AN EPIDEMIC OF ACUTE LARYNGO-TRACHEO-BRONCHITIS

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LATE in December, 1944, acute laryngo-tracheo-bronchitis became prevalent in Ottawa. It rapidly reached epidemic proportions, persisted unabated for 2½ months, then gradually burned itself out. Nothing like it had ever been experienced in the recollection of practising physicians. It was critical enough to receive considerable attention from the local press, which publicity only increased the apprehension of Ottawa mothers.

The first three cases admitted to the Ottawa Civic Hospital died. These infants were all seen early. The first, aged 20 months, was admitted with considerable respiratory distress, eight hours after onset of symptoms. He died suddenly 24 hours later, while being prepared for tracheotomy. He had received 30 grains of soluthiazole on admission and then 15 grains every 4 hours. Penicillin was started when his condition did not improve, but only 30,000 units were administered before death occurred. He was given hypertonic plasma and intravenous glucose. His temperature rose to 107 degrees and death seemed to be due as much to toxæmia as to asphyxia.

The second and third cases, aged 2 years and 1½ years respectively, were admitted three days following onset, having shown no response to adequate sulfonamide therapy at home. On admission, penicillin was administered, 25,000 units every 3 hours to the first, and 15,000 units every 3 hours to the second. Sulfonamides were continued in both instances. Tracheotomy tubes were inserted on admission in both cases. These tubes were changed frequently, the trachea aspirated by suction as necessary, usually with a catheter, but occasionally through a bronchoscope. Penicillin was instilled into the trachea frequently. Despite all these procedures, the infants died, the first 5 days and the second 2 days after admission.

This experience with our first three cases demanded our immediate action. A meeting of the Pædiatric and Oto-laryngological departments was held, to which were invited the bacteriologists of the Laboratory of Hygiene (including the virus division) of the Department of National Health and Welfare. The object of this meeting was to draft a plan for investigating the etiology of acute laryngo-tracheo-bronchitis, and also to revise our treatment.

We all were of the opinion that a virus origin was at least possible. This conclusion was based on two facts. First, there were colds present in other members of the family in most cases that had come under our care, and yet almost invariably, the child with acute laryngo-tracheitis had had a sudden onset of croup with no preceding rhinitis. This seemed to suggest that the acute laryngo-tracheitis was a primary condition, and not due to a secondary invading organism. Second, the poor response to both sulfonamides and penicillin, even in large doses, supported a virus rather than a bacterial etiology.

It was decided, therefore, to investigate subsequent cases for virus infection. Tracheal secretions taken at the time of tracheotomy, and post-nasal secretions from other patients obtained through a West tube, within 24 hours of onset of symptoms, were instilled intranasally into the usual laboratory animals. Due to lack of facilities, egg and ferret inoculations were omitted. For the same reason, *in vivo* and *in vitro* tests for the determination of changes in antibody level due to influenza A. and B. viruses were not attempted. However, 17 specimens of secretions failed to reveal the presence of a virus. This, while disappointing, does not entirely disprove our theory of a virus origin.

Unfortunately, further investigation received very little attention, partially due to a lack of hospital laboratory personnel. Tracheal secretions were cultured in four cases. Two were positive for *Staph. aureus* and non-hæmolytic strep.: one for *Staph. albus*, and one for hæmolytic *Staph. aureus*. White blood cell counts were recorded in 17 children. Ten were over 10,000, three of which showed definite evidence of secondary infections (otitis media and pneumonia). Seven had counts under 10,000 ranging as low as 2,800. Chest x-rays were taken in 8 cases. One showed some irregular congestion, and another a definite pneumonia. The remaining six, including one of those that died, were essentially negative. No further deaths occurred in our series of 103 cases.

As to treatment, the following plan was evolved and put into immediate effect. Enough humidifiers of the fan and wick type were installed in two four-bed wards to produce a cool

atmosphere with a humidity of at least 75%. All cases, private and public, were admitted to these wards.

Teams of special nurses were trained in post-tracheotomy care and placed in these rooms. The expense of these nurses was borne partly by the private patients and the rest by the hospital, so that at all times there were at least two nurses, usually three, and occasionally four, in each four-bed room. Group nursing has not been adopted in Ottawa. However, in treating these seriously-ill youngsters, it was found that during periods of extreme restlessness, associated with respiratory distress, it was often impossible for one nurse to restrain the child, change and aspirate tubes, and call for a doctor, all at one time, so that team work was essential for the efficient care of the patient. Also, when the handling of these sudden, and at times alarming emergencies was the responsibility of one nurse, her apprehension and concern was invariably reflected in her behaviour. It was too difficult for her to maintain that outward calm which is so essential in reassuring and lessening the panic in a child struggling for air. And so it was noticed that with a team of nurses in the room, there was a definite improvement in morale on the part of both nurses and patients.

Coupled in importance with the nursing care was the service of the oto-laryngologists. Tracheotomies and the maintenance of an adequate airway were their responsibility. The insertion of a tube into the trachea was usually delayed until other therapy had failed. The general indications necessitating this procedure were: persistent cyanosis or ashy pallor; marked respiratory difficulty—so-called laryngeal dyspnœa; evidence of fatigue. Although a large number of the cases were potential tracheotomies, it was found necessary to put tubes into only 11 of the 103 children admitted to hospital. The age of these varied from 7 months to 7 years. The last tracheotomy was in the 49th admission, evidence of a diminution in virulence as the epidemic progressed. It was felt that one of our fatalities could have been averted if an oto-laryngologist had been present at the time the respiratory obstruction caused death. With this in mind, it was decided that, when alarming cases were in the hospital, some member of the nose and throat department should be in constant attendance. An adjacent room was assigned to this department, and one of their staff remained in residence during critical periods. To this routine, we credited a great part of our success in reducing the mortality rate.

