THE POSTPHLEBITIC SYNDROME FOLLOWING SHAFT FRACTURES OF THE LEG

A SIGNIFICANT LATE COMPLICATION

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Sixty patients each of whom had a fracture of the lower limb a minimum of five years (median 11 years) previously were studied by photoplethysmography, foot volumetry, popliteal venous reflux and arterial Doppler measurements. The non-fractured limb was used as a control. Postphlebotic symptoms were present in 51% and signs in 49% of fractured limbs compared with 4% and 24% respectively in the control limbs. The incidence was greater in patients who had fractured 15 years or more previously than in those who had fractured 5 to 15 years previously. Eleven limbs had clinically disabling postphlebotic symptoms including venous ulceration.

The postphlebotic syndrome following lower limb fractures in young patients is more common than generally appreciated and develops after a prolonged latent interval. A prospective randomised study using prophylactic anticoagulation for lower limb shaft fractures may be justified.

There is a high incidence of deep vein thrombosis following hip fracture and 5% or more of patients will subsequently suffer a fatal pulmonary embolism (Gruber 1985). The late incidence of the postphlebotic syndrome is unknown because many of these patients are elderly and die before such symptoms develop.

In contrast, tibial fractures, while also associated with a substantial incidence of deep vein thrombosis (Hjelmstedt and Bergvall 1968a), affect predominantly young adults who are likely to live long enough for the fully developed postphlebotic syndrome to become manifest. In two studies, performed three years (Hjelmstedt and Bergvall 1968b) and nine years (Lindhagen et al. 1985) after tibial fractures, no discernible difference in the incidence of the postphlebotic syndrome could be shown between the fractured and non-fractured limb. However, a study performed 13 to 17 years after tibial fractures (Willén, Bergqvist and Hallböök 1982) found

15 of 38 patients (39%) to have clinical evidence of venous insufficiency, though in these patients there was no assessment of the normal limb. The late development of a postphlebotic limb 13 years or more after a fracture is in keeping with the long natural history of chronic venous hypertension (Negus 1970). We are not aware of similar studies after femoral shaft fractures.

If the incidence of chronic venous hypertension following shaft fractures can be shown to be substantial, it would have important therapeutic and medico-legal implications as regards possible anticoagulant prophylaxis at the time of fracture. The aim of this study was to establish the incidence of the postphlebotic syndrome in shaft fractures of the lower limb five years or more after the fracture.

PATIENTS AND METHODS

The hospital records of all patients who had attended the hospital with a lower limb shaft fracture were recalled. Patients who were aged 14 years or more at the time of fracture and who had fractured the leg at least five years previously were invited to attend the vascular laboratory for non-invasive assessment of lower limb venous function. Additional subjects were recruited by notices within the hospital. Details recorded on a prepared pro forma included the nature of the injury, its management, and a full history and examination pertaining to soft tissue and bony deformities of the leg. As far as possible,

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Table I. Distribution of fractured bones in 60 patients

Bone fractured	Unilateral	Bilateral
Femur only	17	1
Tibia only or tibia and fibula	5	1
Femur, tibia and fibula	33	3
Total	55	5

Table II. Frequency of symptoms in the injured limbs

Aches and pains	16
Ankle swelling	15
New varicose veins	6
New venous ulcer	6
Venous claudication	—
Deep vein thrombosis or pulmonary embolism since the fracture	3

Table III. Frequency of signs in the injured limb as compared with the controls

	Fractures (65)		Controls (55)	
	No.	%	No.	%
Ankle oedema	8	12.3	0	
Varicose veins	6	9.2	1	1.8
Ankle flare/liposclerosis	7	10.7	0	
Venous ulcer	6	9.2	0	
Swollen limb (1.5 cm larger in girth)	9	13.8	7*	12.7
Popliteal venous reflux	13	20.0	3	5.4
Deep vein thrombosis in elapsed time	4	6.1	0	
Severe clinical symptoms	11	16.9	0	

* In these patients the control limb was larger than the fractured limb because of muscle wasting or tissue loss in the fractured limb

details were confirmed by reference to the original hospital records and radiographs. All patients were assessed by photoplethysmography (Barnes and Yao 1982), popliteal venous reflux, foot volumetry (Amoore et al. 1983) and arterial Doppler pressures. Patients with abnormal venous function were invited to undergo bilateral ascending venography using hypo-osmolar contrast media. The patient's non-fractured limb was used as a control.

This study was approved by the University of Cape Town Medical School Ethical Trials Committee, and patients undergoing venography gave written informed consent.

Statistical analysis was by Student's *t*-test.

RESULTS

Sixty patients were studied, of whom five had had bilateral fractures (Table I). The majority (47) were males who were riding a motor cycle (21) or were struck by a car (19). The median age at the time of fracture was 25 years (range 14 to 66 years) and the median time since the fracture was 11 years (range 5 to 55 years). There were 24 compound fractures.

