PAPERS AND SHORT REPORTS

Aetiology of pressure sores in patients with spinal cord injury

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Abstract

One hundred consecutive patients admitted to the National Spinal Injuries Centre, Stoke Mandeville Hospital, with pressure sores were studied to assess the relative importance of factors known to predispose to the development of sores. Loss of feeling was critical, because patients were unable to appreciate pain when the sore was developing. Risk of developing a sore increased with age, but duration of the paralysis was of equal importance. After discharge from hospital the presence of a caring relative or friend was essential for survival. Many patients developed sores because of poor facilities at home or inappropriate advice from those who looked after them. An even more distressing factor was the number of patients who developed sores in hospital owing to inadequate nursing care.

There are relatively few paralysed patients in the community, but the lessons learnt in this study may be applied to all patients with orthopaedic injuries and to geriatric patients with limited mobility. Nursing and medical staff must turn patients regularly and ensure that there is proper equipment to relieve pressure on the skin. Patients should not be allowed to sit in a chair if they develop a sacral or trochanteric sore. More effort should be directed towards the appropriate education of patients, their relatives, and all those who are concerned with their welfare.

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Introduction

Any patient who sustains an injury to the spinal cord is at risk of developing pressure sores. The risk is greatest immediately after injury when the paralysis is associated with other lesions.[1] Nevertheless, patients with spinal cord injuries remain at risk of developing pressure sores for the rest of their lives.[2 3] Sores that develop immediately after injury delay rehabilitation and may even result in death. Those that occur after the patient has gone home may endanger his or her health and are costly to treat. Pressure sores are therefore the most important complication of paraplegia by virtue of the high cost of prolonged hospitalisation[4] and they pose a serious threat to life.

Much has been written about pressure sores in paraplegic patients,[5-7] and the major causes of pressure sores are now understood.[8 9] The relative importance of individual aetiological factors has not, however, been analysed. We have conducted a prospective study of 100 consecutive patients who either had pressure sores on admission or developed them while receiving treatment to establish the relative importance of the factors that predisposed to development of the sores.

Patients and methods

The study was carried out on consecutive patients with traumatic spinal cord injuries admitted to the National Spinal Injuries Centre, Stoke Mandeville Hospital, between 1980 and 1982. Patients were considered for the study only if they had pressure sores on admission or if they had developed them while undergoing treatment in hospital. One hundred patients (83 male, 17 female; age range 11-80 years) were included. Twenty five patients (17 female, 8 male) were transferred to the centre for their primary rehabilitation within five weeks after the acute injury. These are referred to as the acute cases. The remaining 75 (all men) were readmitted for pressure sores that had developed some time after their initial discharge. These patients are referred to as the chronic cases.

A questionnaire was completed by one of us (CT) to elicit the following information: age, sex, marital state, neurological state, bowel and bladder function, emotional state, housing, aids and appliances, anatomical site and severity of sore, nursing care, and delay of admission. The questionnaire was checked for clarity by a general practitioner, a rehabilitation nurse, a district nurse, a physiotherapist, and an occupational therapist. In the acute cases the factors underlying the development of the sore were assessed by

examination of the patient and direct questioning of the nursing staff. In the chronic cases they were determined by the patients' answers to the questionnaire.

Results

ACUTE CASES

The pressure sores in the acute group—that is, whose sores had developed within five weeks of the initial injury—were fresh and superficial (superficial was defined as blistering or reddening of the epidermis).[10] Twenty three patients had developed their sores while in hospital and two while on weekend leave. Table I shows the distribution of the sores and the level of the spinal cord injury. All 17 women patients had sacral sores (fig 1). These occurred when the patient was being mobilised and were caused by urinary incontinence.

TABLE I—*Distribution of sores in 25 acute cases*

	Sacral	Trochanteric	Ischial	Multiple	Foot	Spinous process	Breast	Finger
Cervical cord lesion (n = 12)	11	0	0	0	0	0	0	1
Thoracic cord lesion (n = 11)	6	2	1	0	0	1	1	0
Lumbar cord lesion (n = 2)	0	0	0	0	2	0	0	0
Total	17	2	1	0	2	1	1	1

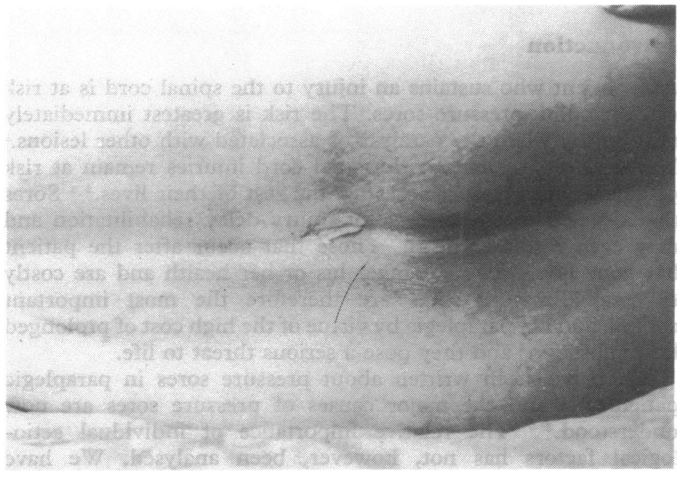

FIG 1—Superficial sacral pressure sore in woman patient due to incontinence of urine.

