considered in isolation but in relation to the whole housing programme of the authority. The use of temporary accommodation will, the Government believes, make it possible approximately to double the number of dwellings which could otherwise be provided with the limited amount of skilled labour available in the first year after building can be resumed. The temporary scheme must not be allowed to stand in the way of the permanent scheme, and it will be for each authority to whom temporary houses are allotted to ensure that both sections of its programme can proceed side by side without stultifying one another. Temporary houses will be provided and owned by the Government; they will be erected on sites acquired and developed by the local authority.

MASSIVE SURGICAL EMPHYSEMA DURING THE COURSE OF GENERAL ANAESTHESIA

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Surgical emphysema occurring during the course of endotracheal anaesthesia has been the subject of several reports in the literature, and much experimental work has been carried out to ascertain the cause of the phenomenon. The first mention of the subject was made in 1912 by Woolsey, who reported one case of surgical emphysema. Luke in 1913 recorded a case of gross emphysema due to obstruction of the return airway, and Georg in 1917 suggested that surgical emphysema was likely to occur if the tracheal mucosa had been traumatized during intubation. He also demonstrated the occurrence of emphysema in dogs after the use of insufflation pressures of 25 to 33 mm. Hg. Marcotte *et al.* (1940) found that an intrabronchial pressure of 24 mm. Hg usually produced mediastinal emphysema in dogs, and had seen it occur after 18 mm. In cats, lower pressures of 16 to 20 mm. Hg were sufficient to produce emphysema. Macklin (1937), by the use of very high intrabronchial pressures in experimental animals, has demonstrated exactly how such emphysema could occur in the lungs and mediastinum. He showed that air could break through in small bubbles from overdistended alveoli to the perivascular connective tissue and could track along the course of the arterioles to the great vessels.

We would like to submit the histories of two cases of massive surgical emphysema that have occurred during endotracheal anaesthesia.

Case I

A girl aged 9½ years was admitted to hospital with a diagnosis of acute appendicitis. The illness had started with generalized abdominal pain about 52 hours previously, and for the last 12 hours this had been localized to the right iliac fossa. The child had vomited on several occasions in the early stage of the illness, when a mild gastro-intestinal upset was considered probable: a dose of castor oil had been administered by the doctor in charge of the case, and the pain subsequently became localized to the right iliac fossa. When she was brought to hospital the signs and symptoms all pointed to a diagnosis of acute appendicitis, with some evidence of local peritonitis. The tongue was dry and dirty; the temperature was 101.4° F., and the pulse varied from 140 to 160 a minute. The only other abnormal physical sign was a loud systolic murmur, which was conducted well into the axilla. The heart was not enlarged. An immediate operation was advised.

Operation.—A preliminary injection of atropine sulphate gr. 1/150 was given and anaesthesia induced with gas, oxygen, and ether by means of a Clover's apparatus. The abdomen was opened by a modified Battle's incision and a large gangrenous appendix was removed without difficulty. The inflammation appeared to be well localized to the right iliac fossa. As the abdomen was being closed convulsive twitchings developed which rapidly became generalized. In an effort to deal with the situation the Clover's apparatus was discarded, and an intratracheal tube was passed with a laryngoscope, and through this tube oxygen containing a small amount of CO₂ was liberally administered. Almost immediately the anaesthetist noticed surgical emphysema of the eyelids. The condition spread with great rapidity over the face, neck, arms, chest, and abdomen. The oxygen was shut off at once and artificial respiration started, as the condition of the patient was now desperate. The convulsions continued, and death occurred within about five minutes of the first seizure. At the time of death the patient was quite unrecognizable as a human being. The features were so completely changed by massive emphysema that the eyes could scarcely be distinguished from the nose and mouth: the neck merged directly into the chin. The appearance was reminiscent of a toy balloon made to represent some animal which had been overdistended. The emphysema extended from the head to the legs. In order to improve these unpleasant appearances an attempt, after death, was made, on the operating table, to deflate the tissues by means of multiple small incisions down to the deep fascia, but no air could be expelled.

