TRACHEOTOMY IN STATUS ASTHMATICUS

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Bronchial asthma, and in particular status asthmaticus, is a disease process usually associated with a high morbidity; fortunately it has a low mortality rate.[2]

Yet, despite the fact that it occurs relatively infrequently, most of us have probably had the frustrating experience of helplessly watching a patient die from the effects of hypercapnia resulting from status asthmaticus, after having tried all the known remedies, new and old.

A case of status asthmaticus is presented in which tracheotomy was probably a life-saving procedure.[1,2]

This procedure has been used in various states of acute pulmonary insufficiency[6] and, considered in terms of pulmonary physiology, its use is rational and logical.

CASE REPORT

T.T.J., a European male (civil servant) aged 45 years, was admitted to Pretoria General Hospital at 6.45 p.m. on 28 November 1956 with a history of a slight, productive cough of a few days' duration. About 3 hours before admission he had begun to experience wheezing and dyspnoea. This patient was known to have been subject to infrequent attacks of bronchial asthma over a number of years. On examination the significant findings were: moderate hyper-resonance of the chest with diffuse, wheezing, rhonchi and a respiratory rate of 20 per minute. Expiration was prolonged and forceful, bringing the accessory muscles of respiration into play. Anxiety was a prominent feature. Blood pressure was 120/85 mm.Hg and the pulse rate was 90 per minute.

During the following 24 hours respiratory distress was unrelieved despite vigorous treatment with various antispasmodics (adrenaline, ephedrine, 'asthmolysin', aminophylline and prantal methylsulfate[5]), sedatives (ether and olive oil retention-enemas, chlorpromazine and pethedine), infusions of ACTH and hydrocortisone, inhalations of 90% alcohol, potassium iodide expectorants, and intravenous nitrogen mustard (0·1 mg. per kg. as a slow infusion). Because of viscous purulent sputum and a moderate polymorphonuclear leucocytosis, antibiotics were given — tetracycline, 500 mg. 6-hourly, and subsequently streptomycin, 0·5 G. *b.d.* and novobiocin sodium, 500 mg. 8-hourly. Cyanosis soon supervened although the patient had been treated in an oxygen tent, but oxygen administration through a face mask caused the cyanosis to disappear. A nasal catheter was later substituted for the face mask.

Instead of improvement, the second 24-hour period produced a rapid deterioration in the patient's condition. Breathing became shallow and rapid, râles appeared at the lung bases, and the patient became irrational and stuporose. Perspiration was profuse, the pulse rate increased to about 170 per minute (sinus rhythm), and the blood pressure rose to 165/110 mm.Hg. The urine, which on admission had shown nothing abnormal, now contained albumin ++++ and some casts. (A specimen examined during convalescence again showed nothing abnormal.)

Towards the evening of the second day, i.e. about 48 hours after admission, the patient became comatose, and could not be roused. Then positive-pressure breathing enabled him to be roused for short periods, after which he again lapsed into coma. His condition now became critical, and about 56 hours after admission he was comatose with shallow breathing at a rate of 44 per minute and a pulse rate varying between 170 and 200 beats per minute. He was still perspiring profusely. Cyanosis could not be abolished entirely although oxygen was administered intermittently.

As a last resort tracheotomy was decided upon and performed under local anaesthesia. The trachea and larger bronchi were immediately cleared of a large amount of tenacious mucus by a suction catheter passed through the tracheotomy opening. A few moments after air was flowing freely through the tracheotomy opening, apnoea supervened,[7] but fortunately only for a few seconds. Very soon after the tube had been secured in position and oxygen administered through the opening, breathing became quieter and the patient assumed a healthy pink colour, having also stopped perspiring. Two hours later he was soundly asleep for the first time in 60 hours, and his pulse rate was down to 98 beats per minute, although wheezing, rhonchi and bronchospasm were still apparent. The respiratory rate dropped to 30 per minute during the following 12 hours.

Twenty-four hours after the tracheotomy it was possible to discontinue oxygen for long periods without ill effects. Signs of bronchial obstruction and infection persisted for another 3 days, after which there was gradual, but definite improvement. Bronchial secretions were regularly aspirated through the tracheotomy tube, and the patient soon learned to cough effectively with his finger placed over the opening in the tube,[4,7] using the same manoeuvre when he wished to speak above a whisper.

Vigorous antibiotic treatment was continued, and antispasmodics as well as prednisone were administered.

On the 19th day of hospital treatment, after the tracheotomy tube had been removed and the patient had been ambulant for almost a week, he developed a severe manic-depressive psychosis. About 3 years previously he had been treated for the same condition. Despite withdrawal of steroids, there was no improvement in his mental state, and he was transferred to a mental hospital on the 25th hospital day. After a few months, however, he was discharged from the institution well enough to resume his work.

COMMENT

It is possible that oxygen was administered too enthusiastically, especially during the initial stages of the attack, thus precipitating and aggravating the hypercapnia through removal of the only remaining stimulant* of the respiratory centre.[3,10] However, when less oxygen was given cyanosis soon manifested itself, and it became a matter of choosing the lesser of the two evils, i.e. CO_2-retention rather than prolonged anoxaemia.[11]

Steroid therapy probably precipitated the psychosis, but the prolonged anoxaemia and CO_2-narcosis must be considered as accessory factors. This possibility stresses the importance of not postponing tracheotomy too long.

