Editorial/Van die Redaksie

Thrombotic thrombocytic purpura — plasma infusion or exchange?

Moschcowitz[1] decribed a rare multisystem disorder, characterised by fever, fluctuating central neurological abnormalities, progressive renal failure, micro-angiopathic haemolytic anaemia and thrombocytopenia. This entity has become known as thrombotic thrombocytopenic purpura and usually occurs without any particular causal event or associated disease process, but may follow influenza vaccinations, the use of oral contraceptives, viral illnesses, legionnaires' disease and, of increasing importance, infections with *Campylobacter* or the human immunodeficiency virus.[2,3] Similar fragmentation syndromes are associated with carcinoma and the administration of a number of drugs, such as mitomycin and cyclosporin A. In addition, there may be associated immune thrombocytopenia and related diseases, exemplified by systemic lupus erythematosus.[4]

Given the varying clinical severity, from a fulminant and rapidly fatal course to a mild or even a relapsing pattern in a disease of unknown cause, uncertain pathogenesis and disputed definition,[5] where treatment is not standardised and plasma exchange is enthusiastically advocated, doctors can be forgiven for what has become the indiscriminate use of this latter procedure. However, considerable perspective and restraint are needed before embarking on an expensive option[6] that carries both morbidity and mortality.[7] A more conservative approach, restricting plasmapheresis to patients where increments in fibrinogen could not be sustained by simple infusion (J.P. Hester — unpublished observation) has been advocated. In view of these contrasting points of view, recommendations for treatment are clearly in need of critical review.

Starting with a high index of clinical suspicion, the first step in management is to confirm the diagnosis using laboratory measurements. These centre on the demonstration of red cell fragmentation that frequently leads to anaemia, morphological changes in platelets and striking increases in lactate dehydrogenase.[8] Thrombocytopenia is prominent due to accelerated intravascular platelet consumption,[9] and bone marrow aspiration and trephine biopsy reveal abundant megakaryocytes with characteristic hyaline lesions in the blood vessels.[10]

Therapy should logically take into account pathogenesis and to this end changes in the circulating blood, amplified to include disturbances in haemostasis,[11] were combined to delineate those clinicopathological features[12] that might provide a better understanding of some final common pathway. Thus, thrombotic thrombocytopenic purpura, although of diverse aetiology, appears explicable on two major, but not necessarily mutually exclusive, primary lesions, both leading to formation of a platelet plug.[13] On the one hand the initial event was considered to be intravascular platelet agglutination, and on the other hand auto-immune vascular injury was incriminated. This issue is by no means settled and evidence in favour of both possibilities, with abnormal levels of plasminogen activator, its inhibitor and protein C, have been reported.[14] Furthermore, molecular defects in the von Willebrand factor[15] and changes in cysteine protease activity, leading to alterations in proteolysis as well as fibrinogenolysis, have to be considered in any scheme that should also involve the presence of lytic antibodies directed against endothelial cells.[16]

These facts and the observation that plasma infusion could ameliorate the course of the disease more consistently than the previous use of cytotoxic drugs and antiplatelet agents,[17] whereas platelet transfusions are potentially dangerous,[18] raise the question whether available evidence justifies the current enthusiasm with which plasma exchange as opposed to its infusion is advocated. Unfortunately, recommendations for therapy in thrombotic thrombocytopenic purpura have produced a morass of anecdotal literature that has attracted the criticism it rightly deserves. Thus, effectiveness has been claimed for individual agents that range from those that act by unknown mechanisms, such as vincristine, to others that favour vasodilatation and platelet disaggregation, exemplified by prostacyclin, in contrast to aspirin and dipyridamole,[19] which impair the integrity of platelet function. These approaches were challenged by the signal observation that plasma infusion was a particularly effective form of treatment,[20] but subsequent publications were difficult to interpret because patient numbers were small or there was the concurrent and confounding use of other modalities.[21] To further confuse the unwary physician, a recommendation for plasma exchange as the primary mode of therapy was made on the basis of a single patient.[22] While such a procedure could remove immune complexes and so prove beneficial,[23] the counter-suggestion was made that it might more reasonably be reserved for those situations where fluid overload restricts the amount of plasma that can be infused.[24] The latter argument is specious and might even be regarded as an indictment of the care with which fluid balance is managed. In all but the most unusual circumstances, careful planning and the judicious use of diuretics combined with central venous monitoring should avoid the iatrogenic complication.