At the necropsy a tiny abrasion of the pharyngeal mucosa was discovered. The emphysema involved the glottis as well as the whole pharynx, the mediastinum, and the subcutaneous tissues. It was

evident that, apart from the convulsions, which in this case were probably the actual cause of death, the patient had suffered from acute laryngeal obstruction.

The striking features of this case, which occurred in 1934, were the speed with which the emphysema developed and the fact that by the time it was first noticed it was already too late to do much about it, and that the emphysema appeared after the introduction of an endotracheal tube. The whole operation was completed in less than 20 minutes, and the anaesthetic was administered by an experienced anaesthetist.

Case II

A coloured boy aged 11 years was admitted to hospital with a diagnosis of "bronchiectasis—for investigation," with a view to surgical treatment of the condition. He had suffered from a cough with foul sputum for some years as well as frequent colds of long duration, and two febrile attacks of more severity which were thought to be bronchopneumonia. On admission he was of average size for his age, with early clubbing of the fingers. Clinical examination of the chest revealed showers of rales over the right lung and occasional rhonchi. He expectorated daily 4 oz. of foul-smelling sputum. The plain radiograph suggested a cystic bronchiectasis in three lobes of the right lung.

In view of the foul sputum, postural drainage was carried out for several weeks, before attempting bronchography, in an endeavour to clear the bronchial tree. When it was finally decided to carry out the examination the sputum had been reduced to 1 oz. daily. His age and temperament were considered to be unsuitable for successful bronchography under local analgesia, and so a general anaesthetic was given.

On Feb. 22, 1944, he was given morphine gr. 1/12 and atropine sulphate gr. 1/100 as premedication. The child was then anaesthetized with CHCl₃ and oxygen. The induction was not uneventful—sputum proving troublesome and cyanosis difficult to eliminate. A Magill endotracheal tube was therefore passed under direct vision and the anaesthetic continued. Cyanosis was still prominent, and on one occasion the pulse faded; oxygen was liberally administered in an attempt to combat this. About 15 minutes after the induction the anaesthetist considered the patient to be more or less settled, and as the cyanosis had improved he felt that the bronchogram could now be made. It was, however, plain that the condition of the patient was not altogether satisfactory. It had been our recent custom in small children receiving a general anaesthetic for bronchography to inject the lipiodol through a fine Monaldi catheter inserted into the lumen of the endotracheal tube. This catheter was so small that, while allowing a flow of lipiodol, it encroached but little upon the airway. This method has not caused us any anxiety. At the moment when the oil was about to be instilled it was noticed that the child's neck was grossly deformed, there being complete loss of the angle between the chin and the neck, and that the neck was alarmingly ballooned right down to the clavicles. Palpation suggested deep emphysema, little or no subcutaneous emphysema being observed. The child was again cyanotic; the CHCl₃ was discontinued at once and the oxygen flow increased. A serum needle, which was fortunately lying on the bronchography trolley, was inserted into the left side of the neck about two fingerbreadths above the clavicle. No diminution in the size of the neck occurred, and the child's condition appeared to be critical. A 20-c.cm. Record syringe was then applied to the needle, but the absolute resistance of the plunger to withdrawal suggested that the needle-point had not reached an air-containing fascial space. The needle was then inserted more deeply until the plunger could be withdrawn with ease. Aspiration was started, the thumb being placed over the hilt on disconnecting the syringe. During the first minute or so there did not seem to be an appreciable reduction in size, but as the plunger's easy withdrawal suggested the continual aspiration of air, the process was continued and the swelling began to recede. At this point a two-way adapter had been procured: the reduction was then more rapid and the aspiration on a better basis. When the contour of the neck seemed almost normal and the condition of the patient much improved, the syringe was disconnected, but the needle was left *in situ*. Within a few seconds the neck ballooned up once again. The process of aspiration was repeated, and the neck returned to reasonable dimensions once more. On disconnecting the syringe a further ballooning occurred. Again aspiration was performed for a period of about 15 minutes, and on this occasion on disconnecting the syringe ballooning did not recur. The oxygen administered in this last 15-minute period was appreciably reduced in amount. It has been estimated that, in all, aspiration was carried out for a full half-hour. While waiting to ensure that no further gross emphysema occurred a radiograph was taken, as the child had been anaesthetized in the x-ray room. The radiograph shows the needle *in situ* and emphysema to be present in the neck—although the neck appeared normal to palpation.

