we had two registrars at the same time who had not the knack of it. The first consideration is to prevent it happening by doing external cephalic version. The next point is to refer to a specialist the few patients in whom turning cannot be achieved, if the doctor does not wish to undertake the breech delivery. That leaves those in whom the diagnosis is not made before labour. There is still a chance that the condition may be diagnosed early in labour, so that if it is desired the patient may be transferred to a specialist hospital. If it is not diagnosed until delivery is starting, the doctor will have to deal with it himself, so that he should have some idea of what Tristram Shandy called "the plain principles of the business." The higher the proportion of successful external cephalic versions the less the experience in breech delivery. And, I should say, a very good thing too. The evidence is that breech delivery is no less dangerous in a multipara than in a primigravida.

Conclusion

I am afraid that I have not kept strictly to my subject throughout. This is probably the result of browsing in *Tristram Shandy*, which sets a shocking example in the matter of digression.

There are at least two types of experience in life, one of which it is more blessed to have occasionally. If I lived in the Lake District, and when I looked out of the window in the morning and could see on every clear day Helvellyn and Catchedicam, I might well soon become unconscious of their presence. But when I see them once a year or less, I get a fresh thrill every time. If I could follow the example of Horatio Bottomley and drink champagne every day for lunch, its fascination might well soon pall. As Shakespeare put it:

So am I as the rich, whose blessed key
Can bring him to his sweet up-locked treasure,
The which he will not every hour survey,
For blunting the fine point of seldom pleasure.

But for experiences that demand technique, like playing bridge or lawn tennis, or practising surgery or obstetrics, practise tends to make perfect or at any rate better, and gives one a sense of pride in achievement.

BIBLIOGRAPHY

- An Obstetric Survey by the South-west of England Faculty of the College of General Practitioners. Torquay, 1957. Supplement to *Research News Letter*, No. 14.
Report of the Maternity Services Committee, 1959. London.
Report on a National Maternity Service. Royal College of Obstetricians and Gynaecologists. 1944.
Report on the Obstetric Service under the National Health Service. Royal College of Obstetricians and Gynaecologists. 1954.
Memorandum from the Royal College of Obstetricians and Gynaecologists submitted to the Maternity Services Committee of the Ministry of Health, supplementing the above Report.
Nixon, W. C. W., and Hickson, E. B., *A Guide to Obstetrics in General Practice*, p. 280, 1953. Staples Press, London.
Redman, T., *Brit. med. J.*, 1950, 1, 814.

British Instruments, a directory and buyers' guide for 1959, is published by the Scientific Instrument Manufacturers' Association and the United Science Press Ltd., 9, Gough Square, London, E.C.4 (615 pp., 42s). It covers scientific and industrial instrumentation for research and production in a wide variety of subjects, and includes glossaries in French, German, and Spanish.

TREATMENT OF TETANUS NEONATORUM WITH INTERMITTENT POSITIVE-PRESSURE RESPIRATION

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Tetanus neonatorum has always carried a high mortality. In the past six years 40 cases have been admitted to Groote Schuur Hospital and only one survived. Many of these infants appeared to die of respiratory failure because, by giving intragastric oxygen, life could at times be prolonged for up to 24 hours after breathing had ceased. This fact, coupled with the successful treatment of tetanus in older patients with intermittent positive-pressure respiration (I.P.P.R.), inevitably led us to consider the application of I.P.P.R. to these infants. The only model of the Radcliffe pump (Russell and Schuster, 1953; Smith *et al.*, 1954) available to us is not, however, designed to deliver the small tidal volumes encountered in neonates. The youngest child treated with I.P.P.R. recorded in the literature (Lawes and Harries, 1956) was aged 10 months and died with surgical emphysema of the lung and a pneumoperitoneum. In this paper is described a simple modification of the Radcliffe pump and its application to the first nine cases of tetanus neonatorum to be treated with I.P.P.R.

The two defects in the standard Radcliffe pump and Stott valve mechanism when applied to infants are the high percentage of the tidal volume which is constituted by the dead space lying between the valve and the patient, and the inefficient activation of the Stott valve by the small expiratory volume of the infant. These defects were overcome (Fig. 1) by increasing artificially

FIG. 1.—Photograph of Case 8 while on intramuscular curare, showing how the Radcliffe respirator was adapted for use in infants. The rubber tube connected with the Y-piece is joined to the manometer.

the tidal volume on the patient's side of the valve. To do this a rubber bag of some 300-ml. capacity (a B.L.B. bag proved satisfactory) was attached to the larger opening of the delivery arm. To the narrower side-arm

was attached a Y-piece, one arm of which was connected to the tracheotomy tube, the other to the manometer. With this arrangement the inspiratory stroke of the pump inflates the bag rapidly, and a smaller volume of air is delivered to the patient, whose lungs become inflated almost at the same time as the bag. On the expiratory cycle, the air from the distended bag and from the infant's lungs passes out via the expiratory side of the Stott valve. The large volume of air plus the elastic recoil of the bag activates the valve very efficiently, and, by dilution, greatly reduces the effect of the dead space in the delivery arm. The only complete dead space is from the side-arm to the endotracheal tube. The inspiratory pressure can be varied according to the weights placed on the bellows of the pump and the tensile strength of the bag.