Unfortunately, the perspective needed for this approach to therapy from controlled clinical trials is lacking; the only such available study[25] is flawed because the regimen itself was complex and included a variety of drugs known to affect platelet function. As is frequently the case, uncritical reading of the literature has led to the statement that *plasma exchange* is the treatment of choice, whereas the authors themselves emphasise that their trial was too small for statistical significance to have been achieved. Resolution of this dilemma will require data from a carefully co-ordinated and prospec-

tive comparison of the two approaches that will also examine different schedules, optimal volumes needed[26] and the confounding issue of high-dose intravenous gammaglobulin.[27]

Finally, there is the question of patients in whom treatment fails[28] and whether they form a distinct entity or a relapsing variant.[29] In this context, splenectomy[30] or additional dextran and steroids[31] are legitimate considerations.

What then might be an approach that is both safe and cost-effective?

In the first instance, reliable diagnosis is mandatory. Thereafter, patients could reasonably be started on 1 mg/kg/d of prednisone, employed for its anti-inflammatory effect on the vascular endothelium, combined with 2 mg vincristine intravenously once a week and infusion of fresh frozen plasma at a dose of 10 ml/kg intravenously every 6 hours. Response needs to be closely monitored and success will be reflected in the gradual fall of circulating red cell fragments, a rise in the haemoglobin level, a drop in lactate dehydrogenase and improvement of the thrombocytopenia over 48 - 72 hours. While it has been suggested that the latter may be a less sensitive index of benefit, this is not consistent with our experience. Impairment of the interaction between platelets and the vascular endothelium, even in the presence of moderate thrombocytopenia, could be achieved with low-dose acetylsalicylate in combination with dipyridamole or Nafazatrom, the latter being used to stimulate prostacyclin synthesis. Patients should be in the care of haematologists with particular expertise in the management of this disease, and the medical-nursing team must have a thorough knowledge of contemporary plasma exchange technology. Where the initial improvement is maintained for between 72 and 96 hours and once the platelet count and lactate dehydrogenase levels are again in the normal range, a combined 15 - 20% decrement in all therapeutic agents would be reasonable on a weekly basis as long as neither haematological nor biochemical deterioration occurs. It should be noted that striking increases in total plasma protein concentration and proteinuria,[32,33] together with marked hypercholesterolaemia, occur. Both of these phenomena reverse once the rate of plasma infusion decreases and neither appear to be associated with short-term or residual abnormalities.

Approached in this disciplined way, plasma exchange could only be justified in those unusual individuals where improvement in platelet count and lactate dehydrogenase levels cannot be achieved using the above regimen. In this context a supportive argument would be the development of significant fluid overload that cannot be corrected despite meticulous monitoring and balancing of intake and output, supplemented by loop-acting diuretics, but it is important that this procedure not be seen as a substitute for the proper control of replacement therapy. Similarly, an expanded plasma volume may be contributed to by continuing steroid administration, with the development of systemic hypertension, but here also escalation of diuretic therapy combined with titrated doses of calcium channel blockers or afterload-reducing agents, even in the face of moderate renal dysfunction, are generally effective and should not be seen as an argument for plasma exchange.

These various considerations support the suggestion that patients with thrombotic thrombocytopenic purpura should be referred, immediately on diagnosis, to experienced haematologists in order that they can be evaluated and entered into established and approved research protocols. Under these circumstances a standardised approach to management will eventually lead to the definition of a small subgroup of patients in whom plasma *exchange* as opposed to *infusion* is needed, thereby avoid bringing the costly but potentially useful former procedure into disrepute.

