MANAGEMENT OF AIRWAY PROBLEMS IN SEVERELY BURNED PATIENTS

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Coincident with significant improvements in the management of shock, fluid management and sepsis in the severely burned patients, respiratory tract damage has emerged as the principal killer in burned patients. Despite the recognized nature of this aspect of the severely burned patients, and despite extensive clinical studies and laboratory research, there remains considerable confusion and disagreement regarding the assessment and management of burned patients with respiratory tract damage.

Very frequently, the Otolaryngologist may be called upon to provide the necessary measures in the management of these patients, especially regarding tracheostomy and problems of tracheo-bronchial toilet.

The Offending Agent

It is important to have an understanding of what is the actual offending agent that brings about the respiratory tract damage. First of all, one would tend to think in terms of a "fire" or respiratory tract "burn" when faced with a patient with extensive body burns. Experimental and clinical studies, however, demonstrate that damage to the respiratory tract in survivors is nearly always caused by the effects of smoke or "Products of incomplete combustion" rather than heat.

Actual thermal burns of the respiratory tract are usually limited to the anterior portion of the nose and mouth due to very efficient heat exchange in the upper respiratory tract. Heat sufficient to produce thermal burns of the pharynx and larynx produces acute obstructive laryngeal oedema incompatible with life before actual thermal burns can be produced in the tracheo-bronchial tree. Even the larynx is usually spared from heat sufficient to cause laryngeal thermal burns, except in already unconscious patients. Accordingly, my thinking in terms of damage to the laryngotracheo-bronchial system should be in terms of the effects of chemical irritation and destruction of tissue by smoke and chemical products of incomplete combustion rather than the effects of actual heat.

Pathology

Pathologic findings demonstrate extensive destruction of bronchial mucosa, dilatation of large and small bronchi, haemorrhagic oedema of the peribronchial connective-tissue, generalized hyperaemia and haemorrhagic oedema of the peripheral bronchi and alteration in the alveolar capillary membrane. By and large, the autopsy findings in patients dying of pulmonary "burns" showed intense tracheitis, bronchitis and necrosis of the tracheobronchial epithelium with diffuse haemorrhagic and focal membranous reaction in the tracheobronchial mucosa. Pulmonary changes are manifested by one/more of the following :

- (I) Pulmonary Oedema
- (II) Atelectasis
- (III) Intra-alveolar haemorrhage.
- (IV) Disruption of the alveolar capillary membrane.

The amount of damage is of course, dependant upon the severity and length of exposure.

Clinical features

It is of utmost importance to obtain a careful history as to the nature of the fire in which the burns were sustained. It has been shown that majority of significant airway damage in burned patients occurs when the burn is a result of flame-type injury in an enclosed space, such as a burning room or building. This again reflects the importance of the influence of smoke and products of incomplete combustion in the respiratory tract damage. This concept is further confirmed by the fact that in the southern parts of the United States and in England, respiratory tract involvement in burned patients is relatively rare. This is because the burns are most often caused by the ignition of clothing by space heaters and unguarded coal fires, wherein the victim usually runs about in relatively clear air rather than inhaling the hot, smoky atmosphere of a burning room or building. Therefore, we must ascertain the situation in which the patient received the burn. Cope has found that persons burned in enclosed spaces stood a 47% chance of suffering respiratory damage. Symptoms

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and signs to indicate the presence of pulmonary damage may be obvious or deceiving, depending upon the severity of respiratory involvement and the state of consciousness and cooperation of the victim. (Table-I).

It must be remembered, however, that cyanosis may be masked by the cherry red colour of blood induced by carbon poisoning. The presence of auscultative changes in the lungs on admission is also a very dire prognostic finding, since physical findings in the chest and X-ray changes in

potential survivors usually do not occur until after a number of hours or even days, following the burn. Another very important point to remember in respect to the signs of respiratory burns is that stupor and confusion are often encountered, and must be recognized as being due to hypoxia from respiratory damage rather than being attributed to a shock or other causes.

Clinical Management

From the otolaryngologist's view point, it is probably best

Table - I

Clinical Aspects	Mild involvement	Severe involvement
I) Historically Burn Agent Environment	- Flame Enclosed	Flame Enclosed
II) Presenting Complaints Sore Throat Cough Hoarseness Dyspnoea	Variable Frequent Infrequent Infrequent	Variable Always Variable Always
III) General Evaluation State of Consciousness Patient Cooperation General Orientation	Alert Good Good	Lethargic to comatose Bad Bad
IV) General Appearance Colour Cough Respiratory effort	Normal Mild Normal to slightly labored.	Cyanotic/Cherry-Red Severe Labored
V) Physical examination Body surface burned Oropharynx Chest	Usually involves respiratory area of face. Injected Normal to decrease breath sounds	Respiratory area of face Injected Decreased breath sounds with rales.
VI) Radiography of Chest	Normal	Normal/or with signs of consolidation and atelectasis or pulmonary oedema

to approach the management of airway problems by utilizing the three stages proposed by Stone.

