screening study and a cut-off point with high specificity in a study of unambiguous groups of normal, depressed, and Alzheimer patients.

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Caffeine and Panic Attacks

SIR: It has not been clearly shown that caffeine alone can cause panic attacks in normal subjects. I report an adverse reaction which occurred during a normal volunteer study in which caffeine was compared against placebo and rolipram (a CNS phosphodiesterase inhibitor).

Case report: A 21-year-old healthy male volunteer with neither a history of panic attacks nor severe anxiety was given 500 mg of caffeine – equivalent to 5 – 8 cups of coffee – in the form of 1 gm of caffeine citrate in an orange drink. After approximately 20 minutes the subject started to feel an intense dread, which was quickly followed by the somatic symptoms of severe anxiety, including sweating, palpitations, and physical restlessness. He was unable to tolerate staying in the experimental room, and he had fears of impending death. Diazepam (15 mg) was given intravenously, which quelled both the physical and the cognitive changes, but did not remove them entirely. After around 1½ hours the anxiety symptoms returned and further diazepam was required.

This adverse reaction has implications both in theory and in clinical practice. Firstly, the interaction between the benzodiazepines and caffeine is not yet fully understood (File *et al*, 1982); Ghoneim *et al*, 1986). It is known that caffeine is an adenosine receptor antagonist at concentrations found in plasma (Daly *et al*, 1981) and not, as had previously been thought, a phosphodiesterase inhibitor. Diazepam interacts at the benzodiazepine receptor, yet this experiment shows that a benzodiazepine will attenuate the symptoms of anxiety induced by caffeine. This is shown by the return of the anxiety after around 1½ hours, when the diazepam is unbound from the receptor; the plasma half-life of caffeine is of the order of 3–6 hours (or longer) in healthy non-smoking men (Axelrod & Reichenthal, 1953). This would imply that the causation of anxiety might in some way be related to adenosine receptor antagonism. Secondly, it shows that large doses of caffeine can cause severe anxiety in normal people, and therefore an estimation of caffeine intake needs to be part of the assessment of panic attacks. Although this may seem a large dose of caffeine it is not uncommon, as Graham (1978) showed, for people in the general population to drink this quantity in a 24-hour period. Finally, this finding also emphasises the importance of caffeine in the generation of anxiety, which does not amount to panic, in the coffee-consuming general population.

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Hyponatraemia and Lofepramine

SIR: Hyponatraemia in psychiatric patients has been variously attributed to compulsive water drinking (Ferrier, 1985), to the primary psychiatric disorder (Singh *et al*, 1985), and to the syndrome of inappropriate secretion of anti-diuretic hormone (SIADH) induced by psychotropic drugs (Sandifer, 1983; Streeten *et al*, 1981). SIADH is a recognised complication of tricyclic antidepressants, and has been reported in association with amitriptyline and desipramine (Sandifer, 1983). We wish to report a case of hyponatraemia which was probably due to SIADH in association with lofepramine.

Case report: A 52-year-old married woman with a history of schizoaffective illness was admitted as an emergency into a medical ward with an acute onset of lethargy, anorexia, vomiting, severe weight loss, and confusion. There were no significant findings on physical examination, but she had repeated abnormal biochemical results. The initial report