ASSISTED RESPIRATION IN THE TREATMENT OF NEONATAL TETANUS.

A THESIS SUBMITTED FOR THE DEGREE OF
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A.K. THAMBIRAN.

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CONTENTS

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CHAPTER I.

INTRODUCTION

Tetanus has been the subject of medical writing since the days of Hippocrates but the "scientific era" only began towards the end of the last century when Nicolaier in 1884 discovered the causative organism, Clostridium tetani. Rapid advances followed and within half a century of this discovery tetanus toxoid was produced for active immunisation (Bloomfield 1958).

Now, almost 50 years after the advent of toxoid, it is disquieting that the disease not only occurs but also presents a therapeutic problem in some parts of the world (Pinheiro 1964, Mollaret and Pocidalo 1965, Adams et al. 1966).

HISTORICAL REVIEW

For centuries the high mortality associated with the disease has been accepted. Theories of causation were as varied and numerous as the forms of treatment. Ice-packs, hot baths, infusions of tobacco, mercury and arsenic were used and the patients who did recover "did so in the hands of physicians bold in the use of opium" (Shackleton 1954).

A new concept of the disease followed the work of Nicolaier (1884) who produced tetanus in animals by injecting soil into them and recovered the bacilli from the pus at the site of innoculation. Shortly afterwards, the bacilli were demonstrated in human lesions (Rosenbach 1887) and in 1890 Behring and Kitasato conducted experiments with tetanus toxin (Bloomfield 1958). In the next century, Bruce (1920) showed that prophylactic immunisation with antitoxin reduced both the incidence and mortality of the disease during World War 1.

Within a very short time, following on the work of Descomby (1924), tetanus toxoid was available. Its efficacy was demonstrated in the Second World War (Long 1943).

TETANUS NEONATORUM:

The disease in infants described by Hippocrates about 460 B.C. and others was first called trismus by Galen around 130 - 200 A.D. (Hartigan 1884).

The aetiology of the disease was not understood but Bartram (1793) was probably the first to relate the condition to the umbilicus, "a theory vaguely referred to by Moschison and Levret in the first century" (Hartigan 1884).

Although Hartigan (1884) gave an excellent clinical description, he discounted umbilical infection as the cause since not all infants with umbilical infection developed tetanus and "no nerves had been traced to the cord". He agreed with Sims (quoted by Hartigan) that the condition was caused by "mechanical pressure exerted on the medulla oblongata..." and both reported dramatic cures by measures to relieve this pressure. Various other writers of the period associated the disease with "cold air", "foul air", poor ventilation, poverty and poor hygiene.

Henoch (1889) associated neonatal tetanus with the separation of the umbilical stump and with the rite of circumcision and he concluded that the disease was a form of epilepsy. He reported three recoveries, all infants, of whom 2 received chloral for sedation.

TREATMENT OF TETANUS NEONATORUM.

TETANUS ANTITOXIN.

Although the value of antitoxin in the prophylaxis of tetanus has been established, its value in treatment has been questioned (Drew 1954, Johnstone 1958). However, Brown et al. (1960) and Patel and his co-workers (1963) showed that serum is beneficial in treatment. Laurence and Webster (1963) in a review concluded that serum was "essential therapy" for clinical tetanus and that a single dose of 50,000 units was adequate.

The use of antitoxin in high doses in adults (Cole and Spooner 1935, Boyd 1946) has been reported but Lucas et al. (1966) presented evidence to show that a dose of 500,000 units may be harmful. Furthermore, the results of clinical trials to determine the optimum dose of serum (Brown et al. 1960, Vakil et al. 1963 and 1964, Lucas et al. 1966) indicate that 20,000 units is an "adequate therapeutic dose" (Lucas et al. 1966).

In infants Tompkins (1958) found no difference in the outcome when serum was given in doses ranging from 10,000 to 100,000 units and felt that a dose of 50,000 units was sufficient. Athvale and Pai (1966) showed that antitoxin was of value and that doses of 10,000, 20,000 and 30,000 units produced similar effects. They stated that a single dose of 10,000 units appeared to suffice.

Human Antitetanus Serum:

Recently, Ellis (1963) reported on the use of human antitetanus serum in the treatment of tetanus.

The advantages of human serum are that it is active for longer periods than equine or bovine serum and that sensitivity tests are not needed (Benhardt and Hickey 1966).

CONSERVATIVE TREATMENT:

Conservative treatment of tetanus consists of sedatives, tetanus antitoxin, antibiotics and, in some patients, tracheostomy. When it became evident that recovery from tetanus was complete, treatment was directed towards adequate control of the reflex spasms (Drew 1954). Constant nursing care, minimal stimulation of the infant and maintenance of nutrition were also emphasized (Pinheiro 1957 and 1964, Tompkins 1958, Wilkinson 1961).

Sedatives and Hypnotics.

Barbiturates, chloral hydrate and paraldehyde, singly or in combination, have been used by Jelliffe (1950), Spivey et al. (1953), Tompkins (1958). Wright (1960a) compared the effects of phenobarbitone combined with either chloral hydrate or chlorpromazine. All these workers reported high mortality rates (Table I). The chief drawbacks were that spasms were not always well controlled and the large doses of barbiturates required caused respiratory depression.

TABLE I: THE MORTALITY OF CONSERVATIVELY TREATED NEONATAL TETANUS.

AUTHOR	No. OF CASES.	% MORTALITY
Jelliffe (1950)	25	96
Spivey et al. (1953)	26	77
Pinheiro (1957)	256	77.3
Tompkins (1958)	141	89.6
Wright (1960a)	217	82.5
Wilkinson (1961)	114	57.8
Pinheiro (1964)	238	77
Adams et al. (1964)	198	92

Relaxants.

Mephenesin, with pharmacological properties of skeletal muscle relaxation without loss of consciousness (Goodman and Gillman 1965), appeared to be a suitable drug for the treatment of tetanus. However, the results obtained by Spivey et al. (1953), Pinheiro (1957 and 1964), Wilkinson (1961) using this drug alone or in combination with others showed no improvement in the survival rates (Table I).

Adriani and Kerr (1955), Veronesi (1956), Creech et al. (1957) reported favourably on a combination of mephenesin and chlorpromazine in adult tetanus.

In neonates the disadvantages were the difficulty of maintaining a continuous intravenous infusion (oral administration was attended by unpredictable action) and the danger of haemoglobinuria.

Recently, Hendrickse and Sherman (1966) tried diazepam which is a benzodiazepene compound. This tranquiliser and muscle relaxant was used alone or in combination with phenobarbitone sodium and chlorpromazine. The results showed that diazepam did not reduce the mortality in neonatal tetanus (55% mortality in both groups). The authors concluded that while diazepam relieved tonic spasm (trismus and opisthotonus), it was not very effective against reflex spasms.

Phenothiazine Derivatives.

Following the experimental work of Hougs and Andersen (1954) in which they demonstrated that chlorpromazine abolished local tetanus in a rabbit, Cole and Robertson (1955), Kelly and Laurence (1956), Laurence et al. (1958) used this preparation in adults and children. Although the results of treatment were poor, the relaxant and sedative effects of the drug were apparent.

Laurence et al. (1958) reporting the results of a clinical trial stated that the management of patients treated with chlorpromazine was easier compared with a group treated concurrently with barbiturates. In a similar trial, Adams et al. (1959) used chlorpromazine and a combination of phenobarbitone and chloral hydrate respectively in 2 groups of infants. The

mortality rate in both series exceeded 90%.

TRACHEOSTOMY IN NEONATAL TETANUS.

The clinical picture of severe tetanus - repeated reflex spasms, trismus, inability to cough resulting in accumulation of secretions in the upper airway, rigidity of the thorax, cyanosis and, on occasions, apnoea, associated with laryngeal or generalised spasms - all point to tracheostomy being the rational approach to provide an adequate airway, to prevent lung infection, and to minimise the risk of death from respiratory failure.

More than a century ago, Curling (1837) suggested the operation but it was only in 1856 that Humphrey performed it on a number of patients (Herzon et al. 1951). As the disease came to be recognised as a respiratory problem (van Bergen and Buckley 1952, Shackleton 1954, Ablett 1956, Adams 1964, Mollaret and Pocidalo 1965), tracheostomies were performed on adult patients (Herzon et al. 1951, Forbes and Auld 1955, Mollaret and Pocidalo 1965). However, in spite of several reports in the literature advocating tracheostomy, "there is no conclusive evidence in its favour" (Adams 1964).

In neonatal tetanus, there are a few reports in the English literature pertaining to tracheostomy as an adjunct to sedative therapy. Laurence et al. (1958) in their series felt that tracheostomy could not be performed due to lack of adequate facilities. Furthermore, the operation carried its own risks and there was no evidence to show that tracheostomy improved prognosis.

Spivey and his colleagues (1953) considered this "formidable procedure in young infants" only as a final resort in 7 critically ill patients. There were no survivors.

Wright (1960a) in a random trial treated 17 infants by sedation and tracheostomy and 20 by sedation alone. All infants had severe tetanus and there were no survivors in either group.

Holloway (1966) in another trial confirmed the findings of Wright (1960a) that severe tetanus was primarily a respiratory problem, some 90% or more of these infants developed respiratory failure and that tracheostomy without respirator assistance did not confer any benefit. Mollaret and Pocidalo (1965) stated that "acute or subacute respiratory insufficiency is the immediate cause of death in tetanus".

CONTROLLED RESPIRATION:

Although Vesalius in 1540 "previewed modern anesthetic and respiratory technics by demonstrating intermittent positive pressure respiration through a tracheostomy in an animal..." (Head 1961), it is only in recent years that this treatment has come to the fore in management of respiratory conditions.

The recent interest has largely been due to the successful application of positive pressure respiration during the poliomyelitis epidemic in Copenhagen (Lassen 1953). Since

then there have been several reports describing the use of total paralysis and controlled respiration in the treatment of tetanus in adults and children (Bjorneboe et al. 1954, Honey et al. 1954, Wilton et al. 1958, Glossop and Low 1957, Powell et al. 1958, Garland 1959, Poulsen and Norby 1959, Purkis and Curtis 1965).

Following the important contributions of Donald and Lord (1953), Donald (1954), several workers have employed controlled ventilation in infants (Benson et al. 1958, Heese et al. 1963, Thomas et al. 1965, Reid and Tunstall 1966) mainly for the treatment of the respiratory distress syndrome. Swensson (1962) obtained good results with controlled respiration in the treatment of respiratory failure in children following abdominal operations.

There are few reports on respirator treatment in tetanus neonatorum (Table II). Andersen (1958) was probably the first to treat neonatal tetanus in this manner. In 1950, using decamethonium iodide as a muscle relaxant, he kept alive a 7 day old infant for 6½ days. In recent studies (Table II) d-tubocurarine (curare) has been used to maintain total paralysis.

TABLE II: RESULTS OF RESPIRATOR TREATMENT IN TETANUS NEONATORUM.

AUTHORS	No. OF CASES.	RELAXANT OR SEDATIVE	% MORTALITY
Andersen (1958)	1	Decamethonium iodide	100
Smythe & Bull (1959)	10	Mephenesin Curare, Paraldehyde, Phenobarbitone	70
Wright et al. (1961)	25	Curare	44
Adams et al. (1964)	114	Curare	36
Adams et al (1964)	37	Chlorpromazine, Phenobarbitone	54
Smythe (1963)	20	Curare	20
Present Series	40	Curare	25

The results shown in Table II represent a considerable advance over the high mortality rates obtained by conservative management.

ASSISTED OR AUGMENTED RESPIRATION.

Assisted, augmented or patient triggered ventilation is a form of positive pressure respiration in which the patient's respiratory efforts are increased by a mechanical ventilator (Donald 1954, Pontoppidan 1965).
Golgan et al. (1960), Delivoria - Papadopoulos et al. (1964) used this form of treatment in the severe respiratory distress syndrome of the newborn.

In tetanus neonatorum, this term is used to refer to treatment by intermittent positive pressure respiration without total paralysis. Sedatives, usually chlorpromazine and phenobarbitone, were given to control reflex spasms. Jackson (1962) used this method to "wean" infants from the respirator when curare had been stopped during the total paralysis regime. Using the same technique, Adams et al. (1964) treated 37 patients when controlled respiration could not be offered to them. The results were a considerable improvement over those with conservative treatment but were inferior to those with controlled respiration (Tables I and II).

SUMMARY - THE RESULTS OF TREATMENT.

From the various reports on conservative treatment, it is apparent that no single drug or combination of drugs influenced the mortality significantly. Tracheostomy combined with sedation did not improve the prognosis in severe tetanus neonatorum.

The use of the total paralysis regime represents a major advance in treatment as the mortality has been reduced significantly.
The results of assisted respiration are considerably better than those with conservative treatment but are inferior to those with controlled respiration.

CHAPTER II.

THE PRESENT STUDY

INTRODUCTION:

This thesis is based on a clinical study which compares the management of severe tetanus neonatorum by two techniques :-

(a) Tracheostomy, sedation and assisted respiration;

(b) Tracheostomy and controlled ventilation,

The design of the trial, the treatment in the two groups and the results of therapy are included in this chapter.

The tracheostomy was adapted in both groups for positive-pressure respiration; several problems resulted because of this modification. As proper care of the tracheostomy is one of the most important factors in reducing morbidity and mortality, this aspect together with the procedure of tracheostomy and the complications encountered are discussed in detail (Chapter III).

The other complications occurring during treatment are considered in Chapter IV while the conclusions based on the results of the trial are the subject of Chapter V.

PURPOSE OF THIS STUDY:

The trial of assisted respiration was designed to evaluate its use in the treatment of severe tetanus neonatorum. Encouraging results obtained in the earlier series (Adams et al. 1964) seemed to indicate that the method had several advantages and it was felt that a trial comparing controlled and assisted ventilation was necessary.

In theory, preservation of cough and laryngeal reflexes in infants receiving assisted respiration is an advantage.

Aspiration pneumonia and retained secretions resulting in collapse of lung segments should occur less frequently than in curarised infants. Conscious babies are able to signify respiratory distress by facial contortions and in the event of machine failure can maintain unaided respiration for some time. Measurements of partial pressure of carbon dioxide (Pa CO_2) need not be done routinely as the infant's facial expression can be an index of respiratory sufficiency. Machine failure which must be detected within seconds when the baby is totally paralysed need not be fatal even after 10 or 15 minutes.

During controlled respiration curare is given for 10 days but a state of partial paralysis exists for a further period of 2 to 3 days because decurarisation in infants is a slow process. Some infants are capable of adequate breathing before this time as spasms can be markedly reduced in severity by the eighth day. Thus a further advantage of assisted respiration is that it obviates the need for prolonged respirator treatment since the patients could be allowed to breathe on their own as soon as they were ready to do so.

If the period of artificial respiration was reduced, then extubation, a procedure often attended by difficulties in the infant could be performed earlier. This form of treatment would make less demands on the nursing and medical staff, both of which are invariably insufficient in areas where tetanus occurs commonly.

The Respiratory Unit at King Edward VIII Hospital, Durban, is equipped for the intensive care of 12 patients but

often twice this number are accommodated because of admissions from peripheral hospitals and clinics. It is not possible to offer controlled respiration to more than 6 patients at a time because of the limited staff. The number of admissions during the period of this study is shown in Table III.

TABLE III: NUMBER OF PATIENTS ADMITTED DURING 1963-1965.

