HAZARDS OF ENDOTRACHEAL ANAESTHESIA

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SUMMARY

The hazards of endotracheal anaesthesia have been reviewed. These have been considered in relation to direct laryngoscopy, intubation and extubation. Prevention of such complications has also been discussed.

The first clinical use of endotracheal intubation was reported by William Macewen in 1880. The work of Magill (1934, 1936) rendered intubation a practical procedure for the ordinary anaesthetist. More recently the introduction of muscle relaxants has further facilitated the technique and nowadays intubation of the trachea is widely and enthusiastically employed when specific indications exist. Occasionally, however, it may be used by the anaesthetist purely for convenience or as a means of demonstrating his prowess. In view of these trends it appears worth while to review the literature concerning complications of endotracheal anaesthesia and their prevention.

These can be conveniently classified as follows:

(1) Complications arising during intubation.
   (a) Acute trauma during direct laryngoscopy.
   (b) Acute trauma during insertion of endotracheal tube.
   (c) Disturbances in cardiac action.

(2) Complications occurring during endotracheal anaesthesia; respiratory obstruction.

(3) Complications arising during extubation.

(4) Complications arising after extubation.

These various dangers will now be dealt with in detail.

COMPLICATIONS ARISING DURING INTUBATION

Acute trauma during direct laryngoscopy.

Injury to the mouth, pharynx and larynx may occur when direct laryngoscopy is rendered difficult by inexperience on the part of the anaesthetist, by inadequate muscular relaxation and reflex suppression, or by the presence of anatomical and pathological factors affecting the teeth, jaw (Bannister and Macbeth, 1944; Cass, James and Lines, 1956; Davenport and Rosales, 1959; Gillespie, 1948a; and Ross, 1963), neck (Gardner and Holmes, 1961) and larynx (Davidson and Aladjemoff, 1963; Fields, 1959). Thus, the lips may be bruised and this may cause considerable discomfort in eating postoperatively (Bamford, 1963). Teeth can be avulsed and then pass into a bronchus (Wasmuth, 1960) producing pulmonary collapse and lung abscess. The mucosa of the pharynx may be bruised or abraded giving rise to postoperative sore throat (Wylie, 1950). During anterior displacement of the epiglottis the vallecula can be stretched, causing dysphagia (Donnelly, Grossman and Grem, 1948). Injury to the epiglottis or the larynx during this procedure may be followed by the development of oedema or a haematoma which can produce respiratory obstruction (Fields, 1959; Shaw, 1946; Verrill, 1963).

Prevention of trauma during direct laryngoscopy.

The incidence of trauma occurring during direct laryngoscopy has probably decreased in recent years. This is possibly due to the more widespread supervision of trainee anaesthetists and also to the frequent use of muscle relaxants to aid intubation. When anatomical and pathological factors render direct laryngoscopy difficult, damage to the dentition may be prevented by rubber guards clipped on to the teeth (O'Leary, 1963) or laryngoscope blade (Blair Gould, 1954; Waddy, 1954) and the use of a curved laryngoscope blade without a vertical component (Bizzari...
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and Giuffrida, 1958). Furthermore, the use of a curved metal endotracheal tube introducer (Cass, James and Lines, 1956) or blind nasal intubation may obviate forceful direct laryngoscopy and its associated traumatic effect.

Acute trauma during insertion of the endotracheal tube.

This can occur when the mucosa of the upper respiratory tract is prodded by the advancing end of the tube or by a protruding metal stilette. It can also arise when excessive friction is produced between the surface of the tube and the mucosa. As a result the mucosa may become oedematous, bruised, abraded or lacerated.