This type of sore was not seen in the male patients. The remaining sores also occurred when the patient was being mobilised and were due to unsuitable or tight clothing (two cases), improperly fitting callipers (two), burns (one), and excessive and abnormal bone formation around the joints (heterotrophic ossification) (three), seen in patients with paraplegia. Although patients in the acute group were aged 11-71 years, the average age was only 33, and 18 were single. Twenty one of the 25 patients had complete spinal cord lesions, and this figure is of note because only about half of patients admitted with acute spinal cord injury have incomplete lesions. Thus patients with total anaesthesia are more likely to develop pressure sores than those who retain some feeling.

CHRONIC CASES

Table II shows the distribution of the pressure sores in the chronic group. In contrast with the sores in the acute group more than half were deep sores—that is, affecting the subcutaneous adipose tissue, muscle, bone, or joints[10]—and required several months of treatment in hospital, including blood transfusions and surgical procedures. Apart from the major sites there was also a high incidence of pressure sores on the foot and ankle. Nineteen patients developed their sores immediately after discharge—that is, within the first year after their initial period of hospitalisation (table III). These patients then remained relatively free of sores until five years after the injury, when they began to develop fresh sores. Fourteen patients remained completely free of sores for 10-15 years before developing a sore for the first time. The incidence of recurrent sores increased with time, and the rise paralleled that of the fresh sores. Recurrent sores represent an enormous drain on the resources of a spinal unit, and this is illustrated by the following two case histories.

Case 1—A single man aged 46 was injured on 8 May 1976 in a fall from a height on a building site. He sustained an incomplete spinal cord lesion below T12. He was first discharged from the National Spinal Injuries Centre on 15 December 1976 and remained free of sores until early 1982, when he was admitted to another hospital with a sore on the left buttock. He was transferred to this centre on 24 February 1982 and discharged on 5 July 1983. Unfortunately, the sore broke down and he was therefore readmitted on 9 August 1983. On 10 January 1984 the right trochanteric sore was excised with a Girdlestone excision, and he remained an inpatient (fig 2). This patient had spent two years and five months in hospital in the seven years and nine months since his injury.

TABLE II—*Distribution of pressure sores in 75 chronic cases*

	Sacral	Trochanteric	Ischial	Multiple	Foot and others
Superficial sores (n = 75):					
Cervical cord lesion (n = 15)	5	4	1	3	2
Thoracic cord lesion (n = 57)	13	14	11	10	9
Lumbar cord lesion (n = 3)	1	2	0	0	0
Total	19	20	12	13	11
Deep sores (n = 39)	6	14	8	8	3

TABLE III—*Time of onset of sores in 75 chronic cases*

	1st year	2nd year	3rd year	5th year	5-10 years	10-15 years	Total
Fresh	19	1	1	1	6	14	42
Recurrent	0	1	3	4	8	17	33

Case 2—An unmarried serviceman aged 26 was injured in January 1973 in a diving accident. He was admitted to Stoke Mandeville Hospital on 18 February 1974 and discharged to another hospital on 12 July but was subsequently readmitted to the National Spinal Injuries Centre for treatment of his sore on six occasions. As a result of osteomyelitis due to the sore his right leg had to be amputated and he remained an inpatient (fig 3). In the 11 years since his injury this patient had spent seven years in bed with pressure sores.

Patients in the chronic group were older (mean age 43·6 years) than those with acute sores, and 50 were single. Fifty seven of the patients had lesions of the thoracic cord (table II), and, of these, 37 had incomplete lesions. This contrasts with patients in the acute group, among whom pressure sores were more common in those who had sustained cervical lesions and who had complete transection of the cord. Only 10 patients were incontinent of urine (the rest, all men, being managed with catheters or condoms), but even in these 10 patients urinary incontinence was not considered to be a major cause of the sore. Nor was faecal incontinence found to be a factor in the development of sores. Forty eight patients developed the sores while they were in their wheelchairs, and 27 developed the sores while in hospital.