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1. Moschcowitz E. Hyaline thrombosis of the terminal arterials and capillaries: a hitherto undescribed disease. *Proc NY Pathol Soc* 1924; **24:** 21-24.
2. Leaf AN, Laubenstein LJ, Raphael B, Hochster H, Baez L, Karpatkin S. Thrombotic thrombocytopenic purpura associated with human immunodeficiency virus type 1 (HIV-1) infection. *Ann Intern Med* 1988; **109:** 194-197.
3. Nair IMG, Bellevue R, Bertoni M, Dosik H. Thrombotic thrombocytopenic purpura in patients with the acquired immunodeficiency syndrome (AIDS)-related complex. *Ann Intern Med* 1988; **109:** 209-212.
4. Neame PB. Immunologic and other factors in thrombotic thrombocytopenic purpura (TTP). *Semin Thromb Hemost* 1980; **6:** 416-429.
5. Herman J. Thrombotic thrombocytopenic purpura. *Ann Intern Med* 1982; **96:** 791.
6. Joneau M, Cordonnier C, Vernant JP, Touzer C, Sobel A. How many plasma exchanges to cure thrombotic thrombocytopenic purpura? *Scand J Haematol* 1985; **34:** 157-159.
7. Huestis DW. Leukapheresis and granulocyte transfusion. *Haematologia* 1982; **15:** 39-55.
8. Crowley JP, Metzger JB, L'Europa RA. The blood counts and lactate dehydrogenase levels in thrombotic thrombocytopenic purpura (TTP). *Am J Clin Pathol* 1983; **80:** 700-702.
9. Neame PB, Hirsh J, Browman G *et al.* Thrombotic thrombocytopenic purpura: a syndrome of intravascular platelet consumption. *Can Med Assoc J* 1976; **114:** 1108-1112.
10. Blecher TE, Raper AB. Early diagnosis of thrombotic microangiopathy by paraffin sections of aspirated bone-marrow. *Arch Dis Child* 1967; **42:** 158-162.
11. Nalbandian RM, Henry RL, Bick RL. Thrombotic thrombocytopenic purpura. *Semin Thromb Hemost* 1979; **5:** 216-240.
12. Kwaan HC. Clinicopathologic features of thrombotic thrombocytopenic purpura. *Semin Hematol* 1987; **24:** 71-81.
13. Lian EC-Y. Pathogenesis of thrombotic thrombocytopenic purpura. *Semin Hematol* 1987; **24:** 82-100.
14. Glas-Greenwalt P, Hall JM, Panke TW, Kant S, Allen CM, Pollak VE. Fibrinolysis in health and disease: abnormal levels of plasminogen activator, plasminogen activator inhibitor, and protein C in thrombotic thrombocytopenic purpura. *J Lab Clin Med* 1986; **108:** 415-422.
15. Murphy WG, Moore JC, Barr RD, Pai MKR, Kelton JG. Relationship between platelet aggregating factor and von Willebrand factor in thrombotic thrombocytopenic purpura. *Br J Haematol* 1987; **66:** 509-513.
16. Leung DYM, Moake JL, Havens PL, Kim M, Pober JS. Lytic anti-endothelial cell antibodies in haemolytic-uraemic syndrome. *Lancet* 1988; **2:** 183-186.
17. Byrnes JJ, Sholtis CM, White P. Plasma infusion in the treatment of thrombotic thrombocytopenic purpura, further experience. *Semin Thromb Hemost* 1981; **7:** 9-14.
18. Harkness DR, Byrness JJ, Lian EC-Y, Williams WD, Hensley GT. Hazard of platelet transfusion in thrombotic thrombocytopenic purpura. *JAMA* 1981; **246:** 1931-1933.
19. Mitnick PD. Aspirin, dipyridamole, and thrombotic thrombocytopenic purpura. *Ann Intern Med* 1982; **96:** 679-680.
20. Byrne JJ, Khurana M. Treatment of thrombotic thrombocytopenic purpura with plasma. *N Engl J Med* 1977; **297:** 1386-1389.
21. Rothberg H, Pachter I, Kosmin M, Barton-Stevens D. Thrombotic thrombocytopenic purpura: recovery after plasmapheresis, corticosteroids, splenectomy, and antiplatelet agents. *Am J Hematol* 1982; **12:** 281-287.
22. Ansell J, Beaser RS, Pechet L. Thrombotic thrombocytopenic purpura fails to respond to fresh frozen plasma infusion. *Ann Intern Med* 1978; **89:** 647-648.
23. Shepard KV, Bukowski RM. The treatment of thrombotic thrombocytopenic purpura with exchange transfusions, plasma infusions, and plasma exchange. *Semin Hematol* 1987; **24:** 178-193.
24. Byrnes JJ, Lian EC-Y. Recent therapeutic advances in thrombotic thrombocytopenic purpura. *Semin Thromb Hemost* 1979; **5:** 199-215.
25. Henon PR. Treatment of thrombotic thrombocytopenic purpura: first results of a controlled clinical trial. *Plasma Ther Transfus Technol* 1986; **7:** 101-106.
26. Newman RS, Ocariz JA. Optimal volume of plasma exchange in thrombotic thrombocytopenic purpura. *Transfusion* 1987; **27:** 445.
27. Staszewski H, Colbourn D, Donovan V, Ludman H. Thrombotic thrombocytopenic purpura: report of a case with a possible response to high-dose intravenous gamma globulin. *Acta Haematol* 1989; **82:** 201-204.
28. Liu ET, Linker CA, Shuman MA. Management of treatment failures in thrombotic thrombocytopenic purpura. *Am J Hematol* 1986; **23:** 347-361.
29. Aviles A, Romero N, Murillo E. High incidence of relapse in thrombotic thrombocytopenic purpura. *Am J Med* 1988; **84:** 983-984.
30. Sturgess AD, Chong BH. Thrombotic thrombocytopenic purpura unresponsive to plasma infusion and plasma exchange, but responsive to splenectomy. *Scand J Haematol* 1986; **37:** 319-322.
31. Cuttner J. Splenectomy, steroids, and dextran 70 in thrombotic thrombocytopenic purpura. *JAMA* 1984; **227:** 397-402.
32. Weening JJ, van Guldener C, Daha MR, Klar N, van der Wal A, Prins FA. The pathophysiology of protein-overload proteinuria. *Am J Pathol* 1987; **129:** 64-73.
33. Waller KV, Ward KM, Mahan JD, Wismatt DK. Current concepts in proteinuria. *Clin Chem* 1989; **35:** 755-765.