Lesions of this nature may occasionally give rise to serious complications. Thus, perforation of the nasopharyngeal mucosa with the creation of a false passage has been reported during nasal intubation (Barnard, 1948; Brown, 1952; Coffin, 1950). Such injuries may lead to the development of retropharyngeal abscess or mediastinitis. Tearing of the mucosa lining the pyriform fossa or sinus may lead to surgical emphysema of the neck and the mediastinum when the lungs are inflated with gases under positive pressure before intubation (Smith, Poole and Volpittro, 1959). This may be followed by the development of tension pneumothorax (Joannides and Tsoulos, 1930). Abrasions of the mucosa in the infraglottic region of the larynx can cause subglottic oedema (Haines and Powell, 1955) or pseudo-membrane formation within the larynx and trachea (Etsten and Mahler, 1951). These conditions may then produce respiratory obstruction.

There is, however, some evidence to suggest that serious sequelae are rare. Thus, although bleeding frequently occurs from the nose during nasal intubation, only two reports could be found in the literature in which this was severe (Dingley, 1943; Scott and Brechner, 1959). Furthermore, Donnelly, Grossman and Grem (1943) found that in a series of 100 patients intubated, 63 showed evidence of oedema, bruising or puncture wounds of the mucosa lining the pharynx and larynx, but none developed serious sequelae.

Prevention of trauma during intubation. Trauma to the nose may be avoided by the use of the oral route for intubation when pre-operative examination reveals obstruction of the nasal channels.

Further safeguards include the selection of a soft and not too large tube, shrinking of the nasal mucosa by spraying with 5 per cent cocaine, and the use of minimum force to overcome resistance to the passage of the tube (Dingley, 1943). Vellacott (1962) suggested that the tip of the tube should be guided past obstructions encountered in the nasopharynx by means of a finger inserted above the soft palate. This procedure may avoid the avulsion of adenoid tissue and also the creation of a false passage in the posterior wall of the pharynx. Prodding of structures within the mouth, pharynx and larynx can be obviated by careful intubation under direct vision and in the presence of adequate muscular relaxation and reflex suppression. The discarding of tubes which have rough surfaces due to the presence of dirt or deflated wrinkled cuffs helps to avoid trauma caused by excessive friction. Further precautions in this respect include the use of a well lubricated tube which passes comfortably through the larynx into the trachea.

Disturbances in cardiac action.

These may occur at the moment of intubation or inflation of the cuff. Disturbances produced can be transient and of no grave consequence or they may lead to cardiac arrest.

Transient disturbances in cardiac action. Evidence exists to suggest that these can be caused by reflex excitation of the vagus nerve or sympathetic system during intubation. Thus Reid and Brace (1940) found that auricular and ventricular extrasystoles, delayed conduction time and slowing of the heart occurred at the moment of intubation or inflation of the cuff in 10 out of 35 patients investigated. The authors suggested that these changes were due to reflex stimulation of the vagus nerve following mechanical irritation of the laryngotracheal mucosa by the endotracheal tube. In support of this, they drew attention to the close time relationship between intubation and the onset of disturbances in cardiac action. Furthermore, they pointed out that similar changes in cardiac action have been produced by the administration of acetylcholine (Scherf, 1929) and pressure on the carotid sinus (Ferris, Capps and Weiss, 1935). Burstein, Lo Pinto and Newman (1950), however, reported that sinus bradycardia occurred in only 4 out of
109 patients who were intubated, and Converse, Landmesser and Harmel (1952) were unable to demonstrate bradycardia during this procedure in 41 patients studied. The evidence to date is therefore contradictory with regard to the production of disordered cardiac action by means of vagal stimulation during intubation.

Burstein, Lo Pinto and Newman (1950) suggested that reflex stimulation of the cardio-accelerator nerves within the sympathetic system or the vagus may possibly be responsible for disturbances produced in cardiac action during intubation. Thus, in a series of 109 patients investigated by these authors, 43 developed sinus tachycardia, 1 auricular fibrillation and 2 ventricular tachycardia at the moment of intubation. Further evidence was provided by Noble and Derrick (1959), who found that in a series of patients during intubation 40 out of 80 developed sinus tachycardia at the moment when the tube was passed through the larynx into the trachea. DeVault, Greifenstein and Harris (1960) also demonstrated manifestations of increased activity of the sympathetic nervous system during this procedure. During intubation in 26 patients, all showed significant increases in the systolic and diastolic blood pressure, and 6 exhibited ventricular extrasystoles shortly after the introduction of the endotracheal tube. In a further group of 10 patients, these changes were prevented by the administration of 5 mg of phentolamine intravenously before intubation.

