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THE NATURE AND TREATMENT OF TROPHIC PRESSURE SORES

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The development of trophic sores in an analgesic part of the body is a feature of many conditions, medical and surgical. While the vast majority can be prevented by simple methods, the cure of an established, penetrating trophic ulcer is a difficult and taxing problem. Their management merits most careful attention. Experience with paraplegic patients reveals the marked contrast in the maintenance of fair general condition and consequent speedy rehabilitation in those in whom this complication has been avoided and the dreadful, steady, septic deterioration in the untreated or badly treated case.

Our experience is based upon the management of a paraplegic unit of 40 beds. While no fundamentally new principles are iterated, it is felt that—since this is the only such unit in southern Africa—experience gained there could well be applied elsewhere in the country.

PATHOGENESIS

While the vast majority of these lesions occur over the sacral, ischial and trochanteric pressure areas, no area of the body is immune if it is anaesthetic. Regardless of the site, extent or underlying aetiology of the individual lesion, the principles of causation, of prevention and of treatment do not differ.

The so-called bedsore which develops is, in reality, an area of gangrene occurring in an anaesthetic part. The absence of the normal neurogenic component of the inflammatory response alters the nature of the reaction of the tissues to injury or insult. As with gangrene elsewhere, the fundamental cause is local ischaemia. This results from the inter-play of 3 *local factors:*

1. *Sustained local pressure*, which obliterates the local blood supply;

2. *A fall in the blood haemoglobin level*, either as a primary phenomenon associated with the initial injury or disease, or secondary to sepsis. It follows that one bedsore causes sepsis which, in turn, provokes secondary anaemia and a vicious cycle is established. The haemoglobin level may drop as much as 1 g.% per day;

3. *Local stasis*, due to paralysis of muscles and total immobility.

In addition to these local factors, the development of paraplegia is associated with *profound general metabolic disturbances.* Among these, as noted by Walsh (1954) is an immediate rise in the level of serum globulin and a fall in that of serum albumin, and an increase in protein catabolism resulting in a rise in urinary excretion of protein metabolites.

Once ulceration has developed, it passes through a stage of activity and extension followed—if properly treated—by gradual resolution and healing. The process may yield one of 2 forms of chronic lesions, depending upon the extent and depth of the original necrosis, viz. (1) the active extending ulcer and (2) the so-called sinus sore.

The active extending ulcer shows a typical picture of spreading necrosis of skin and subjacent tissues—whether fat, muscle or even bone—with margins which are oedematous and sub-acutely inflamed at first, becoming almost punched-out as activity resolves. The affected skin turns the black colour so typical of gangrene while necrotic deeper tissues form a dirty yellow or grey slough. There is commonly a considerable degree of undermining of deeper tissues beyond the actual limit of skin necrosis.

Healing occurs by slow fibrosis except where, as occurs uncommonly, necrosis has not extended deeper than the epidermis. It is a paradox worthy of note that

a lesion which develops within a few days may take as many months to heal.

Where the necrosis has been more extensive in the superficial than in the deeper tissues, the final result is the formation of a *saucer-shaped defect*. This is most commonly seen over the sacrum. Only the floor of the defect becomes covered by granulation tissue which, in turn, is converted into fibrous tissue. In the course of several weeks this fibrosis becomes dense and tough and comparatively resistant to pressure necrosis, while a thin layer of epithelium grows in from the sides of the defect to cover this fibrotic layer.

Alternatively, superficial necrosis may be limited while the process spreads extensively in the deep tissues, even within bone. The outcome is the so-called *sinus sore*, i.e. a small skin sinus overlying a deep, extensive, often branching cavity. This is the common lesion found over the ischial tuberosities. By the very nature of its pathology, spontaneous cure can never occur.