The 27 patients who developed their sores while in hospital had been admitted to their district general hospital for treatment of various medical conditions. Fourteen of these had urinary tract infections and another four chest infections, which may have contributed to their debility (table IV). Eight patients had been admitted for social

TABLE IV—*Reasons for admission to district general hospital of 27 patients with chronic sores*

Urinary tract infection	10	Chest infection	4
Catheter block	2	Jaundice	1
Septicaemia	2	Undiagnosed ailment or social reason	8

TABLE V—*Causes of sores in 75 chronic cases*

	Hospital	Home	Total
No turn	19	7	26
Failure to lift	3	16	19
During transfer	1	10	11
Tight clothing and shoes	0	6	6
Plaster	3	0	3
Periarticular ossification	0	0	0
Cushion and mattress	0	4	4
Sweating	0	1	1
Extravasation of intravenous fluid	1	0	1
Burns	0	2	2
Not known	0	2	2

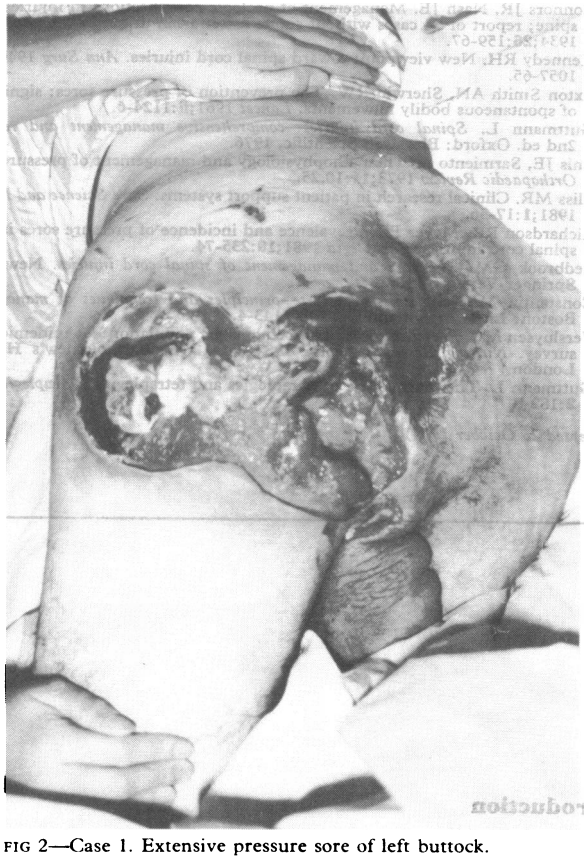

FIG 2—Case 1. Extensive pressure sore of left buttock.

reasons—for example, because there was no one at home to look after them.

The number of patients who developed pressure sores while in hospital was worrying. The main cause for the development of sores is impairment of the blood supply to the tissues, and in 19 of 26 cases the patient was not turned. In the 75 patients of the chronic group as a whole the main aetiological factors were failure to lift themselves or bruising during transfer from bed to chair (table V).

Discussion

These patients were a selected group with severe pressure sores that necessitated admission to hospital. The series does not therefore represent the pattern of sores among all our patients, some of whom do not require inpatient treatment. Furthermore, the National Spinal Injuries Centre gives priority to ex-servicemen, working paraplegics, and patients with life threatening complications, so that we tend to see the more severe cases. This series, therefore, differs from previous reports, with the exception of that of Richardson and Meyer; they, however, did not distinguish between superficial and deep sores.[12]

Patients in the acute group were all young, and the sores occurred predominantly in tetraplegic patients with cervical lesions and complete loss of power and sensation. Such patients

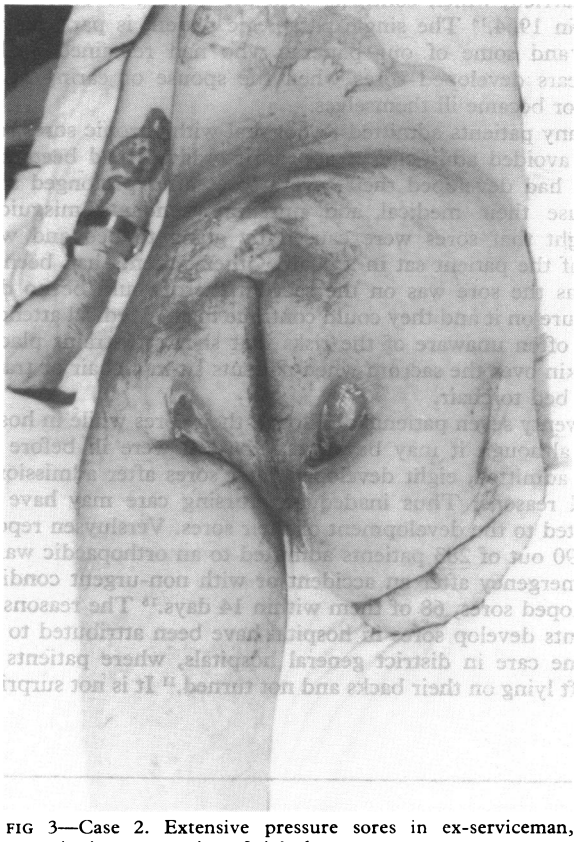

FIG 3—Case 2. Extensive pressure sores in ex-serviceman, necessitating amputation of right leg.