PERIOD	TETANUS		TOTAL
	INFANTS	CHILDREN AND ADULTS	
1963	142	54	196
1964	114	62	176
1965	120	50	170
	376	166	542

DESIGN OF TRIAL:

Selection of Patients.

Only infants with frequent and severe, or continuous reflex spasms (Wright 1960 b), or cyanosis associated with spasms, as judged by 2 or more observers were admitted to the trial. The majority of patients in this series satisfied these criteria on admission. A few infants in whom the disease progressed from the moderately severe to the severe form within 24 hours of admission were also selected for this

study. Premature infants were included although it was realised that prematurity carried its own hazards besides the severe tetanus.

Age, Periods of Incubation and Onset.

Umbilical infection occurring at birth or shortly thereafter was presumed to be the source of infection. The average ages, periods of incubation and onset set out in Table IV show that the infants in both groups were closely matched. Further clinical data relating to each patient are included in the Appendix, page i.

TABLE IV: AVERAGE AGE, DURATION OF INCUBATION AND ONSET IN THE TWO GROUPS.

GROUP	NO. OF PATIENTS	AGE (DAYS)	AVERAGE INCUBATION (DAYS)	ONSET (HOURS)
CONTROLLED RESPIRATION	40	6.9	5.8	11.6
ASSISTED RESPIRATION	20	6.5	5.7	8.7

Randomisation.

When a patient was chosen for the trial, treatment was determined by opening the next of a series of sealed envelopes containing cards randomised between the two treatment groups. Forty infants were assigned to the controlled

respiration group and 20 to the trial of assisted respiration. A provision was made for changing the treatment to controlled ventilation if uncontrolled spasms, cyanosis, apnoea or lung infection occurred during assisted respiration. It was felt that these complications could influence the prognosis adversely. Apnoea was defined as spontaneous breathing not being resumed when respirator assistance was discontinued at intervals during the trial of assisted ventilation.

STANDARD TREATMENT IN THE TWO GROUPS:

Tracheostomy:

Tracheostomy was performed on all patients in the two groups. The operation and its complications are discussed in Chapter III.

Drugs:

Initial sedation for all infants consisted of either 10 or 12.5 mg. chlorpromazine and 66 mg. phenobarbitone. A single dose of 50,000 units of tetanus antitoxin was given (Laurence and Webster 1963). Procaine penicillin 150,000 units was given daily for 10 days but other antibiotics were used when indicated. Chlorpromazine, phenobarbitone, tetanus antitoxin and penicillin were given by intramuscular injection.

Feeding:

The mothers of the patients were admitted as boarders and assisted in their care. The infants were fed with expressed breast milk at 3-hourly intervals through polyvinyl tubes. During the first two weeks 2 oz. per lb. body weight per day was given, thereafter the feeds were

increased to 2½-3oz. per lb. per day. The feeds were reduced during the acute phase of the illness to prevent abdominal distension and gastric stasis.

Before each feed, the gastric aspirate was measured and returned as part of the feed. The remainder was made up of breast milk. If abdominal distension or gastric stasis was present, a reduction in the volume, or omission, of a feed was tried. If no improvement resulted, intravenous fluids were given and the milk feeds discontinued. Poor absorption (high gastric residue) was diagnosed if a third or more of the total 24-hour feed was aspirated.

Intravenous fluids were given through a scalp vein using a short bevelled, fine bore needle attached to an infusion set. The needle was held in place by strips of plaster of Paris. The fluids administered consisted of a 5% glucose - 0.2% saline mixture alternating with one containing 5% glucose, 0.2% saline and potassium (15 mEq./L). A total of 300 cc. was given over the 24 hour period. Gastric aspiration was continued during the period of intravenous feeding. After each 24-36 hour period, milk feeds in small quantities (1 oz. 3-hourly) were given. As absorption improved, the quantity of milk per feed was gradually increased and intravenous therapy was discontinued.

From the second week onwards when severe spasms had ceased the infants were offered milk feeds from a Belcroy feeder (Mann et al. 1963). Both the sucking and swallowing reflexes were impaired at this stage but improved over the following fortnight so that most of the feeds were taken from the bottle. The nasogastric tube was retained until

this time. After removal of the tracheostomy tube, breast feeding was started again and was well established on discharge.

Position of the Infant during Artificial Ventilation:

The infant was placed in a cot with a rolled napkin under his neck so that his head was at a lower level than his trunk (Fig. 1). This position prevented pharyngeal contents flowing into the trachea. The patient's position was changed hourly using the supine, right and left lateral positions in rotation.

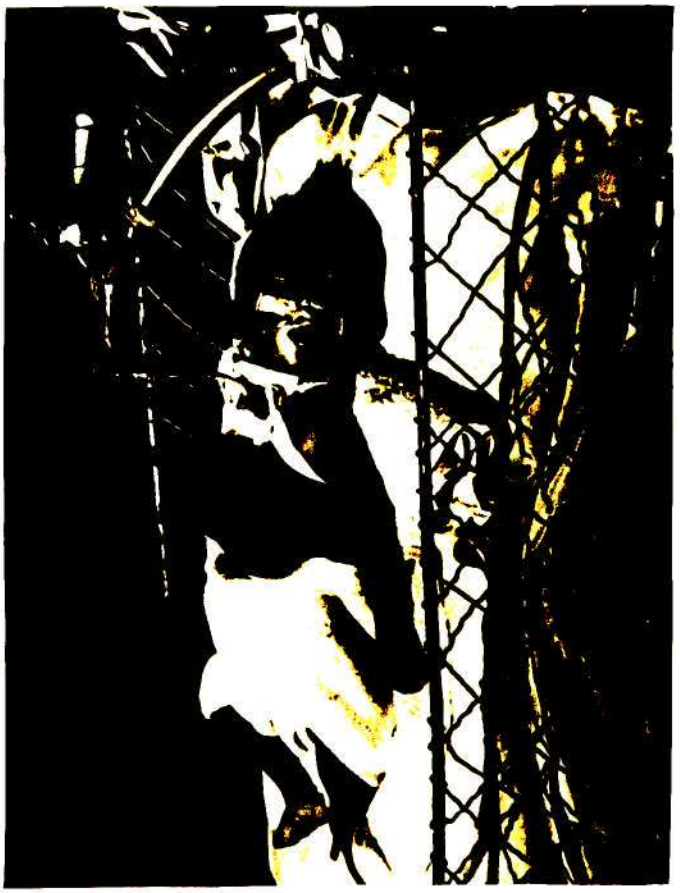
Adequacy of Ventilation During Treatment:

Adequate respiration was judged clinically by good bilateral chest excursions and air entry, and the absence of cyanosis. Measurements of Pa CO_2 and partial pressure of oxygen (Pa O_2) were also used as indicators. The inflation pressure and the rate of the respirator were set to maintain the Pa CO_2 between 25 - 40 mm.Hg. Initially, Pa CO_2 values were determined by a rebreathing method (Campbell and Howell 1960) as modified for infants by Sykes (1960a). Later in the trial blood gas analysis by a microtechnique (Siggard - Andersen et al. 1960) was possible. Pa O_2 estimations were performed using a microtechnique (Desai et al. 1966). These procedures were undertaken regularly during the first ten days of treatment.

Partial pressures of carbon dioxide in the arterial blood were easily maintained but oxygen pressures were low when infants were ventilated with air (Holloway et al. 1966).

Other workers have made similar observations (Campbell et al. 1958, Pontoppidan 1965, Fletcher and Bunker (1966). Because of these observations the oxygen concentration of the inspired air was increased from 21 to 35%.

FIGURE 1 : POSITION OF THE INFANT DURING ARTIFICIAL VENTILATION. THE HEAD OF THE INFANT IS AT A LOWER LEVEL THAN HIS TRUNK.



THE TRIAL OF ASSISTED RESPIRATION.

Assisted respiration was begun in the 20 infants in this group but was terminated in 12 patients because of complications mentioned earlier. The subsequent management of these patients is discussed separately ("Failed" Assisted Respiration). Treatment by assisted respiration alone was possible in only 8 patients.

Technique of Assisted Respiration:

After tracheostomy the respirator was connected to the tracheostomy assembly with the screw cap (Fig. 2, page 28) removed or replaced with a perforated cap. The ventilator was set to generate 15 - 20 cm. water pressure at rates between 25 - 35 cycles per minute. Although the volume of air delivered during each cycle was reduced by the leak in the screw cap, the respirator continued to inflate the lungs. The patient adapted himself rapidly to an imposed pattern of ventilation slower than his own and continued to breathe in between the machine inflations. At intervals during the day, the respirator was disconnected to observe whether the infant could maintain spontaneous respiration.

Chlorpromazine 10 or 12.5 mg. supplemented by phenobarbitone 33 or 66 mg. was given to control the reflex spasms. Chlorpromazine was given regularly at 4-6 hourly intervals but phenobarbitone was used only when spasms were not adequately controlled during the first 5-6 days of treatment. The dose of chlorpromazine was reduced as spasms became less frequent and less severe.

During this time weaning from the respirator was tried. The ventilator was stopped if the patient's breathing appeared to be adequate. He was observed by a nurse sitting at the bedside. If cyanosis, severe rib recession or apnoea occurred, his respiration was aided for another 24 hours before a further attempt at weaning was made.

"FAILED' ASSISTED RESPIRATION.

Reasons for Failure of Assisted Respiration.

Assisted ventilation failed in 9 patients because severe or continuous reflex spasms persisted despite sedation. In addition to the spasms, apnoea, cyanosis, or tachycardia had been observed. Bronchopneumonia in 2 patients and cyanosis, flaccidity and bradycardia in one other were reasons for the commencement of controlled respiration (Table V).

Management of "Failed" Assisted Respiration.

Chlorpromazine and phenobarbitone used during assisted respiration were discontinued. Total paralysis was induced and maintained by intramuscular injections of curare, the frequency of administration being determined by appearance of muscular twitchings. A dose of 7.5 mg was given at about eight hourly intervals and an additional 2.5 mg in between these periods if required. Curare was discontinued on the 10th or 11th day after tracheostomy but for some 36-48 hours after the last dose the infant remained partially paralysed. This was probably due to delayed absorption of curare from the indurated injection sites. Mild or moderate reflex spasms occurred after the effects of curare were no longer apparent and these were controlled satisfactorily by chlorpromazine given orally.

Assisted respiration was commenced as soon as efforts at spontaneous breathing were seen. Weaning from the respirator was usually attempted after about 5-6 days of assisted ventilation.

TABLE V: REASONS FOR FAILURE OF ASSISTED RESPIRATION IN THE PRESENT STUDY.

SERIES NUMBER	DURATION OF ASSISTED RESPIRATION	REASONS FOR FAILURE OF ASSISTED RESPIRATION	FATE
6	10 days	Bronchopneumonia Poor general condition	S
10	4 hours	Continuous reflex spasms Cyanosis	S
18	3 days	Severe reflex spasms Cyanosis	D
21	8 hours	Severe spasms Tachypnoea	S
25	3 hours	Continuous reflex spasms Cyanosis	D
32	4 hours	Continuous reflex spasms	S
34	24 hours	Continuous reflex spasms Cyanosis	S
39	3 days	Cyanosis, flaccidity Bradycardia	D
45	3 days	Continuous reflex spasms	D
50	4 days	Continuous reflex spasms Apnoea	S
56	8 days	Bronchopneumonia, tachypnoea Costal recession	D
57	24 hours	Severe spasms, cyanosis, Bilateral bronchopneumonia, Bradycardia	S

S = SURVIVED
D = DIED

CONTROLLED RESPIRATION:

Forty patients were managed by the total paralysis regime (Mann et al. 1963) instituted immediately after tracheostomy had been performed. The nursing care was identical to that in the assisted respiration group.

RESULTS OF THE PRESENT STUDY: (TABLE VI)

TABLE VI: MORTALITY RATES IN THE PRESENT STUDY.

TREATMENT GROUP	NUMBER OF PATIENTS.	NUMBER OF DEATHS.	% MORTALITY	
ASSISTED RESPIRATION GROUP:				
(i) Sedation and Assisted Respiration	8	5	62	50
(ii) "Failed" Assisted Respiration	12	5	42	
CONTROLLED RESPIRATION	40	10	25	
T O T A L	60	20	33	

The combined mortality in the assisted respiration group was 50% (10 deaths). Of the 8 infants who were treated by sedation and assisted respiration, 5 (62%) died. There were 5 deaths (42%) when the treatment was changed from assisted to controlled ventilation.

In the group treated by controlled ventilation, there were 10 deaths (25%).

The overall mortality in this study was 33% (20 deaths). The difference in mortality between assisted and controlled respiration groups was not statistically significant ($p > 0.05$).

CHAPTER III

THE TRACHEOSTOMY DURING ARTIFICIAL RESPIRATION

TRACHEOSTOMY PROCEDURE:

Patients were given 25 mg. succinylcholine, inflated with oxygen and the tracheas intubated with 3.0 mm red rubber tubes. Controlled ventilation was started using the East Radcliffe, Bennett or Smith-Clarke respirators set to generate 10-15 cm. water pressure at rates between 30 - 40 cycles per minute. By this procedure, the infants were effectively ventilated during tracheostomy without the interference of reflex spasms.

The tissues overlying the trachea were infiltrated with a 1% solution of local anaesthetic. The trachea was exposed by a midline vertical incision. Both high and low tracheostomies were performed. Two or three tracheal rings were divided vertically and the oral endotracheal tube withdrawn a little to allow insertion of the tracheostomy tube. The oral tube was retained until the tracheostomy tube was secured. After introduction of the tracheostomy tube, the metal connector of the delivery tubing from the respirator was connected to the "T" piece of the tracheostomy assembly (Fig. 2). A dressing interposed between the clamp and the wound was changed whenever it became soiled.

