The Management of Acute Croup*

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Croup is an inflammatory condition of the larynx or trachea accompanied by stridor, which is the harsh noise characterizing upper airway obstruction, as opposed to wheezing which characterizes lower airway obstruction. The sound may be of high or low pitch and is due to sound waves set up by the fast flow of air past a narrow point in the airway. This causes stridor of medium or high pitch, chiefly during inspiration. Stridor may however, be of low pitch and rattling in quality when surrounding tissue structures are set into oscillatory motion. This applies particularly to the epiglottis and aryepiglottic folds. Stridor arises primarily from the larynx but may be caused at any point between the pharynx and upper trachea (Fig. 1). The anatomy of this region is crucial to the position and nature of the obstruction, which varies according to the main site of impact of the inflammatory lesion. Anatomy is also important in that there may be underlying structural abnormality, which is discovered by superimposed inflammation and modifies the subsequent course of the disease and its management.

Anatomy and Pathology of the Larynx

There are four levels at which obstruction may occur: at the introitus, the vestibular fold, the vocal fold, and the subglottis. The first three are mobile structures and act as sphincters to close off the airway when the controlling muscles contract. The fourth is a rigid structure with walls formed by the cricoid cartilage.

The first laryngeal sphincter is formed by the aryepiglottic folds with the epiglottis anterior and the arytenoid cartilages posterior (Fig. 1). The aryepiglottic folds are thick, fibromuscular, and relatively mobile structures, especially in early infancy. Closure of the orifice is brought about by contraction of the muscles lying in the lateral wall of the fold (aryepiglottic, thyroarytenoid, and

cricoarytenoid) and posteriorly between the two arytenoid cartilages, the transverse or interarytenoid muscle. Any inflammation may narrow the introitus to a critical degree, especially during the first few years of life when the structures are small but essentially normal, e.g. epiglottitis. When there is an anatomical abnormality, inflammation may readily precipitate obstruction: for example, small, mobile, or backwardly displaced epiglottis; cyst on the aryepiglottic fold; pharyngeal mass (Table).

TABLE

Local Conditions Which May Predispose to Acute Croup

Congenital small larynx (laryngomalacia)
Congenital laryngeal web
Abductor paralysis
Tumour or cyst of pharynx, larynx, or trachea
Compression of pharynx, larynx, or trachea from without

The second pharyngeal sphincter is formed by the vestibular folds (or false cords) of fibroelastic structure. They lie above the vocal cords and are separated from them by the vestibule (Fig. 1). Closure of the airway by approximation of these structures is achieved by contraction of muscles passing around the lateral wall of the larynx in these folds to the arytenoid cartilages (thyroarytenoid and cricoarytenoid muscles). This sphincter is fully effective without the aid of the vocal cords. Inflammation is less critical here but acute oedema due to allergy, or the inhalation of chemical agents, or hot gases evolved during a fire may narrow the orifice and frequently involves both the vestibular and aryepiglottic folds. A foreign body may wedge above the fold or in the vestibule and set up secondary inflammation.

The third sphincter is formed by the true vocal cords which are fibroelastic structures attached to the thyroid cartilage anteriorly and to the vocal process of the arytenoid cartilages posteriorly. Adduction is brought about by contraction of the lower fibres of the thyroarytenoid muscles which

*In the Personal Practice series of articles an author is invited to give his own views on some current practical problem.

FIG. 1—Anatomy of larynx at 1 year of age. Levels at which obstruction may occur: 1, supraglottic obstruction at the introitus (epiglottis and aryepiglottic folds) which form the first laryngeal sphincter; 2, at the vestibular fold and vestibule, the second laryngeal sphincter; 3, at the glottis, the third laryngeal sphincter; and 4, subglottic, at the level of the cricoid cartilage.

lie in the vocal folds (vocalis muscle). Apart from voluntary movement, adduction may be induced reflexly by the presence of irritant or foreign material in the larynx, causing stridor especially on inspiration. Loss of phonation or hoarseness characterizes inflammation of the cords.

All the muscles mentioned are innervated by the recurrent laryngeal branches of the vagus nerve, the centre being in the nucleus ambiguus of the medulla. Unilateral paralysis causes hoarseness but no stridor. Bilateral paralysis causes inspiratory and expiratory stridor with breathlessness due to obstruction. Again, superimposed infection may initiate or accentuate symptoms.

