

## Non-fatal drug overdoses in a region of Birmingham

A.J. BEARD, B.A. HEMSWORTH & N. WRIGHT

*Department of Pharmacy, University of Aston in Birmingham, Birmingham B4 7ET. and Regional Poisoning Treatment Centre, Dudley Road Hospital, Dudley Road, Birmingham B18 7QH.*

The present study was carried out between December 1975 and August 1976 to investigate some of the factors involved in self-administered drug overdose. During this period, 204 patients were admitted to Dudley Road Hospital, Birmingham, following a drug overdose. These figures do not include children under the age of 12 years who are generally admitted to the local Paediatric Department. Of the 204 patients, 56 were males and 148 were females. During the period of the study, 2 males were readmitted and 8 females were readmitted after a second overdose. The information obtained in the study was provided by the patients on the day following their admission by interview conducted with the help of a questionnaire.

The drugs misused were mainly sedatives, tranquilisers and antidepressants. These together accounted for 50% of all cases involving single drugs. Aspirin and other salicylate analgesics accounted for 12%. Of all patients interviewed 58% took drugs prescribed for themselves by their own doctor. The remaining patients obtained the drugs from a variety of different sources.

The peak age range for self poisoning admissions was 15-25 years, although there was a small peak at 40-45 and another peak for the over 60s. The peak for females occurred at 15-20 whereas that for males

was 20-25 years. The peak times for admission to hospital with overdose were shown to be around 12.00 and 20.00 h. Other results from the study showed that four times as many women as men gave disagreements with their parents as a precipitating factor.

There were very few cases where the patients did not freely admit that the act was deliberate. The underlying reasons for the overdose proved to be, in the vast majority of cases, non-psychiatric, these including most often marital strife, boy/girl friend trouble, parental problems and money troubles. Depression was the most common psychiatric diagnosis where formal psychiatric interviewing took place.

The data presented here is of interest because many previous studies (Smith, 1972; Burke, 1976) have been a result of retrospective studies whereas the present information was obtained from the patients during their stay in hospital.

This study was carried out as a pilot of a larger survey which will include approximately 1500 patients and go into greater detail about the social background of the patient and his or her medical treatment while in hospital. The results of the wider study will be analysed using a computer to show up any possible interesting interrelationships of factors.

## References

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## Dopaminergic inhibition of transmitter release as a mechanism of vasodilatation in the dog hindlimb

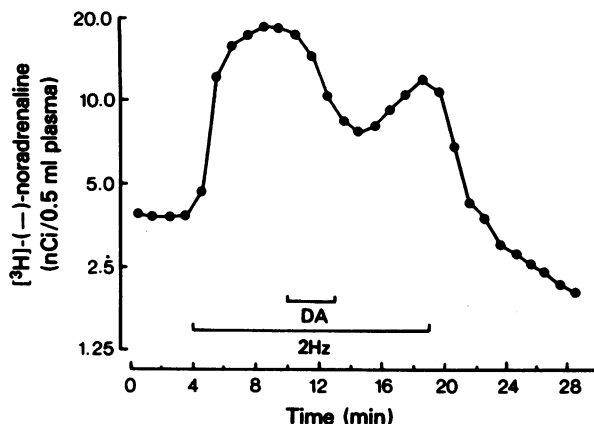
M.G. BOGAERT, W.A. BUYLAERT, A.F. DE SCHAEPRYVER, E.J. MOERMAN & J.L. WILLEMS

*Heymans Institute of Pharmacology, University of Ghent, Belgium*

Injection of apomorphine into the innervated hindlimb of the dog produces a haloperidol-sensitive vasodilatation. We have postulated that this vasodilatation is due to an inhibition of the release of norad-

renaline and that this inhibition is mediated by pre-synaptic dopamine receptors (Buylaert, Willems & Bogaert, 1977). We have now investigated the effect of apomorphine and dopamine on the release of noradrenaline in the canine gracilis muscle perfused at constant flow. The muscle was loaded with [ $^3$ H]-(-)-noradrenaline and the venous overflow of [ $^3$ H]-(-)-noradrenaline, in presence of phentolamine ( $10^{-6}$  M) and cocaine ( $3 \times 10^{-5}$  M), was measured (Rosell, Kopin & Axelrod, 1963). During electrical stimulation of the lumbar sympathetic chain, apomorphine, dopamine or saline was infused into the gracilis artery.

In 5 dogs, infusion of apomorphine ( $0.12 \times 10^{-8}$  mol/min) caused a mean decrease of  $24 \pm 3\%$  of the concentration of [ $^3$ H]-(-)-noradrenaline in the



**Figure 1** [<sup>3</sup>H]-1-noradrenaline in the venous effluent from a perfused canine gracilis muscle. 2 Hz: electrical stimulation of the lumbar sympathetic chain (supramaximal. 1 msec duration). DA: intra-arterial infusion of dopamine ( $0.12 \times 10^{-8}$  mole/min).

venous effluent. In 5 other dogs, infusion of saline provoked a decrease of only  $6 \pm 2\%$  ( $P < 0.05$ ; Mann-Whitney U-test). In 5 dogs infusion of dopamine ( $0.12 \times 10^{-8}$  mol/min) caused a mean decrease of  $33 \pm 5\%$  of the concentration of [<sup>3</sup>H]-(-)-noradrenaline (Figure 1), while in 5 other animals, infused with saline, the decrease was only  $4 \pm 2\%$  ( $P < 0.05$ ).

In 5 dogs, haloperidol (30 µg) was injected into the gracilis artery 5 min before stimulation. In these animals, dopamine ( $0.12 \times 10^{-8}$  mol/min) caused a decrease of only  $8 \pm 5\%$  of the [<sup>3</sup>H]-(-)-noradrenaline concentration. In 5 animals receiving the solvent of haloperidol, i.e. lactic acid, the same dose of dopamine produced a decrease of  $35 \pm 5\%$  of the concentration of [<sup>3</sup>H]-(-)-noradrenaline ( $P < 0.05$ ).

These experiments show that, in the gracilis muscle, apomorphine and dopamine, under  $\alpha$ -adrenoceptor

blockade, inhibit the release of noradrenaline. The antagonism of this inhibitory effect of dopamine by haloperidol suggests that dopamine receptors are involved.

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#### References

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