Editorial

David Taylor

This issue includes a comprehensive review of quetiapine's effects on cardiac conduction prepared by a team largely from Virginia in the United States. Sadly one of the authors, and erstwhile leader of the team, Victor Vieweg died late last year at the age of 79. Dr Vieweg combined (uniquely, I suspect) expertise in cardiology and psychiatry and he was one of the world's foremost academics in the field of cardiac effects of psychotropics. His loss is a substantial one to psychiatry. Our thoughts are with his wife, children and grandchildren.

Victor Vieweg's long held view was that drug-induced QT prolongation *per se* is not a useful indicator of likely toxicity. Taking quetiapine as an example of a psychotropic drug about which warnings had been issued, the authors examined closely 12 cases of QT prolongation occurring in patients receiving quetiapine. It was found that in no case was quetiapine being used in an appropriate fashion in a patient with no other risk factor for QT prolongation – every example was characterised by some permutation of additional risk factors. Understanding this phenomenon greatly simplifies clinical practice and allows more realistic targeting of cardiac monitoring.

Clozapine has numerous cardiac effects, some of them serious. The most common effect is tachycardia which is most often an isolated finding or may be linked to myocarditis or Neuroleptic Malignant Syndrome. Beta blockers are commonly used to reduce heart rate but are not always effective or well tolerated. John Lally and

colleagues describe the successful use of ivabradine in two cases of clozapine-induced tachycardia. Ivabradine is normally used in angina and slows the heart via a direct effect on the sinus node. Its successful use here extends the range of drugs suitable for treating tachycardia in patients on clozapine and may provide a means of reducing the risk of myocarditis.

Two other papers in this issue address the practice of adding extra "augmentation" agents to partially effective first line psychotropics. Imran Chaudhry and colleagues from Manchester examined the use of simvastatin or ondansetron when added to antipsychotics used to treat schizophrenia. Both strategies had, before this, been supported only by case reports. In their 12 week single blind study there was a trend for the addition of both simvastatin and ondansetron to improve symptom scale scores to a greater extent than treatment as usual. These results are not enough to promote these therapies to everyday clinical use but enough to form the basis of a major grant application.

In the other add-on study, Reiji Yoshimura and co-workers examine the relative effects of three augmentation strategies in resistant-depression. In their small study, adding olanzapine or aripiprazole to paroxetine gave very similar results to the addition of lithium. This study adds to previous work suggesting the worthwhile efficacy of additive olanzapine and aripiprazole, and like all of the papers in this issue, edge us towards a greater understanding of the intricacies of clinical psychopharmacology.

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