before there is any feeling of security. In the 1949-50 series 40% of the patients were alive at the end of two years and 34% at the end of three years. If these figures follow the pattern of the other group it would seem reasonable to suggest that a 25% survival at five years might be expected.

Many of those who have died are known to have died from causes other than recurrence of cancer of the lung; suicide, accident, and old age have all played some part in reducing the number of survivors. A number of those who have exceeded the five-year period are known to have lived longer than this arbitrary limit. My longest survivor is 15 years (a case not included in this analysis), and there are four who have lived 10 years, and a greater number who have passed the seven-year mark.

An analysis of any surgical results in the treatment of cancer is bound to be disappointing, and there would seem to be an impression that the results of treatment in lung cancer are so poor that surgery is not worth while. This is suggested by the number of patients who are treated conservatively or are referred for radiotherapy without being seen by a thoracic surgeon, who alone can give the best advice regarding the chances of operation in an individual case. In spite of the many disappointments there is a kernel of success and satisfaction in that at least 21% of the patients operated on survive the five-year period, and indeed in the more recent cases there is every reason to expect that this figure may be appreciably raised.

Summary

An analysis of personal cases of cancer of the lung operated on between 1940 and 1950 is made.

Of a total of 689 thoracotomies, 446 were found suitable for excision—an operability rate of about two-thirds of the cases explored.

A follow-up to the end of 1953 gave for cases operated on prior to 1948 a survival rate of 34% at the end of two years, 30% at three years, 23.5% at four years, and 21% at five years. In the 1949-50 series 40% were alive at the end of two years and 34% at three years.

It is thought that there is some satisfaction that at least 20% of these patients survived the five-year period. Moreover, the more recent cases suggest that this figure may be improved upon.

REFERENCES

- Aylwin, J. A. (1951). *Thorax*, 6, 250.
Stocks, P. (1947). *Studies on Medical and Population Subjects*, No. 1. H.M.S.O., London.
Taylor, A. Brian, and Waterhouse, J. A. H. (1950). *Thorax*, 5, 257.

According to the Annual Report for 1952 of the Chief Inspector of Mines, recently published, the number of persons killed in 1952 was a new low record, being 420, comparing with 487 in 1951 and an average of 583 in the previous ten years. Much of the improvement in 1952 was due to the absence of any great disasters. Deaths due to falls of ground were fewer than in the previous year's, but deaths due to haulage increased by 22 over the previous year's. "The numbers of fatalities in shafts and on the surface have shown no improvement in the last ten years or so—a state of stagnation which constitutes both a reproach and a challenge." The number of accidents required by law to be immediately reported involved 2,109 injured people, as compared with the 1951 figure of 1,942. People injured in accidents not immediately reportable but causing disablement for more than three days numbered 238,100, as compared with 234,400 in the previous year. But as more people were employed there was little change in the accident rate, which was one accident for every three employees. In the last decade fatal mining accidents have fallen by 52% and non-fatal by 25%. The safety organization of the National Coal Board continued to grow, but there were 33 vacancies in the mining inspectorate.

INTERMITTENT POSITIVE-PRESSURE RESPIRATION IN BULBO-SPINAL POLIOMYELITIS

USE OF THE RADCLIFFE RESPIRATION PUMP

BY

J. R. HARRIES, M.D., M.R.C.P., D.C.H.
D.T.M.&H.

Acting Medical Specialist, Colonial Medical Service, Kenya Colony

AND

W. E. LAWES, F.F.A.R.C.S., D.A., D.T.M.&H.

Anaesthetic Specialist, Colonial Medical Service, Kenya Colony

The use of positive-pressure respirators driven either by hand or by machines has been described by Lassen (1953). Smith *et al.* (1954), and Macrae *et al.* (1954) in the treatment of bulbo-spinal poliomyelitis, where postural drainage combined with some form of tank respirator is not sufficient to prevent inhalation of secretions. Recently we have treated three such cases in the poliomyelitis unit at Nairobi, Kenya, all in male adults, and the Radcliffe respiration pump (Russell and Schuster, 1953) was used. One patient died from shock and hyperpyrexia; one died suddenly on the twenty-fifth day of illness, no cause

Recent modifications in the Radcliffe respiration pump (P), and accessories. The pump has alternative motors—one for 230 volts A.C. and the other for 12 volts D.C. The latter is very suitable for transporting patients by air or road. The gas meter (M) replaces the cumbersome spirometer. H=Humidifier. V=Expiration flutter valve. G=Gauge. T=Cuffed tracheotomy tube.

being found at necropsy; while the other was flown to Oxford, still using the Radcliffe pump, at the end of the eighth week and is making satisfactory progress.

Christie (1954) has pointed out the difficulty of clinical assessment and has emphasized the importance of being able to call on the services of the physiologist, the biochemist, the clinical pathologist, and the pharmacologist. Such assistance is generally available only at the larger medical centres, and the aim of this article is to point out to those working in more remote centres that simple clinical methods can be passably successful.

For a general description of the machine and method the reader is referred to the article by Smith *et al.* (1954). This machine has since been modified as shown in the accompanying illustration (Ritchie Russell, 1954, personal communication).

Type of Case.—Intermittent positive-pressure respiration (I.P.P.R.) through a cuffed endotracheal tube inserted into a tracheostome was reserved for those cases having respiratory embarrassment, an ineffective cough, inability to swallow, and pooling of secretions in the pharynx.

Tracheotomy

Andersen and Ibsen (1954) have described how local analgesia was abandoned as unsatisfactory in the Danish epidemic of 1952, and as this agreed with our previous experience it was decided that general anaesthesia should be used. All cases received thiopentone and succinylcholine chloride; atropine, 0.6 mg., was the only premedication. Small doses of thiopentone, 0.15–0.3 g., were used, but the usual dose of succinylcholine chloride, 2–3 ml. in divided doses, as the procedure took ten to fifteen minutes. In one case in which the operation was performed elsewhere and the services of an anaesthetist were not available, the dose of succinylcholine chloride was too small and disaster very nearly followed. The anaesthetist must have full immediate control of the patient, who may be in poor condition.

