A rapid spontaneous rise in temperature is a good prognostic sign; thus all patients who survived had regained a normal temperature within 24 hours. We feel that if rewarming is to be used at all it should be restricted to those patients who have shown no significant rise in temperature by 24 hours. A prolonged period of hypothermia undoubtedly contributes to a very poor prognosis, and in this group active rewarming is justified.

Hydrocortisone was given to all patients during the hypothermic period. Adrenocortical function is impaired in hypothermia and also in myxoedema (Hubble, 1955). Eghdahl *et al.* (1955) found that secretion of 17-hydroxycorticosteroids fell to 25% of control values in dogs cooled to 77–82° F. (25–27.8° C.) and that no increase was produced by infusion of adrenocorticotrophic hormone. It was noticed that in several of the present patients hypotension was more readily controlled after injection of hydrocortisone.

Intravenous fluids were given when indicated, and mephentermine or metaraminol if hypotension was present, but noradrenaline infusion was ultimately required in Cases 2, 4, 7, and 8. Triiodothyronine 10 to 60 µg. daily was given orally in Cases 1, 2, and 3. All patients received penicillin or tetracycline, with digoxin and thiazide diuretics if cardiac failure was present. It has been shown that digitalis is not contra-indicated in hypothermia and is not a factor in the origin of ventricular fibrillation (Angelakos *et al.*, 1958). One patient (Case 5), although not cyanosed, had marked respiratory depression, and positive-pressure respiration was required for 12 hours.

Summary and Conclusions

Nine cases of accidental hypothermia seen in a small hospital within a period of four months are reported. Three patients survived. Electrocardiographic and biochemical abnormalities are briefly reviewed. It is suggested that the condition is by no means uncommon and should be considered in the differential diagnosis of all patients with impaired consciousness during the winter months. It can occur rapidly and is by no means confined to the elderly and neglected. Although there is often a history of exposure during cold weather, hypothermia can develop when the patient is in bed in relatively warm surroundings. The phenothiazine group of drugs should be given to the elderly and hypothyroid patient with caution and the rectal temperature checked if confusion or drowsiness occurs. This should be taken with a low-reading thermometer or, if unavailable, an ordinary lotion thermometer. The prognosis is poor, but is more favourable in the younger patients.

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REFERENCES

- Angelakos, E. T., Torres, J., and Driscoll, R. (1958). *Amer. Heart J.*, **56**, 458.
- Courvoisier, S., Fournel, J., Ducrot, R., Kolsky, M., and Koetschet, P. (1953). *Arch. int. Pharmacodyn.*, **92**, 305.
- Duguid, H., Simpson, R. G., and Stowers, J. M. (1961). *Lancet*, **2**, 1213.
- Eghdahl, R. H., Nelson, D. H., and Hume, D. M. (1955). *Surg. Gynec. Obstet.*, **101**, 715.
- Emslie-Smith, D. (1958). *Lancet*, **2**, 492.
- Fisher, B., Fedor, E. J., and Lee, S. H. (1958). *Ann. Surg.*, **148**, 32.
- Frueman, A. E. (1960). *Arch. intern. Med.*, **106**, 218.
- Fuhrman, F. A., and Crimson, J. M. (1947). *Amer. J. Physiol.*, **149**, 552.
- Hubble, D. (1955). *Lancet*, **1**, 1.
- Knocker, P. (1955). *Ibid.*, **2**, 837.
- Laborit, H., and Huguenard, P. (1951). *J. Chir. (Paris)*, **67**, 631.
- Laufman, H. (1951). *J. Amer. med. Ass.*, **147**, 1201.
- Macdonald, D. W. (1958). *Brit. med. J.*, **2**, 1144.
- Malden, M. (1955). *Ibid.*, **2**, 764.
- Mann, T. P., and Elliott, R. I. K. (1957). *Lancet*, **1**, 229.
- Marshall, R. J., and McCaughey, W. T. E. (1956). *Ibid.*, **2**, 754.
- Mitchell, J. R. A., Surridge, D. H. C., and Willison, R. G. (1959). *Brit. med. J.*, **2**, 932.
- Moyer, J. H., Morris, G., and De Bakey, M. E. (1957). *Ann. Surg.*, **145**, 26.
- Nedzel, A. J. (1952). *J. Aviat. Med.*, **23**, 49.
- Osborn, J. J. (1953). *Amer. J. Physiol.*, **175**, 389.
- Read, A. E., Emslie-Smith, D., Gough, K. R., and Holmes, R. (1961). *Lancet*, **2**, 1219.
- Rees, J. R. (1958). *Ibid.*, **1**, 556.
- Sano, M. E., and Smith, L. W. (1940). *J. Lab. clin. Med.*, **26**, 443.
- Sheehan, H. L., and Summers, V. K. (1952). *Brit. med. J.*, **1**, 1214.
- Summers, V. K. (1953). *Ibid.*, **2**, 366.
- Vandam, L. D., and Burnap, T. K. (1959). *New Engl. J. Med.*, **261**, 595.
- Wynn, V. (1954). *Lancet*, **2**, 575.