The tracheostomy tube was held in a clamp (Fig. 2), the design of which was modified from one used in earlier trials in this unit (Sykes 1960b). The clamp was secured around the neck by two harnesses (made from gauze enclosed in

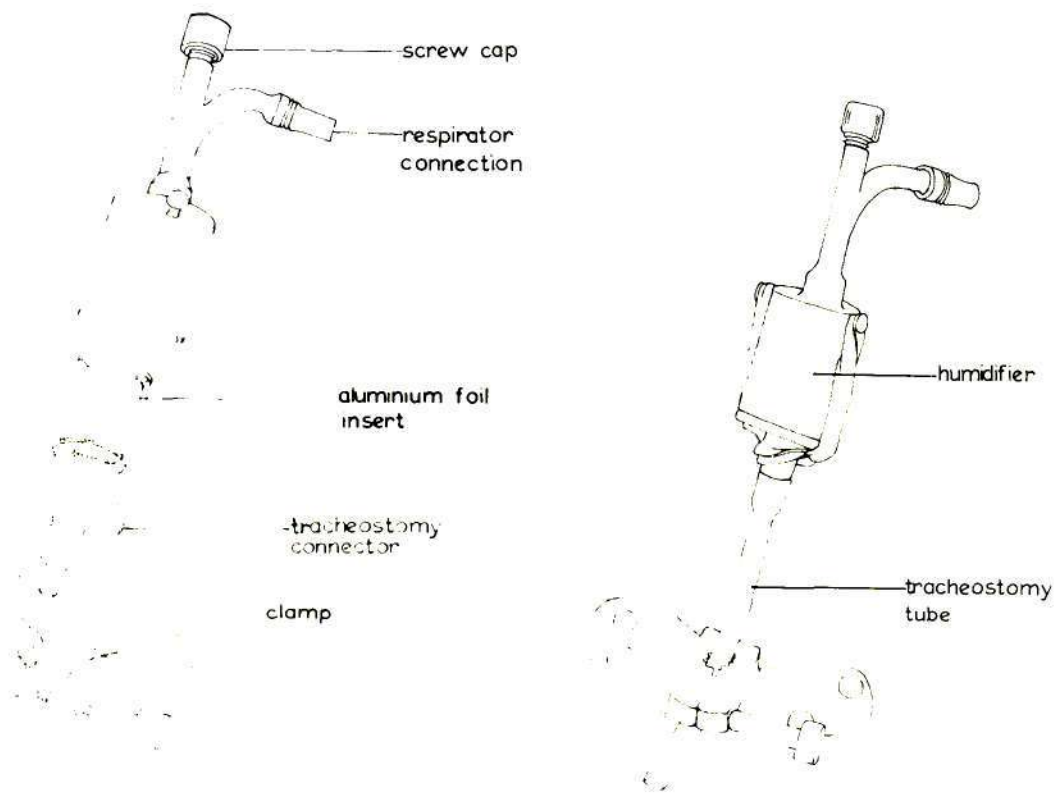
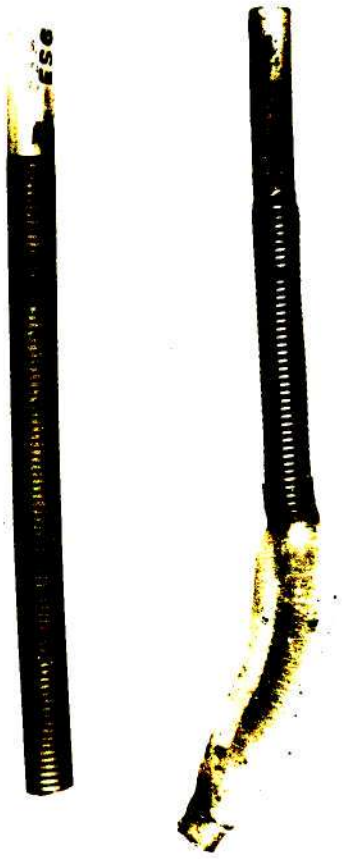


FIG. 2. THE TRACHEOSTOMY ASSEMBLY USED IN THE PRESENT TRIAL.



waterproof strapping), the first passed around the neck and the second over the shoulders and around the front of the chest in each axilla. Linen tape was used to fasten the harness to the clamp.

Position of the Tube in the Trachea.

The nylon reinforced tracheostomy tube (Fig. 3) was 6.5 cm. long. About 2 cm. projected above the clamp, about 2.0 to 2.5 cm. passed through the clamp and soft tissues of the neck and the remaining 2.0 cm. lay in the lumen of the trachea. Thus the length of tube within the trachea could be gauged by measurement between the clamp and the modified humidifier (Fig. 2). The tube at its point of entry into the clamp was circled with ink so that subsequent slipping could be recognised easily and corrected.

The tube was positioned to provide adequate bilateral chest inflations. Careful observation of chest expansion and auscultation confirmed that the tube was correctly placed. Transmitted sounds from the right lung were often heard on the left even when the tracheostomy tube was deliberately inserted into the right main bronchus. With the tube positioned correctly above the carina, breath sounds over the right lung were often louder than those over the left lung.

Tracheostomy Tubes during Artificial Ventilation.

During this period, a short, straight uncuffed nylon reinforced latex tube (Fig. 3) with an internal diameter

of 3.0 mm or 3.5 mm was used (Sykes 1960 b). This tube though not entirely satisfactory was used because it was readily available. Tracheostomy tubes were changed only when necessary; when the upper non-reinforced part frayed, or if it obstructed or if the tube did not form a satisfactory seal in the trachea. In most infants a larger tube was required after about a week because air escaped from the lower trachea into the pharynx.

Tracheostomy tubes in the Recovery Period.

When the patient was able to breathe on his own, a metal tube replaced the nylon reinforced latex tube. A "Parker" 16 F.G. was suitable for the majority of infants. In a few patients the shaft of the tube was shortened to prevent intubation of the right main bronchus. This problem was encountered only in infants on whom a low tracheostomy had been performed. Another disadvantage was ulceration of the anterior tracheal wall because of bad alignment of the tube. The metal tube was retained until extubation was achieved and until then it was changed at weekly intervals.

Tracheal ulcers caused by the nylon reinforced tube healed considerably because the metal cannula overlay the ulcer. The suction catheter also was not inserted for the same distance as during positive pressure respiration because in the convalescent phase, the infants coughed up their secretions and aspiration beyond the tracheostomy tube was unnecessary.

A cuffed tracheostomy tube for infants (Fig. 3).

In an attempt to reduce the complications of the uncuffed nylon reinforced tracheostomy tube, a tube with a self-inflating cuff was designed (Holloway and Thambiran 1966). A loose latex skirt was fixed to a metal reinforced latex tube. The distal end of the tube (enclosed in the cuff) was curved and had multiple perforations which connected the lumen with the skirt. The curve was maintained by making the anterior surface of the cuff shorter than the posterior. The cuff inflated automatically with each respirator inflation and deflated partly during expiration. With this design it was hoped to reduce tracheal ulceration as only the cuff would be in intermittent contact with the tracheal mucosa. Gas escaping from the lower trachea into the mouth during inspiration would also be prevented. The use of this tube is discussed later (page 55).

HUMIDIFICATION:

Humidification was considered important because the normal moistening and warming mechanisms of the upper airway was by-passed. In this series of patients humidification was maintained by passing the inspired air over heated water reservoirs and by a modified heat and moisture exchanger (H.M.E) in the tracheostomy assembly (Toremalm 1960). The H.M.E. consists of 2 layers of rolled aluminium foil, one is flat and the other corrugated. During expiration water vapour condenses on the foil and this is used again to moisten the inspired air. The aluminium foil inserts (Fig. 2) encased in plastic cylinders were changed daily.

After respirator treatment, when a metal tracheostomy tube was in place prior to extubation, humidification was difficult to achieve. Insertion of a fitting for the H.M.E. into the tracheostomy increased resistance to breathing and frequently fell out. It was not possible to nurse all recovering infants in high humidity tents and suitable tracheostomy masks were unavailable. During this period, installation of 1 - 2 cc. normal saline into the tracheostomy proved useful in the aspiration of thick secretions.

PHYSIOTHERAPY AND TRACHEAL SUCTION.

Physiotherapy to the chest is considered essential to assist the movement of mucus and bronchial secretions from the smaller bronchi into the trachea whence they can be removed by aspiration. Physiotherapy consisted of clapping, compression of the chest and tracheal suction to remove secretions (Spalding and Smith 1963). Normal saline introduced into the tracheostomy aided the aspiration of thick secretions. Nurses were trained to use the stethoscope so that they could assess the efficacy of the procedure. Physiotherapy and suction was ordered hourly but the limited staff were able to perform this only 18 - 20 times in 24 hours.

Tracheoscopy (Smythe 1963) was performed if rhonchi persisted after suction or if the breath sounds in one lung (usually the left) were diminished. The tracheostomy tube was removed and an auriscope was inserted into the stoma. By this technique, both main bronchi were easily seen and the

suction catheter could be directed to the obstructed bronchus. Intermittent inflation of the lungs was continued by inserting the tracheostomy tube (still connected to the respirator) into the auriscope held in the stoma.

BRONCHIAL SECRETIONS DURING TREATMENT.

Bronchial secretions were scanty during the first 24 hours. This was probably due to dehydration prior to admission. The secretions aspirated after 24 hours were usually mucoid and thin. Manipulation of the tracheostomy increased the quantity of secretions. Attempts at oral feeding during the recovery period had a similar effect. Purulent secretions indicated infection, either tracheal or bronchopneumonic. Secretions were usually blood stained when ulceration of the trachea (due either to repeated aspiration of secretions or to trauma by the tracheal cannula) was severe.

COMPLICATIONS OF TRACHEOSTOMY. (Fig. 4 and Appendix, page xiv).

The problems encountered in this study were common to both assisted and controlled ventilation.

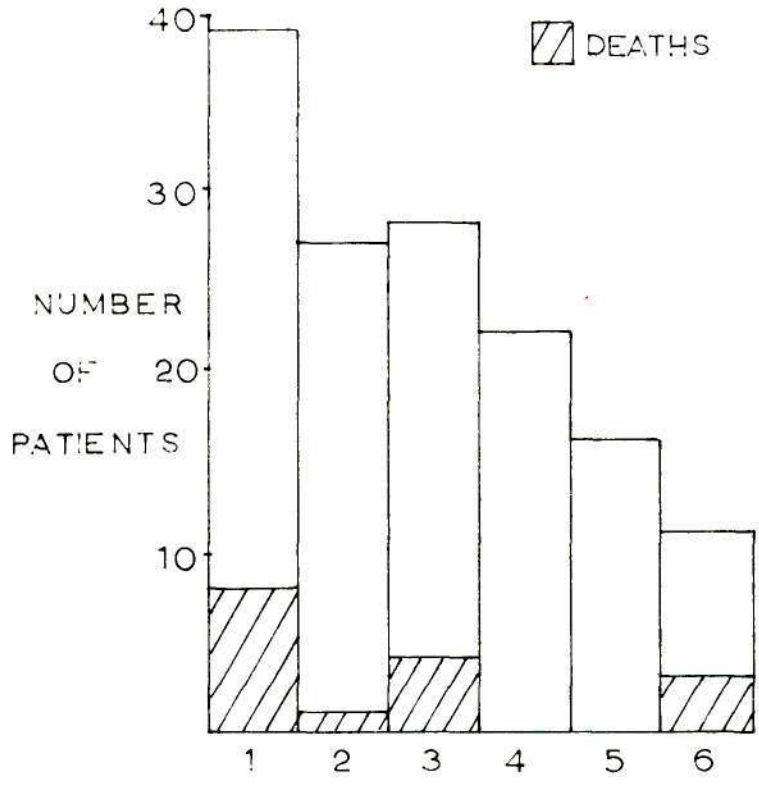
TRACHEAL ULCERATION.

Ulceration Caused by the Tracheostomy Tube.

The clamp held the tracheostomy tube at an angle of 45^{o} to the horizontal; a further angle resulted when the tube curved to enter the trachea (Fig. 5). Because of the induced angle and the tendency for the tracheostomy tube to

FIG. 4. COMPLICATIONS OF TRACHEOSTOMY

During assisted, "failed" assisted and controlled respiration.



1. Respiratory complications
2. Airway obstruction
3. Displaced tracheostomy tube
4. Severe tracheal ulceration
5. Delayed extubation
6. Cyanosis during tracheal suction

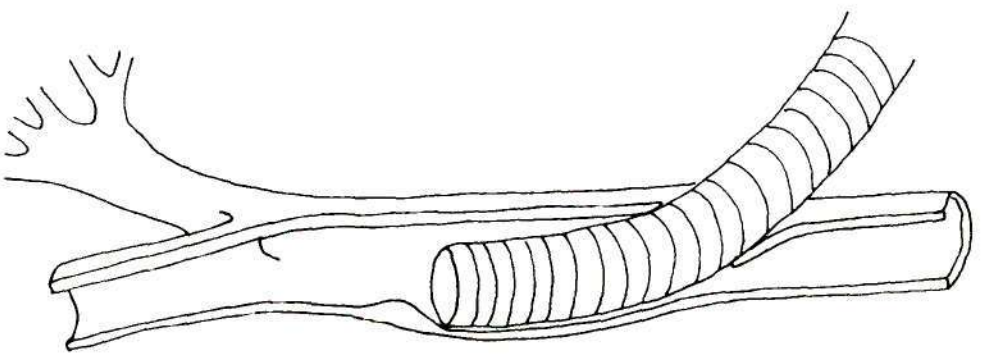


FIG. 5. THE EFFECTS OF THE NYLON REINFORCED TRACHEOSTOMY TUBE

a. INWARD COMPRESSION OF THE ANTERIOR TRACHEAL WALL.

b. INDENTATION OF POSTERIOR TRACHEAL WALL.

c. MUCOSAL "HEAPING."

straighten, the posterior wall of the trachea was consistently ulcerated and indented. These changes were observed during tracheoscopy. Within 24 hours of tracheostomy, erythema was present; this proceeded to indentation and ulceration within a period of 3 to 5 days. The depression was interpreted as the result of continuous pressure on the posterior tracheal wall.

The distal end of the tracheostomy tube rested in the depression and sometimes impaired expiration which was then characterised by a wheeze during this phase of respiration. Another effect was obstruction to the passage of the suction catheter past this depression (Fig. 5). If this was not appreciated by the nursing staff, secretions accumulated beyond the distal end of the tracheostomy tube. If the tube was positioned beyond the depression, then the wheeze disappeared and tracheal suction was easier.

Prolonged intubation resulted in "heaping" of the mucosa beyond the tracheostomy tube (Fig. 5). Squamous metaplasia (Fig. 6) of the ciliated coloumnar epithelium of the trachea was observed on histological examination in several infants.

In very severely ulcerated tracheas, the tracheostomy tube was often found to be lying in a groove aligned with the right main bronchus. This had the effect of isolating the left main bronchus which appeared to be at a higher level than the right.

Another site of trauma was the superior end of the tracheostome. This portion was buckled inferiorly by the tube as it curved to enter the lumen of the trachea (Smythe 1964). This had the effect of weakening the anterior tracheal wall and reducing the lumen in this area (Fig. 5).

Tracheal granulations were encountered usually during the third or fourth weeks, these probably occurred as a reaction to the accumulations of foreign material e.g. infected secretions in areas where the tracheal mucosa was damaged or absent. The common sites for granulomata were the lumen of the trachea at the distal end of the tube and the inferior edge of the tracheostome.

Tracheal weakening, evidenced by severe inspiratory tracheal collapse (observed at tracheoscopy), was seen in several patients especially those in whom decannulation was delayed. In some of these infants, the collapse was severe enough to cause complete obstruction of the airway when the support of the tracheostomy tube was removed.

Severe haemorrhage (either from the tracheal ulcer or from arterial erosion), tracheal perforation or tracheo-oesophageal fistula were not observed in this series.

Ulceration caused by tracheal suction.

In addition to the trauma caused by the tracheostomy tube, the suction catheter can also cause ulceration (Plum and Dunning 1956, Watts 1963, Thambiran and Ripley 1966). If the tube was positioned correctly, any ulceration at the carina or

in either main bronchial orifices was almost certainly due to the suction catheter. Repeated suction because of thick or excessive secretions often caused bleeding.

GRADING OF TRACHEAL ULCERATION:

Ulceration was classified as follows :-

(1) Mild - erythema and minimal ulceration.

(2) Moderate - mild ulceration and indentation of the posterior tracheal wall.

(3) Severe - ulceration of anterior and posterior tracheal walls, moderate to severe indentation of the posterior wall, presence of granulomas, excessive tracheal collapse, buckling of the superior end of the tracheostome.

FIGURE 6: (SERIES No. 16)

Photomicrograph showing the severity of tracheal ulceration after intubation with the uncuffed tracheostomy tube for 14 days.
There is ulceration of the mucosa with an intense inflammatory reaction deep to it. The mucosa adjoining the ulcer is suggestive of early squamous metaplasia. Sara (1965) observed that squamous metaplasia was evident within three days of tracheostomy if humidification was inadequate.

