and recorded two eye injuries. Both patients had a hyphaema and one also had a choroidal rupture. Given that these injuries were caused by the ball, the rate of 9·4 eye injuries per 100 000 competitive playing sessions is no different from our observed rate at the 5% level of significance.

We thank M J Absolon, I H Chisholm, J I McGill, and C B Walker for allowing us to study the notes of patients under their care. We are indebted to Professor W E Waters for his helpful criticism, and we are grateful to the Southampton University Department of Teaching Media for the illustration.

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(*Accepted 9 July 1981*)

Alcohol and Alcoholism

The relation between consumption and damage

RICHARD SMITH

The centrepiece of the alcohol debate for the last two decades has been how the amount of alcohol a society consumes is related to alcohol-associated damage in that society. The initial argument was whether the two were related at all, but now the issues are more how exactly they are related, how they are related in subgroups of the society, and what determines consumption.

Achievements and failure of Sully Ledermann

The idea that alcohol consumption and damage are related in a society has its origin in the work of Sully Ledermann.[1] The mathematical details of his 30-year-old work, which was essentially theoretical, have been severely criticised, but from the ashes of his theory has risen a formidable phoenix. Ledermann made two assertions: firstly, that in a homogeneous population the distribution of alcohol consumption is a logarithmic normal curve (fig 1); and, secondly, that the number of people who drink a certain amount can be calculated if the average consumption is known. Few people interested in alcohol understand the mathematical details of Ledermann's theory just as few understand the criticisms made by Miller and Agnew,[2] Duffy,[3] and Skog.[4] But they do understand the limitations of data gathered by surveys[5] and that many graphs showing the distribution of consumption produced since Ledermann's time are not exactly log normal.[6] People, particularly those who drink heavily, underreport how much they drink in surveys. Nevertheless, the curves produced in various surveys, although not exactly log normal, are similar and are *always* skewed and unimodal.

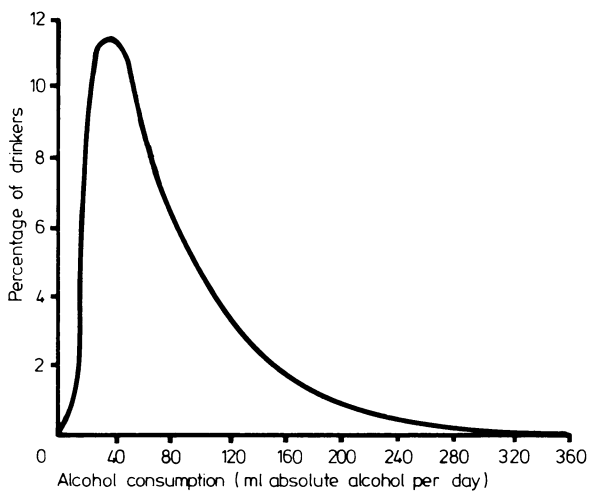

FIG 1—Hypothetical curve proposed by Ledermann that shows alcohol consumption in a homogenous population to be distributed in a logarithmic normal manner.

Empirical evidence

The evidence that consumption and damage are related depends less on theory and more on empirical evidence. Because of tax and excise many countries—including Britain—have good records of national alcohol consumption (they do not record

British Medical Journal, London WC1H 9JR
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homemade alcohol, but in most countries this is only a small part of total consumption). Some also have systems of death certification going back over 100 years. Putting together the figures for consumption and damage produces striking correlations that convince most people of the link between consumption and damage.

Table I gives figures for consumption and alcoholic deaths in Britain for quinquennia from 1885 to 1934[7]: the correlation is clear. As Spring and Buss[8] have described, Britain has had several waves of increased alcohol consumption in the past 300 years—and so far the present rise is small compared with those of the 1750s and 1870s. It is tempting but no doubt an oversimplication to point out that this 120-year cycle will give us our next peak in 1990. Professor R Kendell, chairman of the World Health Organisation Expert Committee on problems related to alcohol consumption, wonders if there is a cycle whereby one or two generations learn to drink heavily, the next then react to the increased damage with controlling measures, but then as the subsequent generations forget the damage caused by alcohol they begin to drink more and start the cycle again.

