

This was not possible, however, for we soon realized we could distinguish the x-ray films of most of the whites from those of the Africans by the different incidence of aortic calcification and the rather characteristic shape of the lower lumbar vertebrae of many of the Africans.

We wish to thank the Medical Research Council for their support and for a grant to one of us (R. C. G.). Thanks are due to many members of the staff of King Edward VIII Hospital, Durban, for making this project possible. Professor E. B. Adams put many facilities at our disposal, including the medical social worker, Miss Ngwane, to whom we are especially grateful. The high quality of the x-ray films was entirely due to Professor B. Sachs and the x-ray department of the Addington and King Edward VII Hospitals, Durban. The extra work we imposed on them was borne in the normal course of their workings. We thank the consultants

of these hospitals for letting us use cases under their care, and also Miss Y. Brown, director of the Lamontville Old People's Home, Durban.

REFERENCES

- Bernstein, D. S., Sadowsky, N., Hegsted, D. M., Guri, C. D., and Stare, F. J. (1966). *J. Amer. med. Ass.*, **198**, 499.
 Nordin, B. E. C. (1966). *Clin. Orthop.* No. 45, p. 17.
 Cooke, A. M. (1955). *Lancet*, **1**, 929.
 Davidson, S., and Passmore, R. (1966). *Human Nutrition and Dietetics*, 3rd ed., p. 400, Edinburgh.
 Dent, C. E., and Watson, L. (1966). *Postgrad. med. J.*, October Suppl.
 Jessorer, H. (1963). In *Osteoporose*, edited by E. Blaschker, Berlin.
 Jonck, L. M. (1964). *S. Afr. J. Radiol.*, **2**, 25.
 Nordin, B. E. C., Barnett, E., MacGregor, J., and Nisbet, J. (1962). *Brit. med. J.*, **1**, 1793.
 Rose, G. A. (1964). *Clin. Radiol.*, **15**, 75.
 Rose, G. A. (1967). *Sci. Basis Med.*, p. 252.
 Schraer, H. (1958). *J. Pediat.*, **52**, 416.
 Seftel, H. C., et al. (1966). *Brit. med. J.*, **1**, 642.

Prognosis in Tetraplegia

J. R. SILVER, M.B., B.S., M.R.C.P.ED.; N. O. K. GIBBON, M.CH., F.R.C.S.

Brit. med. J., 1968, **4**, 79-83

Summary: A total of 141 cases of traumatic tetraplegia were admitted to the Liverpool Paraplegic Centre between 1947 and 1967. Most of the deaths occurred within three months of injury, and comparison with other centres suggests that the early mortality could be reduced by more use of mechanical respirators.

Urological complaints and pressure sores are hazards that can be overcome by careful attention to nursing procedures. Later deaths are more common among patients transferred from the unit to hostels or hospitals than among patients transferred home. Specialist units for these patients improve the quality of their lives as well as their expectation of life. Electronic equipment can also play a large part in helping tetraplegics to play a part in community life.

Introduction

A total of 626 patients were admitted to the Liverpool Regional Paraplegic Centre between its opening in January 1947 and May 1967. Among these were 196 tetraplegics (179 males and 17 females), their ages ranging from 11 to 75 years. The small number of female patients in this series is partly because there has been a four-bedded female ward only since 1961, but it is also because women are less liable to traumatic tetraplegia, as they do not indulge to the same extent as men in the dangerous activities of motor-cycling and outdoor construction work, nor temperamentally are they so prone to reckless driving and diving.

The total numbers of tetraplegics admitted to the centre compared with the total admissions year by year are set out in Fig. 1, which shows that there has been a progressive increase in the total number of patients admitted. Previous to 1953 very few tetraplegics were admitted, but since 1954 the proportion has averaged about 33% of the total admissions, without any obvious trend in either direction. In common with other centres (Walsh, 1967), tetraplegics absorb a relatively high proportion of the resources of the centre, and we have therefore turned our attention recently to the special problems affecting patients with cervical involvement.

