# Preventing thromboembolism in medical inpatients – time to catch up with the surgeons?

Heparin prophylaxis for venous thromboembolism has been considered standard practice for patients undergoing surgical procedures for some time.[1] Only relatively recently has there been a move to adopt the practice more widely among *medical* inpatients, motivated by increasing good-quality evidence of the burden of the problem and perhaps by medico-legal concerns. Some researchers suggest that at least 10% of untreated medical inpatients may develop thrombo-embolic (TE) disease (either deep-vein thrombosis or pulmonary embolus).[2] A number of studies have demonstrated the effectiveness of heparin prophylaxis in this group, with reductions of up to 70% reported in acutely ill medical inpatients.[3,4] The American of College of Chest Physicians 2004 guidelines recommended the use of heparin or low-molecular-weight heparin for prophylaxis in acutely ill medical patients who have been admitted to hospital with congestive heart failure and severe respiratory disease, or who are confined to bed and have additional risk factors.[5]

One patient group at particular risk of TE disease is those with tuberculosis, with or without HIV infection. It is well recognised that TB is associated with a hypercoaguable state.[6] This is probably a consequence of a combination of factors: elevated plasma fibrinogen, thrombocytosis, direct endothelial damage promoted by the tubercle bacillus, and possibly the use of rifampicin. Additionally, a recent study indicated that an HIV-positive patient is 10 times more likely to develop TE disease than his or her uninfected counterpart, the risk being greatest in the recently hospitalised and those with CD4 counts of less than 500 cells/μl.[7] These data come from studies conducted among patients in well-resourced settings and there is very little information available on the incidence of TE among patients hospitalised with TB or HIV-related illnesses in resource-poor rural settings, in large part owing to problems with diagnosis. D-dimer, commonly used to diagnose TE disease in patients who are otherwise well, is raised in sepsis and therefore unhelpful,[8] and whereas a clinically obvious deep-vein thrombosis may be treated presumptively or confirmed by duplex ultrasound, pulmonary embolism is a difficult diagnosis to make in the rural hospital setting. Additionally, poor access to reliable autopsies and their unacceptability to relatives make it difficult to confirm the cause of death in such hospitals. While this under-reporting contributes to a level of ignorance regarding TE as a cause of death, there is no reason not to suspect that the incidence of TE disease among medical patients unwell enough to require admission to South Africa's hospitals is at least that suggested by studies in Europe and America. Indeed, it is probably considerably higher given the high proportion of medical patients admitted with advanced TB and HIV.

In the absence of good descriptive evidence regarding the incidence of TE disease among medical inpatients in South Africa, it could therefore be argued that all patients admitted with TB or AIDS-related illnesses should be started on heparin prophylaxis routinely. The time has come for the physician, like the surgeon, to ask of each inpatient not 'Why *should* they be on heparin?' but rather 'Is there any good reason why they *shouldn't*?'

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1. Caprini JA, Arcelus J, Sehgal LR, Cohen EB, Reyna JJ. The use of low molecular weight heparins for the prevention of postoperative venous thromboembolism in general surgery. A survey of practice in the United States. *Int Angiol* 2002; 21(1): 78-85.
2. Kanaan AO, Silva MA, Donovan JL, Roy T, Al-Homsi AS. Meta-analysis of venous thromboembolism prophylaxis in medically ill patients. *Clin Ther* 2007; 29(11): 2395-405.
3. Lechler E, Schramm W, Flosbach CW. The venous thrombotic risk in non-surgical patients: epidemiological data and efficacy/safety profile of a low-molecular-weight heparin (enoxaparin). The Prime Study Group. *Haemostasis* 1996; 26: Suppl 2, 49-56.
4. McGarry LJ, Thompson D. Retrospective database analysis of the prevention of venous thromboembolism with low-molecular-weight heparin in acutely ill medical inpatients in community practice. *Clin Ther* 2004; 26(3): 419-430.
5. Geerts WH, Pineo GF, Heit JA, *et al*. Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004; 126: 3 Suppl, 338S-400S.
6. Robson SC, White NW, Aronson I, Woollgar R, Goodman H, Jacobs P. Acute-phase response and the hypercoagulable state in pulmonary tuberculosis. *Br J Haematol* 1996; 93(4): 943-949.
7. Ahonkhai AA, Gebo KA, Streiff MB, Moore RD, Segal JB. Venous thromboembolism in patients with HIV/AIDS: a case-control study. *J Acquir Immune Defic Syndr* 2008; 48(3): 310-314.
8. Stief TW, Ijagha O, Weiste B, Herzum I, Renz H, Max M. Analysis of hemostasis alterations in sepsis. *Blood Coagul Fibrinolysis* 2007; 18(2): 179-186.