More recent evidence—from both England and Wales (table II)[7] and Finland (table III)[9 10]—links consumption not only with alcoholic deaths (as always the most easily measured of events) but also with convictions for drunkenness, hospital admissions for alcoholism, drunken driving, alcohol-related accidents, and crimes of assault. Comparing alcohol consumption and deaths from cirrhosis in various countries (fig 2) provides further evidence of the link between consumption and damage. Factors other than consumption—for instance, pattern of drinking—also determine damage but consumption and damage are clearly linked. Indeed, many swallow part of Ledermann's theory and suggest that an increase in consumption will produce a disproportionate increase in damage. Survey evidence also supports this belief: an increase in consumption of a half between 1965 and 1974 in Camberwell was associated with a threefold increase in those in the highest drinking categories.[11]

TABLE I—*Average annual alcohol consumption per caput and average annual alcohol-related mortality per million population: United Kingdom, 1885-1934, by quinquennium*

Quinquennium	Consumption in litres of proof spirit	Deaths certified as due to cirrhosis, delirium tremens, or chronic alcoholism
1885–9	17·3	154
1890–4	18·2	168
1895–9	19·1	182
1900–4	18·6	193
1905–9	16·4	156
1910–14	15·5	131
1915–19	10·5	81
1920–4	10·5	59
1925–9	9·1	55
1930–4	7·3	42

Source: Royal College of Psychiatrists Special Committee.[7]

TABLE II—*Alcohol consumption, convictions for public drunkenness, cirrhosis deaths, and alcohol-related hospital admissions: England and Wales, 1950-76*

Year	Annual per caput consumption of persons aged 15 and over in litres 100% ethanol	Convictions for public drunkenness per 10 000 population aged 15 years and over	Cirrhosis deaths with and without mention of alcohol per million population	Hospital admissions with primary diagnosis of alcoholism or alcoholic psychosis
1950	5·2	14·0	23	
1951	5·3	15·8	25	512
1952	5·3	15·8	26	668
1953	5·1	15·7	26	775
1954	5·2	15·5	26	799
1955	5·3	15·8	26	1 053
1956	5·3	17·4	26	1 385
1957	5·3	19·3	27	1 535
1958	5·3	18·7	26	1 595
1959	5·6	18·6	27	2 044
1960	5·8	19·3	28	2 479
1961	6·2	21·0	30	
1962	6·1	23·3	28	
1963	6·2	22·8	28	
1964	6·5	21·0	28	5 423
1965	6·5	19·8	29	5 774
1966	6·5	19·0	29	6 088
1967	6·7	20·3	28	6 232
1968	7·0	21·2	30	6 391
1969	7·0	21·2	32	6 689
1970	7·3	21·6	28	8 091
1971	7·7	22·9	32	9 230
1972	7·7	23·7	34	10 167
1973	7·9	25·9	37	11 565
1974	8·9	26·8	36	12 495
1975	9·4	27·0	37	12 751
1976	9·7	28·0		

Source: Royal College of Psychiatrists Special Committee.[7]

TABLE III—*Alcohol consumption per caput, arrests for drunkenness, crimes of assault and battery, cases of drunken driving, alcohol-related traffic accidents, deaths from liver cirrhosis, and deaths from alcohol poisoning, per 100 000 population: Finland, 1950-75*

Year	Consumption in litres of 100% ethanol	Arrests for drunkenness	Crimes of assault and battery	Cases of drunken driving	Alcohol-related road traffic accidents	Deaths from liver cirrhosis	Deaths from alcohol poisoning
1950	1·73	3668	148		20		
1951	1·79	3349	148	37	21	2·3	2·2
1952	1·87	3387	145	50	25	2·5	2·5
1953	1·85	3222	139	50	24	2·4	2·5
1954	1·88	3030	142	46	25	3·2	2·1
1955	1·97	3070	133	43	25	3·3	2·5
1956	1·83	2927	123	49	24	3·0	2·8
1957	1·72	2923	121	49	23	3·5	3·1
1958	1·62	2763	119	58	23	3·6	3·0
1959	1·72	2947	127	75	27	3·2	2·7
1960	1·85	2964	125	96	28	3·3	2·4
1961	2·01	3157	126	116	35	3·5	2·9
1962	2·11	2933	125	119	40	3·4	2·9
1963	2·17	3049	120	128	42	3·5	2·4
1964	2·21	2916	119	135	48	3·5	2·7
1965	2·35	3029	126	144	51	3·4	3·0
1966	2·49	3157	131	152	51	3·2	3·0
1967	2·64	3337	139	154	46	3·2	4·8
1968	2·88	3185	155	147	45	3·6	5·2
1969	4·21	2966	212	178	53	4·1	4·3
1970	4·30	3722	237	197	59	4·2	4·6
1971	4·72	4415	251	215	64	4·1	4·9
1972	5·10	4421	265	243	70	4·3	5·0
1973	5·60	4920	279	289	78	4·5	3·7
1974	6·45	6098	289	350	77	5·4	5·5
1975	6·19	5842	277	379	75	6·3	4·3

