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## Sniffing Syndrome

The inhalation of vapours such as anaesthetic agents for their pleasurable effects has been recognized for many years, but the practice spread in the 1950s with the use of petrol fumes as intoxicants,<sup>1</sup> to be followed in the next decade by "glue sniffing".<sup>2</sup> A variety of products have subsequently been sniffed in the quest for pleasure and elation, the most popular being solvents, cleaners, paints, thinners, lacquers, and recently pressurized aerosols.

Glue sniffing, rarely reported in Great Britain,<sup>3,4</sup> has been the most popular sniffing syndrome in America. The practice has caused serious cerebral damage, aplastic anaemia, and even death from asphyxiation by a plastic bag used for inhalation.<sup>5</sup> Recently E. T. O'Brien and his colleagues<sup>6</sup> reported a case showing that there are serious dangers in the sniffing pastime. Acute hepatic and renal damage developed in a 19-year-old glue sniffer after inhalation of vapour from a cleaner, the main constituent of which was toluene. This toxic chemical is present in the vapours of most glues and is probably responsible for the pleasurable effects. It was found in high concentration in the patient's blood and was almost certainly the cause of the toxic symptoms.

Other varieties of the sniffing syndrome may be equally dangerous. The inhalation of petrol fumes has caused severe hepatic damage and lead encephalopathy.<sup>5</sup> Sniffing of a popular spot remover containing trichlorethylene has resulted in acute renal tubular necrosis and acute hepatic necrosis.<sup>7</sup> Possibly the most dangerous practice is the inhalation of fluorinated hydrocarbons from aerosol containers. A recent review<sup>8</sup> reported 110 cases of sudden death during the last decade in American teenage sniffers. These sudden deaths from sniffing differed from previously reported fatalities due to asphyxiation by a plastic bag in that death was usually preceded by a period of hyperactivity or emotional stress. At necropsy no physical abnormalities were found. Deaths followed the sniffing of vapours of glue, solvents, and petrol, but the largest number occurred after inhalation of aerosols in which the fluorinated hydrocarbons act as agents for propelling ingredients out of the can. Severe cardiac arrhythmias, intensified by hypercapnia, stress, or activity, were considered the most likely explanation for sudden death.

The "inert" propellant gases from pressurized nebulizers, which are also fluorinated hydrocarbons, have been shown to sensitize the hearts of mice to asphyxia-induced sinus bradycardia, atrioventricular block, and ventricular T-wave depression.<sup>9</sup> Sensitization is rapid in onset, long-lasting, and potentially lethal. The question has been raised whether sudden death in young people who inhale aerosols and in asthmatics using pressurized nebulizers could be due to a similar cardiotoxic effect, with perhaps sensitization of the heart to endogenous catecholamines, the release of

which may be enhanced by such factors as hypoxia and hypercapnia. Moreover, fluorinated hydrocarbons have recently been found in the blood of volunteers using nebulizers.<sup>10</sup> These findings may not only have a bearing on the sudden death of some asthmatics but also raise questions about the effect on persons with asthma and cardiac disease of aerosol dispensers used for cosmetic, household, and other purposes.

Diagnosis of a sniffing syndrome can be difficult, and, as O'Brien and his colleagues stress, there may be no clinical abnormality when the patient is first seen. The differential diagnosis includes alcoholic intoxication, cerebral disease, gastroenteritis, infectious hepatitis, renal failure, and various psychiatric disturbances, and the syndrome should be considered in any obscure and unexplained illness in teenagers. The characteristic smell of solvent or glue may be present, and the laboratory can be of assistance in isolating the toxic substance from breath or blood, and, with toluene, in the detection of hippuric acid in the urine.

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## Genitourinary Tuberculosis

In contrast to the steady decline in numbers of new cases of tuberculosis of the lung, genitourinary tuberculosis shows little evidence of any falling-off,<sup>1</sup> nor are new cases specially prevalent in immigrants from overseas. On the contrary, it seems to be something that the British migrant takes with him to Australia.<sup>2</sup>

There is no ready method of screening the population for genitourinary tuberculosis like mass radiography of the chest, and effective detection and treatment of the disease depend on the diligent investigation of its early symptoms. These may be slight. The earliest lesion of genitourinary tuberculosis is likely to be a small tuberculous focus in a renal papilla. It will be silent until it ruptures into the renal pelvis and discharges its more or less irritating contents into the urine. At this stage the patient may have irritation of the bladder, frequency, and discomfort on voiding, and cystoscopy may show some inflammation round a ureteric orifice and even some minute tubercles. But often the symptoms are mistaken for those of bacterial cystitis or of a common condition which mimics cystitis but seems to have no bacteriologically identifiable cause. The patient will usually have many polymorphs in the spun deposit of the urine, and the urine will be acid and sterile on ordinary culture. The finding of an acid pyuria should call for the special culture of six early morning specimens of urine for *Mycobacterium tuberculosis*.

At a later stage haematuria may be the symptom which brings the patient to the doctor, and since every case of haematuria should be fully investigated by excretion pyelography and cystoscopy few cases of tuberculosis with this symptom will be missed. Less often it is ureteric colic without obvious haematuria which troubles the patient. In some