H.E. X 25

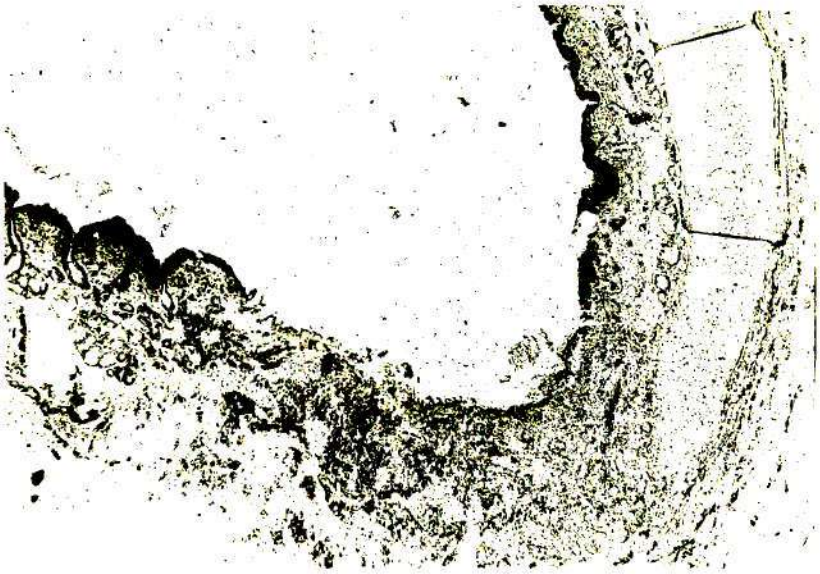


FIGURE 7: ("NON-SERIES" PATIENT)

This photograph shows the extent of ulceration following intubation with the cuffed tracheostomy tube for 7 days. There is pronounced vascularity and a marked inflammatory infiltrate in the mucosa and submucosa. There is a 'pseudo-papilloma' of granulation tissue with a mucosal stalk projecting into the lumen of the trachea.

H.E. X 25

PRECAUTIONS TAKEN TO MINIMISE ULCERATION AND INFECTION:

The nursing staff aspirated tracheal secretions in as sterile a manner as possible. They scrubbed their hands prior to tracheal suction and used sterile equipment. Soft rubber suction catheters (No. 3 F.G.) with side-holes were used for each session. Separate catheters were employed for nasal and pharyngeal aspiration.

The suction catheters were marked 7.5 cm from the distal end to prevent their insertion beyond the tracheostomy tube. In this way the catheter end could not occlude a small bronchus or cause ulceration of the carina and the main bronchi.

Tracheal aspiration can never be a totally aseptic procedure in infants. The use of soft catheters and the need to aspirate the trachea quickly to avoid anoxia precluded the use of a non-touch technique.

Satisfactory asepsis was achieved if two nurses were present during suction; one disconnected the patient from the respirator while the other aspirated the tracheostomy.

The negative pressure on the suction machine was controlled to minimise collapse of lung segments during suction. Efficient suction was obtained with a subatmospheric pressure of 7.5 cm water.

The suction apparatus was designed so that negative pressure was not exerted when the catheter was inserted into the

tracheostomy. A "Y" piece was introduced between the catheter and the latex tube leading to the suction bottles. One limb of the "Y" connection was open to the atmosphere and occluded only during aspiration (Plum and Dunning 1956). This measure was adopted to ensure that negative pressure was applied only during the phase of suction.

COMPLICATIONS OF TRACHEAL SUCTION.

It was impressed upon the nursing staff that prolonged suction could be fatal and they were instructed to limit the duration of suction to 15 seconds. Severe anoxia and death are known complications of tracheal suction (Fineberg et al. 1960, Rosen and Hillard 1962, Hollinger et al. 1965). Anoxia and a sudden increase in venous return to the heart (Rosen and Hillard 1962) are postulated as the mechanisms causing death while vagal stimulation in addition to hypoxia are the mechanisms favoured by Fineberg et al. (1960). Jacoby et al. (1955) showed that severe cardiac irregularities occurred during tracheal intubation in the presence of hypoxia. Similar mechanisms may operate during tracheal suction.

Tracheal suction was associated with cyanosis in 8 patients and with death in another 3 infants. Of the latter 3, bronchopneumonia, patchy collapse and oedema were demonstrated in 2 infants at necropsy. It is felt that fatal anoxia in these patients was contributed to by the abnormal state of the lungs.

INFECTION AS A COMPLICATION OF TRACHEOSTOMY.

Infection around the tracheostome and in the lungs is difficult to prevent in spite of aseptic tracheal toilet (Pearce 1961, Stiles 1965).

LOCAL:

Mild to moderate infection around the tracheostomy was present in every infant during respirator treatment. This improved when the metal tube was in place and later when extubation was done.

PULMONARY INFECTION:

Diagnosis: In addition to the difficulties of auscultation of the lungs described earlier, the diagnosis of bronchopneumonia was further complicated by the absence of fever in many infants. This is not an uncommon observation both in premature infants (Crosse 1957) and in severely ill full term infants (Mann and Elliott 1957). Diagnosis, therefore, had to be made when abnormal chest signs (diminished air entry, crepitations and rhonchi) persisted after physiotherapy and if the tracheal aspirate became purulent. The value of radiography was limited because a portable unit had to be used and many films were not of diagnostic quality. Although two thirds of the patients in this study were subjected to chest radiography, definite evidence of bronchopneumonia, lobar pneumonia and lung cyst (1 patient) was obtained in only 15 patients. In another 9 infants pulmonary infection was confirmed at post mortem.

Predisposing factors: Tracheostomy and intubation of the trachea for varying periods, repeated suction and impaired ciliary action undoubtedly contributed to infection in the lungs. Recently, evidence to suggest that sepsis of the tracheostomy is a common cause of pulmonary infection has been presented (Bolton 1966).

Organisms cultured: Staphylococcus pyogenes, Bacillus proteus and coliform organisms were cultured from the bronchial secretions.

Antibiotics used: In the majority of infants, a satisfactory response was obtained when chloramphenicol, erythromycin or a combination of these preparations with streptomycin was used.

MECHANICAL FAULTS ASSOCIATED WITH TRACHEOSTOMY:

Misplaced tracheostomy tubes: The complication of a partially or totally dislodged tracheostomy tube occurred 42 times in 27 patients in the whole group. Displacement of the tube into the right main bronchus is also included in this section. In all but 6 infants cyanosis of varying degree ensued. The dislodgement was fatal in 2 patients.

In one infant, the humidifier - tracheostomy connection worked loose and resulted in death.

Obstructed tracheostomy tubes: Thick bronchial secretions, associated either with infection or inadequate humidification, resulted in partial or complete obstruction of the tracheostomy and was encountered 39 times in 19 patients. It was fatal on only one occasion. In several infants who died, the possibility of obstruction could not be ruled out because the nurse had already aspirated the tracheostomy during an emergency before a doctor arrived.

Obstruction of the main bronchi: This was observed in 3 patients, in one patient both main bronchi and in 2 the left main bronchus were filled with secretions.

EXTUBATION:

Hollinger et al. (1965) stated that delayed extubation was the most common problem of tracheostomy in infants under one year. Venables (1959) and Oliver et al. (1962) estimated that this complication occurred in about 10% of infants. Because the incidence of other complications increases with prolonged intubation, removal of the tracheostomy tube as soon as the need for it is past is advocated (Oliver et al. 1962). The various causes of difficulty in extubation mentioned by different workers are listed in Table VII.

TABLE VII: REASONS STATED IN THE LITERATURE FOR DIFFICULTY IN EXTUBATION.

AUTHORS		CAUSES
Crooks	(1954)	Tracheal collapse
Bigler et al.	(1954)	Granulation tissue
Reading	(1958)	Stenosis above the tracheostome
Venables	(1959)	Tracheal collapse
Smythe	(1964)	Granulation tissue. Angulation of the trachea. Buckling of tracheal rings above tracheostome

It can be seen that some form of mechanical obstruction of the airway was implicated in every patient (Table VII); in addition the "mental element" (Crooks 1954, Jaffe 1963) probably also plays a role. Breathing through a tracheostomy is easier for the neonate and he experiences difficulty when this is occluded.

Technique of Extubation:

The first attempt at extubation was made some 48 to 72 hours after spontaneous respiration was established. This was done when the infant had settled after a morning feed so that he could be observed closely for the rest of the day.

After suction the tracheostomy tube was removed, the stoma occluded and the patient stimulated to cry. If his respiration was satisfactory, the stoma was closed with a piece of waterproof strapping. Cyanosis, stridor and rib recession before or after stimulation were contra-indications to extubation but mild recession alone was not. The maintenance of a good colour and adequate respiration during crying were valuable criteria of extubation being successful. A good cough reflex usually indicated that airway obstruction was unlikely to occur later.

A sterile tracheostomy tube was strapped to the cot for emergency use. The mother or a nurse observed the patient's progress continuously. Suction of the pharynx and the stoma (if required) was done periodically. A half-hourly record of pulse, respiration and costal recession was made and

used to assess progress. Feeding was carried out by the nursing staff for the next 24 hours, the period during which cyanosis and apnoea occurred frequently. If this period was uneventful decannulation was likely to be successful.

If the first attempt was unsuccessful, further attempts were made at weekly intervals for a month, then fortnightly for a further month and finally at monthly intervals.

This technique of extubation required the infant to adapt rapidly to laryngeal breathing. In older children, insertion of smaller cannulae before extubation results in a more gradual change-over from tracheal to laryngeal breathing. Jackson and Jackson (1959) advocated graded occlusion of the tracheal cannula to encourage laryngeal breathing; when the infant was able to breathe satisfactorily in spite of the completely obstructed tube, it was felt that he was ready for extubation.

This method was not adopted in this trial. Obstruction of the cannula increases the resistance to breathing as only the limited space around the occluded tube is available for laryngeal breathing. If secretions accumulate around the tube the available airway is reduced even more. Furthermore, the ability of the patient to maintain effective respiration in the presence of an occluded tracheostomy cannula does not necessarily indicate that extubation will be successful. Inspiratory collapse of the trachea can still occur when the support of the cannula is removed.

Accidental Extubation:

Dislodgement of the tracheostomy tube when the infant was ready to tolerate extubation (accidental extubation) occurred in 6 infants. Decannulation was successful in all 6 patients.

Emergencies after extubation:

Cyanosis often occurred during or after a feed, during crying or because of secretions in the trachea. It was not unusual to aspirate some milk from the pharynx after a feed. This, together with a depressed laryngeal reflex, made inhalation of pharyngeal contents a potential danger.

The role of residual stiffness in tetanus as a contributory factor in causing cyanosis could not be assessed. Some infants with marked stiffness did not become cyanosed while others with less stiffness had several cyanotic attacks.

Secretions often accumulated in the trachea after removal of the tracheostomy tube. In these infants the cough reflex was not strong enough to expel secretions and the open stoma further reduced the efficacy of this reflex.

Stomal and subcostal recession were often present after extubation. The former was probably due to tracheal weakening and the fact that the trachea was attached to the skin by scar tissue. Thus with every deep or partly obstructed inspiration, the stoma was drawn in. Costal

recession was thought to be due to obstruction of the airway by secretions or granulomata or by tracheal collapse. A strip of adhesive tape applied anteriorly from the stoma to the symphysis pubis minimised the recession in many infants (Jackson 1962). This probably acted by supporting the anterior tracheal wall and thus maintaining a more adequate airway.

When cyanosis or recession occurred, the infant was held face down in the palm of one hand and patted over the back; this usually provoked coughing with restoration of the airway. If this was unsuccessful, the pharynx and stoma (if still open) were aspirated and oxygen given by face mask. If the above measures failed, the tracheostomy cannula was replaced. Respiration was assisted for a few days if apnoea occurred.

Replacement of the tube in an emergency:

The amount of fibrous tissue around the stoma increased with the duration of intubation. Rapid contraction of the fibrous tissue made it difficult to reinsert a cannula which had been removed for some hours (Diamant et al. 1961). Under these circumstances, the metal tube with its introducer was placed over the centre of the scar and pushed backwards towards the vertebral column until it passed through the ostium and then into the trachea. To achieve this considerable force had to be exerted. The creation of a false passage anterior to the trachea was always a danger and this occurred if the tube was directed obliquely downwards instead of at right angles to the spine.

RESULTS IN THE PRESENT TRIAL:

In this study data were collected to assess whether the duration of intubation and the severity of tracheal ulceration influenced decannulation. This information was recorded with the reasons for failure at each extubation attempt. In the controlled respiration group, 20 patients were subjected to high tracheostomy (at the level of 2nd and 3rd tracheal rings) and an equal number to low tracheostomy (at the level of 5th and 6th tracheal rings). A low tracheostomy was performed on all infants treated by assisted respiration.

There were 40 survivors in this trial, 30 in the controlled respiration and 10 in the assisted respiration group. The average extubation time for the two groups is shown in Table VIII.

TABLE VIII: DURATION OF TRACHEOSTOMY - CONTROLLED AND ASSISTED RESPIRATION (SURVIVORS ONLY)

TREATMENT GROUP	NUMBER OF PATIENTS.	AVERAGE TIME OF EXTUBATION (DAYS)	
CONTROLLED RESPIRATION GROUP:			
High Tracheostomy	15	68	61
Low Tracheostomy	15	51	
ASSISTED RESPIRATION GROUP:			
Low Tracheostomy	10	45	
	40	56.5	

FURTHER ANALYSIS:

Infants in whom extubation was successful at the first attempt. (Table IX).

There were 24 infants in whom the first attempt at removal of the tracheostomy tube or accidental extubation succeeded. Tracheal ulceration was severe in 12 patients and moderate in 2. In the remaining 10 patients, the extent of ulceration was not known as tracheoscopy was not indicated during treatment.

TABLE IX: DATA PERTAINING TO INFANTS IN WHOM THE FIRST EXTUBATION (OR ACCIDENTAL EXTUBATION) WAS SUCCESSFUL.

NUMBER OF PATIENTS	TRACHEAL ULCERATION	AVERAGE DAY OF EXTUBATION
12	Severe	35
2	Moderate	
10	Tracheoscopy not indicated	25
24		30

The most likely reason for earlier decannulation in these 10 infants was that they suffered less severe tracheal ulceration in that the complications delaying extubation (granuloma and inspiratory tracheal collapse) did not occur. Thus there were no indications for tracheoscopy.
The average extubation period for the group of 24 infants was 30 days.

Infants in whom decannulation was delayed. (Table X)

Extubation was considered to be delayed if it was not achieved on the first occasion, an average period of 30 days after tracheostomy was performed. Twelve babies treated with controlled ventilation and 4 with assisted respiration presented problems in decannulation. The duration of intubation in the 16 infants varied from 23 to 359 days and the number of extubation attempts from 2 to 8. The average duration of tracheostomy was 45 days in those with moderate ulceration and 106 in those with severe tracheal damage. The mean duration for the whole group was 97 days.

TABLE X: DATA RELATING TO 16 PATIENTS IN WHOM EXTUBATION WAS DELAYED.

NUMBER OF PATIENTS.	TRACHEAL ULCERATION.	NUMBER OF EXTUBATION ATTEMPTS	AVERAGE DAY OF EXTUBATION.
13	Severe	55	106
3	Moderate	7	45
16		62	97

Cyanosis alone or a combination of cyanosis, rib recession and inspiratory stridor accounted for failure of extubation on the first occasion. Cyanosis alone probably indicated failure of the infant to adapt to laryngeal

breathing while the association of rib recession and inspiratory stridor with cyanosis suggested that tracheal weakening was an added complication. Further details relating to each patient are included in the Appendix, (page viii).

