Social phobics reported more blushing and muscle twitching, and less limb weakness, breathing difficulty, dizziness or faintness, actual fainting, and buzzing or ringing in the ears than did agoraphobics.

Analogue studies and open clinical trial suggest greater beta-blocker efficacy in social phobia than agoraphobia (Gorman *et al*, 1985). Tricyclics are quite helpful for blocking panic attacks. Clinically they seem less useful in social phobia. Controlled trials to confirm these impressions, however, are needed. MAO inhibitors appear useful in both conditions.

Plasma epinephrine does not appear to be elevated during *in vivo* or lactate induced panic attacks (Liebowitz *et al*, 1985). A study in progress is demonstrating that at least a subset of social phobics (perhaps 50%) demonstrate plasma epinephrine elevations of 50% or more during performance challenge. Studies in medical house officers suggest two to three-fold epinephrine rise during more stressful real life public speaking (Dimsdale & Moss, 1980). Social phobics are significantly less likely to panic with sodium lactate infusion than are agoraphobics (Liebowitz *et al*, 1985).

Onset and offset seem to differ as well. Social or performance anxiety is triggered only when the individual is faced with interpersonal evaluation or scrutiny, and is probably induced by cognitions that one will perform badly, look foolish, etc. Panic attacks related to panic disorder often occur unexpectedly, especially early on, without obvious cognitive precursors. When feeling anxious, social phobics prefer to be alone to recover their equilibrium; agoraphobics are less likely to panic, and more readily recover, in the presence of a trusted person.

Both agoraphobics and social phobics do fear the onset of severe anxiety experience. Available evidence, however, suggests important differences in pathophysiology, which Solyom's statement obscures.

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Clinical Tests of Memory as Sensitive and Specific Signs of Dementia

SIR: Psychogeriatricians could make greater use of Kopelman's evaluation of clinical tests of memory (*Journal*, October 1986, **148**, 517–525) if his organic and non-organic groups are sub-divided to allow the analysis of Alzheimer and depressed groups. This would extend the validity of several tests of memory, comprehension, and orientation as contributors to the common and problematic differential diagnosis of depressed versus Alzheimer patients. Using the number of subjects failing to score at cut-off point for each test (his Table III), the relative discrimination between these groups was assessed with the two-tail Fisher exact probability test (Armsen, 1955). The Gresham memory and orientation questionnaire, Wechsler logical memory, paired associates, name and address, and anomalous sentences tests all show significant discrimination beyond the 0.05 level of probability. Although test cut-off points were chosen to maximise the discrimination between organic (Korsakoff and Alzheimer) and non-organic (healthy controls and depressed), some show sensitivity exceeding 80% to Alzheimer's disease and specificity exceeding 80% relative to depression and healthy controls: logical memory with 45 minute retention below 50% of immediate retention; paired associates total score less than 14: name and address learnt in more than two trials. This is sufficiently promising to be worth cross-validating. A table of discrimination, sensitivity, and specificity is available from the author.

The publication of revised cut-off points chosen to maximise the discrimination of Alzheimer's disease from depression and normal controls, taking into account the base-rates for these pathologies, would greatly assist the selection and monitoring of cases in clinical trials. The availability of sensitivity, specificity, and misclassification rates as a function of test score (Anthony *et al*, 1982) would permit the selection of a cut-off point with high sensitivity for a

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