The fourth level at which obstruction may occur is the subglottic or cricoid region. Unlike the others, the walls are not mobile and are formed by the cricoid cartilage. It is shaped like a signet ring with a quadrangular lamina posterior and a narrow anterior arch. It forms the narrowest part of the larynx and determines the largest diameter of endotracheal tube which can be inserted. Obstruction at this point may be due to inflammation which is part of a laryngotracheobronchitis infection, or acute oedema following removal of an endotracheal tube which has traumatized the mucosa. Subsequent deposition of debris on an ulcerated mucosal surface may cause obstruction and subsequent postinflammatory stenosis may follow.

Tracheal narrowing due to a variety of causes (Table) may cause stridor which has to be distinguished from laryngeal causes.

Physiological Consequences of Obstruction

Obstruction which is of the vibratory, inspiratory type, and usually due to a mobile epiglottis partly obstructing the entrance to the larynx, causes marked stridor but little or no breathlessness and does not materially interfere with ventilation. A pedunculated polyp may cause similar symptoms but is potentially serious because it may cause complete obstruction with little warning. In contrast, other causes of stridor are accompanied by increase of respiratory effort which maintains ventilation. Tidal volume decreases but alveolar ventilation is maintained by increase of respiratory rate. The exception is the neonate who may respond to airway obstruction with decrease of effort, presumably because his capacity to respond to airway obstruction is not fully developed. Increase in airway resistance results in slight hypoventilation, a small increase of a few millimetres in arterial PCO_2 and decrease in PO_2 . The compensatory increase in the force of contraction of respiratory muscles maintains ventilation and blood gas levels near to normal until a late stage. An airway pressure of 5 to 10 mmHg is associated with the

subjective sensation of breathlessness and the objective appearance of increase of respiratory effort. Appreciably greater airway pressures can be achieved but only for short periods of time (a few minutes) without acute discomfort. A much higher respiratory muscle work load is therefore possible for a short time but cannot be sustained when the obstruction is prolonged beyond a few hours. Indeed there is an approximate inverse relation between the degree of obstruction and the duration for which ventilation can be maintained.

Failure of compensation is produced by decrease of respiratory effort, causing decrease of tidal volume, slowing of rate with irregularity of rhythm. These terminal events are associated with gross hypoventilation, arterial PCO_2 rising to >100 mmHg and PO_2 falling to <50 mmHg. The resulting generalized progressive tissue hypoxia causes lactic acidosis. Hypoxia, hypercarbia, and lactic acidosis together depress cardiac function, the fall in cardiac output causing decrease of cerebral blood flow, depression of the respiratory centre, and cardio-respiratory arrest soon follows. This sequence of events proceeds rapidly once the rate of work output (i.e. power output) of respiratory muscles begins to fall. The exact mechanism of respiratory failure in these circumstances is not clear, but it seems likely that fatigue of respiratory muscles causes critical reduction in their work capacity. If the limit of their power output (work per unit time) is exceeded because the work load is too high, or continues for too long, then hypoventilation will develop sooner or later. The patient should be observed carefully for decrease of stridor. It may indicate improvement, but on the other hand it may signal deterioration. In the former situation the blood gases are normal but in the latter they are abnormal and there is lactic acidosis.

Tachycardia, excessive sweating, and peripheral vasodilatation are the consequence of increase in autonomic activity, stress, and a raised metabolic rate. Increase of oxygen consumption and fever are due not only to the infection, but to the increased work of respiratory muscle. There is an increase of circulating catecholamines. At a late stage, decrease of cardiac output is accompanied by peripheral vasoconstriction with pallor and/or cyanosis, the latter being a reflex attempt to maintain arterial pressure. Dehydration due to impaired fluid intake causes hypovolaemia, and this, together with the above factors, contributes to the precipitation of acute low output circulatory failure. Bradycardia is accompanied by a variable degree of heart block with ST segment elevation or depression.

At this stage the P_{aO_2} is <50 mmHg and the $P_{aCO_2} >100$ mmHg.

Such late signs as these involve imminent danger of cerebral injury due to hypoxia. The crux of management is to relieve the obstruction in time to prevent respiratory and circulatory failure.

In the various lesions to be discussed below, respiratory failure may at any stage be exacerbated by two additional factors. (1) Desiccated secretions accumulating in the lower airway due to an ineffective cough (especially in the young infant) or to dehydration. (2) A pneumothorax produced by the increased respiratory effort caused by airway obstruction.

