

## Editorial Focus

# Does weather influence the development of deep venous thrombosis? The answer may be blowing in the wind

John H. Reid

Department of Radiology, Borders General Hospital, Melrose, Scotland, UK

Those who make the diagnosis and treatment of thromboembolic disease their business occasionally remark on the erratic frequency of this common condition. Behaving like buses, deep venous thromboses (DVT) often come along in little clusters (also turning up late and when you least want one!). But like other apparently chaotic systems in the physical world, when you take a wider view, patterns begin to emerge. The challenge for the scientist is to determine if these patterns represent real, significant and useful observations or are they simply happy coincidences like seeing shapes in clouds? In this issue of *Thrombosis and Haemostasis*, Brown et al. set out a panoramic view of atmospheric variables using meteorological and epidemiological data on a national scale (1). Scotland is perhaps well placed to help investigators extract useful information from a relatively small maritime nation with fastidious public records and the affliction of notoriously changeable weather.

Previous observers have examined a number of climatic variables in relation to the development of venous thrombosis with some agreement over seasonal variation (2). Other meteorological factors have produced conflicting results (3). There is clearly a difference but interdependence between the effects of seasonality, atmospheric pressure, and weather conditions. For example, the observed increased incidence of thrombotic events (including myocardial infarction) in autumn and winter may in part be due to meteorological effects (pressure, wind and rain being interlinked) combined with other factors such as temperature-related procoagulability and reduction in physical mobility. The authors have attempted to strip out seasonality (winter prevalence again confirmed in this study, the second largest to consider the issue) to expose specific short-term meteorological correlations, of which atmospheric pressure, wind speed and rain are the most significant. The current study's findings of a thrombotic peak 7–11 days after a change in pressure also fits with the lag that we might expect in DVT presentation, further reinforcing that the correlation is real.

Hodkinson et al demonstrated in a study previously published in this journal that normobaric hypoxia is not associated with a significant thrombotic risk (4). In a recent study by Toff et al which simulated air travel in healthy volunteers, doubt was also cast upon the relevance of hypobaric hypoxia in thrombosis induction (5). This small study from 2006 specifically excluded patients with known thrombotic tendencies. However, clinical experience tells you that the broad observation by Brown and colleagues of a 10 millibar reduction in pressure leading to a 2% increase in events cannot be linear for everyone, otherwise DVT following long haul flight would be of significantly greater proportions. Therefore it is logical to assume that other prothrombotic factors must be operating in concert with reduced pressure. Schwarz et al demonstrated a doubling of risk of DVT for long haul passengers, and showed that flight-associated DVT occurred exclusively in passengers with well-established risk factors for venous thrombosis (6).

But does knowing any of this confer a diagnostic or treatment advantage? Should someone with varicose veins avoid taking an alpine holiday in bad weather?

Despite the retrospective nature of this study, the authors have persuasively isolated important facts which corroborate and support other work regarding the complex nature of thromboembolism: namely seasonal incidence, association with low atmospheric pressure, and confirmation of the lag time of clinical presentation following convergence of predisposing factors. For me, the correlation with change in atmospheric pressure is particularly helpful when considering the controversy around airline travel. With some authors suggesting that the asymptomatic (mainly below knee) DVT rate in long haul airline passengers could be as high as 10% (7), the findings of Brown et al must surely add impetus to the long haul flight debate and the future design of pressurised aircrafts.

Correspondence to:

Dr. John H. Reid

Consultant Radiologist

Radiology Department

Borders General Hospital

Melrose TD6 9BS

Scotland, UK

Tel.: +44 1896 826 421

E-mail: john.reid@borders.scot.nhs.uk

Received: July 21, 2009

Accepted: July 21, 2009

Prepublished online: July 30, 2009

doi:10.1160/TH09-07-0468

**Thromb Haemost 2009; 102: 611–612**

## References

1. Brown HK, Simpson AJ, Murchison JT. The influence of meteorological variables on the development of deep vein thrombosis. *Thromb Haemost* 2009; 102: 676-682.
2. Gallerani M, Boari B, Smolensky MH et al. Seasonal Variation in Occurrence of Pulmonary Embolism: Analysis of the Database of the Emilia-Romagna Region, Italy. *Chronobiology International: The Journal of Biological and Medical Rhythm Research* 2007; 24: 143 – 160.
3. Clauss R, Mayes J, Hilton P et al. The influence of weather and environment on pulmonary embolism: pollutants and fossil fuels. *Med Hypoth* 2005; 64:1198–1201.
4. Hodkinson PD, Hunt BJ, Parmar K et al. Is mild normobaric hypoxia a risk factor for venous thromboembolism? *Thromb Haemost* 2003; 1: 2131–2133.
5. Toff WD, Jones CI, Ford I et al. Effect of hypobaric hypoxia, simulating conditions during long-haul air travel, on coagulation, fibrinolysis, platelet function, and endothelial activation. *JAMA* 2006; 295: 2251–2261.
6. Schwarz T, Siegert G, Oettler W et al. Venous Thrombosis After Long-haul Flights. *Arch Intern Med* 2003; 163: 2759–2764.
7. Scurr JH, Machin SJ, Bailey-King S et al. Frequency and prevention of symptomless deep-vein thrombosis in long-haul flights: a randomised trial. *The Lancet* 2001; 357:1485 – 1489.