Eight infants in this consecutive series were Coloured and one (Case 2) was European. Their brief case histories follow.

Case 1

A female infant 4 days old was admitted on July 12, 1957, to the Red Cross War Memorial Children's Hospital with tetanus. A.T.S., sedation, and penicillin were given, but in the next 24 hours she deteriorated markedly and was transferred to Groote Schuur Hospital for I.P.P.R. While being transferred she stopped breathing and had to be Eve-rocked by the nurse in attendance. On arrival she was grey and cold (rectal temperature 86°F .— 30°C .), with a slow feeble pulse and breathing only with occasional gasps. An endotracheal tube was passed and I.P.P.R. was started. Spasms were controlled with paraldehyde and luminal. After 15 days of I.P.P.R. the first sign of returning consciousness was observed in that her eyelids contracted to a bright light shone in her face. At this time spontaneous breathing appeared to be adequate, so a tracheotomy was done. This was associated with a second severe anoxic episode. On the 29th day a third anoxic episode occurred when the tracheotomy tube became obstructed. By the 40th day feeds were being taken normally from a bottle. On the 49th day, just when removal of the tracheotomy tube was to be attempted, the child died suddenly and unexpectedly.

At post-mortem examination the brain was shrunken, with atrophic gyri, and on section showed extensive cystic areas of softening. These changes were interpreted as being due to cerebral anoxia.

COMMENT.—This, our first case treated with I.P.P.R., is the subject of a further communication. The length of survival was greater than had been anticipated, and indicated the need to start I.P.P.R. before irreparable anoxic damage had been done to the brain. With the possibility of an infant surviving, prolonged endotracheal intubation was undesirable. As no small cuffed tracheotomy tube was available an alternative was devised as described later.

Case 2

An infant aged 6 days was admitted to hospital on October 25, 1957, with tetanic spasms, which had started the previous evening. Tracheotomy was done under general anaesthesia. Spasms were partially controlled with paraldehyde, 1 ml. four-hourly, and phenobarbitone, $\frac{1}{4}$ -gr. (16 mg.) six-hourly. The following morning breathing became increasingly shallow, and when cyanosis occurred with apnoea I.P.P.R. was started. In the evening hypothermia (temperature 87°F .— 30.6°C .) and flaccidity developed. The heart sounds were faint. The infant was warmed and given digoxin, and all sedatives were stopped. On October 27 spasms recurred, and deterioration and death rapidly followed. The terminal E.C.G. shown in Fig. 2

was taken because of the difficulty in ascertaining whether the child was still alive. At post-mortem examination nothing abnormal was found.

FIG. 2.—Case 2. The electrocardiographic pattern is that of the "dying heart." Auricular activity is absent; ventricular rate is irregular, varying from 45 to 60 a minute; gross widening of the QRS complex is present.

Comment.—This case ran a fulminating course. There was a delay in starting I.P.P.R. and hypothermia was allowed to develop. Relaxants would have been tried had Case 1 not responded to sedation alone.

Case 3

An infant aged 8 days was admitted to hospital on November 18, 1957, with tetanus. Tracheotomy was performed under general anaesthesia and I.P.P.R. started soon after admission. Following this the child appeared to be unconscious. Mephenesin 1 gr. (65 mg.) hourly, and phenobarbitone, $\frac{1}{4}$ gr. (8 mg.) six-hourly, controlled spasms. Consciousness returned on the third day and spontaneous breathing out of phase with the pump on the fifth day, so I.P.P.R. was stopped. Feeds were taken from a Bellcroy feeder on the 19th day and subsequently from a bottle. All sedation was stopped by the 35th day. The infant was now normal in every respect except that repeated attempts to remove the tracheotomy tube were unsuccessful. On the 45th day a staphylococcal tracheitis developed which failed to respond to treatment. The child died three days later.

Post-mortem examination revealed a flap of granulation tissue hanging down from the upper edge of the tracheotomy wound into the lumen of the trachea and capable of obstructing the airway. Thick mucopus occluded the trachea and main bronchi. There was one small area of segmental pneumonia.

Comment.—This infant had recovered from the tetanus, and had it not been for the tag of granulation tissue preventing establishment of normal breathing, complete recovery would probably have followed.