Preliminary Communications

A New Approach to the Treatment of Leg Ulcers

It is generally accepted that the immediate cause of venous leg ulcers is venous hypertension, with the resulting capillary changes due to back-pressure. Chronic venous insufficiency is most commonly a sequel to deep venous thrombosis, which often affects and destroys the valves of the perforating veins in addition to those of the deep veins; the raised capillary pressure and damage so produced result in the escape of protein-containing fluid into the tissues. The limb becomes oedematous and later indurated, and with the interference with the nutrition of the skin and subcutaneous tissues ulceration may eventually occur (Anning, 1954). Many ulcers rapidly become infected, often to a considerable degree.

The logical treatment of venous ulcers of the leg is based on the reduction or correction of these aetiological factors. The traditional means by which this may be affected are threefold.

First, the source can be removed surgically by stripping the varicose long and/or short saphenous vein with division of any defective communicating veins. This may be both prophylactic and therapeutic. After careful and complete diagnostic evaluation the chances of either a primary or a recurrent ulcer developing in a leg so predisposed may be considerably lessened by these means. In addition, in certain intractable, especially post-thrombotic, ulcers, such radical surgery on the veins may succeed in healing the ulcer where other methods have previously failed.

In the second place the patient may be treated in recumbence and by raising the leg above the level of the heart and maintaining this position until oedema subsides and the ulcer eventually heals. This prolonged and often impracticable tedium would seem to be obsolete, for not only does the ulcer tend to recur when normal activities are resumed but there are distinct risks in keeping these patients, many of whom are elderly, obese, and arthritic, in bed for any length of time.

The third method is by the application of external pressure to the leg by means of compression bandaging, in conjunction with which various bacteriostatic or bactericidal preparations can be applied. This can be

done in either of two ways. First, by applying such a preparation directly on the ulcer, covering with gauze over which is placed a polyurethane foam pad, cut to a size larger than that of the ulcer, and finally the compression bandage, either an elastocrepe or an elastic-web bandage being favoured. The dressing is changed daily by the patient or her attendant, ideally before rising. The alternative is by the use of specially impregnated bandages applied directly to the skin of the entire leg, commencing over the metatarsal heads below, to the level of the tibial tuberosity above. Over this a foam pad is applied to cover the ulcer, and finally a firm adhesive bandage. In this clinic we favour the use of one of the "dalzoband" series, most commonly No. 20 (containing zinc paste, calamine and iodo-chlorohydroxyquinoline) or, alternatively, No. 5 (containing zinc paste,

calamine, and urethane). This is covered with a "poroplast" supporting bandage. The dressing is left on for from five days to three weeks, depending upon the condition of the ulcer.