The cause of the tetraplegia was trauma in 141 cases and multiple sclerosis in 45. There were 10 miscellaneous medical conditions, such as angioma of the cord, extradural abscess, and transverse myelitis. The prognosis and management of

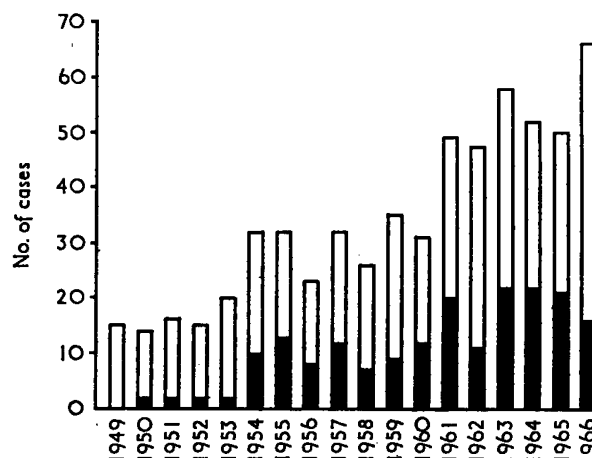


FIG. 1.—Total tetraplegics admitted year by year compared with total admissions. Yearly average is 30%.

multiple sclerosis are dealt with elsewhere (MacAlpine et al., 1955, 1965); this paper is devoted to a consideration of the prognosis in the 141 cases of traumatic tetraplegia.

Breithaupt et al. (1961) have shown that the prognosis differs between complete and incomplete tetraplegia, and so we have taken this into account in presenting our statistics (Table I).

The majority of the patients had been seen for routine follow-up investigations, intravenous pyelography, residual urine check, blood urea, etc., during the last year, and in those who had not (49 patients) a personal follow-up letter was dispatched both to the patient's general practitioner and to the patient. Further information was received on 44 patients, a most gratifying response, so that up-to-date information was available on 135 patients, only six patients being lost to follow-up.

TABLE I

| Group | No. of Patients | Extension Injuries | Age in Years | | Early Deaths up to 3 Months | Average Survival after Injury (days) | Patients Lost to Follow-up | Late Deaths | Time Range (years) | Average (years) | Survivors | Years of Survival | | Total Death Rate |
|------------|-----------------|--------------------|--------------|---------|-----------------------------|--------------------------------------|----------------------------|-------------|--------------------|-----------------|-----------|-------------------|---------|------------------|
| | | | Range | Average | | | | | | | | Range | Average | |
| Complete | 45 | 10 | 15-69 | 35 | 17 (38%) | 17 | 1 | 10 (37%) | 3/12-13 | 3½ | 17 | 1-14 | 6 | 27/44 (62%) |
| Incomplete | 96 | 51 | 11-75 | 45 | 9 (9%) | 15 | 5 | 20 (24%) | 3/12-13 | 4½ | 62 | 1-23 | 6½ | 29/91 (32%) |
| Total .. | 141 | 61 | 11-75 | 40 | 26 (18%) | 16 | 6 | 30 (27%) | 3/12-13 | 4 | 79 | 1-23 | 6½ | 56/135 (41%) |

Complete Tetraplegia

Tribe (1963-4) and Tribe and Silver (1968) have shown that the cause of death in tetraplegia differs very markedly between those who die within the first three months of injury and those who die at a later date. Accordingly this classification has been followed. There were 45 complete traumatic tetraplegics, their ages ranging from 15 to 69 years, with an average of 35 years. The majority were caused by fractures or fracture-dislocation of the cervical spine; only 10 were caused by extension injuries.

The initial prognosis in complete tetraplegia is particularly poor. Seventeen died within three months, in an average of 17 days after injury. The majority died of chest complications (13 of respiratory failure and one of pneumonia), one of pulmonary embolism, and two of pressure sores. Only one patient who was transferred to another hospital and has subsequently returned to Turkey has been lost to follow-up. A further 10 patients died between three months and seven years. Three died of pneumonia, three of renal infection, one of pulmonary embolism, one of suicide, one of pressure sores, and one of causes unknown.

The cases of pneumonia are closely related to the acute group, since one died at five months and one at six months, during their initial treatment. One patient died at 18 months, four days after his discharge to another hospital. The average duration of life after injury in this group was three and a half years. There were 17 survivors living between 1 and 14 years, with an average survival rate of six years. As one patient was lost to follow-up he has been discarded from the statistics in the long-term group. The overall mortality in this group of complete tetraplegics is 62%.