The steps of the procedure were as follows: (1) Aspiration of stomach with the Levine tube: this we regard as important, as regurgitation can occur without warning and a fatal outcome may result. (2) The patient's lungs were inflated with oxygen through a Waters to-and-fro unit from which the canister had been removed. (3) Thiopentone was given in a sleep dose only, followed by succinylcholine chloride, 1–1.4 ml., other syringes containing 0.5–0.7 ml. of succinylcholine chloride being available as required. In only one of our cases was there any weakness of the jaw muscles. (4) Laryngoscopy and laryngopharyngeal toilet. (5) Bronchoscopy and bronchial toilet. (6) Intubation with a plain endotracheal tube. (7) Tracheotomy, which under these circumstances can be done quietly and deliberately. Our surgical colleague commented on the difference between the ease of the operative procedure under this method and the difficulties he had experienced in previous cases under local analgesia. No window was made as suggested by Lassen (1953), and Macrae *et al.* (1954).

General Management and Treatment

Physiotherapy.—All joints were passively moved through their full range once daily. Splints were applied where necessary to prevent deformity. After about a month active exercises were instituted.

Antibiotics.—From admission, antibiotics were given in full doses, the choice depending on the sensitivity of organisms cultured on bronchial swabbing.

Other Drugs.—Phenobarbitone, 32 mg. t.d.s., and ephedrine hydrochloride, 16 mg. t.d.s., were given. Chloral, 1.3 g., was given at night. Paraldehyde intramuscularly or by mouth was used occasionally. Aspirin was used to allay the muscular pains and vitamin-B complex was given daily.

Investigations.—Blood urea estimations were done twice weekly. Bronchial swabs for culture were taken weekly. Urine chlorides were estimated daily. Total blood counts were performed at weekly intervals. Plasma proteins were estimated from time to time.

Fluid Intake and Diet.—Feeding was by means of a Levine tube. The aim was 3 litres of fluid and 2,500 calories daily. The salt intake was governed by the Fantus test for urinary chlorides and the protein intake by the blood urea.

Specific Management and Treatment

The endotracheal tube was changed every three days and deflated four-hourly. Macrae *et al.* (1954) have pointed out the danger of overinflation of the cuff. The cuff should be inflated slowly until air ceases to pass round it; this end-point can be detected by auscultation over the trachea, though sometimes it is not easy. If air enters both apices equally then the tube is not too far in. The management of the tube caused some difficulty at first, as it was not possible for an anaesthetist to be present all the time. The nursing staff quickly learned the dangers of these tubes, as attested by the fact that, during the three months in which the machines have been in continuous use, only on three occasions have emergencies arisen specifically from the cuffed tube. Twice the tube slipped into the right main bronchus, completely blocking the left; on the third occasion an old tube was used, and the very soft cuff herniated over the end of the shortened tube, giving a ball-valve effect. Air could be forced past the herniation but very little could escape. Releasing the cuff had little effect, and the herniation persisted even after the tube had been withdrawn from the trachea. This emergency almost ended in disaster, and since that time no tube has been used more than twice.

Pharyngeal, Tracheal, and Bronchial Toilet.—Suction was performed through the mouth and the endotracheal tube at regular intervals, also before and after turning, and whenever there was any evidence of pulmonary collapse or whenever the stroke of the pump seemed to be impeded. Posturing of the patient, especially into the prone position, with percussion and squeezing of the chest at the end of expiration, brought the secretions within range of the catheters. Bronchial catheters, soft rubber catheters, and Tieman catheters were used; the last-mentioned tended to cause some bleeding unless used very gently. Bronchoscopy was needed during treatment on only four occasions apart from the original surgical procedure: twice when the humidifier had been set at too low a figure and secretions had become dry and sticky; once when it had been thought that a considerable amount of secretion had run down past a deflated cuff; and once for pulmonary oedema, but bronchoscopy in this last condition seemed to do little if any good. The site of the cuff was inspected on each occasion, and appeared to be normal, except on one occasion when the left main bronchus showed a minor degree of excoriation, probably resulting from the too vigorous use of a Tieman catheter. Bi-weekly x-ray pictures were taken to check the clinical findings.

Humidification.—The apparatus described by Marshall and Spalding (1953) proved satisfactory. The temperature of the thermostat was originally set at 110° F. (43.3° C.), but was later increased to 130° F. (54.4° C.), as the sputum became too sticky at the lower figure. A fair amount of condensation occurred in the lagged tube, and care was taken to ensure that it sloped away from the Stott valve towards the humidifier.

Pressure Gauge.—It has been shown that if the pressure in the trachea does not fall to atmospheric level at the end of expiration the circulation will be affected (Cournand *et al.*, 1948; Motley *et al.*, 1948). The gauge at times needed readjusting by disconnecting it from the machine and resetting the pointer. The pressure on inspiration was not allowed to exceed 16 cm. of water. Common sites of leakage, as shown by a reduction of pressure, were round the cuff and at the Stott valve.

Increased Oxygen Percentage.—At first we tended to give extra oxygen through the side tap of the machine. This we later discontinued, as on only a few occasions was oxygen really needed and high flows upset operation of the valve. When oxygen is required it is best to lead it into a reservoir bag attached to the main intake of the inflating bellows.

Control of Respiration.—Spirometer readings of expired air are essential, as the volume delivered each minute by the machine does not all reach the lungs. The difficulty

of deciding the optimum ventilation for each patient has been stressed by many writers (Smith *et al.*, 1954; Macrae *et al.*, 1954; Astrup *et al.*, 1954). Overventilation upsets the circulation and causes alkalaemia. Underventilation causes CO₂ excess with or without anoxia. Some degree of hypertension would suggest CO₂ retention and a rise of urinary pH alkalaemia. Clinical assessment we found to be difficult. Various clinical signs have been reported as being indicative of CO₂ excess. Ibsen (1954) describes a rise in blood pressure, cold clammy skin, gagging and bucking of respiration in cases in which spontaneous respiration is present, and increased secretions in the mouth and nose—all the symptoms being relieved when respiration is increased. Case and Haldane (1941), working on conscious volunteers breathing 6% CO₂, describe hyperventilation, relatively little mental impairment, but some mental distress, slight headache, photophobia, and nausea.