This latter method would seem to be the one of choice, and indeed the results obtained in clinics where it is routinely practised justify this claim (Lorch, 1961; Thorne, 1958). However, the method is not without its problems, the most evident of which are as follows: Firstly, some patients, especially those with grossly infected ulcers and much oedema, find that the initial dressings are very painful and often intolerable. In addition, in such cases the amount of infection makes the dressing exceptionally malodorous after a day or two; this is most unpleasant for both the patient who has to wear it and the nurse or doctor who subsequently

TABLE I

Case No.	Size of Ulcer (cm.)	Pain	Oedema	Visible Infection	Organisms and Antibiotic Sensitivities	Antibiotic Used	Duration of Oral Therapy	Duration of Subsequent Compression Therapy until Ulcer Healed	Remarks
1	2.2 x 1.2	+	++	++	<i>Staph. aur.</i> and <i>E. coli</i> , sensitive to tetracycline, etc.	Tetracycline	5 weeks	3 weeks	Uncomplicated progress to complete healing
	1.4 x 0.7	0	0	0					
2	1.4 x 1.2	+++	+	++	Not ascertained	"	4 "	7 "	" " "
	1.2 x 0.8	0	0	0					
3	3.4 x 2.4	+	+++	++	<i>E. coli</i> , sensitive to tetracycline, etc.	"	10 "	None required	Complete healing with oral therapy
	Healed	0	0	0					
4	2.4 x 2.0	+++	+++	+++	1. <i>Staph. aur.</i> , sensitive to tetracycline and chloramphenicol only 2. <i>Proteus</i> , insensitive to all	1. Tetracycline and then 2. Chloramphenicol (4 days)	4 "	3 months—still unhealed	No benefit from any treatment. Unable to tolerate compression either before or after oral therapy
	2.5 x 2.0	+++	+++	+++					
5	1.0 x 1.0	+++	++	+++	<i>Staph. aur.</i> and <i>Str. haem.</i> , sensitive to tetracycline	Tetracycline	4 "	10 weeks	Uncomplicated progress to complete healing
	1.0 x 1.0 (scabbed)	0	0	0					
6	2.5 x 1.2	+++	++	++	<i>Str. haem.</i> sensitive to penicillin + + +, and tetracycline +	1. Tetracycline and then 2. Penicillin V (5 days)	3 "	4 "	Unable to tolerate compression prior to oral therapy. After this compression at first painful, but tolerable
	2.7 x 0.8	+++	+	0					
7	R. 2.5 x 1.5 L. 1.7 x 2.0	R. L. + + + +	R. L. + +	R. L. + + + +	<i>Ps. pyocyanea</i> and <i>Proteus</i> , insensitive to all tested	Tetracycline	2 "	R. 12 weeks L. 10 "	No specific benefit from oral therapy
	2.5 x 1.2 1.8 x 2.0	+ +	0 0	+ +					
8	R. 0.9 x 0.6 L. 1.3 x 1.0	R. L. + +	R. L. + +	R. L. + + + +	<i>Staph. aur.</i> and <i>E. coli</i> , sensitive to tetracycline, etc.	"	3 "	5 "	Uncomplicated progress
	1.4 x 1.0 1.7 x 1.5	0 0	0 0	0 0					
9	6.5 x 5.5	++	++	++	<i>Staph. aur.</i> and <i>Str. haem.</i> , sensitive to tetracycline, etc.	"	4 "	10 weeks—still unhealed	Only slow healing with compression
	6.0 x 5.5 (scabbed)	0	0	0					
10	Extensive—encircling in places > 20 cm. width	++	+++	+++	Not ascertained	Tetracycline (2 courses)	5 "	7 weeks	Remarkable initial improvement with oral therapy maintained with compression to complete healing
	12 x 4.5	0	+	0					
11	2 ulcers (1) 1.7 x 1.7 (2) 1.2 x 1.0	++	++	++	"	Tetracycline	2 "	12 "	Compression prior to oral therapy very painful, but much more comfortably tolerated after oral therapy
	1.4 x 1.2 1.0 x 0.5	0	0	0					
12	3 ulcers 8.0 x 6.4; 3.4 x 2.8; 5.3 x 2.8	+	++	+++	<i>Staph. aur.</i> , <i>E. coli</i> , and <i>Str. haem.</i> —all sensitive to tetracycline, etc.	"	4 "	All healed in 6 weeks	Uncomplicated progress to complete healing
	3.7 x 1.7; healed; 5.0 x 1.0 (scabbed)	0	0	0					
13	2.5 x 1.6	+	++	++	Not ascertained	"	3 "	8 weeks—nearly healed Then broke down again	Compression more effective after oral therapy than before it
	2.0 x 1.6	0	0	0					
14	3.0 x 2.5	+	++	+++	1. <i>B. coli</i> , <i>Staph. aur.</i> , and <i>Str. haem.</i> —all sensitive to tetracycline 2. <i>Ps. pyocyanea</i> —sensitive to chloramphenicol only	"	1 week	10 weeks—still unhealed	Worse after than before oral therapy. Only slow progress with compression. Oedema persists minimally
	5.0 x 4.0	+	++	++					
15	3.0 x 2.3	+	+	+++	<i>Staph. aur.</i> and <i>Str. haem.</i> —sensitive to tetracycline, etc.	"	5 weeks	None required	Complete healing with oral therapy
	Healed	0	0	0					