Diagnosis

It is essential to establish a diagnosis and in particular to define whether the partial obstruction causing symptoms has arisen in an airway which was formerly normal or whether there is pre-existing disease (Table). The latter group requires a radically different approach to treatment.

History. A history of previous attacks suggests laryngotracheobronchitis, whereas this history is usually absent in epiglottitis. Stridor dating back to birth or present between respiratory infections suggests an underlying abnormality. There may be no suggestive history when a foreign body partly obstructs the upper airway, but the absence of associated infection or persistence of stridor, should arouse suspicion. The history is usually clear cut when the cause is thermal or due to chemical burns after inhalation of noxious gas or vapour. Inquiries should be made regarding past infectious fevers and recent exposure to infectious cases.

Examination. The mouth, nose, and pharynx should be examined carefully for evidence of inflammation, trauma, and Koplik spots. An inflamed epiglottis may be visible over the back of the tongue when this is depressed. The pharynx should be inspected for distortion due to a mass. An enlarged tongue, retrognathos, a short neck, or other external deformity may suggest an underlying abnormality of the upper airway. The neck should be inspected for the presence of masses or tracheal displacement.

Epiglottitis

This is an acute inflammation of the epiglottis and aryepiglottic folds which occurs mainly in the 1- to 7-year age group. It is caused by the *Haemophilus influenzae* type B in most instances. This organism may be isolated from the pharynx and frequently also from the blood stream (Rabe, 1948).

There is an accompanying polymorphonuclear leucocytosis. The septicaemic spread and the fact that *H. influenzae* type B is the organism which also causes meningitis have been stressed by some authors. However, in my experience of 30 cases, clinical manifestations have been confined to the larynx, with signs of airway obstruction. When steps have been taken to relieve this in good time, the illness has been no more severe than tonsillitis.

The disease has been recognized for many years (Sinclair, 1941; Alexander, Ellis, and Leidy, 1942; Rabe, 1948; Miller, 1949; Jones and Camps, 1957; Jones, 1958), but only recently has its clinical importance and potentially lethal nature been appreciated (Johnstone and Lawy, 1967; Gardner *et al.*, 1967; Andrew, Tandon, and Turk, 1968; Jones, 1970). Baxter (1967) estimated that it comprised 8% of admissions with upper airway infection to the Montreal Children's Hospital, and Phelan and Williams (1968) reported 22 cases seen during a 3-year period in Melbourne.

There is usually no history of previous attacks and the mode of onset is acute, with malaise, fever, dry cough, loss of appetite, and the onset of stridor. Signs of obstruction may occur within a few hours; a rapid onset usually heralding a severe attack. It is this feature that distinguishes supraglottic laryngitis from the predominantly subglottic laryngo-tracheobronchitis. The disease runs a course of 4 to 7 days, fever subsiding after 48 to 72 hours. Stridor is predominantly inspiratory. The pharynx may appear normal, yet enlargement of cervical glands suggests infection. This should prompt an examination of the pharynx and on depression of the base of the tongue the epiglottis may be seen as a red oedematous structure. A more detailed laryngoscopic examination may be necessary to establish the diagnosis with certainty. The characteristic appearance of the supraglottic region is shown in the photograph which was taken from a fatal case (Fig. 2).

Management. The patient is best nursed well propped up with pillows. Reassurance is important, but sedation should be used with caution. Light sedation is likely to be ineffective whereas heavy sedation will produce stupor (difficult to distinguish from the effects of hypoxia) and depress respiratory effort. Trimeprazine tartrate (Valler-gan) 2 mg/kg is suitable. Ampicillin is given intramuscularly. Streptomycin may be added but is usually not required. If stridor occurs early, i.e. within 12 hours, hydrocortisone 100 mgIM is indicated and may well be repeated 6-hourly for 24 hours.

The child should be transferred to hospital if there is appreciable increase of respiratory effort and restlessness sufficient to cause interference with sleep and feeding. Evidence of cyanosis is of serious significance. When there is any doubt about the severity or possible course of the disease over the next few hours, admission to hospital should be advised.

In hospital, the child is nursed in an oxygen tent into which humidified oxygen-enriched gas is passed to give a concentration of 30 to 40% oxygen. It is important that the tent be transparent and the humidity insufficient to cause a fog. The child must be clearly visible at all times and be under constant surveillance so that the nurse can give early warning of deterioration. The face should be observed for colour of lips, and the chest for respiratory rate and effort. Instruments for endotracheal intubation or tracheostomy should be at hand. Swallowing may be difficult and drooling may occur. Pharyngeal suction is indicated as often as required to remove secretion which may accumulate in the pharynx.