Tracheal ulceration was severe in the majority of infants who were not extubated at the second attempt. Prolonged intubation appeared to aggravate existing tracheal ulceration. Granulomata and excessive inspiratory collapse of the trachea occurred frequently in this group of patients (see Appendix, page xx). The infants' dependence on the tracheostomy also increased with the delay in extubation. In many infants removal of the tube resulted in rapid onset of severe costal recession, distressed breathing and cyanosis.

HIGH VERSUS LOW TRACHEOSTOMY:

Fifteen patients with high and 15 with low tracheostomy survived in the controlled respiration group. The operation was varied to assess its effects on extubation. There was no difference in the ease of extubation between the two groups. The pattern of tracheal ulceration was similar in both groups (Table XI).

TABLE XI: TRACHEAL ULCERATION AND EXTUBATION FOLLOWING HIGH AND LOW TRACHEOSTOMY.

GROUP	TRACHEAL ULCERATION.		EXTUBATION 1st ATTEMPT	EXTUBATION 2nd ATTEMPT OR LATER
HIGH TRACHEOSTOMY (15 infants)	Severe	7)	9	6
	Moderate	3)		
	Tracheoscopy not indicated	5)		
LOW TRACHEOSTOMY (15 infants)	Severe	8)	8	7
	Moderate	3)		
	Tracheoscopy not indicated	4)		
		30	17	13

EXTUBATION ANALYSIS IN "NON-SERIES" PATIENTS.

Data relating to 81 patients, all treated by controlled respiration (but not included in the present trial) and extubated in the manner described earlier, were analysed to compare the results obtained with the nylon reinforced tube with those of the cuffed tracheostomy tube. The patients are considered in 3 groups :-

A: 31 infants in whom the uncuffed nylon reinforced latex tracheostomy tube was used

B: 17 infants in whom either the uncuffed or a cuffed tube (described earlier) was used. These babies were the survivors of a group of 24 who were the subjects of a preliminary random trial comparing the two tracheostomy tubes.

Extubation analysis in "non-series" patients (continued)

C: 23 infants in whom the cuffed tube was used.

Results in "non-series" patients (Tables XII, XIII and XIV).

TABLE XII: RESULTS IN GROUP A. (NYLON REINFORCED TRACHEOSTOMY TUBE).

GROUP A	NUMBER OF PATIENTS	TRACHEAL ULCERATION		AVERAGE EXTUBATION DAY
EXTUBATION FIRST ATTEMPT	13	SEVERE MODERATE TRACHEOSCOPY NOT DONE	6 6 1	27
EXTUBATION SECOND ATTEMPT OR LATER	18	SEVERE MODERATE	13 5	62
	31			51

TABLE XIII: RESULTS IN GROUP B. (CUFFED AND UNCUFFED TRACHEOSTOMY TUBES).

GROUP B	NUMBER OF PATIENTS	NUMBER OF EXTUBATION ATTEMPTS	TRACHEAL ULCERATION.	AVERAGE EXTUBATION DAY
CUFFED LATEX TRACHEOSTOMY TUBE	7	9	SEVERE 2 MODERATE 5	28
UNCUFFED NYLON REINFORCED TRACHEOSTOMY TUBE	10	18	SEVERE 7 MODERATE 3	30
	17	27		29

TABLE XIV: RESULTS IN GROUP C. (CUFFED TRACHEOSTOMY TUBE).

GROUP C	NUMBER OF PATIENTS.	TRACHEAL ULCERATION		AVERAGE EXTUBATION DAY
EXTUBATION FIRST ATTEMPT	14	SEVERE MODERATE TRACHEOSCOPY NOT DONE	4 5 5	31
EXTUBATION SECOND ATTEMPT OR LATER	9	SEVERE	9	40
	23			35

The results in each "non-series" group were similar to those in the present study. A proportion of infants with severe tracheal ulceration were extubated when the attempt was first made while in others the procedure failed on the first occasion. Most of the babies in whom moderate ulceration was diagnosed and all those in whom tracheoscopy was not indicated were successfully extubated at the first attempt.

In group B, when the uncuffed tube was used, 7 infants suffered severe tracheal ulceration and in 4 of these extubation was delayed. In contrast, using the cuffed tube, there were fewer infants with severe ulceration and only one in

whom extubation was difficult. However, there was no significant difference in the average extubation time between these two sub-groups (Table XIII).

In Group C (cuffed tracheostomy tube), the average extubation period (40 days) when decannulation was difficult was lower than in group A (62 days) or than in the present study (97 days, Table X).

When the extubation time was calculated for all the patients (Group A, B, C and the present study) according to the tracheostomy tube used, the results were in favour of the cuffed tube (Table XV). The results were statistically significant ($p < 0.05$).

TABLE XV: AVERAGE EXTUBATION TIME - CUFFED AND UNCUFFED TRACHEOSTOMY TUBES (GROUPS A,B,C AND PRESENT STUDY). THE DIFFERENCE IN THE EXTUBATION TIME BETWEEN THE TWO GROUPS WAS STATISTICALLY SIGNIFICANT ($p < 0.05$).

TRACHEOSTOMY TUBE	NUMBER OF PATIENTS	AVERAGE EXTUBATION DAY
UNCUFFED	81	51
CUFFED	30	32

PROLONGED NASOTRACHEAL INTUBATION.

Several workers have recently resorted to nasotracheal intubation as an alternative to tracheostomy because of the complications of the latter operation (Sykes 1960 c, Allen and Stevens 1965, Crul and Wolffensperger 1965, Thomas et al. 1965, McDonald and Stocks 1965). The technique of nasal intubation does not dispense with the meticulous care required for tracheostomy. Both tracheal suction and fixation of the tube are more difficult, the former because of the longer length of the tube. Trained personnel must always be available to replace the tube quickly if obstruction or displacement occurs. Although less severe ulceration has been reported, some infants have suffered severe laryngeal damage and subglottic stenosis (Sykes 1960 c, Allen and Stevens 1965, Pracy 1966).

Extubation after nasotracheal intubation is not always easy; a certain number of infants require re-intubation while others may need tracheostomy. In a series of 50 patients, McDonald and Stocks (1965) reported that 20% required tracheostomy, the indications being persistent laryngeal oedema or ulceration, airway obstruction, or prolonged respirator assistance.

Nasotracheal intubation as advocated by Davenport (1964) and McDonald and Stocks (1965) for the management of "failed" extubation was performed on 3 infants in the present study and on several non-series patients with good results.

SUMMARY.

When the tracheostomy is adapted for intermittent positive-pressure respiration, the complications of displaced or obstructed tracheostomy tubes bear a grave prognosis especially during controlled respiration.

Infection around the tracheostomy and of the lungs is commonly encountered. If arterial oxygen tensions are reduced because of infection combined with collapse of the lungs, further anoxia during tracheal suction is likely to be fatal.

Severe tracheal ulceration was observed in 50% or more of the infants during positive-pressure respiration irrespective of the type of tube used. The cuffed tube causes circumferential ulceration of the trachea but severe indentation is seen in only a few infants. Tracheal perforation, tracheo-oesophageal fistula or haemorrhage was not encountered. These complications occurred in a few "non-series" infants when the uncuffed tube was used. The cuffed tube facilitates artificial respiration because air leaks do not occur and inflation of the left lung is easier since severe indentation is not a common occurrence.

Extubation at the first attempt is easy in about 50% of the infants with tracheal ulceration while problems occur in the remaining 50%. The average duration of tracheostomy in both groups was about 30 days when decannulation was first attempted. The reasons for the occurrence of

tracheal weakening (which delays extubation) in only one of the two groups intubated for similar periods are not easy to assess. However, with prolonged intubation the incidence of severe inspiratory tracheal collapse, cyanosis and granulomata increased.

Prolonged intubation is an alternative to tracheostomy if it is needed for periods of three weeks or less (Pracy 1966). This time limit and the report of sub glottic and laryngeal ulceration probably make prolonged intubation unsuitable in the treatment of tetanus neonatorum. Respiratory assistance in tetanus is often needed for more than three weeks. Nasotracheal intubation, however, is of value in managing respiratory distress which often occurs after the removal of the tracheostomy tube.

The poor attendance at the follow-up clinic in this unit does not permit any conclusions about the long term effects of the complications of tracheostomy. However, data collected on 71 infants (both clinical and radiological, including tracheal x-rays) over a period of 3 years indicate that only a small number suffer serious disability as a result of tracheostomy.

CHAPTER IV

DISCUSSION.

The complications occurring frequently during assisted and controlled respiration are discussed in this chapter. The problems in the two treatment groups are considered together because they were, with one exception, common to both groups. The one exception was the difficulty in achieving satisfactory control of reflex spasms during assisted respiration.

Brief case histories illustrating some of the problems encountered during treatment are included in the Appendix, pages v - xii.

CONTROL OF REFLEX SPASMS.

Reflex spasms which were abolished during the total paralysis regime by the use of curare were not well controlled by chlorpromazine and phenobarbitone during assisted respiration. Although the dose of chlorpromazine (Table XVI) in these patients was almost ten times that recommended for infants, tetanic spasms were not adequately suppressed. Adams et al. (1959) in their series used twice the dose of chlorpromazine given in the present study without achieving satisfactory control of spasms.

TABLE XVI: SEDATION DURING ASSISTED RESPIRATION.

SERIES NUMBER	AVERAGE DAILY DOSE (mg.)	
	CHLORPROMAZINE	PHENOBARBITONE
1	48	94.5
9	37	44
16	36	77
29	44	89
40	43	100
47	46	100
52	60	100
54	70	60

The combination of chlorpromazine and phenobarbitone in high doses to suppress convulsions resulted in drowsiness and flaccidity. This made it impossible to assess respiratory distress by the infant's facial expression. Severe spasms occurred even when the infant was flaccid. Physiotherapy, manipulation of the tracheostomy, feeding, collection of blood and gas samples and injections induced spasms in addition to those occurring spontaneously. Physiotherapy and tracheal suction were probably the most potent and frequent stimuli. Consequently these procedures were often inadequately performed or delayed until spasms abated after additional sedation.

Spasms occurring during a feed were dangerous. The possibility of inhalation of pharyngeal contents or milk regurgitated past the indwelling gastric tube into the pharynx was always present during such attacks. Since the tracheostomy tube was uncuffed pharyngeal contents could be aspirated easily.

Assisted respiration in the earlier trial (Adams et al. 1964) was commenced after periods of conservative treatment varying from one to 9 days (calculated by the author). During this period these infants suffered several attacks of cyanosis and apnoea. Many were flaccid when assisted respiration was started and thus artificial ventilation without curare was facilitated.

In contrast, in the present study, patients were treated by assisted respiration on admission or within 24 hours of admission when frequent, repeated, reflex spasms occurred. Continuous convulsions hindered mechanical ventilation and as a result cyanosis was observed in many infants during these episodes.

Effects of Chlorpromazine and Phenobarbitone on respiration.

Laurence et al. (1958) favoured chlorpromazine during conservative treatment of tetanus because clinical respiratory depression was not observed. However, Dobkin et al. (1954) noted a consistent fall in tidal volume (and minute volume) when chlorpromazine was given to normal subjects.

That phenobarbitone depresses repiration is well known but it is used in tetanus because, combined with chlorpromazine, better control of reflex spasms is achieved (Wright and Adams 1960). In this study, as large doses were used during assisted respiration, depression of the respiratory centre due to both drugs was very likely. However, this could not be measured as respiratory assistance was given to all patients. Apnoea, as defined for this trial, was observed in only one patient.

ABDOMINAL DISTENSION AND GASTRIC STASIS: (See Appendix, page xiii).

Abdominal distension and gastric stasis were frequent complications during both controlled and assisted respiration. Ileus as reported by Wright et al. (1961) and Smythe (1963) was seen as a terminal event only in those patients with severe lung infections. A similar observation was made by Sykes (1960 b).

In the present trial, abdominal distension or gastric stasis was usually evident when signs of respiratory infection were present; both conditions improved as the lung lesion resolved. In a few patients, stasis or distension heralded the onset of a respiratory complication which was only diagnosed some 24 to 36 hours later. Distension in 3 infants was apparent after bouts of severe reflex spasms or cyanosis.

Aetiology of abdominal distension and gastric stasis.

The aetiology of both conditions is not clear. Abdominal distension has been reported as a feature of respiratory infection (Cross 1957, Nelson 1964). In this trial, the association of respiratory infection, severe spasms and cyanosis with distension or stasis suggests that some degree of hypoxia may be a factor in the aetiology. Curare in the dosage used during controlled respiration probably did not play a dominant role in producing ileus since it was used only in small doses. Furthermore, the infants with gastric distension or stasis were severely ill and often did not require curare during these periods. Low body temperatures were frequently observed in this group of patients.

OBSERVATIONS ON BODY-TEMPERATURES:

Spaeth (1940), Jelliffe (1950), and Tompkins (1958) observed that fever was a bad prognostic sign in tetanus neonatorum on conservative treatment. Spaeth (1940) correlated fever with the complications of treatment while Tompkins (1958) found that the mortality was significantly higher in infants whose temperature did not fall below 98°F. Sykes (1960 b) and Wright (1960 b) found that hypothermia occurred frequently during the total paralysis regime. In the present trial, subnormal temperatures (between 93°F. and 98.4°F.) rather than hypothermia (below 93°F.) were encountered.

Body-temperatures during the height of the illness (the first 10 days - when curare or maximum doses of sedatives were given) were analysed for all the patients in the present study. The temperatures were measured rectally.

