manage to cope and keep working after many stressful life events, and yet have work disability and develop post-traumatic stress disorder after minor motor vehicle collisions. Perhaps it is as Sir John Collie remarked long ago:

> 'In short, the essential quality of a thing is its worth to the individual, and its value to him is its power to serve his private ends. On one occasion, when examining a working-man for an injury to his thumb, he observed me examining the terminal phalanx of one of his fingers, which had been partially removed, obviously as the result of a former accident. "That," said he, "is of no importance; it was done at home"!' (Collie, 1917).

Collie, J. (1917) *Malingering and Feigned Sickness* (2nd edn), p. 15. London: Edward Arnold.

Karlsborg, M., Smed, A., Jespersen, H., *et al* (1997) A prospective study of 39 patients with whiplash injury. *Acta Neurologica Scandinavica*, **95**, 65–72.

Mayou, R. & Bryant, B. (2002) Psychiatry of whiplash neck injury. *British Journal of Psychiatry*, **180**, 441–448.

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Regarding Mayou & Bryant's study (2002), it is interesting that the predictors of pain at 1 year are 'feeling not to blame for the accident', claiming compensation and 'anger cognitions'. With multivariate analysis, only claiming compensation at 3 months is a predictor of pain at 1 year. This means that feeling not to blame for the accident, initial anger or anger cognitions are predictors of pain only in claimants, otherwise not. Thus, of two patients, both not-at-fault, and both equally angry, it is the one who chooses litigation that will have the worse outcome. Why?

Does litigation/claims create a psychosomatic phenomenon that allows anger and victimisation to express itself as pain? Or are litigants more likely to be compelled to focus on all sources of aches and pains in their life (even pre-accident sources) by keeping pain diaries more often and by being instructed to see more physicians and therapists, to withdraw from more activities that hurt, to take more medications, to develop poor physical fitness, postural problems, medication adverse effects and anxiety?

It is further interesting that 14% of accident victims with no injury had bodily pain at 3 months! How does this happen? Is it a manifestation of psychological distress, or perhaps does pain occur as part of life, even if not injured (or, for that matter, even if not involved in an accident)? The percentage of accident victims with pain at 1 year in the 'no injury' group is half that of whiplash injury victims with pain at 1 year (27%). As one does not expect whiplash injury to create an immunity from whatever is affecting the 'no injury group', half of the whiplash injury group was going to have pain at 1 year, even if they had had no injury, or had fully recovered from their injury, because the 'no injury' group gets pain anyway. Not all of the pain at 1 year in whiplash victims can thus be due to physical effects of the initial injury, since then there would be at least some additional burden of pain from whatever factors also cause pain in the 'no injury' group as well. Statistically, half of the chronic pain that exists in whiplash patients is independent of having had an initial physical injury.

The findings of this study also suggest that when a physician encounters a patient who is not to blame for an accident and who is feeling angry, the physician should very clearly advise that entering a claim will adversely affect the patient's health and is more likely to lead to chronic pain.

Mayou, R. & Bryant, B. (2002) Psychiatry of whiplash neck injury. *British Journal of Psychiatry*, **180**, 441–448.

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Authors' reply: Drs Kwan and Friel make familiar general points about the interpretation of prospective research. However, they underestimate the practical, methodological and ethical difficulties of obtaining and using medical records and of qualifying information about life events. It is also worth noting that in medico-legal practice it is very common for medical experts and lawyers to disagree about the significance of medical histories and of life events following the identified trauma.

Dr Ferrari's first paragraph over-interprets multivariate analysis dependent on statistical significance in concluding that initial anger or anger cognitions are early predictors of pain in claimants. Although there are some differences between claimants and non-claimants, our overall experience in this study, and in a previous paper which followed up claimants for 6 years, is that the two groups are very similar (Bryant *et al*, 1997). The research findings, together with clinical experience, indicate that litigation is one of a number of reminders of the accident which do result in subjects focusing on their aches and pains. Further accidents, continuing medical complications and persistent financial difficulties are probably other important factors acting in a similar manner.

Fourteen per cent of accident victims with no recorded injury in the emergency department had pain at 3 months which was attributed to the accident. Perhaps the most likely explanation is that these people suffered minor musculo-skeletal injuries but that the symptoms did not become significant for hours or days after the accident. This is well described in relation to whiplash neck injury. It is therefore incorrect for Dr Ferrari to use our evidence to draw conclusions about the extent to which pain reported by whiplash patients may be independent of physical injury.

I also strongly disagree with Dr Ferrari's final conclusion that patients who are not to blame but angry should be advised not to enter a claim. The financial and other losses may be considerable and compensation desirable and even necessary. The more appropriate conclusion is that medical and legal procedures should take account of the patient's reactions and beliefs, avoid increasing distress and attempt to provide a sympathetic and rapid resolution of both the medical and the legal issues.

Bryant, B., Mayou, R. & Lloyd-Bostock, S. (1997) Compensation claims following road accidents: a six-year follow-up study. *Medicine, Science and the Law*, **37**, 326–336.

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Regional selectivity of novel antipsychotics

Xiberas *et al* (2001) measured D_2 receptor occupancy in striatum, thalamus and temporal cortex in patients treated with haloperidol, risperidone, amisulpride, clozapine and olanzapine. On the basis of their findings, they conclude that in the striatum and in the thalamus atypical antipsychotics induce a significantly lower D_2 binding index than haloperidol does. Their results are consistent with previous studies showing only small differences between striatal and temporal cortex blockade by traditional compounds and relatively selective D_2

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