Symptoms and signs are detailed in Tables II and III. Intermittent or trivial symptoms were ignored in the clinical assessment. When the circumference of the non-fractured limb was more than 1.5 cm greater than that of the fractured limb it was assumed that muscle wasting or tissue loss in the fractured limb accounted for the difference. Residual bone deformity was found in 15 patients and tendon shortening in four.

Photoplethysmography. The mean recovery time (and standard deviation) for all the fractured limbs was significantly shorter than that in the controls (25.3 ± 14.4 s v. 36.4 ± 16.4 s; $p < 0.001$). Twenty-one fractured limbs (32%) and seven non-fractured limbs (12.7%) had a recovery time of 17 seconds or less ($p < 0.05$). The half-recovery time (Willén et al. 1982) also was significantly shorter in the fractured limb (9.7 ± 4.7 s v. 13.6 ± 8.18 s; $p < 0.001$). If the individual fractured bones (rather than the fractured limbs) are considered separately this difference was significant only for tibial fractures (recovery time 25.0 ± 14.9 s v. 36.6 ± 15.7 s, $p < 0.001$; half-recovery time 9.3 ± 4.8 s v. 15.0 ± 8.9 s, $p < 0.001$).

Twenty-one fractured limbs were assessed 15 years or more after the fracture and had a significantly shorter

photoplethysmography recovery time than the uninjured control limbs (23.2 ± 12 s v. 38.0 ± 13.2 s; $p < 0.001$) but assessment of individual bones confirmed that this difference was limited to tibial fractures only (22.81 ± 14 s v. 38.3 ± 13.0 s; $p < 0.01$). Limbs fractured 5 to 15 years before assessment also had a shorter recovery time, but this was less pronounced (27.2 ± 17.0 s v. 34.8 ± 17.3 s; $p < 0.01$). When tibial fractures occurring 5 to 15 years previously were considered separately they also had a shorter recovery time than their non-fractured control, but this did not reach statistical significance (26.9 ± 17.6 s v. 35.4 ± 17.5 s).

Popliteal venous reflux was found in 13 fractured limbs (20%) and three non-fractured limbs (5%) (Table III). The *foot volumetry* half-recovery time was 21 ± 13 s and 27 ± 12.7 s in the fractured and non-fractured limbs respectively; this difference was not significant. The mean volume of the fractured foot was 23 ml more than that in the non-fractured foot. Arterial *Doppler studies* were normal in all patients except one who had peripheral vascular disease.

Postphlebotic symptoms. There were 10 patients (11 limbs) with clinically disabling postphlebotic symptoms; six of these patients had developed a venous ulcer. Three of the 10 patients were aged 60 years or more at the time of assessment; only two were aged 50 years or more at the time of fracture. The patient's age at the time of fracture, prolonged immobilisation, external splintage, whether or not the fracture was compound and the severity of the trauma did not appear to predispose to the development of a postphlebotic limb.

Four patients with clinically significant postphlebotic limbs and evidence of chronic venous hypertension agreed to undergo bilateral ascending venography; in three of these the tibia had been fractured and in one the femur. The venograms showed evidence of previous venous thrombosis in the calf with multiple collaterals. However, in no patient was there evidence that the thrombosis had ever extended beyond the popliteal vein and the anatomy of the superficial and common femoral veins appeared normal.

DISCUSSION

The relationship of deep venous thrombosis to chronic venous insufficiency and the subsequent development of a postphlebotic limb is well recognised (Bauer 1942) but normally 5 to 10 years is required before significant clinical features (sometimes including venous ulceration) develop (Negus 1970; Linton 1953; Owens 1978). In our study we have demonstrated that patients who fractured a leg at least 15 years prior to assessment had a significantly greater incidence of venous insufficiency than in the non-fractured limb; this was confirmed by finding that 6 of 10 patients with clinically disabling postphlebotic symptoms had fractured the limb at least

15 years before our assessment. These findings assume importance because the majority of our patients were young at the time of fracture with a life expectancy in excess of 50 years. It is likely that a further proportion of those with an abnormal photoplethysmogram and venous reflux will develop clinical manifestations of a postphlebotic limb with the passage of time.

The incidence of deep vein thrombosis following tibial fractures has been well-documented (Hjelmstedt and Bergvall 1968a; Lindhagen et al. 1985; Willén et al. 1982) and at least two of these studies have concluded that the high incidence merits anticoagulation. However, few centres routinely employ prophylaxis, presumably because the incidence of fatal pulmonary embolism is low. The postphlebotic limb is a largely incurable condition and causes considerable emotional, social and financial hardship. The studies referred to have revealed an increasing incidence of postphlebotic limbs with the passage of time: 13%, 35% and 39% at 3, 9 and 14 years respectively. This increase (in keeping with the long natural history of the development of the postphlebotic syndrome) is confirmed in our study which assesses venous physiology rather than relying predominantly on clinical and venographic criteria as in previous studies.

