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Title

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Permalink

<https://escholarship.org/uc/item/5498r116>

Journal

The Medical Journal of Australia, 146(3)

ISSN

0025-729X

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Publication Date

1987-02-01

DOI

10.5694/j.1326-5377.1987.tb120148.x

Peer reviewed

LEADING ARTICLES

Smoking and drinking problems in young Australians

Although tobacco and alcohol are legal drugs in our society, their use by young persons has been the subject of continued concern, and has been described lately as the major public health problem that faces Western society today.¹ The habit of smoking tobacco has been clearly associated with cancers in almost all sites in the body and with cardiovascular diseases.² The effects of smoking on health represent a major financial burden to the community. There is strong evidence that the habit of cigarette smoking becomes firmly entrenched in the adolescent years, and that it is one of the most difficult addictions to treat later in life. Prevention of the uptake of cigarette smoking in adolescents is surely the most effective way to reduce the prevalence of smoking in the community.

All major health authorities in Australia currently are undertaking interventions in schools that are aimed at a reduction in the proportion of adolescents who experiment and then take up the habit of smoking. In New South Wales alone, the "Quit. For Life" project in schools had a budget of \$200 000 between 1984 and 1986. How successful are these efforts in changing trends in the uptake of cigarette smoking in adolescents? National surveys, such as the 1984 survey of smoking and drinking in Australian secondary school students that is reported by Hill et al. in this issue of the *Journal* (page 125), provide valuable information to enable answers to this question. The large sample size (23 000 students) allows small trends to be detected on a state-by-state basis. The 1984 data show clearly why interventions to reduce the uptake of smoking are greatly needed; just under 15% of 3484 12-year-old children reported themselves as smokers, and this proportion increased to over 30% of 2741 adolescents at the age of 17 years. (The authors note that their estimate in the older age group is artificially low because of the lower rates of smoking in those who stay on at school.)

The paper by Hill et al. compares these results with those of previous surveys that were undertaken by the same research group in similar populations in 1967 and 1973. This comparison indicates that the prevalence of smoking has increased in girls and decreased in boys. However, the interval between 1973 and 1984 is too long to conclude confidently that the rate of smoking among girls is still rising. Because other surveys have used different questions to obtain information on smoking behaviour, and because the reliability of responses (and therefore the estimates of the prevalence of smoking) varies with different types of questions, Hill

et al. are justified in not comparing their results with those of surveys that used different questions. Other evidence suggests that the rate of smoking in girls peaked in 1983, but dropped back markedly in 1984.³ It is important that large-scale national surveys on smoking, that use consistently structured questionnaires, are repeated regularly (at least every three years), if we are to have any reliable indication of the effectiveness of continued intervention.

Hill et al. report data on alcohol use in a similar manner to data on smoking. This suggests that the early consumption of alcohol and the early use of cigarettes are assumed to represent the same type of public health problem. However, the evidence does not support such an assumption. First, the regular use of small amounts of alcohol is associated with better health outcomes than is abstinence.⁴ Further, the consumption of alcohol in our society is normal behaviour, and there is no conclusive evidence that the early onset of drinking is associated with problems with alcohol in later life. The health problems that are associated with the consumption of alcohol (which include both cirrhosis and traffic accidents) come from heavy drinking,^{4,5} and it would appear that the effect of alcohol on health appears only after a threshold level of consumption is reached rather than an increased effect with each level of consumption (dose-response). However, the evidence that is available does not even support the assumption that early heavy drinking will lead to later heavy drinking.⁶ In other words, drinking alcohol, unlike smoking, is not an addictive behaviour for most persons.