Case 4

An infant aged 4 days was admitted to hospital on November 30, 1957, with tetanus. At the time of admission the infant had a heavy discharge of pus from the nose from which a pure growth of *Klebsiella pneumoniae* was grown. Early tracheotomy under general anaesthesia was followed by I.P.P.R. Consciousness was not regained. Spasms were controlled with mephenesin, 100 mg. hourly, and phenobarbitone, $\frac{1}{4}$ gr. (16 mg.) t.d.s. by stomach tube. During the second night, in error, mephenesin was not given and severe spasms occurred, the pulse rate rose to 200, the pupils failed to react to light, and muscular flaccidity developed. No stools were passed during the illness and the abdomen became increasingly distended. There were occasional vomits. On the fifth day gastric suction, together with an intravenous drip of curare and oxytetracycline, was started. On the seventh day the child died. A blood culture done immediately after death grew *K. pneumoniae*. Post-mortem examination revealed extensive focal necrosis of the liver due to the septicaemia. The trachea, bronchi, and lungs showed no disease.

Comment.—This child was heavily infected on admission. Septicaemia, ileus, and an anoxic episode were all factors that could have contributed to a fatal outcome.

Case 5

An infant aged 6 days had a tracheotomy under general anaesthesia and started I.P.P.R. soon after admission on December 12, 1957, with tetanus. Mephenesin, 2 gr. (130 mg.) hourly, was required to control spasms. The child never recovered consciousness. The pupils were first contracted and then dilated and fixed. There was a dusky cyanosis. The radial pulse was not palpable. Cortisone, digoxin, and methylamphetamine hydrochloride ("methedrine") were given. On the third day for a short period the pulse became palpable and urine was passed for the first and only time. The next day the child died. No stools were passed during the illness.

At post-mortem examination nothing abnormal was found. The cause of death appeared to be circulatory failure.

Case 6

This child was 8 days old when admitted to hospital on February 5, 1958, with tetanus, and had a tracheotomy under general anaesthesia. On her return from the theatre the pulse rate was over 200. I.P.P.R. was started. Mephenesin, 100 mg. two-hourly, and phenobarbitone, $\frac{1}{4}$ gr. (16 mg.) t.d.s., were given by stomach tube. Spasms were not adequately controlled. The infant remained slightly cyanosed, with contracted fixed pupils. On February 6 the mephenesin was increased to 100 mg. hourly. Although spasms were now controlled there was no improvement in the heart rate, which remained over 200. The pulse became weaker, and the heart sounds softer. As no urine had been passed the infant was catheterized and 25 ml. of urine obtained. At 4 p.m. on February 7 all therapy except I.P.P.R. was stopped. An E.C.G. taken at this time is shown in Fig. 3. The infant remained flaccid and

Fig. 3.—Case 6. The electrocardiogram shows auricular standstill. The ventricular rate is 70 a minute, with marked widening of the QRS, producing a gross right bundle-branch block pattern. The record is in keeping with severe hyperkalaemia. It also occurs preterminally before the onset of ventricular fibrillation.

unresponsive until death occurred at 11 p.m. on February 8. No stools were passed during the illness. A blood culture taken soon after death was sterile.

At post-mortem examination there was congestion of the lungs; otherwise nothing abnormal was found. The cause of death appeared to be circulatory failure.

Case 7

An infant aged 6 days was admitted to hospital on March 16, 1958, with tetanus. Tracheotomy was performed under general anaesthesia. Spasms were partly controlled with phenobarbitone, $\frac{1}{4}$ gr. (16 mg.) t.d.s., and mephenesin 1 gr. (65 mg.) hourly by stomach tube. This child was given a trial with tracheotomy alone, but gradually deteriorated with increasingly shallow respirations and cyanosis so that I.P.P.R. had to be started on March 18. Despite some

initial improvement the child died on March 23. An immediate post-mortem blood culture grew *Escherichia coli*.

At post-mortem examination the cause of death was found to be a severe bronchopneumonia.

Comment.—In this infant there was difficulty with a collapsed lung and superimposed infection occurred.

Case 8

A female infant aged 7 days was admitted to hospital on April 22, 1958, with tetanus. There was considerable infection of the umbilicus with a surrounding zone of erythema about 2 in. (5 cm.) in diameter. Tracheotomy and I.P.P.R. were carried out soon after admission. Gamma-globulin, 2 ml., was given intramuscularly as well as penicillin, streptomycin, and A.T.S. Whenever spasms occurred, which was about every 12 hours, 10 mg. of tubocurarine chloride with 0.5 ml. of hyalase was given intramuscularly. This was found more effective than any previous method, and produced complete relaxation. During the first few days the pulse volume was good and urine was passed; but slow deterioration followed. By the 9th day the infant appeared slightly cyanosed and comatose; the left upper lobe of the lung had collapsed and the radial pulse was barely palpable. Her condition appeared critical. Quite unexpectedly on the 10th day of I.P.P.R., no spasms having occurred for 17 hours after the last injection of curare, she opened her eyes. Within 24 hours she was weaned off I.P.P.R. and passed her first stool since admission. Spasms were now controlled with mephenesin, chloral, and phenobarbitone.