Published studies have largely concentrated on patients with tibial fractures and have not considered the sequelae of isolated femoral shaft fractures. Although we were not able to demonstrate a significant incidence of associated postphlebotic syndrome after femoral shaft fractures in our study, this may be because of small numbers.

Recent emphasis in venous thrombosis research has concentrated on prophylactic regimens capable of reducing the incidence of both thrombosis and fatal pulmonary embolism. Low-dose subcutaneous heparin is now widely used in general surgery (Kakkar 1978), but appears to be less effective in elective and emergency orthopaedic procedures in the lower limb (Bergqvist et al. 1979). Prophylactic regimes which have shown the greatest promise in prospective, randomised studies in orthopaedic surgery include dextran (Bergqvist et al. 1979), heparin plus dihydroergotamine (Kakkar et al. 1979), adjusted doses of heparin (Leyvraz et al. 1983) and warfarin (Sevitt and Gallagher 1959; Francis et al. 1983).

Our findings may have some medico-legal importance. In cases where compensation is payable, successful claims are normally concluded in "full and final assessment", which precludes legal redress should a postphlebotic limb develop later. We advise patients to be cautious and to ensure that settlement does not prejudice revision of their claim should a postphlebotic limb develop.

Finally, since the incidence of the postphlebotic limb following tibial fractures in young adults is clearly substantial, we believe that a prospective, randomised study employing anticoagulant prophylaxis is both justified and needed.

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REFERENCES

- Amoore JN, van Oudenhove LB, Immelman E, Jeffery P. The assessment of the post-thrombotic limb by foot volumetry and pedal venous pressure. *J Med Eng Technol* 1983;7:180-5.
- Barnes RW, Yao JST. Photoplethysmography in chronic venous insufficiency. In: Bernstein EF, Ed. *Non-invasive diagnostic techniques in vascular disease*. St Louis: CV Mosby, 1982:514-21.
- Bauer G. A roentgenological and clinical study of the sequels of thrombosis. *Acta Chir Scand* 1942;Suppl 74:1-116.
- Bergqvist D, Efsing HO, Hallböök T, Hedlund T. Thromboembolism after elective and post-traumatic hip surgery: a controlled prophylactic trial with Dextran 70 and low-dose heparin. *Acta Chir Scand* 1979;145:213-8.
- Francis CW, Marder VJ, Evarts CM, Youkoolbodi S. Two-step warfarin therapy: prevention of postoperative venous thrombosis without excessive bleeding. *JAMA* 1983;249:374-8, 2021 (correction).
- Gruber UF. Prevention of fatal pulmonary embolism in patients with fractures of the neck of the femur. *Surg Gynecol Obstet* 1985;161:37-42.
- Hjelmstedt Å, Bergvall U. Incidence of thrombosis in patients with tibial fractures: a phlebographic study. *Acta Chir Scand* 1968a;134:209-18.
- Hjelmstedt Å, Bergvall U. Phlebographic study of the incidence of thrombosis in the injured and uninjured limb in 55 cases of tibial fracture. *Acta Chir Scand* 1968b;134:229-34.
- Kakkar VV. The current status of low-dose heparin in the prophylaxis of thrombophlebitis and pulmonary embolism. *World J Surg* 1978;2:3-18.
- Kakkar VV, Stamatakis JD, Bentley PG, Lawrence D, de Haas HA, Ward VP. Prophylaxis for postoperative deep vein thrombosis: synergistic effect of heparin and dihydroergotamine. *JAMA* 1979;241:39-42.
- Leyvraz PF, Richard J, Bachmann F, et al. Adjusted versus fixed-dose subcutaneous heparin in the prevention of deep-vein thrombosis after total hip replacement. *N Engl J Med* 1983;309:954-8.
- Lindhagen A, Bergqvist D, Hallböök T, Berndtsson L. Venous function after fracture of the lower extremity: a 9 year follow-up of 150 patients. *Acta Orthop Scand* 1985;56:110-4.
- Linton RR. The post-thrombotic ulceration of the lower extremity: its etiology and surgical treatment. *Ann Surg* 1953;138:415-32.
- Negus D. The post-thrombotic syndrome. Arris and Gale Lecture on 28th May 1970. *Ann R Coll Surg Engl* 1970;47:92-105.
- Owens CJ. The postphlebotic syndrome: management by construction means. In: Bergen J, Yao JST, eds. *Venous problems*. Chicago: Year Book Medical Publishers, 1978.
- Sevitt S, Gallagher NG. Prevention of venous thrombosis and pulmonary embolism in injured patients: a trial of anticoagulant prophylaxis with phenindione in middle-aged and elderly patients with fractured necks of femur. *Lancet* 1959;ii:981-9.
- Willén J, Bergqvist D, Hallböök T. Venous insufficiency as a late complication after tibial fracture. *Acta Orthop Scand* 1982;53:149-53.