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First use of heroin: changes in route of administration over time

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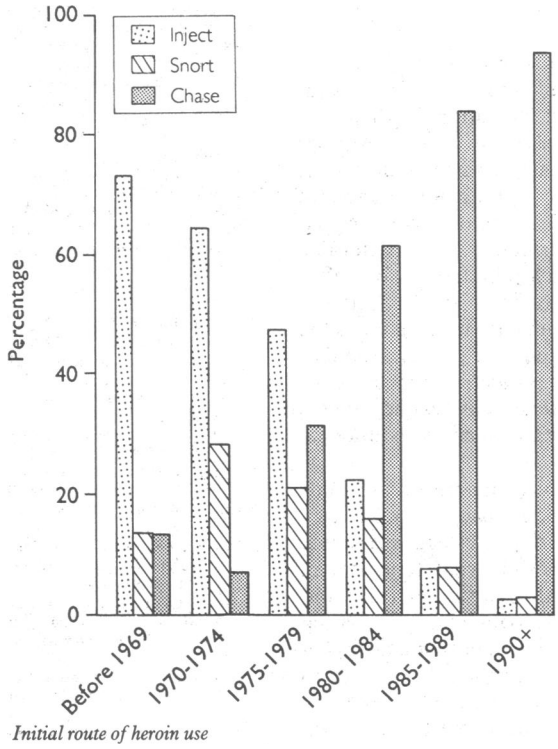
AIDS and drug misuse are linked mainly by the injection of many drugs. Major changes in the methods of heroin use, however, have fundamentally altered the importance of heroin use in the transmission of HIV. Recent reports describe the extent of "chasing the dragon" (inhaling sublimated heroin after heating it on tinfoil) as a new route of heroin use but give no information on the emergence of this pattern.[1,2] During the 1960s heroin use was by injecting.[3] What events occurred (and when) to account for this substantial change in the nature and the link with HIV of the heroin epidemic?

Subjects, methods, and results

Four hundred heroin users were contacted and interviewed by trained peer group interviewers through a structured and tape recorded interview. A total of 204 (51%) were currently out of contact with any treatment service, 100 (25%) were currently attending a drug clinic, and 124 (31%) were currently attending a needle exchange scheme. A total of 136 (34%) had never had contact with either treatment services or an exchange scheme. Their ages ranged from 17 to 53 (mean (SD) 27·6 (6·3) years); 248 (62%) were male; 96 (24%) were in current employment. There was wide variation in first year of use of heroin use (1954 to 1991): 16 (4%) started during the '60s, 28 (7%) during the early '70s, 76 (19%) during the late '70s, 124 (31%) during the early '80s, 120 (30%) during the late '80s, and 36 (9%) during the '90s.



Initial route of heroin use

Three different routes of initial drug use were identified: injecting, snorting, and "chasing the dragon." Analysis of these data by year revealed a major change in the annual proportion who were initiated by either injecting or chasing (figure).

"Chasing" was a route of initiation for a minority of users up to the late 1970s but has become an increasingly common route of initiation since 1975. By 1979 there were as many initiations by chasing as by injecting, and by 1981 more than half of the initiations into heroin use were by chasing (with the annual proportion remaining above half since 1981). By 1985 more than three quarters of initiations were by chasing, and since 1988, 87 out of 93 initiations (94%) were by chasing. During most years, a tenth to a quarter of users were initiated by snorting.

Comment

Heroin use today is not what it was yesterday. Initiation no longer occurs by injecting but by the new route of "chasing the dragon." The emergence of new non-injecting routes of heroin use may partly explain not only the major heroin epidemic in the United Kingdom during the 1980s but also its apparent continuation[4] despite the addition of AIDS as a potential consequence. Perhaps the protective societal taboo against injecting was circumvented and a less fettered epidemic has developed. In the 1990s virtually all initiations into heroin use in our London sample were by "chasing the dragon," even though heroin use in other countries (for example, the United States) and even in other British cities (for example, Edinburgh)[5] continues to be by injection. Should the change in London be regarded as an isolated development in a few "chasing" cities, or is it an indication of likely future changes on a wider scale? And what is the significance for tomorrow's prevention and treatment programmes?

Our level of ignorance about changing routes of drug administration is not only scientifically disturbing but also interferes with the development of prevention and treatment programmes. Effective primary prevention strategies depend greatly on the adequacy of knowledge about the gateways into drug use, and yet our understanding of the phenomenon is informed. largely by

study of past casualties. For example, our data indicate that a programme of preventing heroin use in the 1990s will be ineffective if it focuses on injecting: initiation into heroin use is now by chasing, and the progression (or non-progression) to injecting occurs at a later stage through influences of which we know little.