Recent advances in intractable pain control

Intractable pain is a frequent cause of disability, and constitutes a major health and economic problem world-wide. Even more important is the cost in human suffering. However, a better understanding of opioid kinetics and the development of new routes of administration have revolutionised opioid analgesia in intractable pain. Opioids can now be given transdermally (fentanyl patch),[1,2] buccally (morphine), sublingually (buprenorphine), transnasally (sufentanil, butorphanol),[3] epidurally, intrathecally, and into the cerebral ventricles. The introduction of sustained-release morphine formulations (such as MST), has improved patient compliance. New drugs (ondansetron) that block S_3 receptors for 5-hydroxytryptamine can control drug-induced nausea and vomiting.[4] Patients with severe pain not due to malignancy, whose quality of life has been greatly reduced and for whom no other therapy has been successful, are now being treated by long-term opioid therapy,[5] provided that a firm diagnosis has been made, that there are no psychological contraindications, and that the pain is opioid-sensitive. The combination of long-acting local anaesthetics and opioids, given either as intermittent boluses or as constant infusions into the epidural and intrathecal spaces (in acute pain), still needs to be evaluated. Opioid antagonists are used for treating intractable pain of central origin (post-stroke).[5] High-dose intravenous naloxone, followed by oral naltrexone or nalmefene for maintenance, is used.