has to remove it. Finally, it is time-consuming: this is a great deterrent to its wide use in general practice, in which the treatment of leg ulcers can be so interesting and rewarding.

It was with these disadvantages in mind that I have attempted to formulate an alternative therapeutic regime, based upon the principle that reduction or elimination of the oedema and control of the infection constitute the prime requisite of healing a venous leg ulcer. It was therefore decided to attempt to do this by means of oral therapy alone, using a combination of diuretics and antibiotics in those cases in which infection and oedema were predominant. The following is a preliminary report of this work in a series of 15 patients seen both at a hospital clinic and in my general practice.

CASE SELECTION AND METHODS

Cases were regarded as suitable to be included in the series where evidence of infection in the ulcer was visible and where leg oedema was marked, irrespective of whether this was unilateral or bilateral. Initially, a swab was taken from the ulcer and sent to the laboratory for bacterial culture and antibiotic sensitivity tests. In four cases, however, this was either unobtainable or inadvertently omitted. Treatment was then begun with an oral diuretic in suitable dosage, and a broad-spectrum antibiotic—in each case a five-day course of tetracycline being given in a dosage of 1 g. per day. At this stage local treatment to the ulcer consisted entirely of gauze smeared with ung. paraffin molle *B.P.C.*, to prevent its adherence to the ulcer, and covered with an elastocrepe bandage applied from the foot to just below the knee. This was changed daily by the patient.

One week later the patient was seen again and reassessed, with special regard to the swab report. If any bacteria present were insensitive to tetracycline a further course of the correct antibiotic was given. In the event of visible infection persisting or recurring after cessation of the antibiotic, a fresh swab was taken for re-examination, and the results were recorded. In these cases a further course of antibiotics was not thought justifiable. Diuretics and local therapy were prescribed as above until it became evident either that progress was no longer occurring or that treatment was proving ineffective. Further treatment was then continued with compression bandaging as detailed previously, diuretics being given where necessary, and supplemented with potassium if used for periods in excess of a fortnight.

RESULTS

The results are shown in the accompanying Tables. In Table I the distinctive features of the ulcer—namely, size, infection, amount of oedema, and pain—both immediately before and after oral therapy, are placed in the same columns as each other. The amount of pain, infection, and oedema is graded from "0" to "+++", according to severity. Table II shows the age and sex of each patient, and includes additional details, such as the history and duration of the ulcer, and any relevant associated features.

There were three outright failures (Cases 4, 7, 14), including the only male patients in the series, in both of whom diuretics had no beneficial effect upon the oedema, which was unilateral in each case. In Case 4 the oedema was a direct result of a deep venous thrombosis which preceded the appearance of the ulcer

by one week. In each of the three patients the offending organisms were either *Ps. pyocyanea* or *Proteus vulgaris*, either initially or eventually insensitive to every antibiotic whose use could be considered justifiable.

In Case 8 the ulcers became larger during oral therapy, but the other features assessed indicated improvement.

In the remaining 11 patients definitive improvement was evident with oral therapy alone, and in two (Cases 3 and 15) complete healing of the ulcer occurred without the subsequent use of additional local therapy. Two patients (Cases 6 and 11) who were previously unable to tolerate compression owing to intense pain were able to do so after oral treatment. One patient (Case 10) showed a particularly dramatic improvement, with conversion from a grossly oedematous leg, with a malodorous discharge from an immeasurably large ulcer, to a much smaller and relatively dry ulcer within three weeks.