are completely dependent and unaware of painful stimuli and thus unable to move themselves to prevent the development of sores. This finding agrees with a study of acute and chronic cases in spinal centres in the United States.[4] Sacral sores were common in the acute cases and occurred only in women. They developed during the early stages of mobilisation when the patients were undergoing bladder training but spending most of their time in bed, when urine leaks backwards and macerates the skin. Once they had progressed and were up all day sores were less common, even though the patients were still incontinent. This emphasises that the damage to the skin is predominantly one of maceration rather than friction between skinfolds, as has been suggested.[13] The use of a temporary urethral catheter allowed these superficial sores to heal within a few days.

Most of the sores in the chronic cases occurred in patients with thoracic lesions, a finding which has been reported before,[14] and in both the acute and chronic groups sores were more common in patients with complete cord lesions. Several patients in the chronic group developed sores around the foot and ankle, indicating that they were active and had knocked themselves during their daily activities. Neither urinary nor faecal incontinence was considered to be an important aetiological factor in the chronic cases, most patients developing sores within a year after their initial discharge. This may reflect poor training before discharge or poor adaptation to home surround-

ings, or both. Once, however, this dangerous period had passed patients remained relatively free of sores until 10-15 years after injury. Older patients are known to be at greater risk of developing sores,[15] but we have shown that the duration of the paralysis is also important. Thus a 40 year old patient who has been paralysed for 15 years and is suffering from urinary tract infection, osteoporosis, immobility, obesity, and atrophy of the tissues due to continuous sitting is plainly not as fit as the 40 year old who first becomes paralysed at this age.

The social state of the patient is another important factor. In our series of patients with chronic sores 50 were single and 25 married, which contrasts with the pattern recorded at this unit in 1964.[16] The single paraplegic patient is particularly at risk, and some of our patients who had remained well for 15 years developed sores when the spouse or caring relative died or became ill themselves.

Many patients admitted to hospital with chronic sores might have avoided admission if appropriate advice had been given. Most had developed their severe sores after prolonged sitting because their medical and nursing attendants misguidedly thought that sores were caused by going to bed and would heal if the patient sat in a chair. Other patients had been told that as the sore was on the sacrum there would be no direct pressure on it and they could continue to sit. Medical attendants were often unaware of the risks that shearing strains place on the skin over the sacrum when patients sit in a chair or transfer from bed to chair.

Twenty seven patients developed their sores while in hospital and, although it may be argued that all were ill before they were admitted, eight developed their sores after admission for social reasons. Thus inadequate nursing care may have contributed to the development of their sores. Versluysen reported that 90 out of 283 patients admitted to an orthopaedic ward as an emergency after an accident or with non-urgent conditions developed sores, 68 of them within 14 days.[15] The reasons why patients develop sores in hospital have been attributed to poor routine care in district general hospitals, where patients may be left lying on their backs and not turned.[11] It is not surprising, therefore, that paraplegic patients develop sores, because they cannot turn themselves; furthermore, because their skin is numb they are unable to appreciate that they have developed a sore and do not complain of pain. Clearly, all the medical staff who are responsible for the management of these patients must be aware of the factors which predispose to the development of sores and ensure that routine nursing care takes these into account. Similarly, both the patients and those responsible for looking after them at home must be well briefed so that the risks of developing this distressing complication may be minimised.

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β Blockers and loss of hearing

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Abstract

Loss of hearing in a 43 year old man during treatment with metoprolol was dose related and disappeared within a few months after the drug had been stopped. The hearing impairment was of mixed type, with an air bone gap without any disorder of the middle ear observable by conventional clinical methods. Similar scattered reports from international sources on loss of hearing during treatment with β blockers are also presented.

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Introduction

β Adrenoceptor blocking drugs play a great part in the treatment of common cardiovascular diseases such as angina pectoris and hypertension.[1] The efficiency and limitations of these drugs are well known. One side effect, however, seems to have been forgotten since a thorough report appeared on practolol and deafness.[2] Here we report loss of hearing during treatment with metoprolol and discuss similar scattered reports from international sources.

Patient and methods

A 29 year old man was admitted to hospital in 1969 for hypertension and glucosuria. Blood pressure was 160/120 mm Hg despite treatment with chlorthalidone 50 mg daily during the previous two months. Intra-arterial blood pressure was normal, and glucosuria disappeared when the drug was stopped. In 1970 he was investigated further. *X* ray films of the heart were normal. Angiography showed minor, possibly hypertensive, changes of the intrarenal arteries. Funduscopic examination and tests for phaeochromocytoma and primary hyper-