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Urinary 5-hydroxyindole acetate concentration in pregnancy induced hypertension

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The role of pressor agents, and particularly serotonin,[1] in the pathogenesis of pregnancy induced hypertension has been proposed. Abnormal circulating concentrations of serotonin have not been detected,[2] but serotonin concentrations in platelets of pre-eclamptic women are reduced.[3] A recent report of the successful treatment of an eclamptic patient with widespread cerebral ischaemia with nimodipine,[4] a potential inhibitor of serotonin, promised further study. Twenty four hour specimens of urine from patients with pregnancy induced hypertension and controls were analysed.

Patients, methods, and results

The study involved 13 women with pregnancy induced hypertension recruited from the antenatal ward over two weeks and 19 control patients of similar gestation. All but one patient had proteinuria as determined by an Albustix reagent strip. However, only five patients had proteinuria greater than 500 mg/24 h collection. In these five cases the proteinuria ranged from 0·65 to 3·64 g/24 h with a mean of 2·01 g/24 h. The clinical details are shown in the table. None of the patients with pregnancy induced hypertension were receiving treatment at the time of analysis.

Patients had a 24 hour specimen of urine collected into a bottle containing hydrochloric acid, which was frozen until analysed. The concentrations of 5-hydroxyindole acetate, a urinary metabolite of serotonin, was measured by gas chromatography and mass spectrometry without knowledge of subject group.

Clinical and laboratory details of subjects studied

	Normotensive group (n=19)		Hypertensive group (n=13)		(Mann-Whitney U test)
	Median	Range	Median	Range	
Total 24 hour urine volume (l)	1·43	0·56-2·9	1·06	0·62-1·56	NS
Gestational age at time of sampling (weeks)	33	29-39	37	30-39	NS
Maternal age (years)	26	18-31	28	19-36	NS
Systolic blood pressure (mm Hg)	120	100-130	150	140-177	
Diastolic blood pressure (mm Hg)	70	60-80	100	88-114	
5-Hydroxyindole acetate concentration (μmol/l)	18·0	8·5-51·1	30·6	17·9-44·6	<0·001
Total 24 hour excretion of 5-hydroxyindole acetate (μmol)	25·9	13·1-44·5	30·7	25·3-49·9	<0·005
5-Hydroxyindole acetate: creatinine (μmol/g)	21·8	14·1-32·5	23·6	20·0-45·4	<0·05
No of primiparous women	4		9		
No of multiparous women	15		4		

Results between the two groups were compared by the Mann-Whitney U test. Urinary excretion of 5-hydroxyindole acetate was significantly greater in the patients with pregnancy induced hypertension than in the controls (table). The difference was most marked for absolute concentrations, but was also apparent for excretion rate and concentration in relation to creatinine. There was no significant difference between the proteinuric hypertensive patients and the non-proteinuric hypertensive patients when comparing the 24 hour total urinary output of 5-hydroxyindole acetate or the μmol/g of creatinine ratio.

Comment

Serotonin is a neurotransmitter and is also found in platelets, being released into the circulation during the platelet release reaction. It is extensively metabolised, 5-hydroxyindole acetic acid being the major metabolite. We have found substantially more 5-hydroxyindole acetate in the urine of women with pregnancy induced hypertension than in normotensive controls of similar gestation. A possible contributory factor to this could be related to the significant increase in embolic trophoblastic fragments found in the venous circulation of women with pregnancy induced hypertension compared with non-hypertensive controls.[5] The trophoblastic fragments could attract platelets which in turn could release platelet serotonin. This trophoblastic factor is unique to pregnancy.

It is likely that the increased urinary excretion of 5-hydroxyindole acetate reflects increased circulating concentrations of serotonin, possibly related to trophoblastic fragmentation, which is significantly increased in women with pregnancy induced hypertension. Though this assumption contrasts with an earlier report,[2] it is doubtful if the methodologies used in previous studies were of sufficient sensitivity. We would also propose that the increased circulating serotonin is derived from platelets which have undergone aggregation as part of the pre-eclamptic process. Our observations were made when hypertension was already established and we cannot therefore draw inferences about the possible contribution of serotonin to the initiation of the pre-eclamptic process. However, we suggest that increased circulating concentrations of serotonin could contribute to at least some of the features of pre-eclampsia or eclampsia. In particular, serotonin alone or in combination with other vasoconstrictor agents could contribute to the cerebral vasospasm which seems to be a feature of eclampsia.[4]

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