Water was swallowed from a Bellcroy feeder on the 26th day. Milk feeds were not taken from a bottle until the 31st day. All sedation was stopped on the 39th day, although muscular tone was still considerably increased. Repeated attempts were made to remove the tracheotomy tube, but these invariably resulted in such marked costal recession and cyanosis that the tube had to be reinserted. On the 56th day a Parker tracheotomy tube was inserted in which a hole had been made to communicate with the larynx. Occlusion of the ostium of the tube was sometimes followed by a period of adequate breathing, but ultimately always resulted in a severe cyanotic attack.

The following day breathing sounded obstructed and the tube had to be forcefully removed. The cause of the obstruction was a piece of granulation tissue (confirmed by histology) about 1 by 0.5 cm. which had herniated through the hole in the tube and had been guillotined off as it was removed. It was not clear whether this was another granulomatous polyp, as was found in Case 3, or simply granulation tissue off the wall of the tracheotomy opening. A fairly small area of attachment favoured the former explanation. Hopes were entertained of being able to remove the tube, particularly as the tone of the muscles was now normal, but further attempts were equally ineffective. On the 60th day, laryngoscopy under general anaesthesia failed to reveal any obstruction. On the 67th day after admission the tube was successfully removed, and the infant was subsequently discharged home apparently quite normal.

Comment.—This appears to be the first recorded case of tetanus neonatorum successfully treated with I.P.P.R. It seems to have been a severe case because of the intensity of the early spasms, the length of apparent coma, and the subsequent duration of muscular hyper-tonicity. In muscular curare was used because of the difficulty previously experienced in the severer cases of obtaining satisfactory control of spasms with mephenesin.

Case 9

A male infant aged 11 days was admitted to hospital on July 11, 1958, with a one-day history of tetanus. Tracheotomy was done under general anaesthesia and

spasms were fairly well controlled with phenobarbitone, $\frac{1}{4}$ gr. (16 mg.) t.d.s., and chloral, 1 gr. (65 mg.) two-hourly. Twenty-four hours later an episode of apnoea with cyanosis occurred, so I.P.P.R. was started. Mephenesin, 1 gr. (65 mg.) hourly, was given by stomach tube and the chloral was stopped. The pulse volume was satisfactory and urine and stools were passed normally. By the sixth day spontaneous breathing out of phase with the pump had started, but I.P.P.R. was continued for another day as the infant seemed quite comfortable. On the seventh day I.P.P.R. was stopped. The mephenesin dosage was reduced to 65 mg. two-hourly and chloral, 1 gr. (65 mg.) two-hourly was recommenced. Except during spasms this infant always swallowed his saliva quite well. Sucking movements were made on the 14th day. Water was taken from a Bellcroy feeder on the 15th day, but swallowing was not quite satisfactory as some water leaked out of the corner of the mouth until the 22nd day, after which all feeds were taken by bottle. Hypertonicity of muscles diminished and by the 29th day, when the tracheotomy tube was removed, he was partially unclasping his fingers and moving his arms around freely. He appeared quite normal when discharged home two days later.

Comment.—This case, of moderate severity, ran a fairly uneventful course.

General Management

On admission 1–2 ml. of intramuscular paraldehyde was given to control spasms. 10,000 units of A.T.S. intravenously and 20,000 units intramuscularly were given. 2 mg. of vitamin K was given to minimize bleeding at tracheotomy.

Tracheotomy was done under ether-and-oxygen general anaesthesia, with an endotracheal tube passed. No cuffed tracheotomy tube was available for infants, so a tube was made from a piece of Magill No. 1 endotracheal "portex" tubing $1\frac{1}{2}$ in. (28.6 mm.) long. This tube was passed through a window cut in the trachea and, without any cuff, fitted snugly enough to maintain positive pressure and prevent much leakage of saliva down from the pharynx. It was secured only by tape around the neck. The chest had to be auscultated frequently to ensure that the air passages were clear and that the tube was well placed, as small changes in the angle or depth could improve or impair air entry to the lungs. A brass connecting-piece inserted at one end attached the tube to the Radcliffe pump via the delivery arm. This arm was kept at an angle, with the Stott valve at the lowest point to ensure that water of condensation did not run into the infant. Dressing was not applied to the tracheotomy wound, with the result that there was far less subsequent trouble from infection.

With the exception of Cases 2, 5, and 9, which were given a trial period of tracheotomy and sedation alone, I.P.P.R. was started as soon as the infants returned from the operating theatre. The respirator was run at 26 cycles a minute and the pressure at 10–12 cm. of water. In Case 7 this was reduced to 8–9 cm.