Incomplete Tetraplegia

There were 96 incomplete traumatic tetraplegics, their ages ranging between 11 and 75 years, with an average of 45. This higher average age was due to the larger number of extension injuries, 51 in this group. This can be attributed to the fact that the elderly patient is particularly liable to degenerative changes of the cervical spine. The remainder were due to fracture or fracture-dislocation. Despite the older average age the initial prognosis in the incomplete lesions was much better; there were only nine deaths within three months of injury. The time (average 15 days) and cause of death are similar to the complete group. In eight of the nine cases the cause of death was respiratory failure. Only five patients have been lost to follow-up, and they too have been omitted from the statistics in the long-term group of patients. A further 20 patients died between three months and 13 years, with an average survival of four years and three months.

The causes of death are similar to those of the late deaths in the complete group, there being two cases of pneumonia, nine of pressure sores, and three of uraemia. There are many more survivors (62) than in the complete group, the average duration being six years and four months. The overall prognosis taking into account the acute and late deaths is much better in this group than in the complete ones.

Causes of Death

Respiratory Failure

There were 21 deaths from respiratory failure among these patients, all within three months of injury: 13 were in complete and eight were in incomplete lesions (Table II). They presented a remarkably uniform picture, dying on an average 16 days after injury. The patients had usually a high cervical injury at the C5 or C6 level, and they were confused or apprehensive, breathless, and cyanosed. They had tachycardia in contrast to the bradycardia normally found in tetraplegic patients. Their extremities were warm. Frequently they lost consciousness and had an epileptic fit following a change in position. Examination of the chest showed multiple scattered crepitations and only a little frothy non-purulent sputum could be obtained. They died choking and coughing. In the majority of cases the neurological lesion had not ascended, though in a small number the lesion might ascend a segment or half a segment. At necropsy there was remarkably little to find. The lungs were uniformly red and oedematous, the larger bronchi were patent, and histologically there was a little inflammatory reaction.

TABLE II.—Causes of Early Death

| Cause: | Respiratory Failure | Pulmonary Embolism | Pneumonia | Sores |
|---------------|---------------------|--------------------|-----------|-------|
| Complete .. | 13 | 1 | 1 | 2 |
| Incomplete .. | 8 | 1 | — | — |
| Total .. | 21 | 2 | 1 | 2 |

The mechanism of death has been discussed elsewhere (Silver, 1967-8). In the patient with the complete cord transection at the C5 level important respiratory muscles, the intercostals and the abdominals, are paralysed. Ventilation is entirely supported by the diaphragm; this too may be partially paralysed, and the vital capacity may be as little as 200 ml. This is very close to the patient's anatomical dead space; any further reduction in the efficiency of the lungs by a head injury, an aspiration pneumonia, the injudicious use of drugs causing respiratory depression, or overtransfusion will precipitate the patient into respiratory failure.

Pneumonia

There were six deaths from pneumonia in this series (Table II and III). The distinction between this group and those who died of respiratory failure is rather arbitrary and

TABLE III.—Causes of Late Death

| Cause: | Unknown | Related | | | | | Unrelated | | |
|---------------|---------|-----------|---------|----------------------------|-------|--------------------|-----------|---------|----------------------------|
| | | Pneumonia | Uraemia | Uraemia and Pressure Sores | Sores | Pulmonary Embolism | Suicide | Cardiac | Tuberculosis and Carcinoma |
| Complete .. | 1 | 3 | 2 | 1 | 1 | 1 | — | — | |
| Incomplete .. | 1 | 2 | 3 | — | 9 | — | 4 | 1 | |
| Total .. | 2 | 5 | 5 | 1 | 10 | 1 | 4 | 1 | |

there is obviously some overlap. The distinction was largely made on pathological grounds. Previous to injury the patients frequently had a history of chronic bronchitis with a productive cough. The paralysis of the expiratory muscles made it impossible for them to void their sputum, and at necropsy the large bronchi were filled with purulent sputum and histologically there were destructive changes in the lung. They died much later than those considered in the previous section.

The rapidity with which such a pneumonia can develop is reflected by the fact that two patients died of pneumonia within four days of discharge, one to another hospital and one to his home.