Source: Österberg E.[9 10]

Consumption and damage in subgroups

The general rule that consumption is related to damage continues to apply in smaller groups, but the pattern is not so neat. Martin Plant in his work with Fiona Pirie[6] and Suha Kilich[12] has illustrated this with studies carried out in Britain. The first was of four Scottish towns—Inverness, Aberdeen, Glasgow, and Ayr—and, although the towns with the higher consumption had more damage, the study produced examples of exceptions to the general rule. Firstly, supporting the mathematical erosion of Ledermann's formula, the town with the highest average consumption—Inverness—did not have as many heavy drinkers as Glasgow. This was partly explained by Glasgow having the most abstainers. Secondly, small variations in consumption were associated with large variations in problems. For example, average consumption in Inverness was only 49% higher than in Ayr and yet crime rates were more than 1000% higher, hospital admission rates were about 800% higher, and mortality was twice as high. Many factors must be involved to explain these differences, but to anybody who has spent Hogmanay in Inverness they may not come as a great surprise: if most of the alcohol is consumed on the two nights when the boats are in then violence, crime, and accidents are likely to be the result. Consistent heavy drinking each day—as in France—gives high cirrhosis rates, while binge drinking—as in Inverness—leads to more problems associated with drunkenness and less cirrhosis.

The second study of regional variations in alcohol-related problems in Britain also showed some inconsistencies.[12] In England and Wales alcohol-related mortality, crime, and admissions for alcoholism were all positively correlated and

related in turn to unemployment. In Scotland, however, mortality was negatively correlated with the other three measures. Martin Plant and Suha Kilich's conclusion was that this probably reflected both the poor quality of the data and Scottish idiosyncracies in recording deaths, but it does illustrate the complexities of alcohol epidemiology. Another interesting paradox is that although Scotland has higher rates of alcohol problems than England and Wales alcohol consumption does not appear to be higher.[13] As Kreitman notes: "It has been well said that the epidemiologist's primary concern with alcoholism is to escape from the field before his reputation is helplessly tarnished."[5]

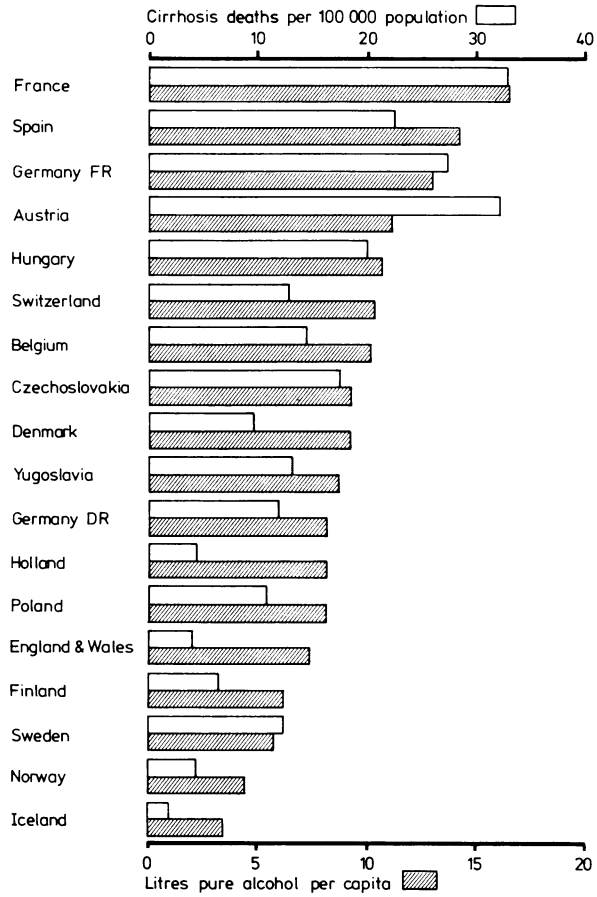

FIG 2—Liver cirrhosis death rates and alcohol consumption in various countries in the mid-1970s (source: Office of Health Economics[14]).