Cardiac arrest during intubation. From time to time reports have appeared in the literature of cardiac arrest occurring at the moment of intubation (Dale, 1952; Elton, 1961; Fleming et al., 1960; Stevenson, Reid and Hinton, 1953). In many of the cases reported by these authors hypoxia, hypercarbia or both were thought to have been present at the moment of intubation and in 3 instances the patients suffered from severe burns. It is therefore important to consider the effect of these factors on cardiac action when reflex excitation of the vagus nerve or sympathetic system occurs during intubation.

Hypercarbia has been demonstrated at the moment of intubation after the induction of anaesthesia with diethyl ether, cyclopropane or a thiopentone-relaxant sequence (Lachman, Long and Krumperman, 1955). This may cause an increase in sympathetic nervous discharge (Carey, Schaefer and Delgado, 1955; Gellhorn, 1953; Tenney, 1956) and associated excitation of the cardio-accelerator nerves. As a result, catecholamine levels within the myocardium may rise (Price et al., 1958). Further increases can be brought about by reflex stimulation of the cardio-accelerator nerves during intubation and this can cause ventricular fibrillation.

Elton (1961) and Fleming et al. (1960) have reported cardiac arrest during intubation of patients suffering from severe burns. The authors believed that this was possibly due to the occurrence of reflex vagal stimulation during intubation which was potentiated by existing metabolic disturbances. It is possible that hyperkalaemia was present, due to the existence of a metabolic acidosis (Mudge, 1953; Keating et al., 1953), and also to the administration of suxamethonium chloride (Galindo and Davis, 1962) which was used to aid intubation. This could have led to cardiac arrest by potentiating the action of acetylcholine released from cardio-depressor nerves (Fulton, 1950).

Drugs used in the premedication of the patient or the induction of anaesthesia may predispose to the production of cardiac asystole by the mechanism discussed. Thus, the vagotonic action of morphine (Cohn, 1913), pethidine (Johnstone, 1951), thiopentone and pentobarbitone (Redgate and Gellhorn, 1956), chloroform (Embley, 1902;
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Waters, 1951), cyclopropane (Price, 1960), and halothane (Burn et al., 1957) may potentiate reflex vagal stimulation occurring during intubation. Sensitization of the carotid sinus pressor receptors by chloroform, diethyl ether and trichloroethylene (Robertson, Swann and Whitteridge, 1956) could also produce a similar effect. Furthermore, diethyl ether, cyclopropane and halothane may possibly prevent the establishment of idopathic ventricular rhythm after vagal inhibition of the heart has occurred. In support of this, Smith and associates (1962) found that these agents raised the diastolic threshold for electrical excitation of the ventricle in the dog.

Factors which increase sympathetic nervous discharge or sensitize the heart to the action of adrenaline could facilitate the production of ventricular fibrillation by the mechanism discussed. Thus, sympathetic nervous discharge can be increased by pre-operative anxiety, premedication of the patient with atropine (relative increase) or the administration of chloroform (Beattie, Brow and Long, 1930; Brow, Long and Beattie, 1930), cyclopropane, diethyl ether (Price et al., 1959), and cocaine (Burstein, Lo Pinto and Newman, 1950; Wylie and Churchill-Davidson, 1960). Furthermore, the heart may be sensitized to the action of adrenaline by chloroform (Levy, 1911, 1912), cyclopropane (Meek, Hathaway and Orth, 1937) and halothane (Hall and Norris, 1958; Johansen, 1962; Johnstone and Nisbet, 1961; Raventós, 1956; Rosen and Roe, 1963).

Prevention of cardiac arrest during intubation.