The problem of CO₂ could not be investigated by elaborate methods as described by Astrup *et al.* (1954). A simple CO₂ meter of German manufacture (Dräger) was obtained from Messrs. Garthur, and was used to measure the CO₂ content of the expired air. Sampson Wright (1945) gave a figure of 4% for CO₂ in expired air. It was decided that a figure of 4% should be regarded as the maximum permitted for samples collected in a bag at the respiratory vent. As the meter gave slightly different readings from the same sample, the mean of three readings was used. There were, however, occasions when the figures seemed somewhat bizarre, and the meter readings were used only as a guide. On a few occasions attempts were made to obtain figures for air at the carina, but the figures were very variable, possibly owing to turbulence, and this method was abandoned. Routine urinary pH examinations were done as a check against hyperventilation.

The respiration pumps were used at speeds of 20 to 26 a minute, and the readings for expired air on the spirometer varied from 9 to 14 litres a minute. The higher figures were used on a heavily built man with an axillary temperature of 42.2° C. (108° F.) in whom it was exceedingly difficult to get the CO₂ readings down to 4%. These spirometer readings are higher than those recommended by other writers (Smith *et al.*, 1954; Astrup *et al.*, 1954; Macrae *et al.*, 1954), but on only one occasion was the urine alkaline.

Case 1

A European air-pilot aged 33 was admitted to the Infectious Diseases Hospital, Nairobi, on September 4, 1954. He had been flown from Mweiga, a distance of some 300 miles (480 km.). One week before admission he had suffered from epigastric pain, which continued for five days, when he developed frontal headache and aches across the shoulders and behind the knees. He had lost his appetite and had had several bouts of vomiting.

On admission he was found to have stiffness of the neck and back, horizontal nystagmus, and slight weakness of the flexors of the right hip. On the following day the nystagmus had increased and there was weakness of the left masseter and palate. He was cyanosed and no movements of the intercostals were detected. The flexors and extensors of the elbow-joints on both sides were weak. He was unable to raise his legs from the bed. The respiratory embarrassment increased, and at 1.50 p.m. he was put into a tank respirator. The next day his condition remained about the same. On September 7, 36 hours after being put into the tank respirator, he was found to be cyanosed, and the left side of the face and the left masseter were paralysed. He was unable to swallow, and secretions had collected in the hypopharynx. Both the intercostals and the diaphragm were paralysed, and all the limb muscles were paralysed to a greater or lesser degree. It was decided at this stage to institute intermittent positive-pressure respiration by means of the Radcliffe pump. The operative procedure and routine management employed have already been described.

His progress was marked by the frequent occurrence of pulmonary collapse. On one occasion this was caused by the endotracheal tube slipping down the right bronchus. On all other occasions plugs of mucus were the causative factor. These were dealt with rapidly and satisfactorily by turning, tipping, percussion, and squeezing of the chest combined with bronchial suction. The relief of collapse in these cases could only be referred to as dramatic. In almost all cases the collapse occurred in the left lung. On two occasions early on it was necessary to have recourse to bronchoscopy.

His temperature took about three weeks to settle. The first week it slowly fell from 103 to 101° F. (39.4 to 38.3° C.), the second week there were occasional spikes to 100° F. (37.8° C.), and the third week to 99° F. (37.2° C.).

On September 19 he was able to make short attempts to breathe alone, but not for more than 30 seconds at a time. On the 21st the Levine tube was removed, as ability to swallow was completely recovered. On the 22nd an attempt to replace the I.P.P. respirator by the Paul-Bragg pulsator was unsuccessful. After three hours in the pulsator the CO₂ in his expired air had risen to 6.2% and his blood pressure had increased from 135/80 to 172/105. Normally his CO₂ levels for expired air were between 2.5 and 3% and his blood pressure was about 135/80.

The usual ventilation given was 10 litres a minute, but this was increased to 12.5 litres a minute on October 11, by which time his blood-pressure level had slowly risen to 150/100. The increased ventilation caused a slow decline to the previous level.

On October 5 weaning to a Monaghan type of cuirass respirator was tried. This again was unsuccessful. After five minutes in the cuirass his blood pressure had risen to 225/105 from 130/85, his pulse had fallen from 100 to 40 and had become irregular, and the CO₂ in his expired air was 5%. Both blood pressure and pulse returned to their previous levels eight minutes after restarting I.P.P.R. Similar results were obtained on repeating the procedure, with elevation of blood pressure to 220/120, pulse 60, and CO₂ 4.2%. In an attempt to explain the mechanism of these changes, we carried out the following experiment.

The patient was inflated with pure oxygen at a slow rate (2.5 litres a minute) in order to produce CO₂ accumulation. After eight minutes the blood pressure showed a slight rise to 160/105, the pulse remained constant, and the CO₂ was 4.1%. The oxygen was then stopped and he was allowed to breathe by himself. After one and a half minutes his blood pressure had further risen to 190/115, and his pulse had fallen to 60. The Radcliffe pump was restarted and after about 10 minutes the blood pressure, pulse, and the CO₂ readings settled to 135/85, 96, and 2.5% respectively. After a rest, the patient was then allowed to breathe by himself, but this time he was given pure oxygen. After eight minutes there was a slight rise in blood pressure to 158/105, but the pulse remained constant. However, after stopping the oxygen and allowing him to breathe air, the blood pressure rose slowly during the first three minutes to 162/110, and the pulse dropped to 84. In the next three minutes the blood pressure rose rapidly to 190/115 and the pulse fell to 60. No cyanosis was observed.