The duration of I.P.P.R. in the four patients who achieved spontaneous respiration was largely determined by trial and error. The three infants on mephenesin were not completely paralysed, and spontaneous respirations out of phase with the pump indicated the time for a trial of unassisted breathing. In the child treated with curare (Case 8) attempts were made to lengthen the interval of about 12 hours between injections. Although spontaneous respirations occurred, and at times continued without the pump, muscular twitchings and

spasms would necessitate more curare. Quite abruptly this interval became prolonged and spontaneous breathing without the pump appeared to be adequate. Weaning from the pump was achieved over 24 hours by increasing the duration of spells of unassisted respiration. If there was a rising pulse rate, shallow breathing, or slight cyanosis a further period of assisted respiration would be given, and it was noticeable that the pump would take over respiration if it was required. If the pump was applied when it was not necessary the infant seemed to be uncomfortable and salivated excessively.

Once normal respiration was firmly established the plastic tracheotomy tube was changed for a No. 16 metal Parker tracheotomy tube. This tube was changed every four days. The infant was now nursed in a plastic oxygen box into which fresh humidified air was pumped by the Radcliffe respirator. This maintained humidity high enough to prevent trouble with concretions in the tracheotomy tube. With the shorter and narrower Parker tracheotomy tube the need for a cuff appeared to be greater, as a lot of saliva was aspirated at times from the trachea. Removal of the tracheotomy tube was not attempted until all saliva and feeds were swallowed normally and there were no attacks of laryngospasm. It is not clear why there was such difficulty in the final removal of the tube in Case 8. Possibly granulation tissue growing into the lumen of the trachea was the cause, and that may be an important complication of prolonged tracheotomy at the age of this infant. If hypertonia of the laryngeal muscles persists as long as it does in the limb muscles (in Case 8 for eight weeks) it may cause some degree of laryngeal obstruction, such as occurs in spastic infants. It is always possible that a bolder policy might have got the tube out earlier.

Relaxation with mephenesin in doses of $\frac{1}{2}$ –2 gr. (33–130 mg.) an hour, given by stomach tube, had the advantage of ease of administration and of relaxation without total paralysis, so that in the event of a mechanical failure of the respirator spontaneous respiration might still be possible. The control of spasms was, however, less certain than with the tubocurarine chloride given with hyalase intramuscularly. For a neonate the dose of 10 mg. of soluble curare is many times larger than that usually given in anaesthesia to produce muscle paralysis. It produced adequate relaxation in Case 8 for approximately 12 hours, and, so far as could be judged by clinical observation, there were no effects other than myoneural paralysis. Nor did the rapid rate of recovery of adequate respiration and muscle tone, following injections twice daily for 10 days, suggest that there was any cumulative effect. After stopping I.P.P.R., spasms were still moderately severe but rarely so prolonged as to cause cyanosis. They were adequately controlled with chloral, 1 gr. (65 mg.) and mephenesin, $\frac{1}{2}$ –1 gr. (33–65 mg), two-hourly, and phenobarbitone $\frac{1}{4}$ gr. (16 mg.) t.d.s. given by stomach tube. As the spasms decreased these drugs were reduced, being finally discontinued four to five weeks after admission.

Every effort was made to prevent pulmonary infection. A tube was run from the air inlet port of the Radcliffe pump out of the window, so that only fresh air was inspired by the infants both while on positive-pressure respiration and while in the oxygen box. Nurses and doctors wore masks, and hands had to be washed before aspirating the tracheotomy tube. "Polythene" tubes of

1 mm. diameter were used for suctioning the trachea and were kept in benzalkonium chloride. After the Parker tracheotomy tube was inserted, a size 3 rubber catheter was also used for suctioning, and was boiled before being used again. Rubber catheters were used for suctioning saliva from the mouth. 100,000 units of procaine penicillin was given intramuscularly daily to control infection; also, in the later cases, 125 mg. of streptomycin intramuscularly twice a day. Chloramphenicol was given in some cases.

Whether to use prophylactic antibiotics in an infant of this age with a tracheotomy is a difficult problem, but in Cases 8 and 9 all antibiotics were stopped after 14 days, with no trouble from infection, whereas in Case 3 continuous antibiotic cover resulted in a suppurative tracheitis due to a resistant staphylococcus. A good aseptic technique and efficient aspirations of the trachea and large bronchi are the more important factors in preventing infection. In Cases 8 and 9, to assist in the control of infection, 2 ml. of gamma-globulin was given intramuscularly on admission, this being repeated in Case 8 after six weeks. This procedure will be given a further trial in any future cases. Hydrogen peroxide compresses only were applied to the umbilicus.

Pressure sores on the scalp and actual depression of the bones of the skull developed in Case 1. Subsequently these were avoided by nursing the infants supine with the head lying free over the edge of a pile of blankets, most of the weight being taken by the nape of the neck supported on a roll of gamgee. This posture allowed full expansion of the chest, and, even though the infants were not turned at all for periods of up to 10 days because of the difficulty of maintaining a stable posture when lying on the side, no pressure sores developed, nor were there any hypostatic pulmonary complications.