It is apparent that the presence of hypoxia and hypercarbia during intubation may predispose to the production of cardiac arrest. Lachman, Long and Krumperman (1955) showed that changes in arterial blood gases of this nature could be prevented by spontaneous hyperventilation with 100 per cent oxygen. This was carried out for a period of 3 minutes before anaesthesia was induced and rapid intubation was performed using a thiopentone-relaxant sequence. Furthermore, Wycoff (1960) found that passive hyperventilation with 100 per cent oxygen for a period of 1 minute prior to intubation obviated hypercarbia during this procedure. Additional precautions include the prevention of laryngeal spasm, coughing and "bucking", during insertion of the endotracheal tube, by the use of a muscle relaxant or a deep level of anaesthesia before intubation is attempted. Blocking of autonomic reflexes by means of a topical anaesthetic applied to the laryngotracheal mucosa or a deep level of anaesthesia may also obviate reflex disturbances in cardiac action during intubation (Burstein, Lo Pinto and Newman, 1950; Johnstone and Nisbet, 1961). Pre-operative medication with atropine may offer some protection against the occurrence of cardiac asystole after vagal stimulation during insertion of the endotracheal tube. If, however, sympathetic stimulation predominates during this procedure, then atropine may predispose to the production of dangerous ventricular arrhythmias (Gillies, 1948; Johnstone, 1952; Johnstone and Nisbet, 1961).

Some evidence has been put forward which suggests that hyperkalaemia may predispose to the production of cardiac arrest when intubation is aided by the administration of suxamethonium chloride. In view of this it would appear safer to facilitate intubation using tubocurarine (Dowdy and Fabian, 1963) or an inhalation agent when hyperkalaemia is present and intubation is considered essential.

COMPLICATIONS OCCURRING DURING ENDOTRACHEAL ANAESTHESIA: RESPIRATORY OBSTRUCTION

Although endotracheal tubes are used to ensure the airway, they by no means exclude the possibility of respiratory obstruction occurring. Obstruction may result from occlusion of the lumen of the endotracheal connector or the tube itself by foreign bodies. Other causes include abnormal inflation of the cuff of the tube, kinking or disruption of its walls, and misplacement.

Rollason (1956) and Forrester (1959) each described a case in which respiratory obstruction was caused by imperforate suction unions; Forrester's patient died from cerebral hypoxia. Hewer (1956) mentioned a patient in whom airway obstruction was produced by the plastic spigot of a face mask being lodged within a Cobb's union. Haseluhn (1958), Jenkins (1959) and Dutton (1962) also reported cases of occlusion of the lumen of the endotracheal tube, due respectively to the rubber tip which is used over the adaptor of an "Abbott Venopak", the broken...
half of a Magill's endotracheal tube cleaning brush, and a rubber bung.

Abnormal inflation of the cuff may occur as a result of a local weakness in the balloon sleeve or the wall of the tube which lies adjacent. Factors which may cause these weaknesses include entrapment of air bubbles or dirt within the rubber during manufacture (Forrester, 1959), repeated boiling or autoclaving (Stark and Pask, 1962), or the application of oily lubricants (Hewer and Lee, 1957). Robbie and Pearce (1959) and Forrester (1959) reported cases in which respiratory obstruction was caused by internal herniation of the cuff which occluded the lumen of a latex armoured tube. Similar incidents have been reported by Hayes (1961) and Clarke (1962) respectively, in which the airway of a Carlens and a White double-lumen tube was obstructed by inward pressure from the inflated tracheal cuff. Pryer (1960) also described an instance of the bevel of the tube being pushed up against the wall of the trachea by a cuff which inflated asymmetrically. Uneven inflation may further lead to herniation of the cuff over the distal end of the tube. This can also be produced by upward traction of heavy anaesthetic hoses. Waters and Gillespie (1944) and Forrester (1959) each reported a fatality which followed respiratory obstruction produced by this mechanism.

Respiratory obstruction may be caused by kinking of mineralized rubber or plastic tubes in situ. This may arise at points where the tube undergoes marked changes in direction. Such sites are where metal connectors are attached to the tube, the oropharynx (Duckworth, 1962) and the nasopharynx (Gillespie, 1948b). Acute flexion of the head, torsion produced by heavy anaesthetic hoses, and softening of the wall of the tube by repeated boiling or autoclaving (Stark and Pask, 1962) are additional factors in the production of this hazard.