For patients on long-term non-steroidal anti-inflammatory drugs, misoprostol, a synthetic prostaglandin E_1 analogue, has been developed. This inhibits gastric acid secretion, and markedly decreases gastric ulceration in these patients.[6] Two more recently introduced non-steroidal anti-inflammatory drugs (etodolac and sulindac) have shown no marked effects on the renal prostaglandin enzyme systems.[5]

Secondary analgesics are being successfully and increasingly used in intractable pain, and now have a wide application in its management. These drugs include psychotropics (antidepressants, anticonvulsants, anxiolytics), hormones (corticosteroids, calcitonin), muscle relaxants (baclofen, dantrolene), catecholamine antagonists (clonidine, bretylium), catecholamine precursors (L-tryptophan, L-dopa), cytotoxics, calcium channel blockers (diltiazem, nifedipine, felodipine), and enkephalinase inhibitors (*d*-phenylalanine).[7] Recently introduced selective 5-hydroxytryptamine blockers (fluoxetine, fluoxamine) are less potent secondary analgesics than those antidepressants which exert their effects on a wider spectrum of catecholamine neurotransmitters.[5] The α_2-agonists (clonidine) are used epidurally and intrathecally to produce analgesia.[8,9] Calcitonin, L-dopa, mithramycin, and the diphosphonates offer analgesia in disseminated bone metastases.[10] Calcitonin has been used intravenously for early phantom limb pain,[11] and as a long-term intrathecal infusion to manage chronic, intractable, non-malignant pain.[12] For the pain of sympathetic dystrophy, bretylium is used in the isolated limb and opioids (sufentanil) can be injected over the sympathetic ganglia.[13]

Ketanserin (5-hydroxytryptamine-2 antagonist) is used in the treatment of algodystrophy and in Raynaud's disease.[5] Intrathecal baclofen and the opioids (buprenorphine) can be used to treat paraplegic pain and spasm.

Aminoglycosides (streptomycin) injected locally have recently been successfully used to treat trigeminal neuralgia, intercostal neuralgia, and scar pain.[14] A suspension of acetylsalicylic acid and ethyl ether provides a new topical treatment in post-herpetic neuralgia.[15]

More advanced drug delivery systems are now available. These range from sophisticated patient-controlled analgesic machines (even for epidural usage)[16] to permanent intrathecal pumps (such as the Deltic pump, the Cordis Secor, and the Infusaid continuous infusion pumps).[7] Interpleural blocks with bupivacaine are used successfully in chronic pancreatitis and in reflex sympathetic dystrophy.[17,18]

Chemical neurolysis has progressed through the use of glycerine, with fewer resultant side-effects.[19] Glycerine is used to cover the dorsal root ganglion in segmental pain, the trigeminal ganglion in Meckel's cave in trigeminal neuralgia, and the greater splanchnic nerve in malignant upper abdominal visceral pain and in chronic pancreatitis.

Recent indications for transcutaneous electrical nerve stimulation and dorsal column stimulation (with epidural electrodes) are angina and ischaemia of the lower limbs.[5] Stereotactic deep-brain electrode insertion may in the future be replaced by cellular implant techniques. A recent application of cryotherapy is pituitary ablation for non-malignant pain.[19]

An important advance has been the recognition that the psychological component needs to be assessed and treated along with the organic pathology.[20] Psychological therapy includes psychotherapy, operant conditioning, and behavioural therapy. Progress has also been made in assessing a patient's pain more accurately by the use of a pain diary. This consists of a daily visual linear analogue pain scale, a daily history of medications used and of activities performed.

