sharing in mind, whereas in the hospital sector electronic records are rarely useable as the master clinical record.[11] The NHS wants this to change. In theory, an appropriate and effective information infrastructure has much to offer, providing information to underpin commissioning, quality control, clinical governance, and performance management as well as being the prime clinical information source for individual patient care. But it needs to be provided in the context of local multidisciplinary teams involved in the care of patients in complex care pathways, tailored to local facilities and resources.[12]

With a workforce of over 1.3 million, the NHS is one of the world's largest employers. Its national programme for information technology is the largest and most ambitious public sector information technology procurement project to date. The rest of the world watches our progress with interest, as this kind of record sharing technology, although familiar in web based commerce, is novel in a large scale health environment. So far the new team running the national health technology programme has proved pragmatic and effective, and the reality of NHS wide clinical record technology is closer than ever. But involving front line clinicians in the evolution of their workplace information systems is essential. If safeguards to confidentiality and accuracy of patient information prove insufficient, then both patients and the caring professions will not use the spine, and the money will have been wasted.

Competing interests: None declared.

Nick Booth *principal clinical research associate*

School of Population and Health Sciences, Centre for Health Services Research, University of Newcastle, Newcastle upon Tyne NE2 4AA
n.s.booth@ncl.ac.uk)

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Rhabdomyolysis

Has many causes, including statins, and may be fatal

The dramatic title—Rhabdomyolysis: the hidden killer—given to a recent review of this condition emphasised that dissolution of striated muscle fibres, with leakage of muscle enzymes, myoglobin, potassium, calcium, and other intracellular constituents, can occur in anyone under particular circumstances and that the consequences can be severe and sometimes fatal.[1] There are no prospective studies of the incidence of rhabdomyolysis and many mild cases probably go unrecognised.

Rhabdomyolysis is defined as an acute increase in serum concentrations of creatine kinase to more than five times the upper normal limit—and when myocardial infarction has been excluded as a cause (CK-MB fraction less than 5%). Visible myoglobinuria (tea or cola coloured urine) occurs when urinary myoglobin exceeds 250 μg/ml (normal <5 ng/ml), corresponding to the destruction of more than 100 g of muscle.[2 3] Myoglobinuria can be inferred by a positive urine dipstick test for haem, in the absence of red cells on microscopic examination of urine.

The causes of rhabdomyolysis are legion, but all lead to a critical increase in sarcoplasmic calcium and intracellular damage by activation of calcium dependent proteases and phospholipases. Risk is increased by pre-existing metabolic factors such as hypokalaemia, hypophosphataemia, and hyponatraemia. Single episodes are most commonly caused by infections (viral, bacterial, or other), drugs, or physical factors such as compartment syndromes, ischaemia, reperfusion (including surgical procedures), and pressure from hard surfaces in comatose patients. Bywaters and Beall described the development of acute renal failure following crush injuries sustained in the London blitz,[4] and trauma remains an important cause, although the incidence of rhabdomyolysis after the attacks on the World Trade Center on 9 September 2001 was low, reflecting the high fatality rate.[5]

Severe or unaccustomed exertion, particularly in extremes of heat, is a common precipitant and has been reported in long distance runners, bodybuilders, and military recruits, and may also follow prolonged seizures, certain involuntary movement disorders, and rigors. It is also known to occur in polo ponies and racehorses.[6 7]

Alcohol and opiates are the drugs implicated most often,[8] but all potentially myotoxic drugs (particularly mixtures of drugs) can induce rhabdomyolysis, as can drugs that induce states of extreme agitation—as in the serotonergic syndrome caused by amphetamines and ecstasy. Rhabdomyolysis is also an important component of the neuroleptic malignant syndrome, induced by dopaminergic blockade or withdrawal of dopaminergic agents.