Armoured tubes are by no means immune from kinking which may occur at the proximal or distal end. In the former position, this can arise when metal connectors are not pushed down to the level at which the wire spiral commences. In the latter site, the soft rubber tip can be kinked when the tube is inadvertently pushed up against the wall of the trachea (Ballantine and Jackson, 1954). Marked kinking of the armoured portion of the tube may also be produced when the patient bites on this region or when excessive pressure is exerted by Cheatle's forceps (Robbie and Pearce, 1959).

Disruption of the tube wall may add to the list of bizarre causes of respiratory obstruction during endotracheal anaesthesia. Thus, Burns (1956) reported a case in which this developed as a result of swelling of the inner wall of a flexometallic tube. This was thought to have been caused by the absorption of trichloroethylene into the inner layer of latex, which was partly detached from the main body of the tube. Robbie and Pearce (1959) also mention a patient in whom airway obstruction was caused by puckering of the inner wall of a latex armoured tube; the puckering occurred during insertion of the metal connector and also during the withdrawal of a metal stilette.

Misplacement of the endotracheal tube may arise when the right main bronchus is inadvertently intubated. This can produce hypoxia and hypercarbia as a result of inadequate ventilation of the left lung (Gillespie, 1948c; Keating, 1961) which may subsequently collapse (Bamford, 1963). Hypoxia and hypercarbia may become more marked if the cuff of the tube is inflated producing obstruction of the right eparterial bronchus. This can also arise when a White double-lumen tube is inserted too far into the right main bronchus and the cuff is inflated (Clarke, 1962). Complete respiratory obstruction may occur when the bevel of the tube is pushed up against the medial wall of the right main bronchus (Bamford, 1963).

Prevention of respiratory obstruction.

Detection of foreign bodies may be assisted by the use of transparent material in the manufacture of endotracheal connectors and tubes (Haselhuhn, 1958). Further safeguards include testing of such apparatus for patency after cleaning and again before use (Jenkins, 1959), and disposal into receptacles specifically reserved for this purpose.

Respiratory obstruction produced by abnormal inflation of the cuff may, to some extent, be avoided by manufacturing tubes without inherent weaknesses in the wall or the cuff. This may be
achieved by preventing the entrapping of air bubbles or dirt within the rubber and the production of chemical bonding between the latex layers of armoured tubes (Davies, 1963; Robbie and Pearce, 1959). The discarding of tubes which have been weakened by boiling or autoclaving on more than six occasions (Stark and Pask, 1962) and the inspection of the inflated cuff before use to detect an external aneurysmal swelling, peripheral extension or internal herniation, are additional safeguards.

Kinking can be avoided by discarding endotracheal tubes with weakened walls, appropriate positioning of anaesthetic equipment and by the use of a mouth-prop to prevent biting on the tube when anaesthesia becomes light. The use of an Oxford non-kink or latex armoured tube affords additional protection against this hazard when the head is placed in an abnormal position or subjected to excessive movement during surgery (Duckworth, 1962; Ballantine and Jackson, 1954). Other precautions include pushing of metal connectors slightly beyond the beginning of the wire spiral of flexometallic tubes and cutting off the soft rubber tip (Ballantine and Jackson, 1954).

Puckering of the internal layer of latex armoured tubes can be prevented by avoiding the production of local weaknesses within the tube wall during manufacture. Further safeguards are careful insertion of metal connectors and the use of a lubricated stilette which is curved so as not to impinge against the wall of the tube during withdrawal (Robbie and Pearce, 1959).

Inadvertent intubation of the right main bronchus may be obviated by passing the tip of the tube to approximately 3–4 cm beyond the true vocal cords in the adult. Auscultation of the chest after intubation and positioning of the patient provides additional protection against this hazard (Bamford, 1963) and also against obstruction of the right eparterial bronchus by the inflated bronchial cuff of a White endobronchial tube (Clarke, 1962).