Indications to relieve obstruction. In most instances these measures will result in diminution of obstructive symptoms within a few hours. Persistence of obstructive signs, namely appreciable increase of respiratory effort, sufficient to cause restlessness, interference with sleep and feeding, together with any evidence of cyanosis in the oxygen tent, are indications to relieve obstruction. A raised arterial PCO_2 (>50 mmHg) is also an indication to relieve obstruction, but a figure within the normal range does not contraindicate relief when the clinical evidence is present.

The passage of an endotracheal tube by the oral

FIG. 2.—Acute epiglottitis. Appearance at necropsy of the epiglottis and aryepiglottic folds which are grossly congested and oedematous, larynx unopened. A child of 6 years who sustained hypoxic brain injury due to acute low-output circulatory failure before admission to hospital.

or nasal route, or a tracheostomy, are both effective methods of relief. When the appropriate skill is available the passage of an endotracheal tube is preferable. It has the advantage that since the technique is simple, it can be implemented earlier giving a greater margin of safety and thus minimizing the possibility of collapse with the great likelihood of disastrous sequelae due to cerebral hypoxia. The presence of the tube does not aggravate the inflammatory lesion. The technique of intubation through the inflamed entrance to the larynx has not proved to be a problem in skilled hands. Once the tube is in place the child experiences marked relief and usually goes straight off to sleep. The swallowing of fluids may be possible but when there is reluctance or any difficulty, a fine plastic tube should be inserted through a nostril into the stomach, and via this appropriate hydration and nutrition can be ensured. The tube should be left *in situ* for 48 hours after which it is removed and the patient carefully observed for evidence of obstructive symptoms. If these are present it may be necessary to reintubate for a further 24 or 48 hours. Once the subject has been intubated, the general course of the disease is very similar to that of tonsillitis.

When the skill required to insert an endotracheal tube is not available, tracheostomy is indicated. It provides effective relief but because it is an operative procedure with residual scar formation, the tendency is to delay longer with increase of attendant risks.

Laryngotracheobronchitis

This common respiratory infection in the 1- to 5-year age range may also be associated with obstruction at laryngeal level. It is a virus infection in most instances but secondary bacterial involvement occurs. There is usually a clear history of recurrent attacks with malaise, cough, and fever, followed after 24 to 48 hours by stridor. Sometimes stridor occurs early in the attack and there may or may not be preceding upper respiratory tract infection. Stridor is inspiratory and expiratory and of variable degree. A cough is prominent and may be productive. On examination, the signs of airway obstruction are the same as those found in epiglottitis. The obstruction in this disease is predominantly subglottic. The walls of the trachea are red and oedematous. There may be a deposit of exudate which tends to dry and encrust. A variety of organisms may be isolated from this exudate, including nonhaemolytic and haemolytic streptococci, and staphylococci (Davison, 1966). There may be rales over the lung fields due to bronchitis.

The general principles of management are similar to those advocated in epiglottitis. Adequate humidification with an ultrasonic humidifier is particularly important to liquefy and prevent further drying of secretion.

Mucopurulent secretion accumulates in the lower airway and may also cause obstruction. Intermittent pharyngeal suction removes material at the entrance to the larynx, and also stimulates coughing which causes secretions to be expectorated from the major airways. This is particularly important in the child under 2 years who is less co-operative and cannot otherwise be encouraged to cough and remove secretion. When the child will tolerate it, the right and left lateral position with head dependent should be adopted 4 times daily with the object of aiding removal of sputum. During these sessions physiotherapy and coughing are encouraged.

Despite the fact that virus infection initiates the disease, bacteria are frequently also present, especially after intubation which increases the risk of contamination of the respiratory tract. Hence wide spectrum antibiotic cover with ampicillin and cloxacillin is required in the first instance.

Corticosteroid therapy is advocated by a number of authors (Novik, 1960; Pennington, 1964; Davison, 1966). It is probably of help in relieving obstructive symptoms during the 48 hours after admission and is advised as for epiglottitis.

The indications for relief of obstruction are the same as in epiglottitis. An endotracheal tube is again preferable to tracheostomy. The exception is the subject in whom the larynx as a whole is structurally smaller than normal due to a localized narrowing in the subglottic region which will not admit an endotracheal tube of adequate size.