Lederman hypothesized that the distribution of alcohol consumption in a population could be described by a log-normal curve, and that the average consumption is the only important variable in predicting the proportion of heavy drinkers in the population.⁷ While the validity of this hypothesis is still debated, empirical observations support an association between the frequency of alcohol-related problems and per capita consumption.⁸ According to this model, if the proportion of non-drinking teenagers increased, then there would be fewer teenagers who were classified as heavy drinkers, and we would expect to see a reduction in road accident deaths, which is the major consequence of heavy drinking in young persons. However, evidence exists that among teenagers the heavier drinkers are the ones who are initiated into drinking later in life than are the more moderate drinkers.⁹ Therefore, the promotion of abstinence in

young persons may not have the desired effect. It would seem that we should concentrate on the problem of heavy drinking in young persons, rather than, as is done by Hill et al., on any drinking behaviour.

Other information indicates that the number of adolescent heavy drinkers in our society represents a significant public health problem.¹⁰ There is a common feeling among many health and education workers that the proportion of teenagers who drink heavily has escalated significantly in recent years. In New South Wales during 1986, the National Drug Offensive mounted a major campaign ("Stay in Control") which used social pressure in an attempt to reduce heavy social drinking and drunkenness in young people.¹¹ However, at about the same time, the volume of alcohol advertising seemed to increase enormously. Much of this advertising was targeted at young persons in particular, without even a hint that moderation in drinking should be exercised. The advertising code for alcohol should be scrutinized by public health authorities. Many current practices are clearly against the interests of the health of the community, although advertisements that concentrate on the restriction of alcohol consumption to below the legal limit for blood alcohol levels could be considered to be the exception.

Advertising that emphasizes the pleasure of drinking (so-called life-style advertising) is of greatest concern to the interest of public health. One of the goals in our attempts to prevent alcohol-related problems should be to have this type of advertising either banned completely or much more keenly regulated. The recently released draft National Health Policy on Alcohol in Australia presents several strategies for the control of the advertising of alcohol.¹² We recommend strongly that the implementation of these strategies should be given the highest priority.

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Guillain-Barré syndrome

*We draw attention to a clinical syndrome which we have observed in two cases, a syndrome characterized by motor disturbances, loss of tendon jerks, with preservation of cutaneous reflexes, paraesthesiae with slight disturbance of objective sensibility, tenderness on pressure of the muscles, little change in the electrical reaction of the nerves or muscles and noteworthy hyperalbuminosis of the cerebrospinal fluid in the absence of cytological reaction.*¹

This was the original description in 1916 by Guillain, Barré and Strohl of the disease that is now named after them.¹ However, Landry had already described the disorder in 1859,² but since this was before the introduction of the method of lumbar puncture by Quincke in 1891, the cerebrospinal fluid change, the "*dissociation albumino-cytologique*", that was emphasized by Guillain and his colleagues was omitted by Landry.

Since then, and probably because its pathogenesis remains uncertain, the syndrome, has been described under various synonyms, such as acute infectious polyneuritis, febrile polyneuritis, acute postinfective polyneuritis, acute idiopathic polyneuritis, acute immune-mediated polyneuritis and acute inflammatory polyneuropathy. Although it is to honour Landry and Strohl in spirit rather than by name, there is much to commend the use of the well known and widely employed eponym, Guillain-Barré syndrome, to describe this acute/subacute disease of the peripheral nervous system, with its characteristic clinical, electrophysiological, pathological and immunological features which set it apart from other types of peripheral neuropathy.

It is helpful to regard the Guillain-Barré syndrome as being at one end of a spectrum of neuropathy, that of the acquired inflammatory demyelinating neuropathies, with chronic inflammatory demyelinating polyradiculoneuropathy being at the other end; the two neuropathies are linked by partial forms and variants of the disease. This important spectrum of neuropathies accounts for some 40% of all adult neuropathies; they have in common the pathological features of segmental demyelination, elevation of the protein content of the cerebrospinal fluid at some stage in the course of the disease, and slow nerve conduction velocities.³ A further common

