

155 - QT prolongation

QT prolongation

Schizophrenia and related psychoses CHAPTER 1 ECG changes – QT prolongation Introduction Many psychotropic drugs are associated with ECG changes and some are causally linked to serious ventricular arrhythmia and sudden cardiac death. Specifically, some antipsychotics block cardiac potassium channels and are linked to prolongation of the cardiac QT interval, a risk factor for the ventricular arrhythmia TdP, which is sometimes fatal.¹ Case-control studies have suggested that the use of most antipsychotics is associated with an increase in the rate of sudden cardiac death.^{2–8} This risk is probably a result of the arrhythmogenic potential of antipsychotics,^{9,10} although schizophrenia itself may be associated with QT prolongation,¹¹ QT interval is longer in patients with schizophrenia than in controls (e.g. 418ms vs 393ms in one study)¹² and in one study prolonged QTc was identified in 7.6% of psychiatric in-patients who had an ECG.¹³ A more recent systematic review¹⁴ suggested that 4% of people with schizophrenia and receiving an antipsychotic had a prolonged QTc. Antipsychotic-related risk is probably dose-related and, although the absolute risk is low, it is substantially higher than, say, the risk of fatal agranulocytosis with clozapine.⁹ One report of cases gathered by a national database put the risk of TdP at between 0 and 19.2 cases per 100,000 patient years, depending on the individual antipsychotic and age of patients.¹⁵ The effect of antipsychotic polypharmacy on QT is somewhat uncertain,¹⁶ but the extent of QT prolongation is probably a function of overall dose.¹⁷ ECG monitoring of drug-induced changes in mental health settings is complicated by a number of factors. Psychiatrists may have limited expertise in ECG interpretation, for example, and still less expertise in manually measuring QT intervals. Even cardiologists show an inter-rater reliability in QT measurement of up to 20ms.¹⁸ Self-reading, computerised ECG devices are now widely available and compensate for some lack of expertise, but different models use different algorithms and different correction formulae.¹⁹ ECG machines may not be as readily available as they are in general medicine. Also, there may be insufficient time for ECG determination in many areas (e.g. out-patients). Lastly, ECG determination may be difficult to perform in acutely disturbed, physically uncooperative patients. ECG monitoring is nonetheless essential for all patients prescribed antipsychotics. An estimate of QTc interval should be made on admission to in-patient units (in the UK this is recommended in the NICE schizophrenia guideline)²⁰ and yearly thereafter. QT prolongation ■ ■ The cardiac QT interval (usually cited as QTc – QT corrected for heart rate) is a useful but imprecise indicator of risk of TdP and of increased cardiac mortality.²¹ Different correction factors and methods may give markedly different values.²² The most widely used formula is Bazett's. This tends to overestimate QT length, especially when heart rate is increased.²³ ■ ■ The QT interval broadly reflects the duration of cardiac repolarisation. Lengthening of repolarisation duration induces heterogeneity of electrical phasing in different ventricular structures (a phenomenon known as dispersion), which in turn allows the emergence of

early after-depolarisations, which may provoke ventricular extrasystole and TdP. Measures have been developed (QT dispersion ratio, dispersion transmural repolarisation time) which may better predict arrhythmia.¹²

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