A stomach tube was passed through the nose, preferably while the infant was still under the general anaesthetic. It may be difficult or impossible to pass a tube in the presence of tetanic spasms, possibly because of spasm of the soft palate or pharynx. If the tube had to be changed after relaxants had been discontinued, 1 ml. of paraldehyde was given intramuscularly to obtain adequate relaxation. Glucose water only was given by tube for the first few feeds and the stomach was aspirated four-hourly to ensure that there was no residue. Subsequently milk feeds were given, but aspiration of the stomach four-hourly was continued because of the frequency of ileus, with the risk of vomiting. Feeding by mouth, with water from a Bellcroy feeder, was attempted only after trismus had diminished enough for the infant to make sucking movements and pharyngospasm had been relieved enough for saliva to be swallowed. About five days' practice with a Bellcroy feeder was necessary before milk was taken normally from a bottle.

Hypothermia developed insidiously and rapidly. Four-hourly temperatures were taken with a low-reading thermometer and the child was warmed whenever necessary. Unlike older children with tetanus, these infants did not develop hyperpyrexia.

No praise can be too high for the nursing staff, who were all student nurses and without any special training. It is clearly desirable that changes of staff should be as infrequent as possible, so that the nurses may have the opportunity of familiarizing themselves with the technique of tracheal suction and with the appearance of the patient, and, by recognizing minor changes,

undertake corrective measures before serious retrogression has time to occur.

Discussion

There may be some doubt about the accuracy of a diagnosis of tetanus in the first few days of life, particularly as swabs from the umbilicus were cultured in all of the present series of cases, both on admission and at necropsy, without revealing tetanus bacilli. Excision of the umbilicus was not considered justifiable either as a therapeutic or as a diagnostic measure. It is felt, however, that the clinical picture even at this age is sufficiently characteristic. The early appearance of trismus in an infant that has thrived for the first few days of life, refusal to suck, followed by a risus sardonicus with pursing of the lips and frequently expression of saliva, together with generalized opisthotonic spasms provoked by stimuli, and board-like rigidity of the abdominal muscles, are as diagnostic in the infant as they are in the adult.

Although only two children recovered out of nine in this series, two deaths—one from cerebral anoxia and one from the tracheotomy—occurred after the infants had recovered from the effects of tetanus. At least one, possibly two, other infants died with a septicaemia, indicating the added hazard of umbilical sepsis and infection. Furthermore, the length of survival appeared to be prolonged, as all cases survived more than two days (see Table). This is in contrast to the average

Summary of Results of Treatment of Tetanus Neonatorum With I.P.P.R.

Case	Age on Admission (Days)	Days of I.P.P.R.	Days of Survival	Relaxant and Sedative	Cause of Death
1	4	15	49	Paraldehyde, phenobarbitone	Cerebral atrophy due to anoxia
2	6	1	2	" "	Circulatory failure
3	8	3	48	Mephnesin phenobarbitone	Granulomatous polyp in trachea and staphylococcal tracheitis
4	4	7	7	" "	Septicaemia, circulatory failure, anoxia
5	6	4	4	Mephnesin	Circulatory failure
6	8	3	3	Mephnesin, phenobarbitone	" "
7	6	5	7	" "	Bronchopneumonia
8	7	10	Recoverd	Intramuscular curare with hyalase, followed by mephnesin, phenobarbitone, chloral	
9	11	7	"	Mephnesin, phenobarbitone, chloral	

survival of 2.2 days among the 39 dying previously at this hospital from tetanus neonatorum following treatment with various combinations of different sedatives, chlorpromazine, cortisone, and hypothermia, but not with tracheotomy; two infants survived seven days, no others survived longer than four days. No fewer than 16 of the infants died within 24 hours of admission. It would appear, therefore, that tracheotomy with I.P.P.R. is an improvement on previous methods of treatment of tetanus neonatorum; and it is clear that this procedure is practicable even in these small infants.

Tompkins (1958) has described some of the factors that influence the severity of tetanus in the neonate. Two other factors were found useful in this series. They were brain-stem involvement with signs of respiratory or

circulatory failure, ileus, and coma, which always indicated a severe form of the disease; and the severity and duration of muscular spasm, especially of the pharynx. In milder forms the infants may swallow all saliva and even their feeds; with increasing severity of the condition saliva was extruded from the mouth, but only during spasms, and choking was likely to occur if feeds were given. In the most severe form no swallowing was possible at all, and saliva drooled continuously out of the mouth or lay pooled in the pharynx. During recovery the sequence was reversed and the number of days of treatment before, firstly, all saliva was swallowed and, finally, all feeds were taken normally from a bottle, was greater in those with a more severe form of the disease.