The acute deaths which are largely attributable to respiratory failure are set out in Fig. 2. It is salutary that, despite the instigation of regular postural drainage, prophylactic antibiotics, and inhalations, in all the cervical injuries since July 1965 there has been no obvious reduction in the mortality in this group (see Fig. 2), and the results compare unfavourably with other series of tetraplegic patients (Cheshire and Coats, 1966-7). Only since the introduction of positive-pressure respiration in these high injuries in 1968 has some change in the prognosis appeared.

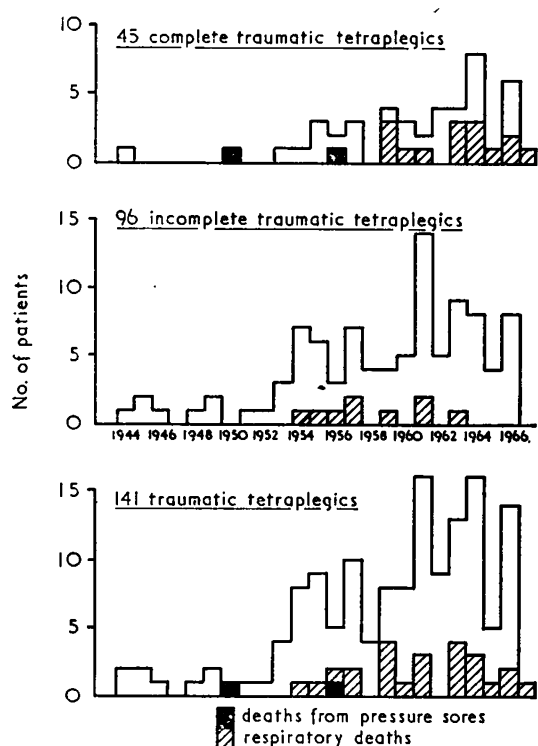


FIG. 2.—Early deaths from respiratory failure compared with deaths from pressure sores in tetraplegic patients.

Pressure Sores

Twelve patients died of pressure sores: two within three months of injury and a further four while undergoing their initial treatment during the first year in the spinal centre. All these deaths occurred before 1965, when the staffing of the centre was not at its present level and less was understood about the treatment of tetraplegic patients. Since July 1965, with the extra nursing staff, a full-time physiotherapist, a registrar, a consultant, and the provision of additional nursing aids for the positioning and turning of patients, there have been no pressure sores among the tetraplegic patients. Any tetraplegic patient admitted with a pressure sore has had it healed, and consequently there have been no deaths from this cause during the past three years.

There were many more late deaths from sores among the incomplete tetraplegics than among the complete ones. This is partly a reflection on the better initial prognosis of these patients, as there were more of them surviving the initially dangerous few weeks after injury to develop sores, but this group is much more liable than the complete ones to develop spasms and contractures, since their long descending tracts which mediate spasticity from the brain stem are intact. This makes them very difficult to nurse and so prevent a sore. Once a sore has occurred, especially around the hip, it is difficult to heal up, since the shearing stresses of the spasms tend to undermine the skin. Attempts to relieve the spasticity by subarachnoid alcohol block did not always prevent the occurrence of further sores.

Since all pressure sores are preventable (Munro, 1943; *Lancet*, 1967), the occurrence of a sore is a direct index of the quality and quantity of nursing care available to the patient. It is most interesting in this context to study the fate of the tetraplegic patients once they had reached a definitive stage of treatment. The fate of the complete tetraplegic and the severely paralysed incomplete case depended only to a limited degree on the physical extent of their paralysis and was much more dependent on social factors. When there was a devoted relative to look after him the patient could be discharged home. Those who were not in this fortunate position either stayed in the centre or were discharged to a long-term hostel or hospital. Of the 29 patients discharged to another hospital or hostel 13 are dead; of the 79 discharged home only 10 are dead.

There are several reasons for the poor prognosis of patients discharged to long-term accommodation. When a patient is admitted to such accommodation it is a failure of rehabilitation and patients often give up their wish to live. This is not unique to tetraplegic patients; other disabled people admitted to long-term accommodation have a high initial mortality. A further significant factor is that if a patient is discharged home there is at least one full-time person to look after him, and what these relatives lack in skill they make up for in affection. When a patient is admitted to long-term accommodation the average nursing care that is available is only three-quarters of an hour a day, and this is not sufficient to keep a tetraplegic in good health (Fine, 1967).