We concluded that these anomalies of elevation of blood pressure and fall in pulse rate were probably due to a certain degree of anoxia. We thought it possible that he had a hypertensive vasomotor centre or carotid sinus which responded to hypertension by causing a marked slowing of the pulse rate. In an attempt to wean the patient from the Radcliffe pump, he was allowed to breathe pure oxygen on his own, and when he was evacuated to England he was able to breathe for 13 minutes at a time. By December 2 we understand that he could breathe without assistance for eight hours a day.

When he left Nairobi recovery from the cranial nerve palsies had occurred, he had a fair amount of power in all the arm and shoulder muscles, and there was movement in

all the muscles of the left leg and in the hamstrings and gastrocnemius of the right leg. The sacrospinalis showed some muscle recovery.

On October 24 he was flown to the Radcliffe Infirmary, Oxford, a distance of some 4,500 miles (7,240 km.), in a pressurized plane to the Respiration Unit directed by Dr. W. Ritchie Russell. He was accompanied by a team consisting of a physician (J. R. H.), a hospital superintendent, and a nursing sister. The machine was run from batteries by means of a dynamo to provide 240 volts. Oxygen was carried but not used. The journey of 21 hours' flying time was uneventful, apart from a collapse of the entire left lung due to secretions. The collapse was rectified by suction, posturing, and slapping. The team was met at London Airport and the patient transferred to Oxford by ambulance under the care of Dr. Crampton Smith. The latest Radcliffe respiration pump has since been modified so that it may be run from 12-volt batteries (Ritchie Russell, 1954, personal communication) (see Fig.).

Case 2

On October 15 a male Chinese electrician aged 46, working at Musoma, Tanganyika Territory, developed fever, headache, and a sore throat. On the following day the left leg became paralysed. On the 17th the right leg followed suit, and he was unable to pass urine. On the 18th he was transferred to Nairobi by plane—a distance of some 400 miles (640 km.).

On admission he had slight difficulty in swallowing; the right side of the chest moved poorly and paradoxically. Both legs were paralysed with the exception of the dorsi and plantar flexors of the right ankle-joint. His bladder was distended. At this time there was no involvement of the arms or of the diaphragm. He was nursed in the lateral positions with the foot of the bed raised. He would not tolerate the semi-prone position. The following day (October 19) he developed nystagmus, and had marked impairment of deglutition and weakness of the shoulder girdles. His respiratory rate was 40, with abdominal respirations. The intercostals on the left side were now involved. Some secretion was accumulating in the pharynx. At this time his temperature was 100° F. (37.8° C.), pulse 132, and blood pressure 115/85. A Levine tube was passed into his stomach at 2.15 p.m. This had to be done under anaesthesia (thiopentone and succinylcholine chloride). At 11 p.m. his temperature rose to 105° F. (40.6° C.), pulse 162, and respirations 38. At 1.30 a.m. he was noticed to have periodic respirations, his pulse being difficult to palpate, with blood pressure 85/70, and pulse 162. The pharyngeal secretions had increased and he became very restless. Cyanosis was not observed. The Paul-Bragg pulsator was found to be of little help, so it was decided to institute I.P.P.R. by means of the Radcliffe pump rather than the tank respirator—compare experiences quoted by Macrae *et al.* (1954).

Three hours after starting the I.P.P. respirator his blood pressure had risen to 104/80, and pulse to 158. He became very restless after recovering consciousness and was given 6 ml. of paraldehyde intramuscularly three and a half hours after the tracheotomy had been performed. He lapsed into unconsciousness after the paraldehyde, and did not recover until 15 hours later. At 10.30 a.m. on October 20 his temperature was 103.2° F. (39.6° C.), and he was given 25 mg. of chlorpromazine hydrochloride intramuscularly. In the afternoon of the same day his blood pressure fell to 60/50, and a noradrenaline drip was set up, which maintained the pressure at about 105–120 mm. Hg systolic, with occasional drops to 80. He had pulsus paradoxus, the fall in pressure during inspiration being about 10 mm. Hg. This improved when the stroke volume of the machine was reduced and the rate increased.

In the evening his temperature again rose to 105° F. (40.6° C.). The noradrenaline was stopped after nine hours, but had to be restarted as his systolic pressure fell to 45 mm. Hg. After restarting at a slower rate, his blood pressure was maintained at about 80 and gradually rose to 90, when

the drip was finally discontinued after 17 hours. At 9.30 p.m. he was given 25 mg. chlorpromazine hydrochloride intramuscularly in an attempt to reduce his temperature. Later the same day the machine was discontinued for two intervals of ten minutes as he was fighting against it. Shallow abdominal respirations were present, but in a short time the blood pressure rose to 145/100, and the CO₂ was 4.3%, so the machine was restarted.

By October 26 his temperature had gradually settled to normal and his blood pressure averaged about 115/70, pulse 100. From this day onwards he was tried for gradually increasing periods without the machine. On the 28th he developed pulmonary oedema for some 16 hours.

On November 2 he suddenly collapsed and became cyanosed, and was so for 20 minutes until medical aid was at hand. The cuff tube was at once removed, with dramatic recovery. The accident was thought to be due to a herniation in the cuff covering the opening of the endotracheal tube, producing a ball-valve effect. A bronchoscopy was performed, when a large amount of pus was found in the right bronchus. Two days later he began to bring up frothy sputum, several ounces a day, with signs of pulmonary oedema, which continued until his death.

On November 9, after breathing by himself for 16 hours, he became a little restless and the I.P.P. respirator was restarted at 10 p.m. He was given 6 ml. of paraldehyde as a sedative by mouth and 0.6 mg. of atropine subcutaneously, as he was bringing up a fair amount of frothy sputum. At 1.15 a.m. his condition was recorded as good. At 2 a.m. his pulse was said to have become thready. Medical aid was summoned, but the patient was found to be dead, with post-mortem mottling of the skin.