Statins are of particular concern because of their widespread and increasing use. Myotoxicity occurs in about 0.1% of cases, although cerivastatin was withdrawn in 2001 because the incidence of myotoxic-

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ity with this drug was some 10 times greater.[9] Drug interactions particularly with fibrates or drugs that interfere with cytochrome p450, the main isoenzyme involved in the metabolism of statins, seem to account for most instances. Reassuringly, fatal rhabdomyolysis due to statins is now rare and occurs in less than one per million prescriptions.[10] Inflammatory myopathy ("myositis") is rarely a cause of rhabdomyolysis, and although routine muscle biopsy may show fibre necrosis and degeneration, it may be entirely normal.

A history of recurrent episodes, a family history of attacks, or episodes precipitated by exertion or starvation, increases the probability of a genetically determined metabolic myopathy. Of these, carnitine palmitoyl transferase II deficiency is probably the commonest, but rhabdomyolysis can occur with any of the glycolytic enzyme deficiencies, with fatty acid oxidation disorders, and with many of the mitochondrial cytopathies. Susceptibility to malignant hyperthermia may also account for some cases. However, many cases of recurrent myoglobinuria are deemed idiopathic (Meyer-Betz disease). No doubt they represent undiagnosed or as yet undefined forms of metabolic myopathy.[2 3]

The immediate consequences of rhabdomyolysis include hyperkalaemia, which may cause fatal cardiac dysrhythmia, and hypocalcaemia due to calcium binding by damaged muscle proteins and phosphate.

Acute renal failure results from renal vasoconstriction, intraluminal myoglobin cast formation, and haem protein nephrotoxicity.[11] No randomised trials of treatment have been conducted, but by consensus the fundamental management principle is intravascular volume expansion by using saline and sometimes mannitol to maintain urine output at more than 200-300 ml/hour, with careful monitoring of sodium and calcium concentrations. Alkalinising the urine by using sodium bicarbonate can reduce the risk of tubular obstruction by myoglobin casts. However, myoglobin is also intrinsically nephrotoxic and can precipitate acute tubular necrosis through iron dependent inhibition of oxidative phosphorylation and iron independent inhibition of gluconeogenesis.[12]

In some experimental models, haem protein cytotoxicity could be blocked by iron chelators and glutathione,[11 12] but this has not been evaluated clinically. Dantrolene sodium blocks the release of calcium from the sarcoplasmic reticulum and can reduce calcium mediated myolysis. Occasionally fasciotomy may be required to prevent irreversible peripheral nerve injury by muscle swelling in tight fascial planes.[2 3] Disseminated intravascular coagulopathy is rare in uncomplicated rhabdomyolysis but may occur in more complex cases—for example, with associated sepsis. When renal failure ensues despite these measures, continuous haemofiltration or haemodialysis will be required. The prognosis should be excellent providing the causative mechanism for the rhabdomyolysis is identified and reversed where possible.

Russell Lane *consultant neurologist*
Malcolm Phillips *consultant nephrologist*

Charing Cross Hospital and Imperial College, London W6 8RF
(r.lane@imperial.ac.uk)

Competing interests: None declared.

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Acute psychiatric day hospitals

Are not in fashion, but evidence shows that they provide feasible and effective care

Acute day hospitals are among the earliest forms of psychiatric community care, but they are definitely not in fashion. In the NHS Plan they are not targeted for investment,[1] whereas in the United States, under managed care, they have been in steady decline since the 1980s.[2] Yet acute day hospitals did not fall from favour because they were ineffective, and emerging social trends may yet restore them to favour.

Day hospitals were invented in Russia in the 1930s, spread to America and Europe in the 1940s and 1950s, and reached their peak in the 1970s, when they provided the main alternative to hospital admission.[3] Paradoxically, the success of acute day hospitals as an alternative to inpatient care was a major factor in their decline, since it begged the question of whether hospital care was necessary at all. In the 1980s new radical approaches to community care, such as assertive community treatment and acute home based care, made day hospitals look old fashioned, stigmatising, and, worst of all, expensive.[4] As day hospitals began their fall from grace, hard questions were asked about their evidence base, which experts admitted was perplexing.[5 6]

However, a recent systematic review has shown that the answers to the hard questions were better than generally believed.[7] The evidence for acute day hospitals was not lacking, but it was complex. The review identified nine randomised controlled trials of acute day hospital treatment including 2268 patients;

BMJ 2003;327:116–7