What influences consumption?

Working out what determines how much a society drinks is easier than working out what determines how much an individual drinks. This superiority of knowledge about societies is another argument used by pragmatists to advance the case for a societal rather than an individual approach to alcohol problems. Genetics, personality, attitudes, beliefs, religion, culture, age, sex, occupation, social class, experience, exposure, and area of residence all determine how much an individual drinks, how he drinks, and how and whether he is damaged by his drinking.[7] No doubt many factors also determine how much a society drinks,[5] but certain controllable factors—price, availability, and advertising—emerge as important.

Figure 3 shows that as the real price of alcohol has fallen over the last 30 years in Britain the amount of alcohol consumed has risen.[14] Other factors have changed too over these years, of course, but looking further back into British history shows that when the price rises consumption invariably falls and vice versa.[8] Table IV shows the strikingly close correlation between

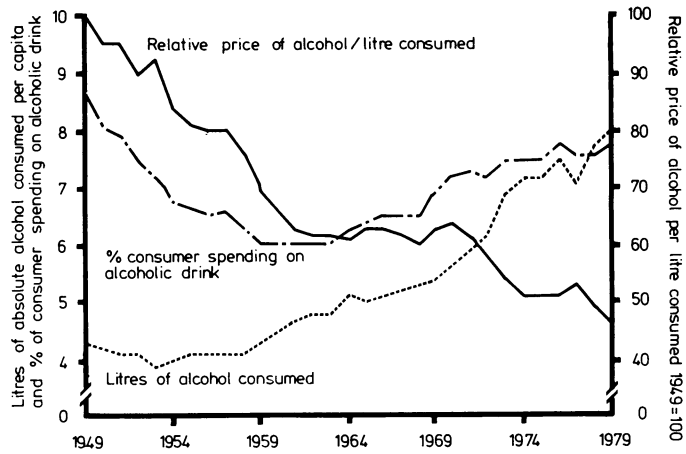

FIG 3—Relative price of alcohol in Britain related to alcohol consumption in 1949-1979 (source: Office of Health Economics[14]).

TABLE IV—*Consumption of alcohol, relative price of alcohol, and deaths from liver cirrhosis: Ontario, 1928-67*

Year	Per caput alcohol consumption in litres of absolute alcohol	Relative price	Deaths from liver cirrhosis per 100 000 population over 20
1928	2·81	0·102	4·4
1931	2·64	0·112	4·0
1934	2·09	0·137	4·2
1937	3·36	0·086	4·5
1940	3·64	0·074	5·0
1943	4·91	0·064	4·8
1946	5·82	0·069	5·4
1949	7·18	0·058	7·2
1952	7·32	0·051	7·7
1955	7·55	0·047	8·8
1958	7·96	0·043	11·0
1961	8·14	0·043	11·6
1964	8·73	0·039	11·9
1967	8·91	0·035	13·2

Source: Popham RE, Schmidt W, de Lint J.[15]

TABLE V—*Cost of beverage, alcohol consumption, and liver cirrhosis mortality in various countries*

Country	Per caput alcohol consumption in litres of absolute alcohol	Relative price	Deaths from liver cirrhosis per 100 000 people over 20
France	24·66	0·016	51·7
Italy	18·00	0·027	30·5
Portugal	17·57	0·023	48·0
Austria	14·47	0·025	38·5
W Germany	13·63	0·026	29·0
Australia	10·71	0·029	7·8
Czechoslovakia	10·27	0·080	14·8
Canada	8·95	0·029	11·6
Belgium	8·42	0·022	14·2
United Kingdom	7·66	0·057	4·1
Rep Ireland	7·64	0·092	5·0
Denmark	7·50	0·069	11·6
Netherlands	6·19	0·028	5·7
Finland	4·16	0·117	5·4

Source: Popham RE, Schmidt W, de Lint J.[15]

consumption, relative price of alcohol, and deaths from cirrhosis in Ontario from 1928 to 1967.[15] Popham *et al* studied all the data available in various jurisdictions and concluded: "Almost universally relative price was found to be very closely associated with indices of consumption and alcoholism."[15]

International comparisons (table V) of relative price and consumption of alcohol provide further evidence.[15] McGuiness[16] in his widely quoted study commissioned by the Scottish Health Education Unit (now group) concluded that price was important but that availability was also very important.