Urological Complications

Tetraplegics run the usual risks associated with the neurogenic bladder. They are also at a special disadvantage in several respects, especially when the lesion is complete.

(1) They are relatively immobile, and this may encourage urinary stasis and prevent ready access to the W.C. or commode, a point of particular importance in the female.

(2) There is total sensory denervation of the bladder, which may become grossly overdistended without the patient's knowledge, apart from occasional manifestations of autonomic hyperreflexia.

(3) The tetraplegic cannot exert effective manual or abdominal pressure on the bladder. Any procedure which interferes with reflex bladder contractions is therefore contraindicated. This is emphasized by the results of subarachnoid alcohol block carried out for skeletal spasticity or autonomic hyperreflexia in 10 of our tetraplegics—three complete and seven incomplete. In no case was retention relieved by the injection. We have previously drawn attention to the frequent failure of subarachnoid alcohol block to relieve obstruction at the level of the external sphincter (Ross *et al.*, 1967). Furthermore, in only two cases out of the 10, both incomplete, could bladder emptying subsequently be achieved as a result of multiple operations designed to reduce urethral resistance.

(4) There is difficulty in handling appliances, and this adds to the problem of urinary incontinence and prevents self-catheterization.

These special features make the management of the bladder even more exacting in tetraplegia than is the case with lower lesions. Fortunately, in many cases the cord damage is incomplete and the vesical involvement slight or temporary. Our 141 traumatic tetraplegics included 96 with incomplete lesions. Nine of the latter retained normal micturition while 25 regained normal or almost normal control after a period of retention. Thus in over a third of these incomplete cases no permanent urological attention was required.

The tetraplegic who is admitted to a special centre is not specially prone to lower urinary tract complications; in any case, these have been virtually eliminated in the field of paraplegia by modern techniques of intermittent and continuous drainage. Renal complications, however, remain a serious danger in spite of recent advances in prevention and treatment. Uraemia is usually due to a combination of infection and dilatation of the upper urinary tracts, and these changes may arise as a result of a series of acute attacks or develop insidiously.

Acute pyelonephritis (the "flare-up") is due to vesico-ureteric reflux of infected urine. This occurs during washouts, cystometry, or cystography if care is not taken to avoid rapid or excessive filling, but the commonest cause is certainly a blocked catheter or an acute retention. The majority of tetraplegics have lost all sensation of vesical overdistension, and the first sign of trouble is likely to be a rigor, or headache and sweating due to autonomic hyperreflexia. We have observed stiffening of our fine plastic drainage tubes to be an early sign of cessation of urinary flow. The temperature drops by about 5° C. within a quarter of an hour of a blockage, and this should be ample to allow operation of a monitoring system, with which we are now experimenting.

In chronic renal failure, infection and dilatation of the upper tracts may still be aggravated by calculus formation, though this complication is now rare in special centres (Damanski, 1963-4). Occasionally the patient presents the picture of a nephrotic syndrome due to renal amyloidosis, and in our experience a negative rectal biopsy does not exclude this condition. Regular follow-up examination, including intravenous pyelography, is the only safeguard against insidious development of renal failure. Dilatation and irregularity of the lower ureters should be taken as early signs of inadequate bladder function with persistent infection. In our view an acceptable residual urine is one which is compatible with a sterile specimen and normal pyeloureterograms, and we pay little attention to absolute figures or percentages.

Among the 45 complete tetraplegics there have been two predominately urological deaths. One occurred at two years in a chronic hospital despite normal pyelograms three months previously; the other at 13 years, also in a chronic institution. When the latter was followed up after a delay of five years he had advanced hydronephrosis. In a third patient, who died with extensive sores, urinary infection seems to have been contributory, though he was known to have had normal pyelograms seven months previously.

An indication of the urological condition of the survivors is given by the normal pyelograms found in all 12 patients checked up in the year ending May 1967. Two of these were on drainage, one after a subarachnoid alcohol block which left him with a straining pressure of only 20 cm. of water. Rhizotomy, sparing the sacral segments, seems preferable to alcohol injection in these complete cases. The other patient had a low residual urine but preferred a catheter for convenience.

This satisfactory state of affairs was not achieved without a good deal of surgical intervention. If we exclude the early deaths, 58% of the complete tetraplegics have had operations

on the bladder-neck, the external sphincter, or both, presumably due partly to low voiding pressures.