COMPLICATIONS OCCURRING DURING EXTUBATION

Raffan (1954) and Shumacker and Hampton (1951) reported a total of five cases of cardiac arrest which were directly associated with aspiration of secretions from the trachea and extubation. Potentiation of reflex vagal stimulation during extubation, by hypoxia produced during tracheal suction (Kergin, Bean and Paul, 1948), was thought to be the cause. It would therefore appear safer to ventilate the lungs with 100 per cent oxygen after tracheal suction and also before extubation (Martin, 1963).

Throat packs may be left in situ inadvertently after extubation, thus causing respiratory obstruction. This hazard can be avoided by tying the throat pack to the endotracheal connector (Martin, 1963) or by clipping a metal disk marked “throat pack” to the catheter mount (Evans and Gray, 1959), and also by leaving the end of the throat pack protruding from the mouth.

Laryngeal spasm is a further cause of respiratory obstruction after extubation. This may be initiated reflexly by painful stimuli from the site of operation and stimulation of sensory laryngeal receptors by secretions, or withdrawal of the tube (Murtagh and Campbell, 1954). The application of a local anaesthetic to the mucosa of the larynx and trachea before intubation and the aspiration of secretions before extubation may assist in preventing this complication. Laryngeal spasm may also be prevented by removing the endotracheal tube at a deep plane of anaesthesia or after the administration of suxamethonium chloride (Lambie and Pfaff, 1956). This, however, increases the danger of regurgitated gastric contents being aspirated. Atropine appears to have little effect in the prevention of laryngeal spasm when given in doses commonly used in premedication (Harrison, 1962; Harrison and Vanik, 1963; Rosen, 1960).

COMPLICATIONS OCCURRING AFTER EXTUBATION

Sore throat is the most common complication of intubation. Wylie (1950) reported that the incidence was 46 per cent and the findings of Gard and Cruickshank (1961) agreed closely with this figure.

A consideration of the aetiology of postintubation sore throat indicates that trauma is an important factor. Wylie (1950) found evidence of oedema and bruising of the throat in 87 per cent of patients who complained of sore throat after endotracheal anaesthesia.
Various mechanisms exist whereby the throat can be traumatized during intubation. Thus, trauma may be produced when direct laryngoscopy and intubation are attempted in the absence of profound muscular relaxation and reflex suppression. In support of this, Wylie (1950) found that the incidence of trauma was 47 per cent in a series of patients who were intubated using cyclopropane or diethyl ether anaesthesia. However, in a further group of cases the incidence decreased to 36 per cent when intubation was carried out with the aid of a muscle relaxant.

A further cause of pharyngeal trauma may be excessive friction between the endotracheal tube and the mucosa of the pharynx. Gard and Cruickshank (1961) reported that the postoperative incidence of sore throat was 58 per cent when frequent movement of the head occurred during surgery and 43 per cent when movement of the head was slight or absent. Moreover these authors also showed that repeated application of lubricant jelly to the surface of the tube during anaesthesia lowered the incidence of sore throat from 54 to 38 per cent. Further evidence was provided by Conway, Miller and Sugden (1960), who found that the incidence of postoperative sore throat was reduced when intubation was facilitated by tubocurarine instead of suxamethonium chloride, and also when a cuffed tube was used. They suggested this was due respectively to the prevention of coughing and "bucking" immediately after intubation by the more prolonged action of tubocurarine and also to the anchoring of the tube within the trachea by the inflated cuff. The same authors further pointed out that the use of throat packs is followed by a higher incidence of sore throat. This may be explained by excessive friction between the pharyngeal mucosa and the throat pack during insertion.

Laryngitis is a rare complication of intubation. Wolfson (1958) reported an incidence of 3 per cent in a series of patients who were intubated with the aid of a muscle relaxant. It is possible that the low incidence found by this author was accounted for by the avoidance of trauma to the larynx when laryngoscopy and intubation were carried out in the presence of profound muscular relaxation and reflex suppression. However, static pressure of the endotracheal tube on the posterior commissure of the larynx (Dwyer, Kronenberg and Saklad, 1949) may have been the causative factor in those patients who did develop laryngitis in this series. In support of this, Baron and Kohlmoos (1951) found evidence of varying degrees of redness of the arytenoid cartilages, and some roughening of the posterior third of the vocal cords, in a group of patients who complained of hoarseness following intubation.