Another advance has been the input from several disciplines for the purpose of diagnosis, treatment, and rehabilitation of patients in pain relief units. This input is best co-ordinated by the consultant in charge of the pain relief unit. The type of treatment, as well as the dosage, is individualised.

The assessment and treatment of patients with intractable pain is an actively developing field. In southern Africa, with its heterogeneous Third-World population, there is a unique opportunity to study ethnic influences on the understanding of intractable pain. On a group level, treatment may have to be selective. (For example, neurolysis instead of sophisticated pharmacotherapy for malignant pain in a Third-World population group.) There is, however, a great need in southern Africa to establish regional pain relief units, and integrate their various services on a national level.

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1. Zech D, Dauer HG, Stollenwerk B, Lehmann KA. PCA and TTS fentanyl in the treatment of cancer pain. *Pain* 1990; suppl 5: S356.
2. McLeskey C, MacRae J. Transdermal fentanyl for postoperative pain following lower abdominal surgery. *Anesth Analg* 1990; **70**: S264.
3. Harshaw DH, Schwesinger WH, Reynolds JC, Fox KM, Crook F, Fite F. Transnasal butorphanol versus intramuscular meperidine in the treatment of postoperative pain. *Pain* 1990; suppl 5: S153.
4. Cubeddu LX, Hofmann IS, Fuenmayor NT, Finn AF. Efficacy of ondansetron (GR38032F) and the role of serotonin in cisplatin-induced nausea and vomiting. *N Engl J Med* 1990; **322**: 810-816.
5. Budd K. Recent advances in the treatment of chronic pain. *Br J Anaesth* 1989; **63**: 207-212.
6. Agrawal NM, Dajani EZ. Treatment and prevention of non-steroidal anti-inflammatory drug induced gastro-intestinal mucosal injury. *Int J Clin Pharmacol Res* 1989; **9**: 347-357.
7. Budd K. The pain clinic — chronic pain. In: Nunn JF, Utting JE, Brown BR, eds. *General Anaesthesia*. 5th ed. London: Butterworths, 1989: 1349-1369.
8. Eisenach JC, Dewan DM. Intrathecal clonidine in obstetrics: sheep studies. *Anesthesiology* 1990; **72**: 603-608.
9. Bonnet F, Boico O, Rostaing S, Loriferne JF, Saada M. Clonidine-induced analgesia in postoperative patients: epidural versus intramuscular administration. *Anesthesiology* 1990; **72**: 423-427.
10. Cleton FJ, Van Holten Verzantvoort AT, Bijvoet OLM. Effect of long-term biphosphonate treatment on morbidity due to bone metastases in breast cancer patients. In: Brunner KW, Fleisch H, Senn HJ, eds. *Recent Results in Cancer Research — Biphosphonates and Tumor Osteolysis*. Berlin: Springer-Verlag, 1989: 73-78.
11. Jaeger H, Maier CH. A double-blind study on calcitonin IV treatment in early phantom limb pain. *Pain* 1990; suppl 5: S553.
12. Chiang MF, Weigel K, Mutze M, Brock M. Long-term follow-up of three patients with chronic non-malignant pain treated with continuous infusion of salmon calcitonin. *Pain* 1990; suppl 5: S240.