PREVENTION

The chief factors concerned in prevention are most easily expressed in tabular form:

1. Maintenance of optimal general health and nutrition is essential. Although bed-ridden, such patients must be coaxed to take a very high-calorie, high-protein diet with added vitamins and iron. Tube feeding may be necessary for a time. The mental health being as important as the physical state, the sickest patients should be interspersed among the nearly recovered, and all should be kept profitably occupied. In this regard the work of occupational and physio-therapists cannot be over-stressed.

2. The blood picture must at all times be kept optimal. Regularly repeated blood-counts are necessary and any fall in the haemoglobin level below 15 g.% must be counteracted by immediate transfusion of fresh blood. The quantity of blood administered must be adequate; 2-4 pints per week may be necessary at first. To conserve veins, cut-downs should be avoided. Where allergic responses occur, transfusion of washed cells or the addition of an antihistaminic drug to the drip, should be tried.

3. Any prolonged skin-pressure must be avoided, notably at the sacral, trochanteric, ischial, heel and malleolar areas. The patient is nursed on a foam-rubber mattress laid on a firm base, with additional soft pillows at strategic points. All acute paraplegics, whether the cord lesion is partial or complete, are turned 2-hourly, day and night, from the time of admission; once the acute phase has passed, the frequency of turning may be reduced to 4-hourly. The turning sequence is: back, side, abdomen, other side. In leaner subjects, rings should be applied so as to avoid any direct pressure on bony protruberances; thick orthopaedic felt has been found to be of value for this purpose.

5. Scrupulous hygiene is maintained. The patients should be trained to report any soiling of bed linen, which is changed immediately. The whole body is washed regularly with warm soapy water and the pressure areas are massaged with surgical spirit daily. The use of silicone-base creams as an alternative to this method—as reported by Bateman (1956)—has proved unsuccessful and has been abandoned as an alternative method of skin care.

6. Heat, in any form, is dangerous. The analgesic skin will not tolerate hot-water bottles, heat lamps or even prolonged exposure to direct sunlight.

7. Regular physiotherapy is commenced as early as possible. Daily passive movements to completely paralysed parts serves to prevent local circulatory stasis.

These simple principles should be available under the most rudimentary conditions. Simple as they are, they must be practized most punctiliously. Again it must be stressed that prevention is essential, for cure is difficult. Furthermore, there is a strong impression that the early intervention of sepsis—whether from bedsores or any other source—with concomitant secondary anaemia, retards the recovery process of the spinal-cord lesion.

TREATMENT

The development of any skin abrasion or ulcer, however trivial, must be regarded as a matter of grave consequence. Untreated, such small lesions invariably spread in extent and depth at an alarming rate. The patients are, at first, somewhat secretive in this respect, for they know that the development of a minor abrasion necessitates immediate confinement to bed. (Later they come to appreciate the true significance of this complication; in fact, a minor trophic lesion is the commonest reason for rehabilitated paraplegics seeking, and gaining, re-admission.) As a prophylactic measure, one should make a complete visual examination of all patients, including the ambulant ones, once weekly.

Fundamentally, the treatment of all lesions follows exactly the principles laid down for their prevention. The patient is confined to bed absolutely until healing is complete and stable; the general nursing and hygenic regime described is instituted and is maintained strictly until such healing has occurred.

The patient must be nursed in such a position that no pressure whatever is allowed to play upon the affected skin area. With the predominant incidence upon buttock and trochanteric sores, the majority need to be confined to bed in the prone position.

(*a*) *The active, extending ulcer*

The basic aim of treatment is a reversal of the factors responsible for the ulceration. The patient is confined strictly to bed and is nursed in a position which avoids any pressure upon the affected area. A mild antiseptic dressing, e.g. gauze soaked in Eusol, is applied locally and is covered with cotton wool. The routine of hygiene and nutrition mentioned above is enforced strictly.

Above all, the blood picture must be maintained at a normal level. The blood haemoglobin is checked twice weekly and any fall below 15 g.% is treated by immediate transfusion of adequate quantities of fresh blood. With extensive ulceration the level may fall as rapidly as 1 g.% daily at first and 2-4 pints of blood may be required weekly for several weeks.