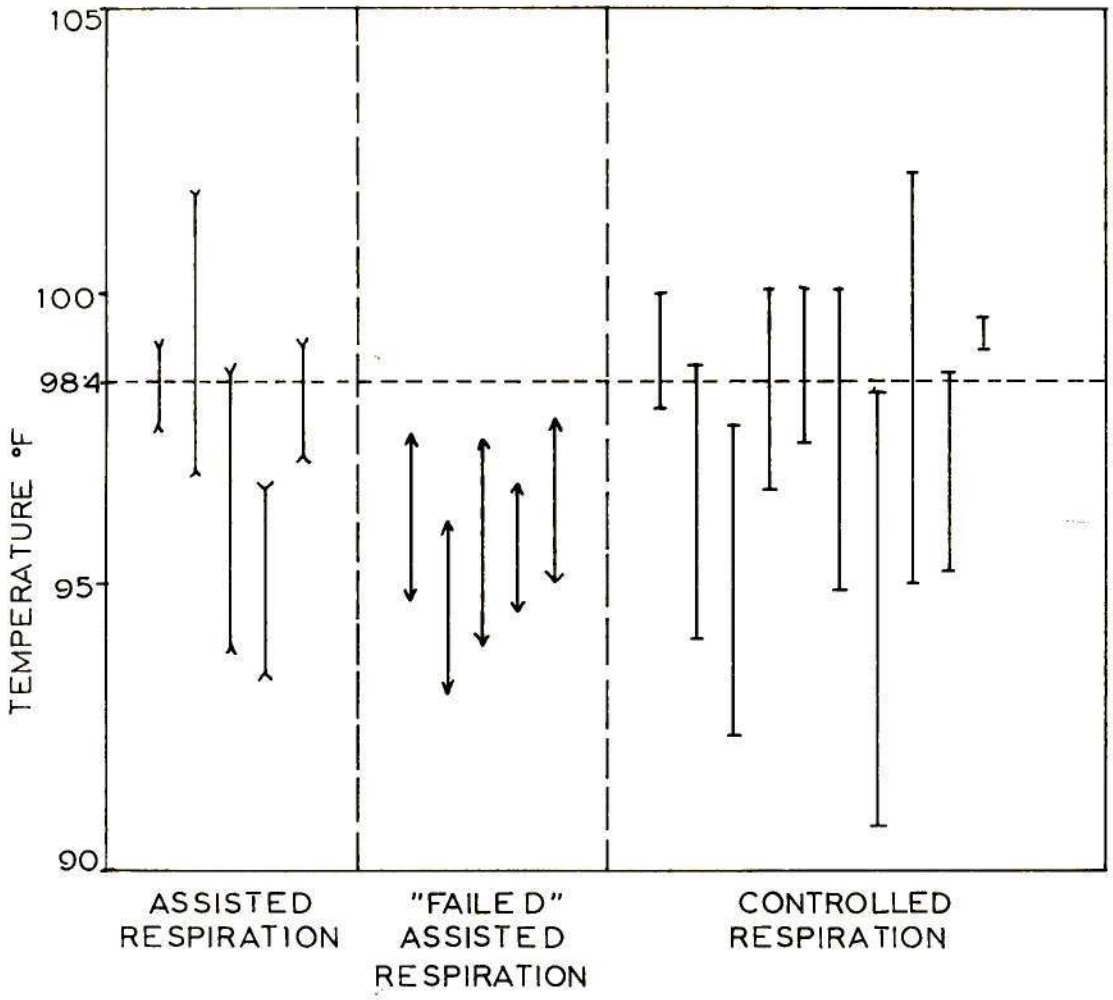
Observations in the Present Study:

(1) Although the majority of infants had subnormal temperatures, it was found that the survivors in all three groups had significantly higher temperatures (Table XVII) than those who died. Figure 8 shows that all the lowest mean daily temperatures and several of the highest mean daily temperatures of those who died were below 98.4°F.

TABLE XVII: MEAN DAILY TEMPERATURE FOR 38 SURVIVORS AND 20 DEATHS DURING THE PRESENT STUDY. THE DIFFERENCE BETWEEN THE MEAN DAILY TEMPERATURES IS STATISTICALLY SIGNIFICANT, $p = < 0.001$ (TEMPERATURE RECORDS FOR 2 SURVIVORS WERE MISLAID).

GROUP	NUMBER OF PATIENTS.	MEAN DAILY TEMPERATURE OVER TEN DAYS.
SURVIVORS	38	98.0°F S.D. 0.88
DEATHS	20	96.8°F S.D. 1.34

FIG. 8 SUBNORMAL TEMPERATURES IN INFANTS WHO DIED (IN PRESENT STUDY)



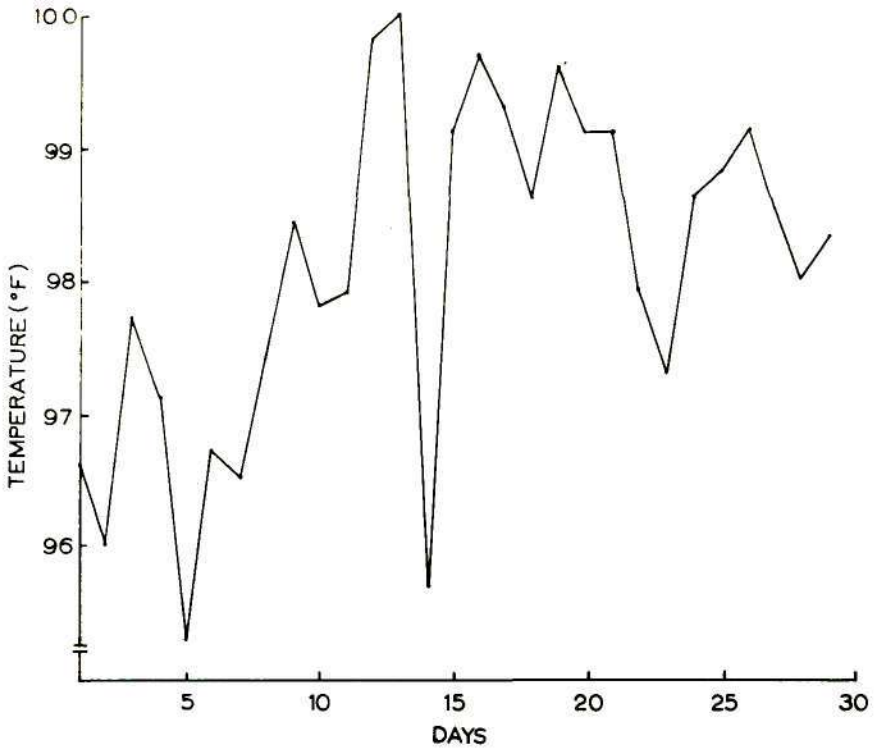


FIG 9 (SERIES Nº. 42). GRAPH SHOWING SUBNORMAL TEMPERATURES DURING CONTROLLED VENTILATION (FIRST 10 DAYS) AND A RISE IN TEMPERATURES THEREAFTER.

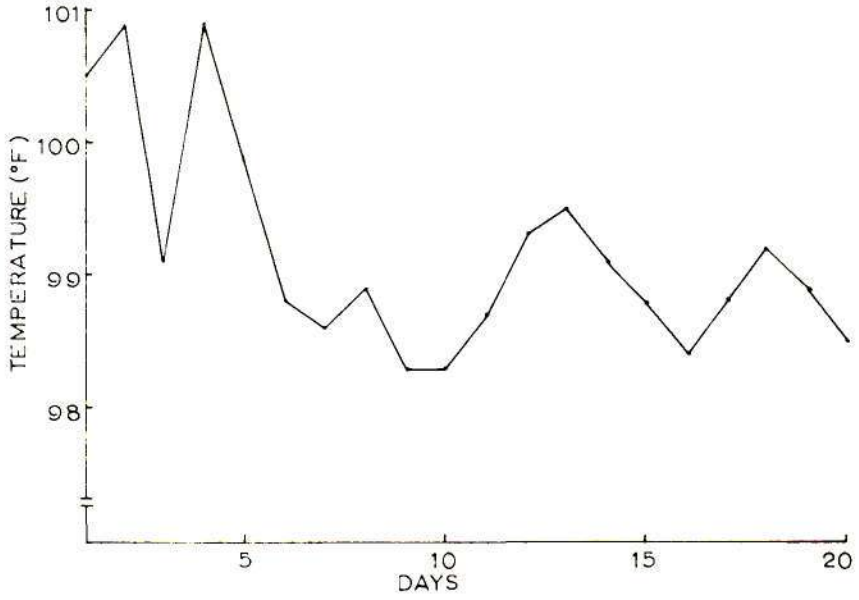


FIG. 10 (SERIES N°10) NORMAL OR ELEVATED TEMPERATURES DURING "FAILED" ASSISTED RESPIRATION

(2) Body temperatures were elevated on admission (99°F to 104°F). Within 24 hours of commencing treatment, the temperatures were reduced and in many infants were subnormal (Figure 9). The temperatures rose to about normal levels during the second week (Figure 9) in the majority of patients. Subnormal temperatures persisted for longer periods in a few infants (excluding premature infants) who suffered severe respiratory infections.

(3) Body temperatures were maintained at normal or above normal levels (Figure 10) in a small group of patients. The clinical courses of these patients were characterised by the absence of complications or if these occurred, they were mild.

(4) In those infants who received curare, it was observed frequently that the dose of curare was directly related to the temperature. Table XVIII shows this association for patients in the "failed" assisted respiration group.

TABLE XVIII: THIS TABLE SHOWS THAT MOST INFANTS WITH HIGHER TEMPERATURES (NOT NECESSARILY NORMAL) REQUIRED LARGER DOSES OF CURARE THAN THOSE WITH LOWER TEMPERATURES. NOTE ALSO THE LOWER TEMPERATURES OF THOSE INFANTS WHO DIED.

SERIES	MEAN DAILY TEMPERATURES (^{o}F)	AVERAGE DAILY CURARE DOSE (mg.)	FATE
6	96.7	15.0	Survived
10	99.4	22.25	Survived
21	97.3	14.3	Survived
32	98.3	15.9	Survived
34	97.2	15.8	Survived
50	97.1	17.5	Survived
57	96.4	7.1	Survived
18	96.1	15.0	Died
25	94.6	11.25	Died
39	95.6	2.5	Died
45	95.7	11.2	Died
56	96.4	6.2	Died

Observations on "non-series" Patients.

Since the number of patients in the present study treated by assisted respiration was small, the temperature records of 37 patients in the previous trial of assisted respiration (Adams et al. 1964) were analysed.

A similar analysis was made for a group of infants treated conservatively during the present trial. The sedatives used were chlorpromazine and phenobarbitone.

The first 3 observations recorded for the patients in the present study were also true for these 2 groups of infants.

Incubator Nursing.

Because of the high incidence of subnormal temperatures, 16 infants treated by controlled ventilation but not included in the present study were nursed in incubators to maintain body temperatures at normal levels. These infants had temperatures ranging from 94°F to 96°F. when nursed at room temperature and all suffered severe respiratory infections due to gram negative bacilli. Maintaining the temperatures at about normal levels did not improve the prognosis; it seemed that the severe infection offset any benefit the infants may have derived from the warm environment.

Aetiology of Subnormal Temperatures.

Temperature control is influenced by several factors. Chlorpromazine lowers body temperature by hypothalamic depression and by peripheral vasodilatation (Goodman and Gillman 1965). The relaxant effects of curare, chlorpromazine and phenobarbitone all produce a lowering of the temperature by rendering the infant immobile. Muscular activity is one of the mechanisms maintaining normal body-

temperature. Hyperpyrexia in tetanus may be due to medullary involvement by tetanus toxin (Montgomery 1961). Whether the toxin by a similar mechanism can produce hypothermia is not known.

Septic processes in the newborn can depress body-temperature (Mann and Elliot 1957) and if these infants are nursed in a cold environment their prognosis worsens. Broncho-pneumonia was present in most of the infants in this study; other infections (gastro-enteritis, meningitis and peritonitis) occurred infrequently.

Association Between Low Body-Temperatures and Mortality.

Premature as well as mature infants (Scopes 1966) have difficulty in maintaining their body temperatures in a cold environment. Clinical trials have demonstrated that the mortality among these infants is lower if they are nursed in a warm environment (Silverman et al. 1958, Buetow and Klein 1963).

The exact mechanism of death in the presence of low temperatures is not clear (Scopes 1966) but it is known that exposure to cold results in an increased oxygen consumption (Bruck 1961) and matabolic acidosis (Gandy et al. 1964). In addition Gandy and his co-workers (1964) have shown that vigorous infants were able to maintain a normal blood pH by increasing CO_2 elimination whereas even mildly asphyxiated infants were unable to compensate and developed severe metabolic acidosis.

In the present study, drug therapy and infection rather than a cold environment probably played a major role in producing low temperatures. Analysis of acid base measurements on 30 infants in the present trial did not reveal metabolic acidosis.

DURATION OF ARTIFICIAL VENTILATION:

For the reasons mentioned earlier (Chapter II) it was hoped that treatment by assisted ventilation might reduce the duration of respirator assistance. The figures in Table XIX show that there was no difference whether infants were treated by assisted or controlled respiration during the present trial. The shorter duration of artificial ventilation (10 days) in the 1964 trial (calculated by the author) was due to the fact that these infants were offered respirator assistance after conservative treatment for periods ranging from one to 9 days.

TABLE XIX: DURATION OF ARTIFICIAL VENTILATION IN TETANUS NEONATORUM.

TREATMENT GROUPS	NUMBER OF SURVIVORS	AVERAGE DURATION OF ARTIFICIAL VENTILATION (DAYS)
SEDATION AND ASSISTED RESPIRATION	3	20
"FAILED" ASSISTED RESPIRATION	7	24
CONTROLLED RESPIRATION	30	20
ASSISTED RESPIRATION (1964 TRIAL)	17	10

MODES OF DEATH:

The modes of death were similar (Tables XX, XXI and XXII) in each of the groups treated during the present trial. It was difficult to determine the exact cause of death in some patients while in others it was likely that a combination of 2 complications were responsible. In the former group were 3 infants who were found dead while being ventilated by normally functioning machines. Post mortem examinations in all 3 infants revealed patchy atelectasis which was thought to be insufficient to account for the fatal outcome. The possibility of fatal brain stem intoxication by tetanus toxin precipitating cardiac arrest could not be excluded (Kloetzel 1963, Adams et al. 1966).

Clifton (1964) and Adams et al. (1966) described the "hypotensive" or "shock syndrome" as an important cause of death in severe tetanus amongst children and adults. The terminal clinical features of these patients - low skin temperatures, poor peripheral circulation, cyanosis and hypotension - are similar to those observed in very ill infants during this study. Since the possible causes of the "shock syndrome" discussed by Adams et al. (1966) could apply to infants as well, it seems reasonable to postulate that this syndrome may also occur in tetanus neonatorum. The association between low body-temperature and mortality has been discussed.

TABLE XX: CAUSE OF DEATH - ASSISTED RESPIRATION.

SERIES NUMBER	DAY OF DEATH	IMMEDIATE CAUSE OF DEATH	POSTMORTEM FINDINGS
1	5	Unknown	Congestion and patchy collapse both lungs
9	11	Unknown	Purulent meningitis
16	14	Unknown	Oedema and patchy collapse both lungs
40	6	Bronchopneumonia	Confluent bilateral bronchopneumonia
52	5	Tracheal suction	Oedema and congestion both lungs. Oedema of the brain

Management of patients by tracheostomy and positive pressure respiration introduces the hazards of the operation and the mechanical apparatus.

TABLE XXI: DEATHS IN THE "FAILED" ASSISTED RESPIRATION GROUP.

SERIES NUMBER	DAY OF DEATH	IMMEDIATE CAUSE OF DEATH	POSTMORTEM FINDINGS
18	39	Bronchopneumonia and severe abdominal distension, preventing inflation of the lungs	Lobar pneumonia
25	3	Anoxia - cause unknown	Consent for necropsy refused.
39	6	Tracheal suction	Bronchopneumonia
45	4	Dislodged tracheostomy tube	Bilateral suppurative bronchopneumonia
56	13	Bronchopneumonia	Consent for necropsy refused

In 10 infants mechanical faults (faulty connections, dislodged tracheostomy tubes, airway obstruction or tracheal suction in the presence of a respiratory complication) were associated with fatality but in only 3 could death be attributed to the fault with reasonable certainty. Respiratory complications (infection and atelectasis which could contribute to the hypoxia caused by the mechanical fault were present in the other infants.

Mechanical faults were the cause of cyanosis and bradycardia in several patients (see Appendix, page XXIV). In a few infants the fault was discovered before complications ensued but this was not always possible when the ward was overcrowded.

During the second half of the trial, a monitor system was designed which sounded an alarm when ventilation was interrupted for longer than 15 seconds (Eaton et al. 1966). This alarm has also proved useful in preventing prolonged tracheal suction.

TABLE XXII: DEATHS IN THE CONTROLLED RESPIRATION GROUP.

