Blasted with ennui

Dangers in another drug fashion

Ecstasy has been much in the news in Britain during recent months. Such a statement does not imply an epidemic outbreak of transcendental joy among a phlegmatic people but rather the arrival of yet another drug of misuse—ecstasy. The pharmacological name for this substance is 3,4-methylenedioxyamphetamine (or MDMA). It is a "designer drug" with both psychomimetic and stimulant properties.[1]

As so often happens with British drug fashions, MDMA achieved earlier notoriety in America: in perhaps 1985 it was beginning to be encountered as a recreational drug on that side of the Atlantic, and in 1987 a survey suggested that two fifths of 369 Stanford undergraduates had used it at least once.[2] There was the usual phase of therapeutic enthusiasm for the drug,[3,4] and it was advocated as adjunct to psychotherapy—providing echoes of lysergide (LSD). Allegedly it helped people to get in touch with their feelings and form close relationships. It was then shown that the drug could induce dependence,[5] which was not surprising given the chemical kinship to methylamphetamine. Next came the news that a man aged 22 had taken the drug, climbed a pylon, and died from electrocution[6]—an incident that might be expected with a psychomimetic substance. Possibly MDMA may also cause cardiac arrhythmias, at least in susceptible people.[6]

There is a sense of tired sameness as yet another drug fashion emerges with the familiar cycle of complacency followed by alarm and probable exaggeration. It was Ophelia who used the phrase "blasted with ecstasy" in relation to Hamlet's sad overthrow of mind; with current drug fashions we are perhaps in danger of being blasted with ennui. But what is new about MDMA and its congeners is the evidence that they cause damage to nerve endings concerned with serotonergic transmission in a few animal species.[7-11] There is an almost biblical vengeance about this finding: the price of ecstasy shall be damage to the capacity to feel pleasure. We seem to be dealing with a subtle and nasty neurotoxin, and whether such changes are reversible in all circumstances is still open. There must be the usual provision about extrapolating from animals to man, and the dosage equivalence also deserves scrutiny—but the comforting deception that this is just an innocent Californian fun drug no longer carries conviction.

Drugs often acquire euphemistic labels: "crack" for cocaine base, "speed" for amphetamine, "angel dust" for the highly dangerous phencylidine hydrochloride, and whisky, usquebaugh, or the spirit of life for ethanol. We should resist complicity and refer to this particular drug only as MDMA rather than giving it the benefit of the further advertising that goes so perniciously with the popular soubriquet.

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Botulinum toxin: a new ally of an old adversary

Minute doses useful for some muscle disorders

Although toxins of *Clostridium botulinum* are among the most lethal known,[1] minute doses can be used in treating ocular-facial disorders. The toxin blocks the release of acetylcholine from motor neurones, inducing paralysis lasting about four months.[2] The toxin was originally developed for treating concomitant squint, the rationale being that injecting it into the overacting muscle would cause transient paralysis; the position of the eye would be altered and hence the eye muscles would change their length. When the treated muscle recovered its function after four months the strabismic eye could be straightened and the normal sensory fusion mechanisms would maintain this position.

Unfortunately injections often have to be repeated for concomitant strabismus and the treatment has been more successful in preventing the contracture of the antagonist muscles during recovery from paralytic strabismus.[3] Limited benefits have also been reported in the treatment of dysthyroid ocular myopathy[4,5] and infantile esotropia.[6] Botulinum toxin has also transformed the miserable prognosis of patients with essential blepharospasm,[7] hemifacial spasm,[8] and to a lesser extent Meige syndrome.[9] Although patients often require injections ever four to six months, these are a welcome alternative to the uncertainty of surgery to the facial nerve or the orbicularis muscle or to the side effects of drugs.

Ptosis is sometimes a complication of toxin injections, but it may act as a natural "bandage" to the eye and hence be useful in protecting the cornea from exposure. Ptosis may be induced by a direct injection of toxin into the levator palpebrae,[10] and similar treatment may improve the unsightly upper lid retraction of dysthyroid eye disease or entropion—in this case by injecting the orbicularis of the lower lid.[11] Side effects of the injection of botulinum toxin include paresis of other extraocular muscles, but this may be prevented or reversed by injecting antitoxin.[12]

Though this treatment has been successful mostly for the extraocular and finer facial muscles, larger muscles (such as