Antibiotics are administered, usually penicillin to-

gether with streptomycin or a sulphonamide in the first instance. A course of 7-14 days is usually adequate, for these drugs have no essential effect upon receding or chronic ulceration.

Once the extent of the slough becomes demarcated, it should be excised freely into healthy tissues. The procedure being a septic one and the affected area anaesthetic, excision may be performed quite conveniently in a side ward. The procedure may have to be repeated.

The method of treatment is unvaried, regardless of the extent of the active ulceration when first seen. With strict application of these measures, it is unusual for the ulceration to extend in the otherwise uncomplicated case.

(*b*) *The healing ulcer*

As mentioned above, healing occurs by a process of very slow fibrosis. Throughout the healing process, nursing in a position which completely avoids any pressure of the lesion, is continued. The haemoglobin level is checked regularly. In certain cases, even, where healing is extremely tardy despite a normal or nearly normal blood picture, an occasional transfusion of a pint of fresh blood is of inestimable value.

Two forms of dressing are used, depending upon whether the ulcer is dirty, with exudate and slough, or whether it is covered by clean, dry granulations. Dirty ulcers require Eusol dressings 4 times daily; clean ulcers require simple *tulle-gras* dressings every 3rd or 4th day. The entire affected area is kept covered with several layers of gauze and cotton-wool and standard care of the healthy surrounding skin is observed.

Where the lesion is peripheral, sympathectomy may be of value in accelerating healing. This applies notably to chronic or recurrent pressure-sores on the feet occurring in cauda-equina lesions, where a localized lower lumbar sympathectomy has been found to accelerate healing.

(*c*) *The chronic sinus-sore*

This lesion, by its very anatomy, cannot heal spontaneously. Not infrequently the cause of persistence of discharge from the sinus is the presence of infected bone at the depths of the ulcer. The method of treatment, now a fairly standardized procedure, follows the practice of Guttmann and his colleagues at Stoke Mandeville. The method is described fully by Walsh (1954).

Briefly, the entire cavity is fully and firmly packed *via* the sinus with ribbon gauze, thereby converting it into an artificial tumour. The tumour is lined by the dense fibrous tissue forming the limits of the original cavity. A fusiform incision is made around the sinus and the tumour is excised by knife or diathermy into healthy tissue on all sides, without entering the gauze pack. Where the cavity extends into bone, any diseased bone is freely curetted away. Absolute haemostasis is affected and primary suture is carried out around a drain. Where the cavity is extensive, the resultant wide skin-flaps should be tethered to the underlying tissues, as described below. Finally a firm pressure-dressing is applied.

Antibiotic cover is given pre-operatively and is maintained for several days thereafter. The drain is removed after 48 hours and the skin sutures after about 10 days. Any accumulation of blood or exudate which forms should be aspirated through a wide-bore needle.

Primary healing is usual. The unhappy alternative is the formation of a far smaller, more localized chronic sinus-sore. This does not tend to extend and may be re-excised in exactly the same way.

(*d*) *The healed scar*

This results from an extensive area of fairly superficial necrosis. The saucer-shaped defect becomes covered eventually by a thick and comparatively stable fibrotic scar. Where it is restricted in extent and is removed from any area subject to pressure or friction from sitting, lying or appliances, it may well be left alone. Otherwise, full-thickness skin cover must be obtained.

Experience has shown that the application of split-skin grafts or the swinging of skip flaps has no part whatever to play in the treatment of the vast majority of cases. One should make use of skin immediately surrounding the defect. With healing of the lesion, this tends to become thickened and immobile. Considerable mobilization can be obtained by daily oil-massage, and before proceeding to formal surgery one should nearly be able to cover the defect by simply pushing the supple surrounding mobilized skin over it manually.

The operative procedure, again with antibiotic cover, consists of total excision of the scar up to the margins of healthy skin. The healthy skin and subcutaneous tissue is undercut as widely as is necessary to obtain full cover without tension. Thorough haemostasis is obtained by diathermy coagulation.

