acidosis, but preceded clinical hepatic failure. Unlike our patient, however, they were seen 48 hours after ingestion and also had circulatory disturbance and hypoglycaemia.

Loss of consciousness early with paracetamol alone is not recognised; however, profound lactic acidosis from whatever cause may be associated with coma. In our patient rapid correction of the acid base state produced only slight improvement in conscious level.

Dixon[3] proposed paracetamol poisoning as a cause of sudden death before histological hepatic damage. The ventricular arrhythmias possibly due to the acidosis noted in this case could provide a mechanism.

Plasma paracetamol concentrations of the magnitude recorded here would normally be expected to cause hepatic failure.[4] Remarkably only minor hepatic damage occurred. Evidence suggests that intravenous acetylcysteine offers maximum advantage when given within eight hours after ingestion.[1] In our patient drug absorption may have been delayed or perhaps severe acidosis interfered with the mechanism associated with toxicity. In such circumstances acetylcysteine may be of more than anticipated benefit. In late treatment (10-24 hours) Winchester *et al*[5] suggested that both acetylcysteine and haemoperfusion are of value in offsetting hepatotoxicity. This case supports their view.

We thank Mr W B Yeoman and the staff of the Regional Toxicology Laboratory, Dudley Road Hospital.

[1] Prescott LF, Illingworth RN, Critchley JAJH, *et al.* Intravenous N-acetylcysteine: the treatment of choice for paracetamol poisoning. *Br Med J* 1979;ii:1097-100.
[2] Record CO, Iles RA, Cohen RD, Williams R. Acid-base and metabolic disturbances in fulminant hepatic failure. *Gut* 1975;**16**:144-9.
[3] Dixon MF. Paracetamol hepatotoxicity. *Lancet* 1976;i:35.
[4] Prescott LF, Roscoe P, Wright N, *et al.* Plasma paracetamol, half-life and hepatic necrosis in patients with paracetamol overdosage. *Lancet* 1971;i:519-22.
[5] Winchester JE, Gelfand MC, Helliwell M, *et al.* Extracorporeal treatment of salicylate or acetaminophen poisoning—is there a role? *Arch Intern Med* 1981;**141**:370-4.

(*Accepted 11 June 1982*)

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Effect of two hypnotic drugs on actual driving performance next morning

Most drugs that affect the central nervous system impair driving, at least temporarily.[1 2] Furthermore, many hypnotic drugs of the benzodiazepine group have some "hangover" effects next morning and have been shown to impair performance in (experimental) psychomotor tasks, though the degree of impairment depends on the dose of the hypnotic, its plasma half life, and individual variability.[3] Such impairment is taken to mean that morning driving might be impaired: but is it? We could find no evidence that other workers had looked at the effect of these drugs on actual driving so we decided to do so. We chose a drug with a relatively short half life, temazepam, and one with a longer one, flurazepam.

Methods and results

Twelve professional women were recruited (women more often take hypnotics than men). All were fit and well, drove regularly, and had passed their driving tests at least two years before. All gave informed consent and avoided other drugs and alcohol over the test period. Each subject served as her own control and took a single dose of either placebo, flurazepam 15 mg, or temazepam 20 mg in a balanced-order design (a double-placebo technique was used, as the temazepam and its placebo were in soft capsules). Doses were taken a week apart and, for each subject, at the same time of night. Exactly 12 hours later the subject arrived at the test centre, all tests being carried out between 9 and 11 am. After objective and subjective tests of arousal and mood (to be reported elsewhere) she walked to a nearby car park, where the driving-test course was laid out. A standard Datsun saloon was used: none of the subjects had driven the particular model before. Two minutes' practice with the car over the course before testing was allowed for each subject.

Each subject drove three times over the first test course, a weaving task. She had to drive as fast as possible, weaving in and out between plastic bollards over a measured course, turn round, and come back to the starting point without hitting any of the bollards. Subjects were told there were financial penalties for going too slowly or striking bollards. Note was made of the time taken to complete the test and of the number of bollards hit. The second test was a gap-acceptance task, in which the subject drove 10 times round a circular course: at one part of the course she was presented with a gap formed by two bollards (the width between them was varied each time by the experimenters), and at 30 m she had to decide instantly whether the gap was wide enough to drive through (in which case she did so) or too narrow (in which case she drove past it). Note was made of the time taken to complete the whole task, the number of passable and non-passable gaps attempted or rejected, and the number of hits occurring while passable gaps were negotiated. Both tests were similar to tasks in other driving experiments.[1 2 4] Results were analysed using appropriate non-parametric statistical techniques.[5]

After taking flurazepam subjects hit significantly more bollards in the first test than under placebo conditions (table). In the second test subjects taking both drugs hit the side of the passable gaps significantly more often than when taking placebo, without a concomitant increase in speed or decrease in ability to recognise a passable gap; this suggested carelessness rather than increased risk taking. Out of 144 possible non-passable gaps in the second test, only five were attempted.

Number of hits in two tests

	Weaving test: bollards hit	Gap test: "passable"* gaps hit in passing through
	Placebo	
Total	21 (36 attempts)	18 (72 attempts)
Mean	1·75 ± 2·14	1·5 ± 1·2
	Flurazepam	
Total	41† (36 attempts)	33‡ (72 attempts)
Mean	3·42 ± 2·9	2·75 ± 1·4
	Temazepam	
Total	29 (36 attempts)	35‡ (72 attempts)
Mean	2·4 ± 2·0	2·9 ± 1·4

*"Passable" gap was wider than car: all passable gaps attempted.
†Significantly more than placebo ($p < 0.05$; randomisation test).
‡Significantly more than placebo ($p < 0.025$; randomisation test).

Comment

A single night-time dose of both hypnotics caused changes in driving behaviour next morning that increased the chance of a road accident. Whether the effect wears off in the day (and if so, when), whether subjects adapt to repeated dosing, whether the effect is dose dependent, and whether men would be affected we cannot say; we suggest, however, that doctors should advise patients to avoid morning driving for the first few days of taking one of these hypnotics. The effect of temazepam was unexpected, as it has a short half life and has little effect on psychomotor tests the next morning[3]: the study should be replicated.

We thank Alan Wilkinson and Alison Crowe for help in the rest of the experiment, and Peter Harvey for statistical advice. The help of Dr E H L Harries, of BioMedical Services, is also acknowledged, and we especially thank our subjects.

[1] Betts TA, Clayton AB, Mackay GM. Effects of four commonly used tranquillisers on low speed driving performance tests. *Br Med J* 1972; iv:580-4.
[2] Clayton AB, Harvey PG, Betts TA. The psychomotor effects of atenolol and other antihypertensive agents. *Postgrad Med J* 1977;**53**:157-61.
[3] Bond A, Lader M. After effects of sleeping drugs. In: Wheatley D, ed. *Psychopharmacology of sleep*. New York: Raven Press, 1981:177-97.
[4] Clayton AB. The effects of psychotropic drugs on driving related skills. *Hum Factors* 1976;**18**:241-6.
[5] Siegel S. *Non parametric statistics for the behavioural sciences*. New York: McGraw-Hill, 1956.

(*Accepted 29 June 1982*)

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