Among our 96 cases of incomplete tetraplegia only two are known to have died of renal insufficiency—one at four years and one at nine years. The latter patient succumbed despite a suprapubic cystostomy done two years previously for progressive hydronephrosis with reflux. Urinary infection may also have contributed to the death of a third patient who had congenital absence of one kidney.

The urological condition of the surviving incomplete tetraplegics may be indicated by the findings in 27 of them who were re-examined in the year ending May 1967. These patients had survived two months to 25 years (average four years) and 24 had undergone regular pyelography. No major radiological changes were found in any of these patients and minor changes in only four of them. Only four were on continuous drainage. If we exclude the early deaths the rate of operation on the bladder outlet in the incomplete tetraplegics was 21%, or only one-third of that applying to the complete cases.

The frequency of surgical intervention in our series is related to our policy of early elimination of the catheter. We often operate within three months for persistent retention, and only 9% of our tetraplegics have finished up on drainage. Even this figure is twice our overall rate for paraplegia (Damanski, 1965). One effect of this active policy has been to reduce the average stay of all our paraplegics to just over four months. Reports from other centres (Comarr, 1958; Harding, 1961) indicate relatively long periods of catheterization, followed by a final drainage rate in all paraplegics of 30%. While much is to be said for early elimination of the catheter, we admit freely that good drainage is preferable to poor bladder function, and also that operation might have been avoided in some of our cases by a more prolonged period of conservative management.

Discussion

Our overall uncorrected mortality rate for tetraplegia (39.7%) is much higher than that of Nyquist and Bors (1967-8) (12.4%). As they have pointed out, very few patients were admitted to their unit within two months of injury. The time of admission of our own traumatic cases is shown in Table IV: 79% of the patients arrived within three months of injury and 60% within three weeks. We know of several cases in which death has occurred within a few days, while arrangements were being made for the transfer of the patient, and there is no doubt that prompt admission to a paraplegic centre transfers the early mortality to that institution. For this reason it is impossible to compare meaningfully the mortality rates in different centres without knowing the percentage and mode of selection of all the tetraplegic patients admitted and the time of admission in the respective areas. The only available figures that we have found are those of Guttman and Frankel (1966-7) and of Cheshire and Coats (1966-7).

TABLE IV.—Time of Admission After Injury of Tetraplegic Patients

| | Time after Injury | | | | | | | | | Total |
|---------------|-------------------|----|---|---|----|-------|----|---|---|-------|
| | Weeks | | | | | Years | | | | |
| | 1 | 2 | 3 | 4 | 12 | 26 | 1 | 3 | 5 | |
| Complete .. | 27 | 5 | 1 | 1 | 6 | 0 | 3 | 1 | 1 | 45 |
| Incomplete .. | 35 | 10 | 6 | 4 | 15 | 7 | 8 | 3 | 8 | 96 |
| Total .. | 62 | 15 | 7 | 5 | 21 | 7 | 11 | 4 | 9 | 141 |

Of 608 traumatic patients admitted within 14 days to Stoke Mandeville Hospital 62.5% of whom were transferred within 48 hours, there were 229 cervical patients and 27 of these

were dead within three months, giving an early mortality rate of 11.8% compared with our own figures of 78 tetraplegics admitted within 14 days with a loss of 23 patients, a mortality rate of 29.5%. Inspection of the causes of death in our early cases shows that they were nearly all due to respiratory failure—21 out of 26; it is significant that at Stoke Mandeville respirators have been used in the management of these cases for the past five years and an intensive care unit is being constructed in the middle of their centre. Cheshire and Coats reported a fall in respiratory mortality among their complete tetraplegic patients from 50% in 1959 to 12½% in 1964 with the institution of full respiratory care. It is only since 1968 that a patient has been placed on a respirator at this centre, and there is clearly a need for improved intensive care facilities for our patients.

Our late deaths have been due not to lack of effective therapy but to poor liaison with the general practitioners and chronic hospitals leading to a failure of follow-up or a lack of emergency readmission. As many of these patients are very unhappy when transferred to a chronic institution there is much to be said for provision of special hostels with easy access to specialized medical and nursing care. One such hostel has already been built associated with Stoke Mandeville Hospital.