The child made an uneventful recovery from this operation with no further treatment; but a full course of sulphathiazole was given

as a precaution against infection of the fascial planes in view of the foul expectoration.

On examination of the anaesthetic apparatus it was apparent that the expiratory valve was not working at the maximum of efficiency and that the pressure of gases in the pharynx must have been unduly high. The anaesthetic was administered by an anaesthetist of skill and experience in the procedure.

Summary and Conclusions

Two cases of massive surgical emphysema which occurred during the administration of general anaesthesia are recorded. The common features are: the rapid onset of the condition; the alarming rate of spread; and that in both cases oxygen was liberally administered to combat an emergency—convulsions in one and cyanosis in the other. It is suggested that incision or the mere insertion of needles into the superficial tissues is not sufficient to control the emphysema, and that aspiration is necessary; that if the cause remains the emphysema returns with alarming rapidity when aspiration of air is discontinued. Finally, if aspiration is attempted the needle may have to be passed through the deep fascia of the neck.

In both these cases it is probable that the condition was caused by a minute perforation of the pharyngeal mucosa, through which gas, under pressure, was forced into the fascial planes of the neck and mediastinum. It is therefore conceivable that, once such a process has been started, it might continue, even after the primary cause has been removed, if the patient develops an obstruction to the airway and struggles for breath.

REFERENCES

- Georg, C. (1917). *Amer. J. Surg. (Anaes. Supp.)*, **31**, 77.
 Luke, H. C. (1913). *Surg. Gynec. Obstet.*, **16**, 204.
 Macklin, C. C. (1937). *Canad. med. Ass. J.*, **36**, 414.
 Marcotte, R. J., Phillips, F. J., Adams, W. E., and Livingstone, H. (1940). *J. thorac. Surg.*, **9**, 346.
 Woolsey, W. C. (1912). *N.Y. St. J. Med.*, **15**, 93.

AN UNUSUAL CASE OF GENERALIZED TUBERCULOSIS

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The following case of generalized tuberculosis seems to us to be well worth recording.

Case History

An American seaman aged 26 was suddenly taken ill on Nov. 22, 1943, complaining of headache and chills. He ran a continuous fever from the onset to his death in March, 1944. During the course of the illness, which was difficult to diagnose, he developed erythema nodosum. The interest of the case is in the x-ray and post-mortem findings, which are as follows:

Summary of X-Ray Reports.—23/12/43:—Marked increase in left hilar shadow, continuous with a fan-shaped opacity spreading into left mid-lung zone; consistent with a localized area of consolidation and associated glandular enlargement. 30/12/43:—No appreciable change. 14/1/44:—Increase in size of opacity in left hilum and mid-lung zone; consistent with tuberculous infiltration and glandular enlargement of the type usually seen in children. 20/1/44:—Increase in the spread. 9/2/44:—Increase in size and density, associated with widening of the mediastinum. The possibility of lymphadenoma or neoplasm, with secondary lung involvement, cannot be excluded. 21/2/44:—Slight increase of the glandular enlargement to the right of the midline.

Post-mortem Examination.—**Lungs:**—There was approximately half a pint of deep-yellow fluid in the left pleural cavity. There were adhesions at the base on the left side, and a large breaking-down caseous peribronchial lymph gland in the left side of the mediastinum approximately the size of a tangerine. The lower lobe of the left lung was solid, very little caseation being apparent. There were some scattered tubercles throughout the rest of the left lung and also the right lung. The condition in the left lower lobe seemed to be a direct spread from the affected peribronchial gland. Trachea and bronchi were not involved in the disease process. **Heart:**—There was a large amount of fibrinous pericarditis. Numerous tubercles (up to 1/4 in. in diameter) were present in the pericardium: these did not extend into the myocardium. However, a solitary tubercle (approximately 1/2 in. in diameter) was found in the myocardium of the right ventricle. There was healthy myocardium between this tubercle and the pericardium. The valves were normal