SERIES NUMBER	DAY OF DEATH	IMMEDIATE CAUSE OF DEATH	POSTMORTEM FINDINGS
4	3	Unknown	Patchy atelectasis both lungs
20	4	Died during insertion of a larger tracheostomy tube	Patchy collapse both lungs
53	7	Humidifier - tracheostomy connection worked loose	Bronchopneumonia Prematurity
22	8	Died after insertion of a larger tracheostomy tube. Anoxia.	Bilateral bronchopneumonia
49	16	Abdominal distension preventing inflation of the lungs	Fibrinous peritonitis
12	21	Tracheal suction	Congestion and patchy atelectasis both lungs
35	21	Anoxia due to blocked humidifier in tracheostomy assembly	Consent for necropsy refused
30	26	Cyanosis probably due to inhalation of a feed	Patchy collapse and bronchopneumonia - both lungs
19	29	Severe anoxia ?due to airway obstruction	Consent for postmortem refused
43	40	Airway obstruction	Small abscesses both lungs

It was hoped that infants treated by assisted respiration would be at an advantage to those treated by controlled ventilation if mechanical faults occurred (see Chapter II). However, during assisted respiration the heavily sedated infant was incapable of maintaining unaided ventilation for long periods, and mechanical faults resulted in cyanosis and bradycardia. These complications occurred readily if respiratory infection was already present.

Although mechanical faults were numerous in the controlled respiration group, it cannot be concluded that total paralysis contributed to death in all because only 3 patients were curarised at the time of death. The remaining 4 fatalities occurred between the 21st and 40th days.

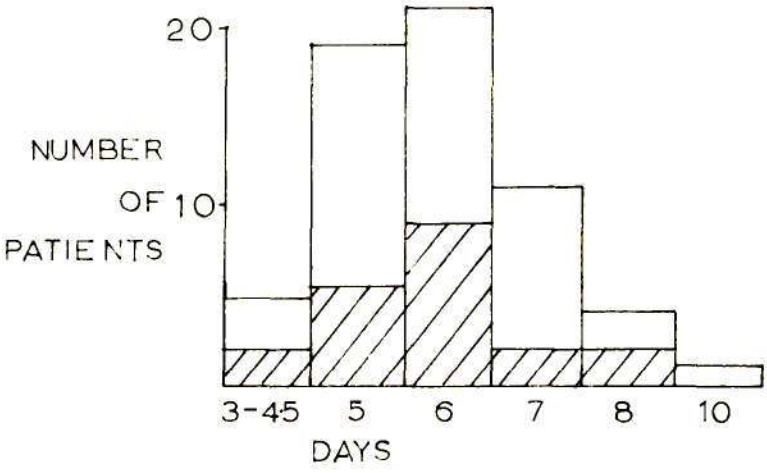
DAY OF DEATH.

Fifty percent of the deaths occurred between the 11th and the 40th days (Tables XX, XXI and XXII) when the effects of etatnus were minimal or absent. Thus, the complications of treatment accounted for some of the deaths.

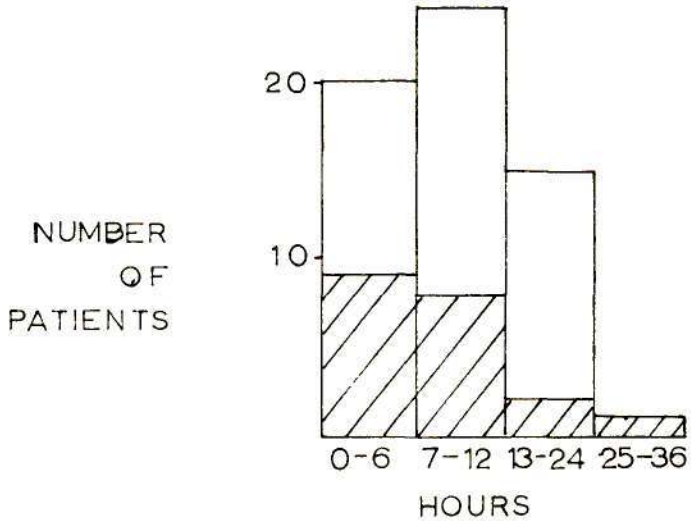
MORTALITY IN RELATION TO SEVERITY OF THE DISEASE.

Cole (1938 and 1940) and Spivey (1953) showed that prognosis is related to the severity of tetanus. An incubation period of less than 7 days and a period of onset less than 48 hours were considered to be indicators of severe tetanus.
Wright (1960 b) and Adams et al. (1966) also made similar observations using different criteria to assess severity.

FIG. 11 MORTALITY IN RELATION TO INCUBATION PEROID AND PEROID OF ONSET



INCUBATION PERIOD



PERIOD OF ONSET

DEATHS SURVIVORS

The criterion adopted by these workers was the presence of severe and frequent tetanic spasms.

All the infants in the present trial had severe tetanus when judged by both the methods mentioned above. The mortality was highest in those infants whose incubation periods were less than 6 days and the onset times less than 12 hours (Figure 11). This was so despite the fact that all infants were exposed to a variety of complications, some which could have caused death independent of the disease.

SUMMARY.

The management of infants with severe tetanus neonatorum by assisted and controlled ventilation was similar in many respects. The one problem peculiar to assisted respiration was the control of reflex spasms.

Gastro-intestinal complications, low body-temperatures, respiratory infections, and mechanical faults were common to both groups of patients. An association between gastro-intestinal and respiratory complications was observed and the probable mechanisms are discussed. It is felt that hypoxia may be an important factor in causing gastric stasis and distension.

The incidence of subnormal temperatures was high in both groups and the patients who died were shown to have significantly lower temperatures than the survivors. Drug therapy and infection probably played a dominant role in producing low temperatures.

The causes and modes of death were similar in both groups. In a few infants, the exact cause of death was not known but in others death was associated with a combination of mechanical faults and respiratory infection. Fifty percent of the deaths could be attributed to the complications of treatment.
The possibility of the "shock syndrome" is postulated in the severely ill infants.

An alarm system has proved useful in minimising the effects of mechanical faults.

CHAPTER V.

SUMMARY AND CONCLUSIONS.

Favourable results obtained in the earlier study of assisted respiration (Adams et al. 1964) suggested that this technique might be easier than the total paralysis regime for the treatment of severe tetanus neonatorum. It was felt that infants managed by sedation and assisted ventilation would need less medical and nursing attention as they were not paralysed. It was also hoped that the incidence of pulmonary complications, the effects of machine failure and the problems of extubation would be decreased. A shorter period of respirator assistance was also envisaged because infants could be allowed to breathe spontaneously sooner than those treated by controlled ventilation.

The present clinical trial was therefore undertaken to assess whether assisted respiration was a suitable form of therapy in severe neonatal tetanus. Sixty infants were allotted at random for treatment :

(a) with sedation and assisted respiration, or

(b) with controlled ventilation.

The management, with the exception of drugs related to the two techniques, was standardised for both groups. The results suggested that controlled ventilation might be superior to assisted respiration but the difference in mortality between the two groups was not statistically significant.

It was difficult to control reflex spasms adequately with chlorpromazine and phenobarbitone in the group treated by assisted respiration. This problem was encountered in 12 of the 20 patients in this group.

Other complications observed during treatment were common to both groups. It is emphasised that meticulous tracheostomy care is essential but that technique itself is not entirely successful in preventing tracheal ulceration and infection. The hazards of prolonged tracheal suction and the precautions taken to avoid these are listed. Severe tracheal ulceration occurred both during controlled and assisted ventilation but did not always prevent early extubation. The observations in this study suggest that tracheal weakening as a result of ulceration is probably the most important cause of delayed extubation.
On the evidence presented, it seems unwise to advocate prolonged nasotracheal intubation as an alterative to tracheostomy but the former procedure is useful in alleviating respiratory distress occurring after the removal of the tracheal cannula (Davenport 1964). Tracheal and laryngeal ulceration occur as a result of tracheostomy and prolonged nasotracheal intubation respectively; difficulty in decannulation may be a complication of both procedures (McDonald and Stocks 1965). Although less severe ulceration has been reported by some workers, severe laryngeal ulceration and subglottic stenosis have been observed (Allen and Stevens 1965, Pracy 1966). Nasotracheal intubation is recommended for periods of three weeks or less (Pracy 1966).
Respirator assistance in tetanus neonatorum is often required for periods longer than three weeks.

The aetiology of low body-temperatures and the influence of subnormal temperatures on morbidity and mortality as reported in the literature are reviewed. It is suggested

that drug therapy and infections were the chief causes of subnormal temperatures in the present study. It is shown that those infants who died had significantly lower body-temperatures than those who survived. This observation was also true for infants in the earlier trial of assisted respiration (Adams et al. 1964) and for those infants treated conservatively during the present study. The occurrence of subnormal temperatures in infants of the latter 2 groups, all sedated with chlorpromazine and phenobarbitone, indicates that respirator assistance had little or no influence on body-temperatures.

An association between respiratory complications and gastro-intestinal signs is described and it is suggested that hypoxia may be an important factor in the aetiology of abdominal distension.

The modes of death were similar in both groups of patients. Mechanical faults and respiratory infections were the immediate cause of death in many infants but in a few patients the possibility of tetanus toxin being responsible for death could not be excluded.

It is postulated that the hypotensive or the "shock syndrome" (Clifton 1964, Adams et al. 1966) may also occur during artificial ventilation in infants with severe tetanus.

Mechanical faults occurred frequently during assisted and controlled ventilation. Infants managed by assisted

respiration fared no better than those treated by the total paralysis regime when respirators failed, or displacement or obstruction of tracheostomy tubes occurred. Because of heavy sedation, infants in the former group were unable to maintain adequate spontaneous respiration during these times.

Although treatment in both groups involved several hazards which could cause death, it is noted that the majority of fatalities occurred in those infants who had the most severe form of the disease. This observation differs from that of Jackson (1962) who stated that the severity of tetanus "did not appear to affect the results" of controlled ventilation.

THE PLACE OF ASSISTED RESPIRATION IN THE TREATMENT OF SEVERE TETANUS NEONATORUM.

It has been shown that controlled ventilation is the treatment of choice in severe neonatal tetanus (Wright et al. 1961, Mann et al. 1963). However, this technique is costly and requires specially trained medical and nursing staff. The use of mechanical apparatus and the practice of tracheostomy introduce special hazards so that success depends on "making no mistake for 24 hours a day" (Spalding and Smith 1963). Facilities of this standard are rarely available in countries where tetanus occurs frequently.

Although the requirements for carrying out assisted respiration are similar to those for controlled respiration, it was hoped that the former technique would make less demands on the medical and nursing staff. This expectation

was not fulfilled. The other predicted advantages were also not realised in the present study largely because reflex spasms were inadequately controlled. However, the results of this trial and those of Adams et al. (1964) show that the mortality is lower than that with conservative management. On this evidence, it is felt that assisted ventilation merits further investigation for use in those parts of the world where facilities for controlled ventilation are unavailable. The use of preparations other than chlorpromazime and phenobarbitone to control tetanic spasms may be more rewarding. In this regard, a preliminary trial of triethylene choline in this unit has been disappointing (Thambiran et al. 1966).

The investigation into methods of treatment might appear to be the wrong approach since the disease itself is preventable. But the knowledge that there are some countries where "tetanus kills more infants than whooping cough, diphtheria, measles, chickenpox and anterior poliomyelitis together" (Veronesi 1956) and others where the disease ranks second only to prematurity as a cause of neonatal death (Wright 1960 a) is adequate evidence that effective treatment by simpler techniques is needed until public health measures are of the order to eradicate the disease.

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A P P E N D I X

CONTENTS:

a. Series Number, Age, Incubation Period, Period of Onset and Fate.

ABBREVIATIONS USED:

I.P.	:	Incubation period in days
O.	:	Period of onset in hours
S.	:	Survived
D.	:	Died
Afr.	:	African
Ind.	:	Indian
F.	:	Female
M.	:	Male

(a) ASSISTED RESPIRATION.

Series Number	Tetanus Number	Race	Sex	Age (Days)	I.P.	O	Fate
1	1198	Afr.	F.	5	4.5	O	D
9	1225	Afr.	M.	8	7	O	D
16	1271A	Afr.	F.	6	4	12	D
29	1363	Afr.	M.	5	4	9	S
40	1424	Afr.	M.	6	5	16	D
47	1458	Afr.	F.	6	5	O	D
52	1474	Afr.	F.	9	8	12	D
54	1480	Afr.	M.	9	7	15	S
	"FAILED" ASSISTED RESPIRATION.						
6	1218	Afr.	M.	8	7	8	S
10	1230	Afr.	F.	4	3	12	S
18	1275	Afr.	M.	7	7	O	D
21	1288	Afr.	F.	7	6	12	S
25	1310	Afr.	M.	7	6	12	D
32	1380	Afr.	M.	7	6	12	S
34	1385	Afr.	M.	8	7	Few hrs.	S
39	1418	Afr.	M.	7	6	O	D
45	1452	Afr.	F.	7	6	O	D
50	1469	Afr.	M.	6	5	15	S
56	1479	Afr.	F.	9	5	36	D
57	1481	Afr.	M.	5	5	O	S

CONTROLLED RESPIRATION:

Series Number	Tetanus Number	Race	Sex	Age (Days)	I.P.	O	Fate
2	1199	Afr.	F.	7	6	0	S
3	1207	Afr.	M.	6	5	24	S
4	1208	Afr.	F.	6	6	4	D
5	1209	Afr.	F.	6	6	0	S
7	1222	Afr.	F.	6	6	12	S
8	1224	Ind.	M.	6	5	2	S
11	1239	Afr.	M.	7	7	0	S
12	1240	Afr.	F.	8	8	12	D
13	1244	Ind.	M.	6	6	24	S
14	1245	Afr.	M.	6	6	24	S
15	1260	Afr.	M.	8	7	12	S
17	1273	Afr.	F.	7	7	0	S
19	1281	Ind.	F.	5	5	0	D
20	1284	Afr.	M.	6	5	12	D
22	1296	Afr.	F.	5	5	1	D
23	1300	Afr.	F.	8	7	2	S
24	1307	Afr.	M.	7	6	11	S
26	1323	Ind.	F.	5	5	7	S
27	1326	Afr.	M.	6	5	4	S
28	1328	Afr.	F.	8	8	12	S
30	1376	Ind.	M.	8	6	12	D
31	1378	Afr.	M.	7	7	24	S
33	1381	Afr.	M.	7	7	24	S
35	1386	Afr.	M.	12	6	24	D
36	1398	Ind.	M.	6	5	17	S
37	1413	Afr.	M.	8	6	24	S
38	1448	Afr.	F.	11	10	24	S
41	1426	Afr.	M.	7	6	12	S
42	1430	Ind.	F.	7	6	12	S

(continued)

Series Number	Tetanus Number	Race	Sex	Age (Days)	I.P.	O	Fate
43	1436	Afr.	F.	7	6	14	D
44	1439	Afr.	M.	10	8	12	S
46	1455	Afr.	F.	6	5	8	S
48	1460	Ind.	F.	6	5	11	S
49	1468	Ind.	F.	6	6	6	D
51	1472	Afr.	M.	6	5	0	S
53	1477	Afr.	F.	7	6	12	D
55	1483	Afr.	F.	7	6	12	S
58	1484	Afr.	F.	7	5.5	12	S
59	1486	Afr.	F.	6	5	24	S
60	1488	Afr.	M.	6	5	18	S

(b) CASE SUMMARIES:

ASSISTED RESPIRATION.

