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### Rhabdomyolysis due to mania

SIR: Rhabdomyolysis commonly occurs in response to trauma, limb ischaemia, severe exertion or prolonged stasis. It has, however, been associated with wolf attacks, conga-drumming, karate, mechanical bull-riding and deep knee bends (Frucht, 1994). Following injury skeletal muscle enters an accelerated catabolic state releasing myoglobin which is really excreted and precipitates in the renal tubules leading to acute renal failure in up to one-third of cases. Classically the condition presents with myalgia, muscle swelling and tenderness with deep red brown urine. However, incipient deterioration of renal function, haematuria or proteinuria may be the only features. The serum creatinine kinase rises by up to a magnitude of 100 with the MM isoenzyme representing greater than 97% of the total. We report a case of rhabdomyolysis associated with acute mania.

A 67-year-old man presented with a one-week history of profound overactivity, agitation and insomnia. Six months earlier he had been admitted briefly with a similar episode which had been treated successfully with a short course of lorazepam. There was no history of drug abuse and no family history of psychiatric illness. On examination he was profoundly overactive, pacing the room throughout the interview becoming progressively agitated and threatening. He was markedly sexually disinhibited making lurid comments to female staff. Florid pressure of speech with flight of ideas was present although he did not appear deluded or to be hallucinating. Physical examination was normal.

Lorazepam 4 mg per day was commenced and he remained freely mobile on the ward refusing all but minimal quantities of water. His mood settled on the lorazepam but four days following admission he complained of general malaise, worsening bilateral calf muscle pain and darkening of his urine. On examination he was afebrile, moderately dehydrated with a uraemic foetor. There was no evidence of tissue injury although he had extremely tender calves and forearms. Urinalysis revealed 3+ protein with no blood. Biochemistry demonstrated a deterioration in renal function with a serum urea of 20.4 mmol/l and creatinine of 300 mmol/l. Creatinine

kinase was grossly elevated at 7234 iu/l (nr 25–195 iu/l) with MM fraction of 98%. He had myoglobinuria (>100 000 iu/l). He was treated with simple analgesics and intravenous fluids. Urinalysis was negative within five days and the creatinine kinase concentration returned to normal over one week.

To our knowledge, this is the first reported case of rhabdomyolysis due to excessive exertion and dehydration secondary to an acute manic episode though we suspect that mild cases may remain undetected. Patients with psychiatric illness are particularly susceptible to rhabdomyolysis secondary to alcohol abuse of neuroleptic medication but only two other cases of rhabdomyolysis resulting directly from psychotic disorders have been reported. (Coryell *et al.*, 1978; Frankel & Prasad, 1989). In the first a patient with a known psychotic illness developed rhabdomyolysis after assuming a catatonic position for an hour. In the second, a chronic schizophrenic required dialysis following four hours continual jumping up and down during an acute psychotic episode. Rhabdomyolysis is a serious and potentially fatal complication of psychosis and should be considered in all cases of overactivity or catatonia.

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### Cardiovascular and autonomic function in Down syndrome—prescribing implications

SIR: Although modern literature on Down syndrome routinely lists such problems as hypothyroidism, sleep apnoea and atlanto-axial instability, it rarely mentions the evidence of autonomic dysfunction in Down syndrome, which should affect medical prescribing. There has been evidence available for some time, with “the not infrequently fatal idiosyncrasy” of Down syndrome to atropine-related drugs commented on in 1957 by McKusick and with later experimental evidence of hyper-reactivity to atropine (Mir & Cumming, 1971). Since then there has been further evidence of an abnormal sympathetic nervous system in terms of enzyme levels and response to noradrenaline (Lake

*et al*, 1979). There is further evidence from clinical practice in a personal observation that people with Down syndrome commonly have a drop in systolic blood pressure on standing from the supine of over 20 mmHg (which is not a test used in past studies on blood pressure in Down syndrome), often have a poor beat-to-beat variation on ECGs, and that it is difficult to tell by observation whether a person with Down syndrome and insulin controlled diabetes is having a hypoglycaemic (or hyperglycaemic) episode.

The recent enquiry into deaths in psychiatric hospitals has highlighted the potential risks of prescribing high dose neuroleptics which have a marked effect on the autonomic system, coincidental with restraining an angry and hot patient. This risk is probably much higher in people with Down syndrome who are likely to have more cardiovascular side effects from chlorpromazine or thioridazine

than the normal population, and psychiatrists should consider this when prescribing.

Recently I have met a case of a person with Down syndrome having severe cardiac side-effects from less than 100 mg of thioridazine daily. I would be interested to know if others have experienced sensitivity to phenothiazines in people with Down syndrome.

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#### A HUNDRED YEARS AGO

##### The Simulation of Death by Fakirs

Herr Kuhn not long ago presented a communication on this subject to the Anthropological Society of Munich. He had the opportunity of personally observing two cases, as to the genuineness of which he had no doubt whatsoever. One of the fakirs referred to had been buried alive for six weeks, the other for ten days. The condition which the fakir has the power of producing artificially is in all respects identical with the cataleptic trance. The fakirs, who are all hysterical subjects of a very pronounced type, put themselves through a regular course of training before the performance, weakening themselves by semi-starvation, taking internally various vegetable substances known only to them, keeping their bodies motionless in the same position for several hours at a time, etc. The details of this preparation are given in the *Hathayoga Pradīpikā Strātmārāmas*, which has been translated by Walter. When the fakir has by these means got

himself into the proper condition, he has only to lie down in one of the positions enjoined by the sacred books, and fix his eyes on the end of his nose, to fall into a state of trance. The fakirs are also believed to be use haschisch for the purpose of lessening the force of respiration; that hypnotic agent associated with other vegetable substances and used in a special manner is believed by them to supply the want both of air and nourishment. At the beginning of the trance the fakir has hallucinations, hearing heavenly voices, seeing visions, etc. Gradually, however, consciousness becomes annulled, the body becomes rigid, and, as the fakirs themselves say, "the spirit rejoins the soul of the world." In short, the condition is one of auto-hypnosis in hysterical subjects specially prepared for the experiment.

##### Reference

British Medical Journal, May 1895, 1000.

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#### CORRIGENDUM

WEISS, M. G., RAGURAM, R. & CHANNABASAVANNA, S. M. (*BJP*, March 1995, pp. 353-359). The legend for Fig. 1 on page 355 should read: Somatic, depressive and anxiety symptoms reported initially

in response to an open-ended query (■), after symptom specific probes (□), or later in the course of the interview (⊞).