Pulmonary Complications.—Throughout the whole course of the illness large amounts (several ounces) of mucopus were aspirated daily. Only on one occasion, however, did a pulmonary collapse occur—in the left lung. After the advent of pulmonary oedema the amount of mucopus decreased considerably and was replaced by frothy pink-hued sputum. X-ray films of his chest revealed an old quiescent tuberculous lesion of the right apical region and elevation of the left diaphragm.

Mental State.—Great difficulty was experienced in communicating with this patient owing to the language difficulty. His knowledge of English was slight and of Swahili little better. He became very agitated when trying to explain his wants, and suffered from marked depressive phases. At night he used to pull out the Levine tube and pull off the bedclothes and was most irrational. We noticed with European patients that though these phases occurred they tended to be much less marked.

Investigations.—An electrocardiogram was taken on November 4, when the patient was breathing by himself and again while breathing on the machine. The following is an extract of a report which was given by Dr. A. D. Charters, consultant cardiologist to King George VI Hospital, Nairobi: "The electrocardiogram during unaided respiration shows marked clockwise rotation of the heart with low-voltage QRS in all leads, particularly in CR₇, which shows flattened T waves. Rhythm regular, rate 100. P-R interval 0.16 second. QRS 0.08 second. R-T 0.28 second. T wave inverted in VR and upright in the other leads. *Comparison of tracings while in the respirator with those during unaided respiration:* The voltage in the respirator is less in Leads II, CR₁, and CR₄, with smaller T waves in Leads II, CR₁, CR₄, and VL. The voltage is higher in the respirator in VR and CR₇, with a higher T wave in CR₇. P, R, and T waves are all taller in Lead III during unaided respiration."

Case 3

A male European, aged 40, weight approximately 90 kg., was admitted to Kitale Hospital, some 300 miles (480 km.) from Nairobi, on October 22, 1954, suffering from respiratory weakness. Respiration was so embarrassed that he was placed in a tank respirator on admission. He had

been ill for one week beforehand, but had carried on working, as he thought he had "flu." We were later informed that on the day previous to admission he was so ill that he could barely drag himself from his car, and his temperature had been 102.5° F. (39.2° C.).

A team consisting of a physician, a hospital superintendent, and a nursing sister flew to Kitale within a few hours. Bulbar signs had already appeared when the team reached Kitale, and the patient was cyanosed. A tracheotomy was performed and I.P.P.R. started. He was very restless and irrational during the night despite 8 ml. of paraldehyde intramuscularly. The blood pressure fell to below 100 mm. Hg systolic and sweating was profuse. One litre of 0.9% saline was given, as he was considerably dehydrated. No method of tube feeding was available. The temperature rose slowly from 102 to 105° F. (38.9 to 40.6° C.); sponging reduced it to 103.5° F. (39.7° C.).

The Radcliffe pump was hand-operated for two and a half hours during the return flight. The pilot deliberately flew at the lowest possible altitude, 9,000 to 11,000 ft. (2,700 to 3,350 m.). A foot-pump-operated sucker was used during the journey to aspirate the bronchi.

On arrival at the poliomyelitis unit at 9 a.m. on October 23, his general condition was fair and the flight did not appear in any way to have affected him adversely. He was conscious and somewhat restless, he could not swallow, and there was complete paralysis of the intercostals and diaphragm, and of the shoulder girdle and arms; but full movement was present in both legs. His pulse rate was 120, blood pressure 130/90, and temperature 102.3° F. (39.1° C.) in the axilla. Temperature readings from then on were taken every half-hour in the axilla. Chlorpromazine hydrochloride, 25 mg., was given intramuscularly. One hour later bronchoscopy was carried out under 0.3 g. of thiopentone and succinylcholine chloride (the previous evening he had needed 2 g. of thiopentone in addition to succinylcholine chloride). The tracheostoma was enlarged and a No. 10 cuffed tube inserted. His temperature fell slightly after the chlorpromazine, but rose again within an hour and continued to rise sharply to 104° F. (40° C.) that evening, his blood pressure remaining at 140/90, pulse 120. The CO₂ readings were high—just under 5%—in spite of increasing the minute volume from 11.5 litres to 12.7 litres. The CO₂ readings fell to 2.2% when the minute volume was further increased to 14 litres. A second dose of chlorpromazine, 25 mg. intramuscularly, was given, but without much effect on the temperature. A cold sponge shortly afterwards, however, reduced it to 102.6° F. (39.2° C.). No fall in blood pressure followed the chlorpromazine.

The next morning the temperature was up to 105.2° F. (40.7° C.), the blood pressure being 134/90, and his condition remained reasonable. A third dose of chlorpromazine, 25 mg. intramuscularly, was followed by a cold sponge, and the temperature fell to 101.8° F. (38.8° C.). The blood pressure dropped to 100 mm. Hg systolic, pulse 136. The temperature soon rose again, and by midday was over 105° F. (40.6° C.), CO₂ concentration being 4.5%. At this point the patient suddenly collapsed, became unconscious, and remained so until he died 36 hours later. Engström (1954) has described the appearance of circulatory symptoms occurring suddenly at high temperatures followed by rapid death. From now on, the axillary temperature readings were taken every four minutes. His lips and mucous membranes were grey; the blood pressure was 70/50, pulse 160, temperature 105.8° F. (41° C.). The oxygen flow was increased and his general condition slowly improved, the ashen-grey appearance disappearing. The temperature rose slowly to 107° F. (41.7° C.), but the blood pressure remained low, 85/50; the pulse rate was 140. Cold sponging had no effect on the temperature. The humidifier had by this time been turned off.