PATIENT:	V.N.	SERIES NO:	54
INCUBATION PERIOD:	7 DAYS	ONSET PERIOD:	ABOUT 15 HOURS
ADMITTED:	27.8.65	FATE:	RECOVERED

CLINICAL COURSE:

This infant was admitted with severe reflex spasms and cyanosis. Assisted respiration was begun on admission and continued until the 14th day.
Severe spasms which were present until the 6th day prevented adequate physiotherapy.

The tracheostomy tube was removed successfully on the 16th day. Mild spasms were still noted on the 18th day. Apart from diarrhoea, progress was uneventful and the patient discharged on the 26th day.

COMMENT:

This infant was treated successfully by sedation and assisted respiration with diarrhoea as the only complication. The first attempt at extubation was successful. The absence of complications resulted in shortening the duration of aided respiration and in early extubation.

CASE SUMMARIES (continued)

ASSISTED RESPIRATION

PATIENT:	S.L.	SERIES NO:	47
INCUBATION PERIOD:	5 DAYS	ONSET PERIOD:	0 HOURS
ADMITTED:	2.7.65.	FATE:	RECOVERED

CLINICAL COURSE:

Assisted respiration was commenced shortly after admission. Severe spasms, present until the 6th day, hindered physiotherapy but in spite of this, the lungs remained clinically clear.

Sedation was reduced on the 8th day because the infant became flaccid. Progress was uneventful until the 14th day when abdominal distension and bilateral broncho-pneumonia were evident. Gastric absorption was poor over the following two days.

On the 19th day (during assisted respiration) the patient developed subcostal recession and mild cyanosis. Signs of consolidation of the right lung were still apparent. Tracheoscopy revealed two exudate-covered ulcers on the posterior wall and exudate in both main bronchial orifices. Tracheal suction yielded large quantities of mucoid secretions and exudate. Improvement noted on the 21st day was maintained and the respirator was disconnected on the 26th day.

The first attempt at decannulation on the 32nd day was successful. The patient was given a blood transfusion on the 39th day because of anaemia (Hb: 7.1 gm.%). She was discharged well on the 46th day.

(continued)

PATIENT : S.L. SERIES NO: 47 (continued)

COMMENT:

Bronchopneumonia, abdominal distension, severe tracheal ulceration and anaemia complicated treatment of this infant. Successful extubation on the first occasion came as a surprise for difficulty was anticipated because of severe tracheal damage.

CASE SUMMARIES (continued)

"FAILED" ASSISTED RESPIRATION.

PATIENT:	S.M.	SERIES NO:	18
INCUBATION PERIOD:	7 DAYS	ONSET PERIOD:	0
ADMITTED:	4.6.64	FATE:	DIED 12.7.64.

CLINICAL COURSE:

Assisted respiration was started soon after admission but because of severe spasms and cyanosis, controlled respiration was substituted on the 3rd day. On the same day intravenous fluids were given because of abdominal distension which persisted until the 11th day.

Aeration of both lungs was diminished on the 11th day. Tracheoscopy showed occlusion of the left main bronchus by inspissated pus. This was aspirated through the tracheoscope.

Curare was discontinued on the 14th day. Throughout the period of assisted respiration, bilateral rhonchi were present. Spontaneous breathing was adequate on the 24th day but assisted respiration was resorted to again on the 27th day because of bronchopneumonia and cyanosis. Two days later cyanosis was caused by purulent secretions. Inspection of the trachea revealed indentation and ulceration of the posterior tracheal wall with mucosal "heaping" just beyond the ulcer.

On the 17th day while respiration was still being assisted, the infant was found to be cyanosed. After saline

PATIENT: S.M. SERIES NO: 18 (continued)

instillation thick tenacious secretions were aspirated from the tracheostomy. The patient's condition deteriorated on the following day. Pneumonic consolidation and severe abdominal distension appeared to restrict respiratory excursions. In an attempt to inflate the lungs adequately, respiration was controlled and oxygen added to the inspired air but his condition remained unchanged until death on the 39th day.
Severe tracheal ulceration, consolidation of the whole of the right lung and upper lobe of the left lung, and cerebral oedema were demonstrated at postmortem.

COMMENT:

"Failed" assisted respiration was due to severe reflex spasms and cyanosis. Severe abdominal distension, lobar pneumonia and severe tracheal ulceration were complications in this infant.
Most of the temperatures during treatment were subnormal.

CASE SUMMARIES (continued)

CONTROLLED RESPIRATION

PATIENT:	S.M.	SERIES NO:	23
INCUBATION PERIOD:	7 DAYS	ONSET PERIOD:	? FEW HOURS
ADMITTED:	22.7.64.	FATE:	RECOVERED

CLINICAL COURSE:

Controlled respiration was commenced on the day of admission. Scanty aspirate obtained on the 2nd day became mucopurulent on the 3rd day. Signs of bilateral bronchopneumonia were present until the 10th day. Curare was discontinued on the 10th day and assisted respiration begun on the 12th day. The respirator was disconnected on the 16th day but was required again on the 18th day because of poor air entry to the left lung.

Accidental extubation occurred on the 19th day. The tracheostomy tube had been gradually pulled out of the clamp by improperly secured respirator tubing. As the infant's respiration was satisfactory, ventilator assistance was discontinued and the tube not replaced. Apart from mild subcostal recession, convalescence was uneventful and the patient discharged home on the 26th day.

COMMENT:

Although bronchopneumonia complicated the treatment of this infant, the period of respirator treatment was not prolonged. Accidental extubation which occurred on the 19th day was uneventful. Tracheoscopy was not indicated during treatment.

(continued)

PATIENT: S.M. SERIES NO: 23 (continued)

The average daily dose of curare was 27.7 mg; the highest dose recorded during controlled respiration in this trial. Body-temperatures during treatment were maintained at about normal levels during the first 10 days.

CASE SUMMARIES (continued)

CONTROLLED RESPIRATION.

PATIENT:	N.D.	SERIES NO:	53
INCUBATION PERIOD:	6 DAYS	ONSET PERIOD:	LESS THAN 12 HOURS
ADMITTED:	17.8.65.	FATE:	DIED 23.8.65.

CLINICAL COURSE:

Controlled respiration was commenced on this premature infant on the day after admission. Cyanosis occurred after suction on the 3rd day. Bronchopneumonia and mild abdominal distension were evident on the 4th day. On the 6th day intravenous fluids were given because of gastric stasis. The patient died when the humidifier-tracheostomy connection worked loose on the 7th day.

Bronchopneumonia and mild tracheal ulceration were demonstrated at necropsy.

COMMENT:

Bronchopneumonia diagnosed on the 4th day appeared to be improving when a mechanical fault caused death by anoxia three days later.

(c) RESPIRATORY COMPLICATIONS, ABDOMINAL DISTENSION AND GASTRIC STASIS.

Number of Patients.	Respiratory Complication.	Abdominal Distension.	Gastric Stasis.
	ASSISTED RESPIRATION		
2	Bronchopneumonia	Absent	Present
1	Patchy Collapse	Present	Absent
1	Absent	Present (Associated with Spasms)	Absent
	"FAILED" ASSISTED RESPIRATION		
1	Bronchopneumonia	Present	Present
1	Bronchopneumonia	Present	Absent
3	Bronchopneumonia	Absent	Present
2	Bronchopneumonia	Absent	Absent
2	Absent	Present (Associated with Spasms)	Absent
	CONTROLLED RESPIRATION		
5	Bronchopneumonia	Present	Present
2	Bronchopneumonia	Present	Absent
12	Bronchopneumonia (Pulmonary Collapse in 2 patients)	Absent	Present
9	Bronchopneumonia	Absent	Absent
1	Bronchopneumonia	Absent	Present (Associated with severe cyanosis)

(d) COMPLICATIONS OF TRACHEOSTOMY:

Misplaced Tracheostomy Tubes
(Partial and complete dislodgement and displacement of the tube onto the right main bronchus)

Assisted Respiration Group:

Series Number	Frequency	Comments
9	1	Prompt resuscitation - no cyanosis
10	2	Cyanosis due to tube displacement and severe spasms
29	2	Occurred during assisted respiration. No cyanosis.
45	1	FATAL: Bilateral bronchopneumonia at postmortem.
50	1	Accidental extubation (day 35)

Controlled Respiration Group:

3	2	Respiratory distress and cyanosis.
4	1	Cyanosis
7	3	Cyanosis
9	1	Cyanosis
11	4	During spontaneous ventilation. Cyanosed once.
13	1	Accidental extubation
14	1	Assisted ventilation. No cyanosis.
17	2	Tracheostomy tube completely dislodged. Cyanosis on one occasion
20	1	FATAL: Died during insertion of larger tube
23	1	Accidental extubation on the 19th day
24	2	Cyanosis due to dislodged tube and tube in right main bronchus

(continued overleaf)

(d) COMPLICATIONS OF TRACHEOSTOMY:

Controlled Respiration Group (continued)

Series Number	Frequency	Comments
26	1	Total dislodgement. Cyanosis.
27	2	Cyanosis. Pulmonary infection also present
28	2	Cyanosis
35	1	Cyanosis
36	1	Cyanosis - partially dislodged tube
38	1	Poor chest expansion. Tube in right main bronchus
41	1	Infant breathing satisfactory
48	1	Corrected promptly. (Complete dislodgement).
49	2	Cardiac arrest - successfully resuscitated
51	2	Cyanosis. Tube blocked on the one occasion
55	2	Mild cyanosis

Total number of patients : 27

Frequency of misplaced tube : 42

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Partial or Complete Obstruction of the Airway

Assisted Respiration Group:

Series Number	Frequency	Comments
9	1	Cyanosis due to thick secretions
10	3	Cyanosis due to thick secretions
18	2	Cyanosis - thick secretions
29	2	Cyanosis - mucus obstruction
34	6	Exudate blocked tracheal lumen. Thick secretions and bronchopneumonia. Cyanosis.
50	1	Tube kinked
57	1	Cyanosis. Mucus plug obstructing tracheal lumen.

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Partial or Complete Obstruction of the Airway

Controlled Respiration:

Series Number	Frequency	Comments
3	1	Thick secretions. Granulation beyond end of tracheostomy tube.
7	3	Secretions and granuloma
8	2	Cyanosis - thick secretions
11	4	Complete or partial obstruction by thick secretion. Accidental extubation on 359th day
12	1	Cyanosis due to thick secretions
13		Thick secretions - recession only
17	2	Thick secretions - cyanosis and bradycardia
19	3	Cyanosis due to thick secretions and faulty respirator
27	2	Cyanosis due to thick secretions
28	1	Tube completely obstructed by secretions
51	2	Cyanosis due to obstruction of tracheostomy by secretions
59	2	Tube obstruction by thick secretions

Number of Patients : 19

Frequency of Airway Obstruction: 39

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Early Extubation (Present Study)

Series Number	Duration of Tracheostomy Days	Tracheal Ulceration	Comments
Assisted Respiration Group (Low Tracheostomy)			
6	41	Severe	Mild rib recession
10	33	Severe	Mild rib recession
47	32	Severe	
57	25	Severe	3 cyanotic attacks
50	35	Moderate	Accidental extubation
34	30	Moderate	Mild rib recession
54	16	Tracheoscopy not indicated	

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Early Extubation (Present Study)

Controlled Respiration (High Tracheostomy)

Series Number	Duration of Tracheostomy (Days)	Tracheal Ulceration	Comments
27	60	Severe	11 cyanotic attacks after extubation
55	46	Severe	Accidental extubation
59	29	Moderate	
36	32	Moderate	
15	22	Tracheoscopy not indicated	5 cyanotic attacks after extubation
23	19	Tracheoscopy not indicated	Accidental extubation Mild rib recession
31	19	Tracheoscopy not indicated	
42	50	Tracheoscopy not indicated	
44	21	Tracheoscopy not indicated	One cyanotic attack following extubation
Controlled Respiration (Low Tracheostomy)			
2	32	Severe	
5	27	Severe	Mild rib recession
48	42	Severe	
24	33	Moderate	
33	18	Tracheoscopy not indicated	
41	19	Tracheoscopy not indicated	Mild rib recession
46	38	Tracheoscopy not indicated	
60	25	Tracheoscopy not indicated	

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Delayed Extubation (Present Study)

Assisted Respiration Group.

Series Number	Duration of Tracheostomy (Days)	No. of Extubation Attempts	Tracheal Ulceration	Reasons for Failure of Extubation
21	97	6	Severe	Bronchopneumonia, tracheal granuloma
29	72	4	Severe	Cyanosis, inspiratory stridor, recession
32	70	4	Severe	Cyanosis, rib recession, tracheal granuloma

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Delayed Extubation (Present Study)

Series Number	Duration of Tracheostomy (Days)	No. of Extubation Attempts	Tracheal Ulceration	Reasons for Failure of Extubation
Controlled Respiration (High Tracheostomy)				
3	82	5	Severe	Cyanosis, costal recession, inspiratory stridor tracheal granuloma
7	93	4	Severe	Cyanosis, costal recession, tracheal granuloma
11	359	6	Severe	Cyanosis, costal recession, stridor, tracheal collapse and stenosis
28	142	5	Severe	Cyanosis, rib recession
58	35	3	Severe	Cyanosis
37	23	2	Severe	Cyanosis
Controlled Respiration (Low Tracheostomy)				
13	83	2	Severe	Cyanosis, respiratory distress
17	80	3	Severe	Severe rib recession periodic breathing, inspiratory stridor, inspiratory collapse of trachea
26	33	2	Severe	Inspiratory stridor rib recession, cyanosis
38	153	8	Severe	Rib recession, inspiratory stridor and cyanosis

(continued overleaf)

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Controlled Respiration (Low Tracheostomy) (continued)

Series Number	Duration of Tracheostomy (Days)	No. of Extubation Attempts	Tracheal Ulceration	Reasons for Failure of Extubation
51	81	3	Severe	Rib recession, inspiratory collapse of trachea
8	69	3	Moderate	Rib recession
14	44	2	Moderate	Rib recession, severe cyanosis, inspiratory stridor

(d) COMPLICATIONS OF TRACHEOSTOMY (continued)

Cyanosis During Suction

Series Number	Comments
Assisted Respiration Group.	
34	Bronchopneumonia present
52	FATAL
39	FATAL
18	Patient had lobar pneumonia
Controlled Respiration Group.	
12	FATAL
17	Airway obstruction partly contributed to cyanosis
30	Bronchopneumonia demonstrated at postmortem
31	Prolonged tracheal aspiration
42	Bronchopneumonia present
53	Bronchopneumonia demonstrated at postmortem
55	Bronchopneumonia present

(e) MECHANICAL FAULTS (RESPIRATOR and TRACHEOSTOMY ASSEMBLY)

Series Number	Respirator Failure (Frequency)	Faulty Respirator (Frequency)	Comments
Assisted Respiration Group.			
16	1		Infant breathing satisfactorily
21	1		Prompt resuscitation
40		1	Cyanosis and bradycardia
57		1	Cyanosis
Controlled Respiration Group.			
19		1	Cyanosis
20	1		Gross cyanosis
24		1	No cyanosis
26	1		Cyanosis
30	1		Respirator changed before cyanosis occurred
38	1		Cyanosis
44		1	Cyanosis
55	1		Cyanosis
58		1	Cyanosis
59		1	Cyanosis
	Loose Connection in Tracheostomy Assembly		
Controlled Respiration Group.			
44	1		Gross cyanosis
53	1		FATAL

Total Number of Mechanical Faults : 16

Total Number of Patients Affected : 16