SUMMARY AND CONCLUSIONS

A new approach has been made to the treatment of venous ulcers of the leg, initially by means of oral

TABLE II

Case No.	Age and Sex	Duration of Present Ulcer(s)	Response to Treatment (if any) Prior to Oral Therapy	Special Features in History with Associated Complications
1	57 F	6 weeks	Nil to local applications	History of trauma prior to onset of ulcer. First episode of ulceration. Varicose veins. Psoriasis
2	64 F	4 months	Deterioration with local applications	History of recurrent leg ulcers. Varicose veins
3	67 F	2-3 weeks	No previous therapy	Recurrent "superficial and deep thrombophlebitis in both legs. Present ulcer occurred 1 week after deep venous thrombosis. Full investigations have so far not revealed any latent carcinoma
4	62 M	2 months	Poor to bed rest with leg raised, anti-coagulants, and local applications	Trauma to leg prior to onset of ulcer. Varicose veins. First episode of ulceration. Mild ischaemia of both lower limbs. Trauma to leg prior to onset of ulcer. First episode of ulceration
5	68 F	2 weeks	No previous therapy	History of recurrent leg ulcers. Varicose veins
6	73 F	6 weeks	Attempts at compression too painful. No response to local applications	History of recurrent leg ulcers. Varicose veins
7	63 F	R.—7 months L.—3 weeks	Up-and-down progress with compression to R. leg. No previous therapy to L. leg	History of recurrent leg ulcers. Varicose veins
8	66 F	1 week (both)	No previous therapy	History of recurrent leg ulcers. Varicose veins. Simultaneous onset in both legs. No trauma
9	59 F	3 weeks	" " "	First episode of ulceration. Varicose veins
10	65 F	9 months	Up-and-down progress with compression. No treatment for one month resulting in marked deterioration	Very obese lady with history of recurrent leg ulcers. Old deep venous thrombosis. Superadded cardiac oedema
11	58 F	4 months (both)	*Minimal benefit from compression	Varicose veins
12	68 F	2 months (all 3 ulcers)	No previous therapy	History of recurrent leg ulcers. Varicose veins
13	77 F	Over 2 years	Up-and-down progress with compression	History of recurrent leg ulcers. Never completely healed during 2 years' continuous therapy. Subject to repeated trauma to legs
14	59 M	3 days	No previous therapy	History of recurrent leg ulcers. Varicose veins
15	72 F	4 months	Initial slow response to local applications. Nil for 6 weeks prior to oral therapy	History of recurrent leg ulcers. Varicose veins. Present ulcer subsequent to trauma. Sensitization eczema developed due to local applications: this aggravated ulcer

therapy alone, using a combination of a diuretic and a suitable antibiotic. The results of a preliminary trial upon 15 patients have been recorded, and in 11 of these satisfactory progress was obtained. No side-effects have resulted from treatment.

For reasons outlined, the regime is thought to overcome some of the problems of the more recognized forms of treatment, and is a particularly useful means by which the busy general practitioner, who is usually without ancillary aid, can be stimulated to become interested in the treatment of leg ulcers.

Though, fortuitously perhaps, no further treatment was required in two patients, the regime is to be regarded more as a smooth induction to subsequent "traditional" therapy than as an alternative.

I would like to thank Dr. Napier Thorne, without whose advice, help, and encouragement this paper would not have materialized.

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REFERENCES

- Anning, S. T. (1954). *Leg Ulcers and Their Treatment*. Churchill, London.
Lorch, Diana (1961). *Practitioner*, 187, 67-73.
Thorne, Napier A (1958). "Practical Politics in the Treatment of Leg Ulcers, *Nursing Times*.

Medical Memoranda

Post-operative Blindness, with Complete Recovery, in a Patient with Sick-cell Anaemia

Among the various neurological complications in patients with sickle-cell anaemia transient blindness has been rarely reported (Hughes *et al.*, 1940; Margolies, 1951; Greer and Schotland, 1962). The following is a history of a girl with sickle-cell anaemia who post-operatively developed severe neurological symptoms including blindness.