Lassen (1954) reported a mortality rate of 93% in cases of hyperpyrexia and temperatures of over 102° F. (38.9° C.) for five days. It was felt that a further dose of chlorpromazine was justified, and 50 mg. was added to 540 ml. of

glucose and an intravenous drip set up. The temperature fell to 103.8° F. (39.9° C.) when approximately 130 ml. had been run in, but rose again rapidly to 107° F. (41.7° C.) within an hour. The pulse was extremely poor and very rapid—170 to 200—the blood pressure being 60 mm. Hg systolic (by palpation). Covering the patient with chipped ice and allowing a fan to blow over him did not bring down the temperature. He had now been hypotensive for five hours, and we decided to give noradrenaline intravenously. As we had had a poor response with noradrenaline following chlorpromazine previously, we set up a drip of 4 ml. in 500 ml. of 5% glucose, but with little effect. Three pints (1.7 litres) of this solution was eventually run in.

By 7.30 p.m. on October 24 the axillary temperature had risen to 108° F. (42.2° C.) in spite of chipped ice and a very strong breeze from the fan; blood pressure 70 mm. Hg systolic, pulse 128, pupils pin-point. A fourth dose of chlorpromazine, 25 mg. intramuscularly, was given, and within an hour the temperature had fallen to 101° F. (38.3° C.). For the next 16 hours the temperature remained on an average at 100 to 102° F. (37.8 to 38.9° C.), rising to 105° F. (40.6° C.) on one occasion and falling to 98° F. (36.7° C.) for three short periods. During this time the skin, except in the axilla, was cold to the touch, with stagnant cyanosis, but could be made a reasonable colour by rubbing. The capillary refill test was fair. As the noradrenaline had not been effective, methylamphetamine hydrochloride, 20 mg. intravenously, was given at three-hourly to four-hourly intervals, but with no effect. The pulse could not now be felt at the wrist, and the only sign of life, apart from the apex beat, was the screwing up of the face which followed tracheal suction; this reaction to stimulation of the tracheo-bronchial reflexes persisted up to within a few minutes of death.

Towards the evening of October 25 the temperature again started to rise to 105° F. (40.6° C.), and the apex beat could be heard only with difficulty. The pupils became widely dilated and did not react to light towards midnight. An attempt to give an intra-arterial blood transfusion failed, as neither the femoral nor the subclavian artery could be palpated. In a further unsuccessful effort to insert an intra-arterial needle 8 ml. of noradrenaline was added to 540 ml. of 5% glucose and the drip run in fairly quickly for a few minutes. The pulse became palpable, the blood pressure rose to 80/60, and the pupils became half-dilated and reacted to light. The drip was then slowed, but the circulation finally collapsed completely and the patient died within 20 minutes. Necropsy showed the usual changes of poliomyelitis in the cord, medulla, and pons.

Discussion

To date there are reports of five cases of bulbo-spinal poliomyelitis occurring in England (Smith *et al.*, 1954; Macrae *et al.*, 1954), and these three more in Kenya which have been treated with I.P.P.R. Of these eight patients five have died. In the English series one died of the effects of inhalation of vomit (Smith *et al.*, 1954) and two from pulmonary infection (Macrae *et al.*, 1954). In our series the cause of death in Case 2 was unknown, though during the course of his illness he developed the following complications: (a) hyperpyrexia as defined by Lassen—mortality rate 91% (Lassen, 1954); (b) shock—mortality rate 67% (Lassen, 1954); (c) pulmonary oedema—mortality rate 93% (Lassen, 1954). He survived the first two complications, and it would appear from the post-mortem findings that pulmonary oedema was not the direct cause of his death. Case 3 developed hyperpyrexia—axillary temperature, 108° F. (42.2° C.)—and shock, both carrying a high mortality rate as mentioned above.

Shock.—The causes of shock in bulbo-spinal poliomyelitis are: (a) anoxia; (b) CO₂ retention; (c) virus invasion of the brain; (d) interference with venous filling of the heart; (e) central damage due to hyperpyrexia (Christie, 1954; Engström, 1954). Engström points out that high pressures and long inspiratory phases interfere with cardiac output,

and that hyperpyrexia above 105.8° F. (41° C.) is followed by rapid circulatory failure. In Case 2 a fall in systolic pressure of 10 mm. Hg occurred during the inspiratory phase of the machine, indicating interference with the venous return to the heart (Lewis, 1942); this was supported by the electrocardiographic changes. There was no evidence of anoxia or of CO₂ retention, though central damage remained a possibility. It was noted, however, that there was an improvement in the blood pressure and a decrease in the inspiratory fall in pressure on increasing the rate of the machine, so decreasing the time of the inspiratory phase. The pressure recorded on the machine during inspiration remained constant at 16 cm. of water. In Case 3 the shock was presumably due to damage to the vasomotor centre by hyperpyrexia and direct damage by the virus.

Mental State.—All cases showed some degree of mental distress. In Case 1 the patient, who was most co-operative, tended to be very emotional and would burst into tears for no apparent reason. At the beginning of treatment he wanted to pull out the endotracheal tube. He became very intolerant of pain and had marked depressive phases, which were most noticeable about the third week, from which time he became more cheerful. In Case 2 the added difficulty of communication exaggerated the mental symptoms. At all times the patient was restless and agitated, and tended to fight the machine with his remaining respiratory muscles. He slept poorly, and at night, especially latterly, he became very irrational and was always pulling out the Levine tube and pulling off the bedclothes. After the second week he became very depressed and wanted to pull out the endotracheal tube. Case 3 tended to be restless and irrational at night. In none of these cases were the mental changes due to the accumulation of CO₂ or to anoxia. In Case 1 reassurance helped considerably; this has been stressed by Daley and Miller (1952).

Pulmonary Oedema.—This appeared rapidly in Case 2 on three occasions. We are unable to suggest the exact mechanism. Anoxia and CO₂ accumulation did not appear to be relevant. This patient was, however, exceedingly apprehensive and agitated, and had been breathing on his own for considerable periods. The serum proteins were low (total 5.3 g.%), but the oedema persisted even when the total proteins had risen to 6.5 g.%. Treatment with atropine, 0.9 mg. twice daily, aminophylline, 0.5 g. twice daily intramuscularly, oxygen, bronchial suction, and anaesthetizing the bronchi with cocaine were disappointing. The pulmonary oedema on two occasions disappeared suddenly; once for no apparent reason and once 20 minutes after 0.9 mg. of atropine had been given.