Despite frequent changing of tubes (every one to three hours at times) and aspiration with suction, bronchoscopic removal of tenacious secretions was found necessary in most cases to keep open a satisfactory air-way: and at times, this was an emergency procedure. Trays with laryngoscopes, bronchoscopes, bronchoscopic forceps, emergency tracheotomy sets, duplicate tubes, etc., were maintained in the room constantly, as were portable suction machines and oxygen tanks. Following tracheotomies, the patients were usually kept flat. Postural drainage seemed impractical and valueless. Inner tubes were changed every one to three hours, and outer tubes every 12 to 24 hours. Interns and nurses were trained to change outer tubes in emergencies. Tubes were permanently removed as soon as possible, usually between the third and eighth days. In an effort to soften the tracheal and bronchial secretions, saline or 5% soda bicarbonate was instilled frequently through the tube and then aspirated. Penicillin was instilled in the same way in most cases, but no apparent improvement could be attributed to its use.

Penicillin was given intramuscularly to 12 of the 103 cases, including the first three that died, the dose varying from 80,000 to 200,000 units daily. It was our impression that it had very little effect on the course of the illness. All but 11 children received sulfonamides, including all those that were having penicillin. The 11 cases that received no therapy other than cool humidity and nursing care compared favourably with the others, both in duration and severity of the disease.

All those not immunized for diphtheria, and three that were, received 20,000 units of antitoxin, without apparent benefit. Hypertonic plasma was used occasionally, but opinions differed on its efficacy in reducing the dyspnœa. Sedatives were administered sparingly for restlessness, when not associated with too much respiratory distress. Administration of fluids was considered essential, and an adequate intake was maintained by mouth if possible, or parenterally if necessary.

Other treatments which seemed to have little, if any, merit were bismuth suppositories, cough mixtures (expectorant and sedative, and including calcidin), potassium iodide, ice collars and

synapisms. All treatments and medication were grouped to disturb the patient as infrequently as possible.

In evaluating treatment, it seemed to us that the nursing and oto-laryngological services were of first importance; that cool air with a humidity of 75% was particularly beneficial; that adequate fluid intake was important; that hypertonic plasma was of doubtful value; and that penicillin and sulfonamides, while indicated because of the seriousness of the disease, were very disappointing in altering the course.

NASAL MEDICATION IN SINUSITIS*

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IN the light of recent knowledge, nasal medication in upper respiratory infections has undergone a change in the past few years. Greater interest has been stimulated in this subject due in great measure to the contributions of Proetz,[1] Hilding,[2] Lierle and Moore,[3] Walsh and Cannon.[4] When Proetz introduced his displacement method in 1926, the diagnosis and treatment of sinusitis was placed on an entirely new level. He showed how simply fluid could enter the sinuses by posture and intermittent suction. A few years later, Hilding demonstrated the direction and flow of mucus through the nose and its relation to ciliary activity.

Following these important advances Lierle and Moore pointed out that ephedrine, up to 3% in normal saline caused no damage to the cilia or to the epithelium of the nasal mucosa. They also demonstrated the harmful effects on the mucous membrane of nearly all the drugs used in sprays and droppers, including adrenalin, cocaine, silver salts and volatile oils. Even tap water and distilled water caused a definite slowing of the ciliary beat when applied to the mucous membrane of the upper respiratory tract. Seven years ago, Walsh and Cannon drew attention to the fact that many nasal drops or sprays caused permanent damage to lung tissue. They also showed that on aspiration silver salt preparations were more harmful to bronchial epithelium than mineral oil. On the nasal mucous membrane they also have an irritating effect in all probability due to their high alkaline pH value. Although 10% silver solutions do not appear to injure the cilia of the nose, they tend to upset the normal flow of mucus. This is thought to be due to the clumping effect of the precipitated silver protein which interferes with ciliary movement.

Mineral oil preparations containing menthol, eucalyptol and camphor have been prescribed by the medical profession for years in the belief that they clear the head. This was disproved by Fox[5] who showed that solutions as weak as 1% produce a swelling of the mucous membrane which reduces the volume of air through the nose to about one-half. Although there is a sensation of increased patency, this is not actually the case. Since it has been shown that oily solutions containing 5% menthol, eucalyptol and camphor sprayed into the nose daily have a definitely destructive effect on nasal mucosa,[6] their popularity has waned.

Liquid paraffin as a base for nasal medication does not actually reach the cilia but remains on the surface of the layer of mucus.[7] Although it does not cause any chemical harm, oil does not mix with mucus but lies as a heavy blanket on the mucous membrane and by its weight alone interferes with ciliary movement. The danger of pneumonia following the instillation of oily drops in children has also become recognized.

In view of these findings the trend in recent years has been toward nasal medication which does not disturb the normal physiology of the nose. One of the most important physio-chemical properties of nasal secretion appears to be its hydrogen ion concentrations. Eleven years ago Tweedie[8] attempted to show the relation of the bacterial flora of the nose to the pH value of nasal mucus. He found that in all patients in whom the pH value was 6.5 or below, that is slightly acid, cultures from the nose were negative. In the presence of inflammation when the pH was raised to the alkaline side, that is, above pH 7, organisms were usually found. From this he concludes that an acid reaction is inhibitory to the growth of pathogenic bacteria. According to Negus,[9] the beat of nasal cilia is

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Read in part at the Oto-laryngological Meeting of the Medical Chirurgical Society, Montreal, February 7, 1945.