13. Schwartzmann RJ, Tan CM, Santo JL, Grossman KL. Sufentanil sympathetic ganglia injection in patients with refracting reflex sympathetic dystrophy. *Pain* 1990; suppl 5: S490.
14. Gallagher J, Hamann W. Chronic neuropathic pain: aminoglycosides, peripheral somatosensory mechanisms and painful disorders. In: Atkinson RS, Adams AP, eds. *Recent Advances in Anaesthesia and Analgesia*. Vol 16. Edinburgh: Churchill Livingstone, 1989: 191-205.
15. Benedittis G, Lorenzetti A, Besana F. A new topical treatment for acute herpetic neuralgia and postherpetic neuralgia. *Pain* 1990; suppl 5: S57.
16. Sechzer PH. Patient-controlled analgesia (PCA): a retrospective. *Anesthesiology* 1990: **72:** 735-736.
17. Ahlburg P, Noreng M, Molgaard J, Edgebo K. Treatment of pancreatic pain with interpleural bupivacaine: an open trial. *Acta Anaesthesiol Scand* 1990; **34:** 156-157.
18. Reiestad F, McIlvaine WB, Kvalheim L, Stokke T, Pettersen B. Interpleural analgesia in treatment of upper extremity reflex sympathetic dystrophy. *Anesth Analg* 1989; **69:** 671-673.
19. Budd KE. Ablative nerve blocks and neurosurgery. In: Nimmo WS, Smith G, eds. *Anaesthesia*. Oxford: Blackwell Scientific Publications, 1989: 1243-1264.
20. Abram SE. Treatment of chronic pain syndromes. *International Anaesthesia Research Society Review Course Lectures*, 1990: 119-122.

Scorpion stings

Figures on the incidence of scorpion stings in South Africa are hard to come by, as they are not notifiable and most cases are dealt with outside hospital. However, fatalities do occur, and the latest issue of the *Journal of the Medical Defence Union* carries a cautionary tale about criticism of a doctor at an inquest following the death of a 4-year-old child.[1] She had apparently been bitten on the little toe by a scorpion of the genus *Parabuthus capensis*, and although she had been seen by the doctor within an hour of being stung, and had been admitted to hospital for observation, she died shortly after admission.

The doctor was criticised at the inquest for not obtaining the remains of the scorpion to confirm its type, for not removing the clothes of the child to examine her when he was called to the hospital to see her, and for not giving more specific instructions to the nursing staff about what neurological signs they should look for. The doctor gave a satisfactory explanation to all the criticisms, and added that he had not given scorpion antivenom in view of the dangers of anaphylactic shock, and because the child's clinical condition had not seemed to warrant it.

Several lessons can be learned from this episode. The first is that if at all possible, the scorpion should be identified, although this can be difficult if the culprit has already been despatched under the heel of a boot. Scorpions in South Africa can be divided into two distinct families: the frequently dangerous Buthidae, which have thin pincers and thick tails, and the relatively harmless Scorpionidae, which have thick pincers and thin tails.[2] In this case, the scorpion was almost undoubtedly a *P. capensis*, the venom of which is neurotoxic, and which can cause respiratory or cardiac failure, particularly in children. Victims of stings by these scorpions should be admitted to hospital for observation for 24 hours. Although simple analgesics can be used, barbiturates, meperidine, morphine and morphine derivatives should not be used as they can predispose to convulsions and increase the risk of respiratory and cardiac failure.[2]

In view of the risk of anaphylaxis, antivenom should only be used if the victim starts to show generalised symptoms of envenomation. A therapeutic dose of 5 ml (for children and adults) should be given subcutaneously or intramuscularly, and can be repeated after 1 hour if there is no improvement in the patient's condition. Intravenous administration of the serum greatly increases the risk of an adverse reaction, and should only be used in dire emergency. If intravenous administration becomes necessary, the antiserum should be diluted to 1 in 10 with sterile water or normal saline before injection. Anaphylactic reactions can be treated in the usual manner, although the necessary medications should be close at hand before the antiserum is administered. Intubation and assisted respiration may become necessary. Scorpion antivenom is kept in stock at all major provincial hospitals, but can also be obtained direct from the SAIMR or can be ordered from a pharmacy. Before transferring the victim of a scorpion sting to hospital, it is wise to check that in-date antiserum is available there.

Scorpion stings can be rapidly fatal, particularly in young children, as in this case, and rapid and effective action may be necessary if a tragedy is to be avoided.

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1. Hill G. A tale with a sting. *J Med Def Union* 1990; **6:** 69.
2. Prins A, Leroux V. *South African Spiders and Scorpions*. Cape Town: Anubis Press, 1986: 57-60.