Availability and price have some tendency to vary together,

but McGuiness was struck in his analysis by the importance of the number of outlets. Certainly, many people believe that the increased availability of alcohol in supermarkets has contributed to the rise in alcohol problems among women. Licensing hours are one control of availability, and over the years what is probably a disproportionate amount of energy has been devoted to arguments about licensing laws. Evidence on the effects of licensing laws are sparse, and both the Clayson and Errol committees made their recommendations without much hard evidence. Many of the kind of people who inhabit these committees think "civilised drinking" should be encouraged as a way of reducing alcohol damage. By "civilised drinking" they mean the kind of drinking that goes on in a French cafe—with soft drinks, non-alcoholic beverages, and food available, long hours, and a "family atmosphere"—rather than that in a Scottish pub, where furniture, politeness, and women are all at a minimum. This is a subjective rather than objective judgment, and France, we should remember, has a death rate from cirrhosis that is 10 times that of Scotland. "Tavern diversification" in Ontario in 1978 did not lead to any slowing of the increase in consumption over the next five years compared with Manitoba, where previous patterns continued.[15]

Some further historical evidence is available on the influence of licensing. Lloyd George first introduced licensing laws in Britain in 1914 in an attempt to discourage drunkenness among munitions workers. Consumption fell dramatically over the next few years, but the deaths of so many young men—the group who drank the most—may have explained the fall. Also the price was increased at the same time.

In Finland in 1969 a new law intended to relax controls led to a 22% increase in shops selling alcohol, a 32% increase in restaurants with a full licence, opening hours being extended, age limits for sale being reduced, 3000 more cafes selling beer, and 17 000 more shops selling beer. The result was a sudden increase in consumption of 47%.[16] Further evidence from North America showed increased consumption among the young after a lowering of the legal age of purchase.[17] Recent relaxation of the licensing hours in Scotland, however, has not led to any runaway increase in consumption.[13]

Advertising and consumption

The role of advertising in increasing total alcohol consumption is controversial, but little evidence as to its importance is available. The drink trade in Britain spends about £100m a year on advertising, and few people are willing to accept—as the advertisers argue—that this has no effect on total consumption. (The advertisers say that advertising changes only what people drink, not how much.) The example of vodka, which over 20 years has changed from being an almost unknown drink in Britain to one that is now as popular as gin, has convinced many people of the power of advertising. McGuiness in his study concluded that advertising, particularly spirit advertising, did increase total consumption a little, and several independent economists (capable of understanding the complexities of the model) agree with his conclusions. British Columbia introduced a total ban on advertising in 1976 and there were no dramatic changes in alcohol consumption.[17] Interpretation of this is complicated because British Columbians are exposed to the media of both the United States and other Canadian States and the ban did not apply to these media. The same considerations apply to a ban on beer advertising imposed in Manitoba in 1974, when again there was no drop in beer consumption.[20] Nor is much evidence available on the effectiveness of restrictions on the style and content of advertising, but Ogborne and Smart searched for a correlation between the degree of restrictions on alcohol advertising in the 51 States in the US and the consumption of alcohol and did not find one.[19] The importance of advertising remains a moot point, but clearly those concerned to reduce alcohol consumption in Britain should not expend too much of their energy working for an advertising ban.

These studies on the factors affecting consumption have great importance because many people and committees argue that controls on price, availability, and advertising are the best way of dealing with the alcohol problem. But there is a logical step to be made from demonstrating that these factors affect consumption to being sure that policy changes would result in less alcohol-related damage. This will be discussed in the next article—on preventing alcohol problems.

This is the second in a series of articles on alcohol.

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Can the administration of sedative drugs produce an electroencephalogram simulating epilepsy?

Certain psychotropic drugs can activate the electroencephalogram and produce epileptiform abnormalities in the tracing.[1] Chlorpromazine and other phenothiazines are most often associated with this effect, but the tricyclic antidepressants, such as amitriptyline and imipramine, may have a similar effect. In susceptible subjects tonic-clonic fits may be provoked. Benzodiazepine and barbiturate drugs (apart from the anaesthetic barbiturates) do not cause epileptiform abnormalities or fits and, indeed, are used as anticonvulsants. Repeated doses of these compounds may lead to tolerance, however, and rebound fits may occur on withdrawal.—ALAN RICHENS, professor of pharmacology, Cardiff.

1 Chadwick DW. Convulsions associated with drug therapy. *Adverse Drug Reaction Bulletin* 1981;No 87:316-9.