Measles

Upper airway obstruction causing stridor may occur prior to the exanthem of measles. There is malaise, fever, conjunctival and nasal congestion: Koplik spots are usually evident. Obstructive symptoms occur chiefly in the 3- to 6-year age group. In most instances stridor is not severe, but, in a few, relief of obstruction is necessary.

Management is along similar lines to that already described except that antibiotics and steroids are not indicated. Feeding via a nasogastric tube may be necessary. The indications for relief of obstruction are as described for epiglottitis and the method of choice is a nasoendotracheal tube. This is left *in situ* for 1 to 3 days after which it may be removed, the subsequent course being uneventful.

Laryngeal Diphtheria

Laryngeal diphtheria, though rare in this country, should still be mentioned. There may be no membrane visible in the pharynx. Hoarseness is a feature since the cords are involved. Two to three days may elapse before obstructive symptoms occur (Davison, 1966). Antitoxic serum 20,000 to 100,000 units, one half IV, is given after testing for serum sensitivity. Penicillin and erythromycin are effective antibiotics. Tracheostomy for relief of obstruction is advocated (Lang, 1965). A nasogastric feeding tube is required if there is palatopharyngeal paralysis.

Foreign Body

A foreign body may wedge in the larynx immediately above or below the glottis. A less common position, which may easily be missed on laryngoscopy, is in the upper oesophagus where it may obstruct the entrance to the larynx. The danger of obstruction in these situations is that it may suddenly become complete thus asphyxiating the subject. When the previous history is negative and there is no evidence of an inflammatory disease the possibility should always be considered and laryngoscopy undertaken. No further measures may be necessary after removal but when trauma to the mucosa has been sustained hydrocortisone 100 mgIM 6-hourly and an antibiotic are indicated. Should obstructive symptoms follow, tracheostomy is indicated in order to ensure no further injury to the larynx and to allow laryngoscopic inspection from time to time.

Primary Disease of Larynx and Trachea

The possibility of an underlying abnormality of the larynx should always be considered when evidence of upper airway obstruction (1) presents in early infancy; (2) recurs frequently or is severe during upper airway infection; (3) persists in the absence of infection or after it has cleared up.

Obstruction due to infective causes is aggravated by the primary disease and frequently renders the condition potentially serious (Table).

Congenitally small larynx—laryngomalacia.

The larynx as a whole is smaller than normal so that any infection causes a critical degree of narrowing. This condition tends to declare itself during an infection in early infancy. The diagnosis is established by laryngoscopy. Relief of obstruction during an infection by passage of an endotracheal tube is contraindicated since not only is intubation difficult but a tube which can be passed safely

may be too small to provide an airway and readily becomes obstructed by secretion. A tube of adequate size would readily cause pressure necrosis especially in the subglottic area, with the attendant risk of subsequent stenosis on healing. A tracheostomy is therefore required and it is occasionally necessary to retain it for a year or two until the larynx enlarges sufficiently for the danger from recurrent infections to have disappeared.

Other diseases. Tumours and cysts within the larynx or trachea, or causing compression from without, may be symptomless until infection occurs and then cause airway obstruction which demands relief. An accurate diagnosis is essential and in most instances tracheostomy is the method of choice since the problem of management is relatively long term.

Compression of the trachea may be present in a child with a history of recurrent croup. One of the commoner causes is a vascular ring. It is relatively easy to diagnose with its characteristic appearance in the barium swallow x-ray and pulsatile compression of the trachea visible on bronchoscopy.

General Principles of Management

If the patient has collapsed it is necessary to perform oral endotracheal intubation without delay. Anaesthesia is unnecessary and the muscle tone is usually insufficient to prevent laryngoscopy and insertion of a tube. After preliminary removal of secretion from the pharynx, the laryngoscope is passed and an endotracheal tube inserted, avoiding all use of force. The diameter of the external nares is a guide to the diameter of tube required. When muscle tone is present, oxygen and halothane should be administered before laryngoscopy. After passage of the tube, the lower airway is cleared using a thin rubber catheter. The tube is connected to a 'T' piece and rubber bag for supply of an air/oxygen mixture and intermittent pulmonary inflation. At this point inspection of the pharynx and larynx will have established the diagnosis if the obstruction lies above the cords. A decision then has to be made whether an airway is necessary and, if so, whether it should be a nasoendotracheal tube or a tracheostomy.