Even in the less severe forms the mortality remains high when sedation is the only treatment (Tompkins, 1958). It might be anticipated that tracheotomy would improve the prognosis, as anoxia due to laryngospasm or pharyngospasm, with retention of saliva in the pharynx, is common in these infants. In some cases tracheotomy may suffice, and no assisted respiration will be required; but respiratory failure should be watched for, as it can develop insidiously and quickly. In two of these infants shallow breathing and diminished spasms were misinterpreted as indicating good control, whereas they heralded apnoea and cyanosis. Because of this, early trial of I.P.P.R. may be preferable, and it would reduce the chances of any anoxia developing. I.P.P.R. can always be stopped as soon as spontaneous breathing appears adequate or if no improvement results. In the milder cases mephenesin may be the drug of choice as it allows of spontaneous respiration, but the more severe cases, with spasms that are difficult to control, cyanosis, and circulatory collapse, would appear to be better treated with curare.

Many problems require further investigation: in particular, the circulatory failure which dominated the clinical picture in those infants who died of tetanus. This was characterized by tachycardia, a fall in blood pressure (roughly estimated by the impalpability of the radial pulse, then the brachial, and finally the femoral), a dusky cyanosis, coma, and anuria. The abnormal E.C.G.s (see Figs. 2 and 3) taken in the late stages may well have been due to hyperkalaemia associated with anuria which followed upon the circulatory failure. There are many possible causes of the circulatory collapse. Tetanus toxin may have a direct toxic effect on the myocardium, although Brönnimann and Stirnemann (1956) were unable to demonstrate experimentally any such effect in the early stages of the disease. A more likely explanation would be tetanus intoxication of the brain stem causing circulatory collapse, with cardiac anoxia as a secondary effect. Another factor may have been cerebral anoxia due to incorrect ventilation by the pump.

The inspiratory pressure at which to run the pump was judged solely by auscultation of the chest and the clinical picture of the infant. Quite possibly this was too high and may have impeded the circulation by compressing the pulmonary capillaries. Such an effect would be accentuated by the incomplete muscular relaxation obtained by mephenesin, as the lungs would have to be expanded against muscle tone. Curare, by producing total relaxation of muscle, may allow a freer expansion of the lung and a better pulmonary circulation.

Other aspects requiring clarification are the disturbance of bowel function; which varied in severity from prolonged constipation to ileus; the generalized oedema, which was marked and prolonged; and the disturbance of consciousness. Although the level of consciousness was difficult to assess while relaxants were used, there did appear to be a definite moment when the return to consciousness could be ascertained. Furthermore, it may be significant that this always occurred before it was possible to stop I.P.P.R.

One other sign was a source of conjecture. This was the muscular flaccidity that occurred in the terminal phases of some cases. It may well have been due to anoxia (van Liere, 1942); but it is of interest that Brönnimann and Stirnemann (1956) have produced a flaccid palsy experimentally in animals by injecting more than twenty times the single lethal dose of tetanus toxin. This paralysis was fatal if artificial respiration was not provided. If a high concentration of toxin can cause paralysis, it could explain the palsy seen in cephalic tetanus, the flaccid terminal state of severe tetanus often ascribed to exhaustion. It could also explain the fact that in some cases in this series muscular tone appeared to increase for a time during the period of recovery, though previously it had been depressed. The importance of such a concept is that I.P.P.R. may be essential in the treatment of severe cases of tetanus if this type of paralysis occurs.

In future more active steps will be taken to overcome the circulatory failure; and, by estimating oxygen and carbon-dioxide levels in the arterial blood or alveolar air, to try to determine the optimum rate and pressure for the respirator. If, however, the clinical picture of circulatory failure with coma and ileus is largely due to tetanus intoxication of the brain stem, the prognosis is likely to remain gloomy. Should the superimposed effects of anoxia, hypercapnia, and drug therapy determine the outcome, much would still remain to be achieved.

Summary

A modification of the Radcliffe intermittent positive-pressure respirator is described which allows prolonged assisted respiration to be applied to neonates.

Nine cases of tetanus neonatorum which were treated with I.P.P.R. are described. Two children recovered completely. Two others survived for seven weeks and died from causes other than tetanus.

The methods employed to overcome difficulties that arose during treatment are outlined and the problems that still require clarification are discussed.

ADDENDUM.—Since this article was written the following case has been under our care.

Case 10

An infant aged 14 days was admitted to hospital and ran a similar course to that of Case 9. Respiration was assisted with I.P.P.R. for four days. Progress was uninterrupted except that, despite numerous attempts, the tracheotomy tube could not be removed. When the infant was 12 weeks old a bronchoscopy was done and granulomatous tissue was found obstructing the airway. It was extracted with forceps via the tracheotomy stoma. Two days later the tracheotomy tube was successfully removed. This clearly shows, what previously had only been suspected, that with prolonged tracheotomy in neonates granulation tissue may form around the tube in such quantity as to obstruct the airway.