CASE REPORT

An 11-year-old Nigerian girl with known sickle-cell anaemia (SS) was admitted to our wards for repair of a large inguinal hernia. She had been generally well, complaining only of transient vague pains of undetermined cause in her upper and lower limbs. History revealed that she had never had any typical crises. On admission she was a tall thin negro girl in no distress. She was not jaundiced, her blood-pressure was 100/60 mm. Hg, and auscultation revealed a soft apical systolic murmur. X-ray examination of the chest showed moderate cardiac enlargement and clear lung fields. An electrocardiogram was within normal limits. Neurological examination revealed no abnormality. Her haemoglobin was 8 g./100 ml. It was 9.8 g./100 ml. one month before the operation. The operation was performed under general anaesthesia, which was induced with thiopentone sodium ("pentothal") and suxamethonium chloride ("scoline"). She was intubated and anaesthesia was maintained with a mixture of nitrous oxide, oxygen, and halothane. Gallamine triethiodide, atropine, and neostigmine were also given. The operation lasted about 30 minutes and was uneventful, except that the incisional oozing was less than normal for the first three or four minutes. She regained consciousness after one hour and then slept for 12 hours.

On awakening she complained that she could not see. She was jaundiced. Her consciousness alternated between

being orientated and able to respond to simple questions, and refusing to respond or responding inappropriately. She could not see objects or distinguish light from darkness. Both papillary reflexes were sluggish. Nystagmus was not present. The fundi were normal except for a slight tremor of the disk. Our neurological consultant, who noted this, considered the phenomenon to be strongly in favour of organic disease and against hysteria—an alternative we were considering at that point. Her tendon reflexes were equal and active and both plantar responses were flexor. She was generally weak but had no paralysis. Her haemoglobin was 8.2 g./100 ml. and the total bilirubin was 9.7 mg./100 ml. Her blood-pressure was 100/60 mm. Hg.

The clinical picture suggested a possible occlusion of cerebral vessels with aggregated sickle cells. In order to lessen the likelihood of further occlusions, she was given intranasal oxygen and also a 50% solution of magnesium sulphate intravenously six-hourly for three days (H. Lehmann, personal communication). On the second post-operative day she developed a right pleural effusion. A culture of the effusion was sterile. Lumbar puncture was not performed, as it was thought that the procedure might aggravate the neurological condition during the acute stage. On the fourth post-operative day she began to recognize objects placed near her. At the same time her ankle clonus became sustained, but both plantar responses remained flexor. Weakness persisted in all limbs; this was more pronounced in the arms. By the fifth post-operative day her haemoglobin had dropped to 6.5 g./100 ml. She was then transfused with 360 ml. of packed red cells.

Her vision steadily improved and no visual abnormality could be detected four weeks after operation. Concomitantly her mental state returned to normal, the ankle clonus disappeared, and her muscular tone improved. She was discharged five weeks after operation in a satisfactory physical and mental state. The only remaining clinical sign was a slight incoordination of fine movements of the upper limbs. The haemoglobin was 10 g./100 ml.

COMMENT

The neurological manifestations were probably due to cerebral intravascular sickling induced by low oxygen tension during anaesthesia. The transitory nature of the various symptoms and signs as occurred in this case is characteristic of patients with sickle-cell anaemia (Margolies, 1951). The apparent full recovery from the neurological disturbances seen in these patients can be explained on the basis of an initial occlusion of a vessel, whether by thrombus or sludge formation (some authors also maintain that the vessels go into spasm), with resultant impairment of the oxygen supply to a part of the brain, leading to neurological shock. The degree of recovery depends on the availability of collateral supply to the area involved and the reopening of the occluded vessel.

In conclusion the following points should be considered before operating on a patient with sickle-cell anaemia. (1) Elective procedures should be avoided if possible. (2) The choice of anaesthetic should take into account the tendency for red-cell sickling with anoxia, and the hypotensive effect of certain anaesthetics.

We thank Mr. Michael Harmer and Dr. Thomas Stapleton for their help.

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REFERENCES

- Greer, M., and Schotland, D. (1962). *Neurology (Minneapolis)*, 12, 114.
Hughes, J. G., Diggs, L. W., and Gillespie, C. E. (1940). *J. Pediat.*, 17, 166.
Margolies, M. P. (1951). *Medicine (Baltimore)*, 30, 357.