When an endotracheal tube is used, it is essential that the external diameter should be such that a leak around it can be heard during expiration when the tube is obstructed with the finger or after inflation of the lungs with gas. A tube which is too large in diameter very rapidly ulcerates the mucosa, especially in the cricoid region. When *in situ* the tube must be adequately supported. A

tube with a crosspiece* is convenient, and support presents no difficulty. A piece of strapping is sufficient to anchor it and there is no danger of it passing into the trachea. The end of the tube is kept closed with a plastic bung at all times, except when endotracheal suction is being performed (Jones and Owen-Thomas, 1971).

Obstruction may be due to kinking in the pharynx, or to the fact that it is too long, but by far the commonest cause is dried secretion due to inadequate humidification. Humidified gas can be passed through one lateral limb of the crosspiece and an extension tube 3 feet in length attached to the other limb will ensure that all the inhaled gas is adequately humidified. This procedure is much more efficient in achieving humidification (and high oxygen levels, when required), than feeding gas into an oxygen tent and using a simple open-ended endotracheal tube. When the latter procedure is used an ultrasonic humidifier is necessary to provide sufficient humidification in the tent, but under these circumstances the patient is less easily observed.

Oxygen 30 to 40% is used in the inspired mixture and is adequate in all problems of upper airway obstruction unless there is associated lung disease such as bronchitis, bronchiolitis, or pneumonia. Suction to remove secretion from the tube is necessary from time to time but should be carried out at infrequent intervals which will vary depending upon the amount of secretion present. Three or four times per day is usually sufficient but occasionally 1- or 2-hourly suction is necessary. The nurse who is to perform it wears a mask and sterile disposable plastic gloves. The sterile catheter is picked up and the bung removed from the end of the tube. The catheter is passed to approximately the tip of the nasoendotracheal tube, the nurse being instructed to suck only to this depth. The passage of a catheter into the major bronchi should be carried out only by specially trained nursing or medical staff. This is a more skilled procedure, carrying the hazard of mechanical trauma to the mucosa and greater risk from the introduction of infection and the production of hypoxia due to prolonged suction. In general, it should be performed only once or twice a day and is usually not necessary in pure upper airway obstruction.

The duration of endotracheal intubation should be not more than one week. This is sufficient to cater for all inflammatory causes of upper airway obstruction, except when there is a primary laryn-

geal disease, and in these tracheostomy is indicated either initially or as a planned procedure within 24 to 48 hours of intubation.

Extubation is attempted after about 48 hours if the temperature and general signs of infection are subsiding, the amount of secretion from the airway is minimal, the chest x-ray is satisfactory, and the blood gas levels are within normal limits when breathing air. After extubation, the patient is observed closely for evidence of stridor. Should this recur, laryngoscopy is carried out and when necessary the tube is reinserted. Recurrence of stridor may occur between 12 and 24 hours after extubation and in most instances it is not due to the initial lesion but to deposition of debris on injured mucosa in the subglottic region. It may build up to form a diaphragm with a small hole in the centre. Accumulated debris should always be suspected when stridor recurs and when present may be removed with forceps or by suction at laryngoscopy. Laryngeal stenosis, especially in the subglottic region is due to subsequent scarring. It does not occur, however, if a tube of the correct diameter is used and the duration of intubation does not exceed one week.

Tracheostomy care follows exactly similar lines to that described for endotracheal intubation.

Complicating factors. In the great majority of instances once obstruction has been relieved the maintenance of ventilation presents no difficulty. Should this prove to be inadequate three causes should be considered.

(1) A pneumothorax may occasionally occur after a period of considerable increase of respiratory effort, and when suspected an x-ray of the chest should always be taken. When there is evidence of hypoventilation, the air should be removed. It usually does not recur, but an underwater drain may be required.

(2) There is associated lower airway or lung disease. The lower airway may be obstructed by secretion, or the level of tracheal compression may be beyond the end of the endotracheal tube. This may occasionally occur with a mediastinal tumour such as a neuroblastoma or when there is a vascular ring. It is essential to maintain a sufficiently long tube in position in order to keep the airway open, but because of the danger of pressure necrosis, relief of obstruction by operative means should follow without delay.

(3) By contrast with the above two causes in which there is usually increase of respiratory effort, there may be decrease of effort, and this is usually due to hypoxic injury to the brain during a period

*Such as the Jackson Rees endotracheal tube made by Portland Plastics, Hythe, Kent.

of low output circulatory failure due to delay preceding relief of obstruction. Pulmonary ventilation should be maintained by a mechanical ventilator until adequate respiratory effort returns or death occurs.

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