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REFERENCES

- Brönnimann, R., and Stünnemann, H. (1956) *Proceedings of the World Congress of Anesthesiologists, Netherlands, 1955*, p. 282. Burgess Publishing Co., Minneapolis, U.S.A.
 Lawes, W. E., and Harries, J. R. (1956). *Lancet*, **1**, 783.
 Russell, W. R., and Schuster, E. (1953). *Ibid.*, **2**, 707.
 Smith, A. C., Spalding J. M. K., and Russell, W. R. (1954). *Ibid.*, **1**, 939.
 Tompkins, A. B. (1958). *Brit. med. J.*, **1**, 1382.
 van Liere, E. J. (1942). *Anoxia, Its Effect on the Body*, p. 239. University of Chicago Press.

TREATMENT OF SEVERE TETANUS

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Tetanus, a largely preventable disease, remains one of the few bacterial infections for which curative therapy is not available and the precise pathology of which is still disputed (Payling Wright, 1956; Abel *et al.*, 1938; Brooks *et al.*, 1955). However, it has been claimed, with considerable justification, that in most cases tetanus is a self-limited disease provided the patient can be tided over the storms of its assault. In some cases the mode of death is obscure, but many patients die from pulmonary causes, either from asphyxia during spasm involving the larynx and respiratory muscles, or from aspiration pneumonia and atelectasis (Creech *et al.*, 1950; Adriani and Kerr, 1955; Garcia-Palmieri and Ramirez, 1957).

The main aim of treatment, therefore, is to support the patient through the acute attack by minimizing muscular spasm and rigidity and by preventing respiratory infection. This involves a carefully integrated plan of management in which sedation and, more recently, muscle relaxants are used.

Many excellent articles have appeared in recent years, reviewing the conflicting views on the pathogenesis and treatment of tetanus (Drew, 1954; Shackleton, 1954; Forbes and Auld, 1955; Ablett, 1956; Creech *et al.*, 1957). Except for the reports of Adriani and Kerr (1955) and of Veronesi (1956), most of the larger series of cases have come from centres with relatively limited resources (Johnstone, 1958), while the application of newer techniques has been reported in smaller numbers, making assessment of their value difficult. Further difficulty in comparing the results of treatment arises because so many factors affect the outcome of tetanus and cases must be divided into prognostic categories, often by elaborate criteria.

The value of tracheostomy is generally accepted, and more recent methods of treatment which have been advocated include continuous or intermittent intravenous mephenesin (Adriani and Kerr, 1955; Veronesi, 1956), or chlorpromazine, promethazine, and pethidine, which has been used extensively in Europe, and full curarization with artificial respiration (van Bergen and Buckley, 1952; Lassen *et al.*, 1954). Other clinics have further complicated the management by using hypothermia (Hossli, 1956).

In a period of five months from June to October, 1957, 14 cases of tetanus were admitted to the Royal Adelaide Hospital, and nine of these were classified as severe. They were treated by full curarization and artificial respiration. These nine cases are reported here with a description of the problems which arose in the use of this technique, and an assessment of its value in the severe case.

Method of Treatment

Each patient was admitted to a sound-proof room, and after skin tests 300,000 units of antitetanic serum was administered, half of it intravenously. Surgical opinion was then obtained on the question of debridement of the entry wound. When it was indicated, tracheostomy was performed under general anaesthesia at the same time as debridement, and a cuffed rubber tube was inserted. The indications for tracheostomy were essentially those of Ablett (1956), and this operation was performed early rather than late. Assessment of the patient's ability to cough effectively was found to be particularly significant. In order to correct the frequent dehydration, to nourish the patient, and to administer drugs, an intravenous drip was inserted, preferably a "polythene" catheter to prevent extrusion during spasms. By this route, in the earlier cases, mephenesin and later chlorpromazine were administered intermittently. Penicillin was given, and the sedatives found to be useful were promethazine, pethidine, chlorpromazine, paraldehyde, and chloral. If tracheostomy had to be performed, sterile tracheal toilets were done. During this time there was continuous nursing surveillance with frequent medical assessment.

If heavy sedation failed to inhibit spasms, especially of the muscles of respiration, curarization was begun with artificial respiration. This involved continuous medical supervision together with expert anaesthetic assistance, particularly during the periods of induction and of decurarization.

The gases were delivered to the patient through the absorber unit of an anaesthetic machine, maintaining a flow of 6 to 8 litres a minute. During the period of initiation and stabilization a mixture of nitrous oxide and oxygen was used and then a variable mixture of compressed air and oxygen, breathing being maintained by the Beaver respirator adjusted to provide positive and negative pressure. Ventilation was mainly controlled according to half-hourly records of blood pressure, pulse rate, the presence or absence of sweating, and cyanosis. These observations were affected by variations in posture, sedation, muscular tone, and infection, as well as by hypoxia and carbon dioxide retention or lack. Twice-daily estimations of venous carbon dioxide combining power gave warning of gross respiratory alkalosis. The pH of the blood and plasma, was also measured. The latter was found to be more