Chlorpromazine.—This was tried on two of these cases because of its supposed pharmacological actions of (a) protection against shock (Laborit and Huguenard, 1953); (b) potentiation of sedatives (restlessness was a prominent feature of these cases); (c) reduction of temperature and of the oxygen requirements (Laborit and Huguenard, 1951); and (d) depression of respiratory reflexes. Its use in asthma has been reported by Robinson and Zuck (1954). Bronchial suction was frequent, and it was thought that this action would be particularly useful in view of the occurrence of widespread rhonchi in all cases. Chlorpromazine did not seem to have any appreciable effect on the temperature unless ice and a breeze from a fan were used in addition. These two measures in conjunction did bring down the temperature, but the reduction was inconsistent and no definite conclusions could be drawn. Shackman *et al.* (1954), using promethazine as part of a "lytic cocktail," have shown that unless cooling measures are added no fall in temperature occurs in non-pyrexial patients under anaesthesia. The anti-noradrenaline action of this drug was shown, as has been described by Foster *et al.* (1954). In the two cases in which noradrenaline was used to combat the hypotension, large doses were needed and in higher concentrations than are generally recommended. Higher concentrations were used, as we feared that the circulation might be overloaded by larger amounts of 5% glucose

Transport.—We experienced no particular problems in the transport of these cases by air, using the Radcliffe respiration pump. A sucker of some kind is essential. Oxygen cylinders were carried, but were not needed in pressurized planes. The team consisted of a physician, a hospital superintendent, and a nursing sister.

Staff.—It was soon realized that a specially trained team of doctors and nurses was essential. Ideally, a doctor should be in attendance throughout the day. In particular we would emphasize that the nursing is arduous and that, as this is a new technique, every detail must be carefully explained to every member of the team. The members should not be changed if this can possibly be avoided. It is unfair to expect a nurse, new to this type of work, to be able to learn the details "as she goes along." An error of judgment may be fatal, and a call for help must be answered promptly.

It is obvious that this treatment can be carried out only in properly equipped centres, and no one should be tempted to undertake these measures unless the following minimum personnel are readily available: a physician, an anaesthetist, and nine nursing staff. Even with these requirements medical personnel must be prepared for a day's work of 16 hours over long periods.

Conclusions

- (1) Even with the advance of I.P.P. respiration in the treatment of bulbo-spinal poliomyelitis the mortality remains high when complications enter the picture.
- (2) It is important to have a specially trained team of nurses and doctors, and constant supervision for 24 hours a day.
- (3) It is necessary for an anaesthetist to be a member of the team used for the transport of acute cases.
- (4) General anaesthesia is advisable for tracheotomy.
- (5) We would emphasize the difficulty of treating the various complications—shock, hyperpyrexia, and pulmonary oedema—and of ascertaining the exact aetiological factors involved.

Summary

The treatment of three cases of bulbo-spinal poliomyelitis with the Radcliffe respiration pump is described. All cases showed inadequate respiration combined with inability to swallow, ineffective cough, and pooling of secretions.

Experience of these Kenya cases has shown that even acutely ill patients may be flown safely over long distances with this apparatus.

The importance of maintaining a clear airway in the lower respiratory tract coupled with the inflation of air at the correct pressure, humidification, and temperature, and in adequate volume, is stressed. A simple bedside method of measuring the CO₂ content of expired air is suggested, coupled with pH estimations of the urine to detect hyperventilation.

The different features of each of the three cases emphasized the difficulties in the management of this condition. An adequately trained team of doctors and nurses is essential. These patients cannot be left unattended at any time, as critical emergencies may occur without warning.

"Shock," hyperpyrexia, and pulmonary oedema occurred in the two fatal cases. Chlorpromazine and noradrenaline were used to combat "shock" and hyperpyrexia.

Abnormalities of the mental state were prominent in all cases but responded well to reassurance when this was possible.

We wish to thank the matron and the nursing staff of King George VI Hospital and the Infectious Diseases Hospital, Nairobi,

for the long hours of arduous duty given willingly and without complaint. We also acknowledge the invaluable help given to us by Mr. F. Owen, hospital superintendent, the Infectious Diseases Hospital, Nairobi, and Mr. Austen, Government Electrical Engineer. We are indebted to the United States Air Force for their prompt answer to a call for assistance. A cuirass respirator and a technician were flown out to us from Tripoli. We have to thank the Director of Medical Services, Kenya Colony, for his help and permission to publish this article. The Radcliffe pumps were manufactured by H. G. East and Co., Longworth Road, Oxford.

REFERENCES

- Andersen, E. W., and Ibsen, B. (1954). *British Medical Journal*, 1, 786.
 Astrup, P., Gotzsche, H., and Neukirch, F. (1954). *Ibid.*, 1, 780.
 Case, E. M., and Haldane, J. B. S. (1941). *J. Hyg. (Lond.)*, 41, 225.
 Christie, A. B. (1954). *British Medical Journal*, 2, 663.
 Courmand, A., Motley, H. L., Werko, L., and Richards, D. W. (1948). *Amer. J. Physiol.*, 152, 162.
 Daley, R., and Miller, H. G. (1952). *Progress in Clinical Medicine*, 2nd ed., p. 280. London.
 Engström, C. G. (1954). *British Medical Journal*, 2, 666.
 Foster, C. A., O'Mullane, E. J., Gaskell, P., and Churchill-Davidson, H. C. (1954). *Lancet*, 2, 614.
 Ibsen, B. (1954). *Proc. roy. Soc. Med.*, 47, 72.
 Laborit, H., and Huguenard, P. (1951). *Presse méd.*, 59, 1329.
 — (1953). *Ibid.*, 61, 1029.
 Lassen, H. C. A. (1953). *Lancet*, 1, 37.
 — (1954). *Proc. roy. Soc. Med.*, 47, 70.
 Lewis, T. (1942). *Diseases of the Heart*, 3rd ed., p. 180. London.
 Macrae, J., McKendrick, G. D. W., Sefton, E. M., and Walley, R. V. (1954). *Lancet*, 2, 21.
 Marshall, J., and Spalding, J. M. K. (1953). *Ibid.*, 2, 1022.
 Motley, H. J., Courmand, A., Werko, L., Dredale, D. T., Himmelstein, A., and Richards, D. W. (1948). *J. Amer. med. Ass.*, 137, 370.
 Robinson, K. C., and Zuck, D. (1954). *Lancet*, 1, 1349.
 Russell, W. R., and Schuster, E. (1953). *Ibid.*, 2, 707.
 Shackman, R., Wood-Smith, F. G., Grabed, I. G., Melrose, D. G., and Lynn, R. B. (1954). *Ibid.*, 2, 617.
 Smith, A. C., Spalding, J. M. K., and Russell, W. R. (1954). *Ibid.*, 1, 939.
 Wright, S. (1945). *Applied Physiology*, 8th ed., p. 585. London.

