

mortality among cigarette smokers is coronary heart disease. Women who take oral contraceptives are at significant risk for myocardial infarction; smoking during pregnancy increases the risk of spontaneous abortion, fetal death, and other complications—the babies being about 200 g lighter and at risk from long-term physical, intellectual, and emotional ill effects, at least up to the age of 11.

Cigarette smoking also acts as a promoter or cocarcinogen, dramatically increasing the risk to workers in asbestos, rubber, coal, textile, and uranium industries. Pipe smokers also experience a slight but definite increase in overall mortality, having a three to five times higher chance of dying from cancer of the mouth, larynx, or oesophagus. Smoking may induce allergic manifestation, interfere with certain body defences—notably in the respiratory tract—and may cause leucocytosis and eosinophilia. Being a mixture of many noxious substances, it induces enzyme changes and may alter the metabolism and clinical effect of phenacetin, theophylline, caffeine, imipramine, and pentazocine. Given that a lighted cigarette generates about 2000 compounds (separable into a solid or "tar" and a gaseous phase), no wonder that the ensuing physiological effects are quite complex. The main pharmacological agent, however, is clearly nicotine, which releases catecholamines, has a multitude of cardiovascular and metabolic effects, and is the main substance to which dependency has been related.

The report also indicates that much has been learnt about reducing the toxic activity of cigarette smoke, about the behavioural effects of smoking, the receptors for nicotine in the central nervous system, and withdrawal symptoms when people stop smoking. It is now believed that abrupt cessation of smoking is rather more effective than partial abstinence, which may induce a chronic withdrawal syndrome typically leading to relapse. It would also appear that withdrawal symptoms are more severe in women, who therefore may experience more difficulty in stopping the habit. Finally, while current evidence does not implicate smoking in the aetiology of chronic hypertension, it clearly condemns it as an additional risk factor in people who already have established hypertension—a condition that in the past decade has received much attention in the United States.

Pills galore

It is quite likely, indeed, that the vigorous effort to treat hypertension has done more to decrease the death rate from cardiovascular disease than any other change in life style. Much of the incentive for this massive effort came from the Veterans

Administration Co-operative Study, which did not, however, convincingly show the value of treating patients with diastolic pressures of 90 to 105 mm Hg. Because of this, the findings of the recent randomised study of over 10 000 people have attracted considerable attention, and the 17 to 20% lower five-year mortality rate in the treatment group suggests the need to treat even the mildest forms of hypertension.⁵ Yet there are staggering financial implications; for, while generic hydrochlorothiazide (a first-line drug) and hydralazine (a third-line drug) are quite inexpensive, the widely used propranolol, clonidine, and prazosin, when prescribed in multiple daily doses, substantially add to the overall drug bill. A recent interest in the use of the inexpensive drug reserpine is therefore noteworthy, it being declared by several authorities as effective and well tolerated, safe, and not causing breast cancer—leading the *Medical Tribune* to criticise earlier alarmist reports and to suggest that reserpine had been "smeared."⁶ Only time will tell whether reserpine can make a comeback against the more popular but also expensive other antihypertensive drugs.

Still on the subject of pills, Mr Mike Royko recently described how an unknown person decided to hand in to the police a large supply of psychotropic drugs, estimated at a street value of some \$100 000. Whatever the reason for this generous act may have been, it caused the young policeman to set about following—the regulations by counting and writing down the names and quantities of all the recovered pills—until his boss told him to stop wasting time and dump the whole lot in the garbage can. Later, the garbage man recovered the loot in the assumption that the pills might still have some potential value, but, on having a car accident, was arrested for illegal possession and put on a \$100 000 bond. It may all turn out rather embarrassing for the police, who could be accused of being accessories by giving away the material, unless, as predicted, the results of the investigation will also end up in the garbage can.⁷

References

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EDITORIAL

How is the differential diagnosis made between (a) acute alcoholic poisoning, (b) acute lysergide poisoning, and (c) acute schizophrenia when there is no history of any previous illness and a patient presents with symptoms of hallucinosis and amnesia?

The physician must obtain a complete history of the events leading up to the acute illness from the patient after recovery from the acute stages and from those who were with the patient at the onset of the illness. The diagnosis will depend very much on this history. Such conditions as delirium tremens or barbiturate-withdrawal psychosis are more likely to be the cause if the patient drank extremely heavily or took large amounts of barbiturates in the period before admission. If there is no such history they may be instantly excluded. Psychoses due to such drugs as LSD, amphetamines, or phencyclidine may again be relatively excluded if there is a reliable history that the patient never takes illicitly obtained drugs and has definitely not taken LSD or any other such substance. (There have been rare cases of hallucinations due to the accidental ingestion of psilocybin mushrooms, but the history after the event will make this clear.) If there is no history of taking drugs or alcohol, nor of their sudden

withdrawal, schizophrenia or a schizophrenic reaction must be considered a more likely diagnosis.

Drugs and alcohol may be taken by those with other illnesses, and a patient who smells of alcohol may also have ingested amphetamines or have a schizophrenic illness. If there is no history of previous schizophrenia it is unlikely that the symptoms of an acute illness of this type would have been due to the fortuitous onset of a sudden, severe schizophrenic illness in a case where drugs had been taken. Acute alcohol poisoning includes a progression from severe intoxication to coma and death. The history of ingestion of alcohol, the smell of alcohol on the breath, and blood alcohol concentrations will confirm if this is the cause. Urine analysis on admission to hospital may confirm suspicion of a drug-induced psychosis. However carefully the phenomenology, the continuing symptoms of the illness, and the mental state are examined it is unlikely that they will do much to clarify the differential diagnosis, which depends on an accurate history of the events leading up to, and the onset of, the illness. The history is all: if it cannot be obtained from the patient it must be obtained from others.