classroom or reprimanded by the teacher for something over which they have no control. This can lead to low self esteem and emotional difficulties. If the child has tics that are uncomfortable and cause psychological and social distress, then medication may be considered. No drug has predictable and potent efficacy in all children with tics—most studies show a reduction of only about 30% in the severity of tics.¹⁰

Surprisingly few good quality, randomised controlled trials in children are available. Double blind trials have shown tic severity and frequency are reduced using dopamine antagonists, such as haloperidol, pimozide, sulpiride, and the α_2 adrenergic receptor agonist, clonidine.^{6 10 11} Placebo-controlled studies of risperidone in Tourette's syndrome have shown that it is efficacious and has fewer side effects than the older dopamine antagonists.¹²

Other problems such as obsessive compulsive disorder and attention deficit hyperactivity disorder are often present (50-70% of children with Tourette's). The combination can be difficult to treat, and specialist advice from a child psychiatrist may be needed.

Parents often feel helpless and at a loss to know what to do when their children have tics. Helping parents adjust to the diagnosis and manage the negative reaction of peers and public can be empowering to families. A good understanding of the symptoms and their fluctuations is essential. For example, children seem able to suppress tics for periods of time such as at school, followed by a disruptive rebound on returning home.⁶

Most individuals with tics lead highly functional lives, and the tics themselves usually wane in teenage years. Parents should be encouraged to seek support for themselves from various organisations such as the Tourette Syndrome Association (enquiries@tsa.org.uk). With a good understanding of tics and related problems, including acceptance from teachers and

education of peers, most children with tics do not need regular medical follow up.

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Troponin in patients with chest pain

We need to ensure that troponin is only one element of the management algorithm

For several years the measurement of serum troponin concentration in patients presenting with acute chest pain has been routine in most hospitals in the United Kingdom. Guidelines now demarcate myocardial infarction with ST elevation from acute coronary syndromes such as unstable angina and myocardial infarction without ST elevation. Since the early 1990s studies have repeatedly confirmed that troponins are released by some patients with acute coronary syndromes and represent a marker of risk for subsequent cardiac events.¹⁻³ FRISC-II, TACTICS-TIMI 18, and RITA3 all indicate that early angiography and revascularisation can reduce the risk of events in this cohort, thereby improving prognosis.⁴⁻⁶ As a consequence, management of acute coronary syndromes without ST elevation has changed dramatically in the United Kingdom.⁷

Formerly, patients were treated medically and considered for early revascularisation if they had ongoing

ischaemia, which represents only about 10% of such a group. By contrast, current guidelines recommend early invasive treatment in patients not only with ongoing ischaemia but also those with elevated troponins with or without ST depression. In the United Kingdom we are therefore trying to manage a substantial proportion of patients with acute coronary syndromes by early angiography and revascularisation, a strategy that guarantees mismatch between demand and available resources.

This strategy necessitates a wait in hospital for transfer to a tertiary centre for most patients. Furthermore it has led to suboptimal and inequitable management for, perhaps, most patients for the following reasons. Firstly, patients awaiting transfer occupy a huge number of bed days,⁸ even when asymptomatic and mobile. This delay is detrimental to both patients and their families and the efficiency of hospitals. Secondly, as invasive facilities cannot cope with this

unplanned surge in demand, patients are not being treated within the evidence based time period. FRISC-II and TACTICS-TIMI 18 randomised patients to invasive or non-invasive treatment within a few days of presentation, and ISAR-COOL⁹ shows that treating patients earlier than that is beneficial. Perhaps we are simply selecting out a group of patients at lower risk. Thirdly, we are also practising postcode medicine. A patient who has an acute coronary syndrome without ST elevation and is admitted to a hospital with facilities for revascularisation is likely to receive treatment quicker than a similar patient admitted to a hospital without such facilities.¹⁰ Similarly, in one hospital, a troponin positive patient with an acute coronary syndrome may be admitted for a few days and, if asymptomatic, be sent home, whereas in another hospital such a patient may be kept in hospital pending transfer. How can both strategies be correct?

Has measuring troponins in patients with acute chest pain been a clinical success? This measurement is flawed as a clinical tool. Firstly, our interpretation of the data may be overenthusiastic. Of studies showing a correlation between troponin concentration and outcome, the event rate, even in cohorts with the highest band of troponin is around 20%. Many centres currently refer all patients with acute coronary syndrome who have elevated troponin concentrations for early angiography. Thereafter, all these patients (fewer than 70% of whom will have an event during the follow up period) are considered for revascularisation on the basis of a reduction in the six month event rate from 12.1% to 9.4% for death or myocardial infarction in FRISC-II or 19.4% to 15.9% for death, myocardial infarction, or rehospitalisation in TACTICS-TIMI 18.^{4,5} Surely this represents a rather blunt screening tool. The second flaw in measuring troponin is that it is too eagerly perceived as a rule in or rule out test. The test has become, for many clinicians, the main component of their decision making. The final flaw is that elevation of troponin is reported in a myriad of clinical conditions that do not seem to be associated with myocardial ischaemia.

The solution is to refine the way we use the test to ensure that it is only one element of a clinically driven management algorithm. The meticulous derivation of clinical risk scores, particularly GRACE,¹¹ should be exploited. Furthermore, research that focuses on the identification of other markers of risk should be facilitated, in order to target revascularisation at those most likely to benefit. In addition, we should make use of the heterogeneous nature of current management by care-

fully observing outcome in patients who are not put up for inpatient angiography. Do such patients do badly? Recent data from the ICTUS trial¹² call our current strategy of early angiography for all patients with raised troponin into question.

Troponin measurement in patients with acute chest pain is a mixed blessing. We need to refine our current strategy to provide optimal treatment for such patients. Troponin is a blunt screening tool for the assessment of patients with acute coronary syndromes without ST elevation, and we should avoid compounding this by ignoring clinical factors. Above all, we need a realistic national management guideline to avoid inequity of access, pending better risk stratification tools.

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Patenting genes

May slow down innovation, and delay availability of cheaper genetic tests

Patents on genes have been granted for many years. Until January 2001, when the first patent for the first breast cancer gene (BRCA1) was granted by the European Patent Office to Myriad Genetics, a US company, most geneticists in Europe had never worried that genes and genetic tests could become exclusive property of a laboratory or a

company. A patent allows a company to develop commercial laboratory testing services based on the gene sequences and charge other laboratories wanting to run genetic tests. Myriad Genetics holds several patents on the breast cancer genes BRCA1 and BRCA2 and offers a specific test called BRACAnalysis to detect abnormalities in these genes. The company opted to

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