CLINICAL USES OF INTRAVENOUS HYDROCORTISONE

BY

F. DUDLEY HART, M.D., F.R.C.P.

Assistant Physician, Westminster Hospital

Cortisone therapy is now an important part of day-to-day therapeutics. Whatever the argument may be for its use in rheumatoid arthritis, ulcerative colitis, and asthma, there is little difference of opinion when it comes to adrenal insufficiency; for in Addison's disease, Simmonds's disease, and in the adrenalectomized subject it is the most useful drug now available. In acute adrenal insufficiency, however, oral cortisone may not be enough; though the drug is rapidly absorbed from the intestinal tract when taken by mouth, in adrenal crisis a vomiting and dehydrated patient may need rapidly effective parenteral therapy. It is to meet this need that intravenous hydrocortisone has been produced, for the ordinary issue of cortisone or hydrocortisone acetate for oral, intramuscular, or intra-articular use cannot be given intravenously.

The use of intravenous hydrocortisone may be considered in the following conditions:

1. Addisonian crisis.—The dehydrated, vomiting, critically ill patient with Addison's disease may be unable to tolerate oral cortisone, and intramuscular injections may be too slow in action to help him. An intravenous drip of 10–20 mg. hydrocortisone per hour for some six to eight hours will usually effect marked improvement and allow the institution of oral or intramuscular cortisone therapy. This dosage can be increased, if response is unsatisfactory, to 30–40 mg. per hour or even higher.

2. The adrenalectomized patient, usually in the face of infection or some other form of stress, may become ill, weak,

and hypotensive. Such patients may not show the usual signs of adrenal insufficiency, but appear merely to go downhill rapidly and alarmingly. Nausea is frequent and vomiting commonly occurs. The use of intravenous hydrocortisone may be life-saving, and if in doubt the general rule should be to treat as for a probable adrenal failure.

3. The cortisone-treated patient with intact adrenal glands, whether he be a sufferer from rheumatoid arthritis, asthma, or any other condition, may, in the face of infection or any other form of acute stress, experience symptoms of partial adrenal insufficiency. One common form of stress is major operative surgery. A surgical operation may precipitate adrenal failure in a gland suppressed by weeks or months of cortisone therapy, and, if there is no time for 24–48 hours' full pre-operative cortisone treatment, intramuscular cortisone may be augmented by intravenous hydrocortisone. It is well recognized that after stopping cortisone therapy, if such treatment has lasted more than a few days, the adrenal glands may be unable to react normally to operative stress for some weeks or months subsequently. In such patients it is wise to "cover" the operation two days before and a week afterwards by full cortisone therapy. Intravenous hydrocortisone may in these instances also play a part if such "cortisone cover" has been neglected or the dosage insufficient.

4. The Waterhouse-Friderichsen syndrome.

5. Simmonds's disease.—In the face of stress these patients may develop signs of acute adrenal insufficiency. Allott and Simmons (1951) report a case of Sheehan's syndrome which responded only to cortisone. In such cases intravenous hydrocortisone should be considered. Such cases are rare, however, compared with the hypoglycaemic episodes which are common in this condition and which respond usually satisfactorily to intravenous glucose.

6. The above list of indications is not to be regarded as exclusive. Black and Harken (1954), for instance, discussing "the safe conduct of a patient through cardiac surgery," state that in a few cases intractable hypotension after operation appeared to be due to adrenal failure. The patient, alert and orientated despite the absence of a detectable blood pressure, responded to intravenous hydrocortisone though not to intramuscular cortisone or corticotrophin (A.C.T.H.). In severe status asthmaticus, acute disseminated lupus erythematosus, or overwhelming allergic reactions, the use of intravenous hydrocortisone could legitimately be considered.

The following cases are instructive.

Case 1

The patient was a woman aged 47. Bilateral total adrenalectomy was performed by Sir Stanford Cade for metastatic malignant disease secondary to carcinoma of the breast. The immediate post-operative progress was uneventful, no noradrenaline infusion being required. On the seventh post-operative day her condition deteriorated rapidly, swelling and induration became apparent at the medial end of the left incision, and her temperature rose to 104° F. (40° C.), her pulse to 150 a minute. She became drowsy, almost comatose, and her blood pressure unrecordable. As her condition was critical, intravenous hydrocortisone was started that night and 100 mg. was given over a period of six hours. After some two hours her blood pressure rose and her general condition improved dramatically; by morning her temperature had dropped to 99° F. (37.2° C.) and her pulse to 110. Oral cortisone, 25 mg. six-hourly, was given for the next six days and then gradually reduced to the usual maintenance dose of 50 mg. a day. There were no marked changes in serum electrolytes, sodium and chlorides being normal or just below normal throughout.

Comment.—In this case a wound infection rapidly precipitated a degree of adrenal insufficiency which seemed