Symptoms of laryngitis following endotracheal anaesthesia have been reported as ranging from complete aphony to varying degrees of hoarseness, the duration of the complaint being 1 to 7 days (Baron and Kohlmoos, 1951; Wolfson, 1958; Wylie, 1950).

Under certain circumstances the reaction of the laryngotracheal mucosa to intubation may produce serious postoperative conditions. Thus laryngeal oedema may lead to the development of respiratory obstruction in infants and children. This is because the lumen of the larynx is anatomically small and may therefore be considerably reduced in size by slight swelling of the mucosa (Holinger and Johnston, 1950).

Several reports of such instances have appeared in the literature. Gillespie (1948), Haines and Powell (1955) and Shaw (1946) described a total of four cases which were possibly...
caused by traumatic intubation. Crispell and Hampton (1950) reported two in whom the condition was thought to have been precipitated by excessive friction between the wall of the endotracheal tube and the mucosa of the larynx during craniotomy. In the two cases mentioned by Donnelly, Grossman and Green (1948) static pressure from the tube which fitted snugly within the larynx was believed to be the cause. Gillespie (1948a), however, suggested that laryngeal oedema may arise as a result of acute infection superimposed on minimal trauma caused during intubation.

Symptoms and signs of subglottic oedema have been reported as occurring from 40 minutes to 24 hours after extubation. In the initial stage the condition may be characterized by a croupy cough and hoarseness and, as oedema increases, signs of respiratory obstruction develop (Baron and Kohlmoos, 1951; Deming and Oech, 1961; Haines and Powell, 1955).

Occasional reports appear in the literature of membrane formation in the larynx and trachea following intubation. Muir and Straton (1954) described four cases in a series of 1,500 patients who underwent thoracic surgery. The same authors also reviewed a further 23 reports of cases. Lu, Tamura and Koobs (1961) found that 5 out of 23 patients who died a short time after surgery showed evidence of membrane formation within the larynx and trachea.

Various theories have been advanced to explain membrane formation. In a total of 6 cases reported by Belam and Zuck (1953), Dark and Jewsbury (1955), Grimm and Knight (1943) and Turner (1949), it was thought to have occurred as a result of sloughing of the laryngotracheal mucosa, which was believed to have followed pressure from the inflated cuff of the endotracheal tube. In 4 cases reported by Muir and Straton (1954) and 5 by Lu and associates (1961), an acute inflammatory reaction within the mucosa was thought to have been the cause. This may have occurred in the former group of cases as a result of the exacerbation of a chronic low-grade infection by traumatic intubation. In the latter group, the exciting factor may have been necrotic areas of mucosa produced by prolonged static pressure from the tube. General debility of the patient has also been suggested as a factor in the development of membrane formation (Belam and Zuck, 1953; Lu, Tamura and Koobs, 1961; Muir and Straton, 1954).

Clinical manifestations of membrane formation include a high-pitched wheezing cough, hoarseness, stridor and partial respiratory obstruction. These symptoms and signs may arise 12-72 hours after extubation and can be relieved following removal of the membrane by means of bronchoscopic suction (Etsten and Mahler, 1951; Muir and Straton, 1954; Turner, 1949). Acute respiratory obstruction can, however, develop quite suddenly, producing a fatal result if the membrane becomes detached from the wall of the trachea and occludes the airway (Lu, Tamura and Koobs, 1961).

Frequent reports have appeared of intubation granuloma. Griffiths (1932) described 5 cases and Flagg (1951) published data relating to 101 instances reported by American surgeons. In a review of the literature, Wolfson (1958) pointed out that 55 had been described in Britain between 1932 and 1954.