DISCUSSION

The critical measurement of the adequacy of ventilation is really the measurement of alveolar ventilation.[4]

Alveolar ventilation is influenced by variations in respiratory rate, tidal volume, or 'dead space' volume, or by combinations of these factors.[4] When these 3 components are known quantities, the alveolar ventilation rate can be computed.

For practical purposes the equation '(tidal volume − anatomical dead-space volume) × respiratory rate = alveolar ventilation rate' holds more or less true in cases where the anatomical dead-space volume equals the physiological dead-space volume, and where tidal volume is large enough to wash out the respiratory dead space.[4] However, the average figure for respiratory dead-space volume varies between 150 and 200 ml. In bronchial asthma the respiratory dead-space volume may be either normal, increased, or decreased, depending on the degree of emphysema on the one hand, and on the extent of bronchiolar obstruction on the other. It is, however, permissible to use the average figures mentioned if factors such as age, sex, functional residual capacity, and tidal volume are taken into account.[4]

An average value for tidal volume is 500 ml. (400-600) and that for alveolar ventilation is 350 ml. (4·2 litres per minute at a respiratory rate of 12 per minute).[4]

The respiratory rate during the initial stages of an attack of bronchial asthma is slow (if no significant degree of emphysema is present) in an attempt to minimize minute volume, and thus airway resistance, as well as 'gas-trapping'.[4] Due to bronchiolar obstruction (which, of course, is not uniform in degree and extent) uneven alveolar ventilation ensues,[7,8] even though total alveolar ventilation is adequate, with resultant anoxaemia. Increased muscular effort makes a further demand on the already low oxygen reserve.[7] As a result of hyperventilation of the alveoli of non-obstructed bronchioles, CO_2-retention is temporarily postponed.[7] When marked generalized bronchiolar obstruction occurs, however, this compensatory mechanism fails, and CO_2-retention develops with a raised arterial pCO_2. Through respiratory-centre stimulation this results in an increased respiratory rate, which in turn further diminishes the tidal volume,[1] and increases airway resistance (the result of augmented minute volume), creating a greater physiological dead-space volume.[8] The net result of this sequence of events is extreme alveolar hypoventilation with a further increase in arterial pCO_2 (and anoxaemia) until a stage is reached where toxic amounts of CO_2 accumulate in the blood with subsequent depression of the respiratory centre[4] and development of cerebral symptoms — stupor, narcosis and coma. This diminishes the already low tidal volume still more, and a vicious circle is established.

Any procedure which at this stage could increase alveolar ventilation, and thus allow 'escape' of alveolar CO_2, would break the fulminating chain of events.

Again considering the equation [Tidal volume (V_t) − dead space volume (V_{d_s})] × respiratory rate (R) = alveolar ventilation rate (VR_A), it will be seen that with diminishing tidal volumes, alveolar ventilation becomes increasingly insufficient, despite rapid respiratory rates. A few examples will make this point clear:

R	V_T (*ml.*)	V_{DS} (*ml.*)	V_A (*ml.*)	VR_A (*litres per minute*)
12	500	150	350	4·2
12	300	150	150	1·8*
20	300	150	150	3·0*
40	300	150	150	6·0
12	200	150	50	0·6*
40	200	150	50	2·0*

* Ineffective alveolar ventilation

Tracheotomy, by approximately halving the dead-space volume, effectively increases the alveolar ventilation rate,[4,6,9] e.g.:

R	V_T (*ml.*)	V_{DS} (*ml.*)	V_A (*ml.*)	VR_A (*litres per minute*)
40	200	75	125	5·0

Further Advantages of Tracheotomy

Obstruction due to mucus is relieved, since a suction catheter can easily be applied to the trachea and larger bronchi through the opening in the tracheotomy tube.[9] Mechanical obstruction due to the relaxed tongue and pharynx is by-passed.[9]

As a result of improved alveolar ventilation, tachypnoea and violent respiratory excursions are largely abolished. Metabolic rate and oxygen consumption, as well as CO_2-retention, are thus lessened.[7]

Oxygen administration is also made more easy. The disadvantages and some of the dangers of the oxygen tent, the face mask,[10] and the nasal catheter are thus obviated.

CONCLUSIONS

Tracheotomy may be a life-saving procedure in status asthmaticus when conventional methods of relieving respiratory embarrassment have failed.

By diminishing dead-space volume significantly, alveolar ventilation is effectively increased, thus allowing escape of the accumulated CO_2. Clearing of bronchial secretions, also of major importance, is made easier.

Tracheotomy must, however, not be left as a last resort, but should be performed when it becomes evident that the patient cannot clear mucus from his airway, or when symptoms and signs of hypercapnia supervene.[9]

I wish to thank Prof. H. W. Snyman, Head of the Department of Medicine, University of Pretoria, for permission to

* In asthmatic or emphysematous patients the respiratory centre becomes insensitive to CO_2 accumulation. The only remaining stimulant to the centre is anoxia.

publish; Dr. D. J. Franck of the Department of Otolaryngology, who performed the operation; Dr. L. D. Erasmus of the Department of Medicine; and Dr. M. M. Suzman of Johannesburg, for helpful suggestions and criticism in preparing this paper.

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