sleepy drivers (including those with untreated sleep apnea) must be reported to the relevant motor vehicle licensing authority in some provinces. Such reports have the potential to limit an older person's independence because, as pointed out by Wolkove and colleagues,[2] there is marked variability within Canada in the coverage for continuous positive airway pressure devices, the treatment of choice for obstructive sleep apnea. Astonishingly, some provinces do not cover this therapy at all.

Another problem facing Canadian patients with sleep disorders is the variability between provinces in the time patients have to wait for sleep testing. In several provinces patients must wait much longer for testing than recommended by the Canadian Thoracic Society Sleep Disordered Breathing Committee.[4,5] This places these patients at increased medical risk; the increased risk that they will be involved in an automobile accident may also endanger the public.

Meir Kryger MD
Director
Sleep Research and Education
Gaylord Hospital
Wallingford, Conn.
Charles George MD
Professor of Medicine
University of Western Ontario
London, Ont.

Competing interests: None declared for Dr. Kryger. Dr. George is on the Medical Advisory Board for Sleeptech LLC.

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DOI:10.1503/cmaj.1070079

Genetic analysis to prevent warfarin complications

Warfarin is the most widely used oral anticoagulant therapy for patients with thrombosis. Owing to the notoriously narrow therapeutic range of this drug, small variations in dosing may result in hemorrhagic or thrombotic complications. We read with interest the recent *CMAJ* article by Natalie Oake and colleagues, in which the authors concluded that improved anticoagulation control could decrease the likelihood of almost half of all anticoagulant-associated adverse events.[1]

A variety of physiologic and pharmacologic factors modulate the patient's compliance with warfarin therapy, including the pharmacokinetics of warfarin, the bioavailability of vitamin K and the metabolic fate of the vitamin-K-dependent coagulation factors.[2] It has consistently been reported that analysis of the genes encoding cytochrome P450 2C9 and the C1 subunit of the vitamin K 2,3-epoxide reductase complex, 2 pivotal enzymes affecting compliance with therapy, might enable physicians to estimate warfarin dosage more precisely, thereby improving the global efficiency of the titration process and reducing the likelihood of hemorrhagic and thromboembolic events. These 2 genotypes along with age, sex and body weight account for up to 60% of the variance in daily maintenance dose of warfarin.[3–6] Therefore, construction of dense genetic maps based on single nucleotide polymorphisms for both of these genes might be a powerful aid to dissecting the polygenic traits of drug response. When combined with an analysis of specific ethnic, clinical, environmental and psychological factors, such a tool could assist clinicians to define a warfarin dose–response phenotype that could be used to improve the quality of dose management. This might be a crucial step toward individualized medicine.[6]

Giuseppe Lippi MD
Gian Luca Salvagno MD
Gian Cesare Guidi MD
Sezione di Chimica e Microscopia Clinica
Dipartimento di Scienze Morfologico-Biomediche
Università degli Studi di Verona
Verona, Italy

Competing interests: None declared.

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DOI:10.1503/cmaj.1070081

Correction

In a recent News article,[1] the first photograph should be attributed to Lorne Turner. Incorrect information appeared in the print version of the July 17 issue. We apologize for this error.

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DOI:10.1503/cmaj.071001