Intubation granuloma occurs most frequently on the vocal process of the arytenoid cartilages. Out of 50 cases reviewed by Howland and Lewis (1956), 37 were situated on the vocal process, 10 on the middle third, and 3 on the anterior third of the true vocal cords.

It is evident that the formation of intubation granuloma is most frequently initiated by trauma of the vocal process. This is commonly produced by static pressure from the tube on this region when the neck is extended (Campkin, 1959; Howland and Lewis, 1956) or flexed (Epstein and Winston, 1957). Other mechanisms described are hooking of the vocal process by the open end of the tube during intubation and friction between the endotracheal tube and the tip of the vocal process during spontaneous breathing (Jackson, 1953). Rarely, a granuloma may develop on the true vocal cords after trauma has been inflicted by the bevel of the tube (Howland and Lewis, 1956; Wylie, 1950).

Acute ulceration may occur on the vocal process or the true vocal cords following trauma. The ulcer can then become chronic as a result of repeated trauma from the vocal process opposite (Jackson,
1953; Moore and Von Leden, 1959) and superimposed infection. This may be followed by the development of granulations if treatment is not instituted at this stage (Epstein and Winston, 1957).

Howland and Lewis (1956) reported that the incidence of intubation granuloma is seven times greater in the female than in the male. These authors suggested the following explanations: (1) the female larynx is smaller than the male; (2) the mucosa over the true vocal cords in the female is only half as thick as that in the male; (3) the incidence of operations in which the head is placed in an abnormal position is greater in the female than in the male.

The most common symptom of intubation granuloma is persistent hoarseness. This may be noted 1 to 32 weeks after operation (Epstein and Winston, 1957). Other symptoms are pain radiating to the ear, and a sense of fullness in the throat (Brodnitz, 1961).

**Prevention of complications occurring after extubation.**

Trauma of the respiratory mucosa together with superimposed infection are important aetiological factors in the production of complications after endotracheal anaesthesia. The prevention of acute trauma during intubation has been discussed. Trauma due to excessive friction between the tube in situ and the respiratory mucosa may be reduced by using smooth-surfaced tubes, avoiding coughing, "bucking" and spontaneous hyperventilation during anaesthesia, and the firm fixation of the tube with adhesive plaster. Unnecessary movement of the head during surgery is an additional precaution in this respect.

Trauma caused by excessive static pressure of the tube on the respiratory mucosa can be reduced by avoiding overextension or flexion of the head and neck. If this is not feasible, then a tube which moulds to the contour of the respiratory tract should be used (Dwyer, Kronenberg and Saklad, 1949). Flexometallic tubes are satisfactory in this respect and offer an additional advantage over plastic tubes in that they are unlikely to kink in these positions. Cuff inflation should just be adequate to produce a gastight seal when the lungs are inflated using normal inflation pressures. In these circumstances the pressure exerted by the cuff on the trachea is too low to produce ischaemia of the mucosa (Muir and Straton, 1954). Further factors include the use of a tube which is not too wide to pass comfortably through the larynx into the trachea.

Superimposed infection may be less likely if upper respiratory tract infection is treated prophylactically and if laryngoscopy blades, airways and endotracheal tubes are sterilized before use.

**CONCLUSION**

The present review indicates that intubation may lead both to minor and grave complications. It is difficult to ascertain what proportion these represent of the total number of intubations which have been performed or indeed of the total number of accidents which have occurred. Many must have remained unreported. Nevertheless, the fact that calamities have occurred, often in expert hands, is adequate warning that tracheal intubation is not always a harmless procedure. However, many of the complications mentioned may be preventable in the light of recent knowledge, drugs and equipment, and it may well be that similar incidents will be reported in the literature with decreasing frequency. Even so this should not serve as an excuse for the indiscriminate use of a technique which carries both a morbidity and mortality rate.

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LES RISQUES DE L'ANESTHESIE ENDOTRACHEALE

SOMMAIRE

DIE GEFAHREN DER ENDOTRACHEALEN ANÄSTHESIE

ZUSAMMENFASSUNG