(I) Pulmonary insufficiency, (II) Pulmonary Oedema and (III) Pneumonia.

(I) It is the most severe stage, manifested by signs of marked airway obstruction i.e. Cyanosis, retraction, dyspnoea and diminished or absent breath sounds.

It should be recognised that the prognosis is almost completely hopeless in this situation. Although many of the symptoms/signs point to upper airway or laryngeal obstruction, in most cases they are really due to pathology in the entire respiratory tract rather than the larynx. Further, if the main pathology is, indeed, in the tracheo-bronchial tree, tracheostomy may not only be futile but may be fatal. The barrier to ventilation which appears to be of laryngeal origin is primarily due to severe bronchospasm and/or extensive alveolar damage. In this stage of disease process, tracheostomy should only be done if definite laryngeal obstruction can be demonstrated. In this regard, it is important to attempt an assessment of the laryngeal airway, either by direct or indirect laryngoscopy, as soon as the patient is admitted. The general opinion appears to be that unless there is obviously demonstrated laryngeal obstruction, tracheostomy should be deferred due to the dire consequences i.e. increased shock, introduction of sepsis and premature induction of pulmonary oedema. Keeping in mind the high probability of bronchospasm in

this stage in the absence of definitely demonstrated laryngeal obstruction, tracheostomy should be deferred pending on the results of a large dose of corticosteroids in an attempt to break the bronchospasm. If, after steroid administration, the patient is still in respiratory distress, tracheostomy may be considered. It must be recognised that if a tracheostomy is performed during this stage, either because of definite or suspected laryngeal obstruction, a cuffed tracheostomy tube must be utilized and the patient immediately put on positive pressure ventilation.

In view of the grave prognosis and difficulty in determining the exact cause of ventilatory insufficiency, primary treatment consists of the administration of 40% O₂ at 100% humidity via face mask or endotracheal tube (Table-II).

II) The second stage is that of pulmonary oedema. If this is not induced by premature tracheostomy, it seldom develops before the institution of fluid therapy, usually at least 8-10 hours following the burn. The treatment of choice during this stage is that of positive pressure ventilation through a nasal endotracheal tube with sufficient sedation for the patient to tolerate the tube. In patients with pre-existing cardiac disease, fluids should be used cautiously and administration of digoxin and/or digitalis preparation may be of benefit in controlling the tendency to pulmonary oedema but these agents in the absence of pre-existing cardiac disease do not alter the course of pulmonary oedema on the basis of the pulmonary changes

Table -II

Stages	Pulmonary Insufficiency	Pulmonary Oedema	Pneumonia
I) Time of Onset.	0-8 hours post burn	8-36 hours post burn	4-7 days post burn
II) Symptoms & signs	Dyspnoea, sternal retraction, diminished breath sounds	Dyspnoea, frothy sputum & rales	Dyspnoea, areas of pulmonary consolidation
III) Pathophysiology	Marked bronchospasm	Transudation of fluid into the bronchioles & alveoli	Bacterial invasion of lung parenchyma.
IV) Treatment	Intravenous	Intratracheal intubation with intermittent positive pressure ventilation	Large doses of antibiotics, tracheostomy to assist. tracheo-bronchial toilet
V) Prognosis	Grave 80% mortality	Poor 60% mortality	Fair 30% mortality

resulting from the burns. If a previous tracheostomy has been done and the patient has survived to this stage of pulmonary oedema, positive pressure ventilation can be administered via the cuffed tracheostomy tube.

III) The third stage, one which all surviving patients pass through, is that of bacterial pneumonia. This is almost always associated with massive bronchial pulmonary secretions. The onset of this stage may be as early as the second day following the burn, but is usually encountered between the 4th to 7th day. It is during this stage that tracheostomy plays its most significant role in the management of respiratory involvement in the severely burned patient. At this time, the control of the large amounts of bronchopulmonary secretion is difficult by any other means than tracheostomy. Accordingly, tracheostomy for a short period of time is very desirable for adequate tracheobronchial toilet. The pneumonic process itself is managed by massive doses of appropriate antibiotics. It should be pointed out that prophylactic antibiotics in the early stages of "Pulmonary burns" have not been found

to be of any value in preventing this stage of the disease process.

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