The death rate from urinary tract complications in our centre has been kept down only by an unremitting follow-up by a staff with a strong urological bias. Comparable figures from other centres are difficult to trace, but Nyquist and Bors (1967-8) have set 15 urological deaths against 492 surviving tetraplegics to give a "renal mortality" of 3%. On that basis our minimum renal death rate would be 4.8%. Nyquist and Bors have also found deaths from renal causes to be 21% of the total tetraplegic mortality. Our own low figure of 7% is affected by the high respiratory death rate among our earlier admissions. If we exclude admissions within three months of the accident, our renal deaths are still only 13.3% of the total. This means that our total late death rate must be higher than that of Nyquist and Bors.

Our policy has been to eliminate the catheter at as early a stage as possible, and only 9% of all our tetraplegics have finished up with permanent drainage. This figure is about twice our rate for all paraplegics, and reflects the greater difficulty experienced in achieving bladder emptying in the higher lesions. There might appear to be a place for electrical stimulation of the bladder in this group of patients with poor detrusor contractions and we have seriously considered this possibility. Only one patient has so far seemed suitable for such an operation, and he preferred to retain his catheter.

It can be seen that initially there is a high mortality in cervical cord injuries, particularly if they are complete.

Should the patient survive this dangerous period his expectation of life is still less than normal. However, if the patient can be returned home or to a suitable hostel and is not just abandoned to an unsuitable bed in the nearest chronic hospital, and efforts are made to rehabilitate him by all modern methods, including electronic devices, and provided that beds exist at a paraplegic centre to which the patient can be admitted as soon as he develops the smallest complication, there is no reason why he should not lead an extremely happy, healthy, and useful life.

For cases with severe hand involvement electronic devices have been introduced which utilize residual movement of a finger, or mouth movements to actuate systems controlling light switches, telephone, wheelchair, radio and T.V. sets, typewriter, etc.

This equipment is not just an expensive toy. One of our severely disabled complete tetraplegics, using his Possum typewriter, has circularized all users of similar equipment—68 people—throughout the world and found that there are 11 patients in employment—four running their own business, one sales executive, one clerk, two lawyers, one office worker, one copy typist at home, and one mouth painter—seven of whom could not have worked without electronic aid (Beeston and Durr, 1968). This has been substantiated by the large number of tetraplegics who are participating actively in community life by working full time and taking part in sport.

One of us (J. R. S.) would like to thank the National Fund for Research into Poliomyelitis and other Crippling Diseases for a grant, with which part of this work was carried out.

REFERENCES

- Beeston, E. M., and Durr, J. (1968). Personal communication.
Breithaupt, D. J., Jousse, A. T., and Wynn-Jones, M. (1961). *Canad. med. Ass. J.*, 85, 73.
Cheshire, D. J. E., and Coats, D. A. (1966-7). *Paraplegia*, 4, 1.
Comarr, A. E. (1958). In *Proceedings of Seventh Annual Paraplegia Conference*, p. 173. Memphis, Tennessee.
Damanski, M. (1963-4). *Paraplegia*, 1, 149.
Damanski, M. (1965). *J. Urol.*, 93, 466.
Fine, W. (1967). *Daily Telegraph*, 20 July, p. 16.
Guttmann, L., and Frankel, H. (1966-7). *Paraplegia*, 4, 63.
Harding, R. L. (1961). *Plast. reconstr. Surg.*, 27, 235.
Lancet, 1967, 2, 1289.
McAlpine, D., Compston, N. D., and Lumsden, C. E. (1955). *Multiple Sclerosis*, Edinburgh.
McAlpine, D., Lumsden, C. E., and Acheson, E. D. (1965). *Multiple Sclerosis—A Reappraisal*. Edinburgh.
Munro, D. (1943). *J. Amer. med. Ass.*, 122, 1055.
Nyquist, R. H., and Bors, E. (1967-8). *Paraplegia*, 5, 22.
Ross, J. C., Gibbon, N. O. K., and Damanski, M. (1967). *Brit. J. Surg.*, 54, 627.
Silver, J. R. (1967-8). *Paraplegia*, 5, 226.
Tribe, C. R. (1963-4). *Paraplegia*, 1, 19.
Tribe, C. R., and Silver, J. R. (1968). *Renal Tract Disease in Paraplegia*. London. In press.
Walsh, J. J. (1967). *Proc. roy. Soc. Med.*, 60, 1212.