feature is their similarity to experimental allergic neuritis. This paralytic illness, which was produced in laboratory animals with an injection of nerve tissue in Freund's adjuvant, may run an acute, monophasic, mild or severe course as in Guillain-Barré syndrome or a chronic, relapsing, remitting course as in chronic inflammatory demyelinating polyradiculoneuropathy. It appears probable that in this group of conditions there is an immune response against normal peripheral myelin. In patients with the chronic relapsing form (chronic inflammatory demyelinating polyradiculoneuropathy), but not in those with Guillain-Barré syndrome, there is an increased prevalence of histocompatibility locus antigen (HLA) types AW30, AW31, DRW3 and DW3 compared with control subjects; this suggests that HLA-linked genetic factors may influence the expression of the disease and the patient's susceptibility to it.^{4,5}

In this issue of the Journal (page 130), Hankey reports from Perth on the first epidemiological study of Guillain-Barré syndrome in Australia. The annual incidence of the disease in Western Australia for the years 1980-1985 was 1.35 cases per 100 000 population which is comparable with a geographically widespread annual incidence of 0.6-1.9 cases per 100 000 population. Indeed, Guillain-Barré syndrome in Australia resembles in most respects the disease as it is encountered elsewhere. In general, Hankey found that it is more prevalent in men and in white persons than in Aborigines. The incidence of the disease peaks in patients between 16 and 25 years of age, with a smaller peak between 45 and 60 years of age. Two-thirds of all patients with Guillain-Barré syndrome had suffered an antecedent respiratory or gastrointestinal illness, which implies the involvement of viral and sometimes bacterial infective agents. Other risk factors are vaccinations and surgical procedures, while Hankey emphasizes the association of Guillain-Barré syndrome with altered immune function.⁶⁻⁸

From a clinical point of view there is little to add to the descriptions of the disease by Landry² and by Guillain, Barré and Strohl.¹ "*A syndrome characterized by motor disturbances*." The classic onset is the rapid development of an ascending, flaccid, areflexic paralysis. This is usually, but not

always, symmetrical and proximal muscles may be affected maximally. The motor weakness develops over some days but ceases to progress by two weeks in 50% of cases and by four weeks in 90%.³ The progress of symptoms then plateaus, to be followed by a protracted recovery period that may extend over some months.

"*Paraesthesiae with slight disturbance of objective sensibility*." While muscle pains and paraesthesiae are common it is unusual to find significant sensory loss. However, in one variant, sensory loss is marked with little muscle weakness and, in another (the Miller-Fisher variant), positional sensation is lost which results in marked ataxia that is associated with areflexia and oculomotor palsy. In some 50% of cases cranial nerve involvement — usually bilateral facial palsy and bulbar palsy — is present, while 10% of cases show oculomotor palsies.⁸ Rarely, papilloedema may be present. It is uncertain whether this is due to the obstruction of arachnoid villi by the elevated protein level of the cerebrospinal fluid or whether it is due to intumescence of the brain, as in benign intracranial hypertension. Extensor plantar responses occur in up to 10% of cases. Although sphincter function is usually preserved, autonomic features such as hyper- and hypotension, tachycardia and cardiac arrhythmias may develop.^{3,8-11}

"*Little change in the electrical reaction of the nerves and muscles*." More sophisticated techniques have now shown this statement to be in error; slowed nerve conduction velocities occur in up to 90% of cases of Guillain-Barré syndrome with marked slowing or abnormally prolonged distal latencies in 50% of cases.^{12,13} These changes may not be apparent until some weeks after onset but H- and F-reflexes may show early slowing in motor fibres.^{12,13}

"*Dissociation albumino-cytologique*." Although there is no diagnostic test for Guillain-Barré syndrome, an elevated total cerebrospinal fluid protein content with a near normal cell count, in an appropriate clinical setting, strongly supports the diagnosis. This change in cerebrospinal fluid is present after the first or second week of the illness in approximately 90% of cases but may not peak for several weeks later and then remain elevated for some months.^{3,10} The increase in protein level is mainly in the albumin content, with possible increases in