are capable of rapid invasion and often fatal infections typified by well-recognized entities such as meningitis and bacterial endocarditis. Its possible role in producing disease of the respiratory tract is less well defined, and in view of its normal habitat it is surprising that it is not more often encountered as the sole pathogenic agent. One of the difficulties may be due to failure to distinguish between pathogenic and non-pathogenic strains, and between primary and secondary invaders. Pittman (1931) has, however, demonstrated that pathogenic strains are usually capsulated and that the capsules have a specific soluble antigen which renders them capable of differentiation into 6 serological types (A to F). The majority of strains isolated from "influenzal" meningitis are Type B, and Sinclair has shown that the strains recovered from cases of laryngitis with septicaemia also belong to this type. The latter author collected 10 such cases, 8 of them in 18 months, and suggests that the number is increasing.

The clinical picture of severe prostration and acute respiratory distress of abrupt onset, often necessitating tracheotomy, differs from that of laryngeal diphtheria in that the prostration is undiminished by tracheotomy or anti-diphtheritic serum therapy. The disease may run a rapidly fatal course in from 18 hours up to 6 days. Four of Sinclair's 10 patients died, those who recovered having received sulph-anilamide, sulphapyridine, or sulphathiazole.

The diagnosis is essentially a bacteriological one, depending on the isolation of *H. influenzae* by culture from the nasopharynx, larynx, lung, or blood, or by direct typing of nasopharyngeal mucus obtained by swabbing with type-specific serum—the Neufeld technique used by Alexander, Craig, Shirley, and Ellis (1941). Since typing sera are not yet available in this country, diagnosis largely depends on isolation by culture; but uncertainty will arise unless serological typing confirms the strain—otherwise the diagnosis can only be presumptive.

The present case affords an opportunity of discussing the pathology. The organism was first isolated from the throat on the third day of the disease and from the larynx on the fourth, where it had probably been present from the onset of respiratory distress. Evidence of pulmonary disease was first detected on the fifth day, and necropsy proved it to be due to empyema, no inflammatory condition of the lung parenchyma having been found. Post-mortem blood culture was also positive, and judging from 7 of Sinclair's cases in which the organism was recovered from the blood ante mortem, the blood in the present case was probably invaded in the early stages, thus emphasizing the highly invasive property of this particular strain of *H. influenzae*—not unexpected in view of its possessing a capsule analogous to that of type-specific pneumococci. The route taken by the organism was probably the lung—a probability that is suggested by the bilateral empyemata and pericarditis, in which the exudate in both the pleurae and the pericardium was too abundant to have formed as a terminal event secondary to the septicaemia. The absence of any inflammatory disease of the upper respiratory tract at necropsy shows that the initial lesion may resolve and that the fatal outcome in those cases which survive the first few days is due to purulent inflammation of the lungs, pleurae, or pericardium, and septicaemia, or, when the former are absent, to septicaemia alone.

Summary and Conclusion

A case of acute laryngitis and septicaemia due to *H. influenzae* is described.

The strain of haemophilus isolated from the larynx and blood belongs to the well-recognized pathogenic Type B (Pittman).

The diagnosis depends on isolation of the organism from the nasopharynx and blood, employing a suitable medium such as boiled blood agar or by direct typing of nasopharyngeal mucus when typing sera are available.

The pathology of the condition is discussed briefly.

I am grateful to Mr. W. H. Clark, who was responsible for isolating the organism and for its typing.

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ATYPICAL ASPHYXIAL DEATHS

BY

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The remarks of Dr. Eric Gardner in his paper to the Medico-Legal Society described in the *Journal* of April 18, 1942 (p. 502), under "Mechanism of Certain Sudden Deaths," are timely not only from the point of view of correct inquest findings but also in instituting prompt skilful treatment if there is still a chance of life. Dr. Gardner's remarks have been read by the staff of this mental hospital with considerable interest and with a certain degree of relief. Within the past two months it has been my unfortunate lot to have lost three female patients through asphyxiation. The superintendent tells me that within his eleven years' experience here he cannot recall any deaths with similar surrounding clinical and post-mortem evidence.

Case Reports

Case 1.—A woman aged 36 fell on a tiled floor. She was rendered unconscious and was treated for a fractured skull. She regained consciousness within 4 hours, improving rapidly, and was left by the nurse in charge for a few minutes while she attended to duties in the day-room. On her return the patient was dead. It being thought that this sudden catastrophe was inconsistent with the intracranial injury, a necropsy was performed, revealing that the pharynx contained a huge mass of bread impacted above the epiglottis. The bronchial tubes and lungs also contained pieces of inhaled bread. The base of the skull was fractured, but without sufficient trauma or haemorrhage to cause sudden death. The bread could have been given to the patient only by some of her well-meaning friends.

Case 2.—A woman aged 44. This patient, at the breakfast table, devoured her own share of bread-and-butter and, while standing up and about to snatch that of her neighbour, was seen by the nursing staff to slump down into her seat and collapse. Attempts to recover foreign matter from the mouth were unsuccessful, and she died within 5 minutes. The necropsy revealed bread tightly packed in the pharynx, trachea, and bronchi.

Case 3.—A woman aged 65 was noticed by the nursing staff to turn pale and collapse in her seat while eating bread at the breakfast table. Her air-passages were cleared so far as was possible, but she died within 7 minutes. The post-mortem examination revealed soft bread scattered throughout the larynx, trachea, and bronchial tree.

Commentary

The victims were female patients of a mental hospital, and in Cases 2 and 3 were known to be food-snatchers who, not being content with their own ration of bread, would grab at their neighbours' and cram it into the mouth without mastication. The usual signs of choking were absent in the two observed cases: no lividity, cyanosis, coughing, struggling for breath, or convulsive movements.

A feature common to all three cases was a pallid, peaceful facies. The external post-mortem signs of asphyxiation, such as livid patches on the body and extremities, were absent.

The absence of the normal reflexes, through the medulla, of coughing or retching is remarkable in the light of the pathological findings of impaction in the region of the most sensitive receptors for producing them—viz., the larynx, bronchi, and trachea. Considered with the time factor of 5 and 7 minutes in the two observed cases, it is clear that the mode of death could not have been true asphyxia.

Death appears to have been due to syncope set up by shock—the result of a mass reflex through the impaction of foreign matter in the pharynx, larynx, trachea, and bronchial tree, death taking place before the usual signs and symptoms of asphyxiation had become manifest.

I am indebted to Dr. D. N. Parfitt, medical superintendent, for permission to record these cases, to Dr. J. L. Berry for the post-mortem examinations, and to both for their encouragement in my presentation of this paper.