The enormous subcutaneous dead-space formed must be obliterated at all costs. This may be done by placing silk sutures midway along each flap, taking a bite of the deep tissue and being tied over small gauze-pads laid on the flap. An additional safeguard is to fix the line of skin suture to the deeper tissues with a few deep mattress-sutures which take a bite of the deeper tissues.

One or more drains are inserted and a firm pressure-dressing is applied. The post-operative care follows the description given above. The importance may be stressed of prompt aspiration of any collection of blood or exudate.

Primary healing occurs in less than half these cases. Disheartening though this is, all is not lost if the wound does break down partly or completely. One may either perform delayed secondary suture, which sometimes succeeds, or allow the defect to proceed to granulation and fibrosis; the resulting defect is far smaller than the original and may be excised and covered in exactly the same way as before.

LOCAL RESULTS

During the first 8 months of 1956, 34 patients were admitted to the paraplegic ward at Baragwanath Hospital. Among these there were 6 deaths, 4 from complete high cervical cord lesions. During the same

period 25 patients were discharged, fully rehabilitated and free of bed sores.

At the time of writing there were 35 patients in the paraplegic ward. Of these 15 were primary admissions, i.e. patients admitted to the unit immediately after the development of paraplegia and consequently treated *ab initio* by the methods described. Only 5 of this group had, or had had, trophic lesions of any kind, namely:

Two developed friction sores over the lateral malleolus from calipers; both are healing. One developed a pressure sore over the right great trochanter; this remained superficial and has started to heal. One had a sinus sore over the left great trochanter, which has been cured by radical excision. One has a healed friction sore in the anal cleft.

The remaining 20 patients were secondary admissions, i.e. patients referred to the unit for further treatment after a shorter or longer period in other surgical units or hospitals. All but one were admitted with bedsores, there being no less than 51 trophic lesions among the 19 patients. The maximum in any one patient was 7 sores and several had 4, 5 or 6 separate lesions. Of these 31 are now completely healed, 20 are healing satisfactorily and none are extending. A considerable back-log of patients requiring radical excision of sinus sores or healed defects over the pressure areas has been built up from this group of patients.

These preliminary figures indicate clearly the scope of the work and the relative success of the regime of prevention and cure described.

SUMMARY

The trophic lesions which develop in anaesthetic areas are a form of gangrene due to local ischaemia. The local and general factors concerned in the initial causation and in the extension of this necrotic process, are described.

Once necrosis commences, it spreads rapidly in extent and depth, unless promptly and properly treated. Healing occurs by very slow fibrosis, forming either a saucer-shaped defect or a chronic sinus-sore, according to the extent of necrosis of the deep tissues.

The essence of prevention lies in maintaining adequate nutrition and a high blood-haemoglobin, in scrupulous hygiene and care of the skin, and in the prevention of any prolonged local pressure by systematic turning of every acute case.

Cure of commencing or progressing ulceration depends basically upon the strict application of the same principles. Where the end-result is loss of tissue and fibrosis over an area subject to pressure or friction, full-thickness skin cover should be obtained by a relatively simple procedure, which is outlined. Where a chronic sinus-sore forms, it can never heal spontaneously and must be excised radically by the technique developed at the Stoke Mandeville Centre.

Local figures illustrate the rarity of trophic lesions among cases treated in the Baragwanath paraplegic unit from the start, and their great frequency among cases admitted after initial treatment elsewhere. During the first 8 months of 1956, 25 fully rehabilitated paraplegics were discharged from the unit.

SAMEVATTING

Die patologiese proses van trofiese weefselafsterwe en bedseervorming, met verwysing na pasiënte met paralegie, word behandel. Klem word gelê op voorkoming van die bedsere, en die behandeling, indien eenmaal gevorm, word verduidelik.

Syfers toon aan dat behandeling liefs in spesiaal toegeruste hospitale moet geskied, soos byvoorbeeld die paraplegie-kliniek te Baragwanath Naturelle-hospitaal.

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