has also been used as a herbicide and also as a photographic developing chemical. Its use has now resurfaced via the internet.

A 27-yr-old lady was admitted to accident and emergency complaining of fatigue, nausea, and excessive sweating. She admitted to starting a new diet tablet (bought over the internet) a week before her admission. She had doubled the recommended dose for faster results. Past medical history was negative. She was a non-smoker, non-drinker, and had no known allergies. Initial examination revealed an agitated overweight female (BMI 33) with a GCS of 15. Her airway was clear, respiratory rate (RR) 60, oxygen saturation 100% (F\textsubscript{\textsc{e}}\textsubscript{O}2 40%), blood pressure (BP) 122/86, and heart rate 140 beats min\textsuperscript{-1}. Temperature was 38°C. There were no other significant clinical findings.

Toxbase was consulted and she received 2 litre of normal saline i.v. over 2 h and diazepam 35 mg orally. Initial arterial blood gas revealed pH 7.46, P\textsubscript{CO}2 3.9 kPa, P\textsubscript{O}2 13.2 kPa, and base excess (BE) of −5.0. Initial laboratory results including full blood count, electrolytes, amylase, and liver function tests were normal. Creatinine kinase level was 1042. The patient had a further litre of normal saline i.v. over 2 h and 2 mg lorazepam i.v. The intensive care unit was informed. Six hours after admission, GCS was 14 (eyes opening to command), RR 44, BP 146/110, heart rate 150 beats min\textsuperscript{-1}, and temperature of 38°C. Her urine output over 5 h was 146 ml. Repeat arterial blood gas showed pH 7.46, P\textsubscript{CO}2 2.36 kPa, P\textsubscript{O}2 13.8 kPa, BE −11.8, and bicarbonate 16.8. An hour later, she desaturated to <90% and became asystolic. Cardiopulmonary resuscitation was started. Despite succinylcholine 100 mg and vecuronium 10 mg (i.v.), it was not possible to ventilate her due to widespread sustained muscle rigidity. After 14 cycles with epinephrine and atropine, she remained asystolic and was declared dead.

DNP causes a hyper-metabolic state by uncoupling oxidative phosphorylation. Energy is released in the mitochondria as heat. The body attempts to compensate by gluconeogenesis, glycolysis, and lipolysis. Toxic doses will result in uncontrolled thermogenesis leading to hyperthermia and systemic responses to elevated body temperature. Profuse yellow-tinted perspiration may be observed and is pathomonic of DNP poisoning. Renal, hepatic, and neurological sequelae can occur. In cases of severe toxicity, death may be followed by prompt rigor mortis.

Management involves aggressive supportive measures and use of benzodiazepines, but because of a large volume of distribution, DNP is not amenable to dialysis or haemoperfusion. It uncouples oxidative phosphorylation, causing release of calcium from mitochondrial stores. Raised free intracellular calcium causes muscle contraction and hyperthermia. Successful use of dantrolene has been described. By inhibiting calcium release from the sarcoplasmic reticulum, dantrolene reduces intracellular calcium. This is thought to help to facilitate heat dissipation in uncontrolled DNP-related hyperthermia. We stress importance of early aggressive supportive care and level 2 involvement in cases of DNP poisoning. We also propose the early use of dantrolene.

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**Interaction between clonidine and escitalopram**

**Editor**—We report a significant interaction between clonidine and escitalopram in a 66-yr-old critically ill patient. Clonidine was started because of the combination of hypertension and agitation. A few hours later, the patient was calmer and her arterial pressure had decreased to a normal value. The next day, her general practitioner (GP) was contacted to ensure all her regular medication was correctly prescribed. The GP informed us that the patient had been treated for depression with escitalopram for the past year, and this was restarted at a dose of 5 mg. Over the following 3 days, the patient became progressively more drowsy and ultimately almost unconscious. An interaction between the two drugs was suspected and the escitalopram stopped. The following morning the patient was awake and continued to stay alert.

A literature search failed to show any interactions between these two drugs, but one was found with a similar drug, citalopram. This article showed that long-term treatment of rats with citalopram, among other antidepressants, caused a significant decrease in cerebral cortical binding of clonidine accompanied by a functional hyposensitivity of alpha2 adrenergic receptors. This led to attenuation of several central effects of clonidine such as hypothermia and sedation. This is the opposite of what we saw in our
patient. It may be that there is a difference in the interaction of escitalopram with clonidine, as opposed to citalopram. Alternatively, this may represent an additive effect of the sedation seen with both drugs. Whatever the mechanism, caution is needed with all centrally acting drugs used in the critically ill. Drugs may have different effects and interactions in sick patients on multiple other drugs. Restarting drugs that patients were on at home needs careful consideration.

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