dysthymia, for depressed patients in primary care, bipolar patients, drug abusers, and for bulimia. These adaptations, and the clinical trial data, as well as the trials under way, have all been described in our recent book *New Applications of Interpersonal Psychotherapy* (Washington, DC: APP, 1993).

While I agree with Dr Holmes' premise about the importance of Bowlby's theory of attachment as a foundation for psychotherapy, I disagree that Dr Bowlby's work has been ignored in psychotherapy.

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Tricyclic-induced seizures and absent ECT response

SIR: The main point of the report by Drs Byrne & Silverstone (*BJP*, November 1993, **163**, 691–692) was that the patient responded well to a seizure induced by amitriptyline but not to electroconvulsive therapy (ECT).

My personal experience is that seizures induced chemically tend to last longer than those produced by ECT. In this particular case, the amitriptyline-induced seizure lasted 60 seconds, and this is probably two to three times longer than that produced by an average ECT application. Since seizure duration has been reported to be related to clinical outcome (Abrams, 1992) it is possible that the improvement following the amitriptyline-induced seizure was due to its duration. Since this patient had a propensity to develop seizures on amitriptyline, it might have been more appropriate to put the patient on an antidepressant with a low seizure activity profile. Doxepin (Ojemann *et al*, 1983) may be one of the safest antidepressants for treating the depressed patient at risk of seizures. Amitriptyline and fluoxetine (which was also tried) are both associated with a greater propensity to cause seizures in susceptible patients. Krijzer *et al* (1984) examined nine antidepressants and found amitriptyline to be the most proconvulsive. In addition, fluoxetine, when compared with other selective serotonin reuptake inhibitors, has been reported to be associated with more seizures (Ware & Stewart, 1989). Doxepin would have both reduced the risk of seizure and avoided the need to add carbamazepine and thus introduce the risk of neutropenia.

ABRAMS, R. (1992) *Electroconvulsive Therapy* (2nd edn). Oxford: Oxford University Press.

KRIJZER, F., SNELDER, M. & BRADFORD, D. (1984) Comparison of the (pro)convulsive properties of fluvoxamine and clovoxamine with eight other antidepressants in an animal model. *Neuropsychobiology*, **12**, 249–254.

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Cannabis toxic psychosis while on disulfiram

SIR: A 36-year-old man who had been on disulfiram (Antabuse) for a month smoked cannabis as a substitute for alcohol. He immediately developed an acute confusional state (with lowered level of consciousness, disorientation, misperceptions, believing the orange juice he was drinking was wine, and rambling, repetitive speech). After three days this changed to a manic picture, which was unusual in that the overactivity occurred in sudden bursts with relative normality between. This died down after 48 hours, during which he was receiving chlorpromazine. At the request of him and his wife he continued on disulfiram (without cannabis), with no further such problems. He had once taken cannabis, many years before, with no ill effects. The manufacturers of Antabuse had had one previous such report (Lacoursiere & Swatek, 1983).

LACOURSIERE, R. B. & SWATEK, R. (1983) Adverse interaction between disulfiram and marijuana: a case report. *American Journal of Psychiatry*, **140**, 242–244.

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Pain and seasonal affective disorder

SIR: I found the conclusion of Dilsaver *et al* (*BJP*, November 1993, **163**, 672–674) difficult to understand. Why should clinicians single out patients with wintertime depression to question about the experience of pain? Surely it is depression *per se*, no matter what its periodicity, which has been associated with pain. The authors, quoting von Knorring (1975) and others, state that 50–60% of all depressed patients experience pain. Of their winter depressives, 51.2% complained of pain. To my mind, this lack of a