One for the heart

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See editorial by White and McKee

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"An ounce of whisky, please Sister," or was it half an ounce or two ounces? I cannot remember now, but I know that I prescribed some tentatively after having sought the ward sister's opinion when I was called to my first patient with lobar pneumonia as a newly qualified house physician in 1937.

There was nothing else to prescribe unless oxygen was needed. In the 19th century alcohol had been prescribed for many debilitating conditions, but its medicinal use was dying out except for people who were terminally ill, and there was certainly no idea that it might be of any use in preventing disease. Some people must have seen Pearl's report in 1926 of a U shaped relation between mortality and the consumption of alcoholic beverages, but it was totally ignored by the medical profession.[1]

The situation began to change soon after the second world war, with reports of an unusually low prevalence of coronary artery disease in patients found to have cirrhotic livers at necropsy.[2 3] Necropsy series were, however, subject to many biases, and these reports excited little interest. Even in the 1970s, when case-control studies of people with and without myocardial infarcts[4] and cohort studies of people with different personal characteristics[5] began to report a reduced relative risk of myocardial infarction in people who drank small or moderate amounts of alcohol in comparison with non-drinkers, scant attention was paid to them. The belief that alcohol was bad for health was so ingrained that the idea that small amounts might be good for you was hard to envisage, and it is only in the past 10 years that cardiologists and specialists in preventive medicine have begun to take it seriously.

Summary points

- The consumption of small and moderate amounts of alcohol reduces mortality from vascular disease by about a third
- The effect on a person's risk of death depends on the relative risks of vascular disease and of the causes of death that are aggravated by alcohol
- In middle aged and elderly men in Britain the beneficial effects on total mortality outweigh the harmful effects up to at least four units a day, in women up to somewhat less
- The beneficial effect is due to the content of ethanol, not to the characteristics of any particular type of drink

Prophylactic value of alcoholic beverages

The evidence for a beneficial effect is now massive.[3 6 7] It includes not only a reduction of about a third in the risk of vascular disease but also, because vascular disease is such an important cause of death in middle and old age, a reduction in total mortality. That the reduction observed reflects a true causal relation was resisted by some specialists for many years. In many studies lifelong non-drinkers and ex-drinkers had been classed together, and Shaper et al argued that the higher mortality in non-drinkers than in light drinkers was chiefly because some who were ill, and hence at greater risk of death, had given up drinking specifically because of their illness.[8]

This explanation was not tenable, however, because studies that distinguished between lifelong non-drinkers and ex-drinkers also showed J shaped or U shaped mortality curves for vascular disease and for total mortality. An example is provided by the observations by Klatsky et al on 20 000 men and women who were examined in a prepaid health plan in California and were subsequently followed up for eight years.[9] The results are summarised in table 1. Similar relations have also been found in men and women without any major disease—in the American Cancer Society's study of 490 000 Americans aged 30 years and over[10 11] and in our study of 12 000 middle aged and elderly British doctors, 5000 of whom had specifically stated that they were free from any form of vascular disease before follow up began.[12]

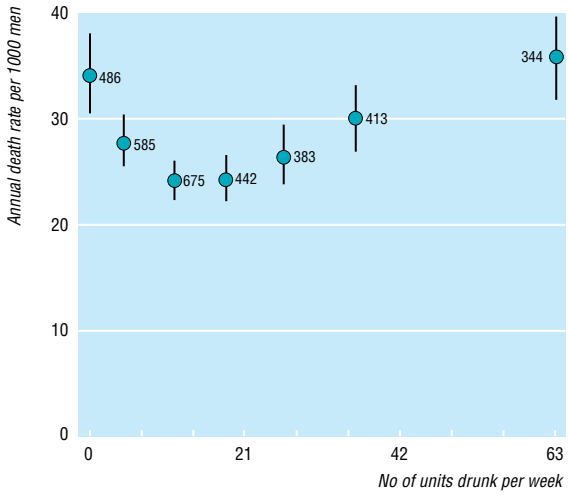
The suggestion by Shaper that the reduced mortality from ischaemic heart disease was due to confounding was also unsatisfactory,[13] as most factors that are known to increase the risk of the disease are either independent of alcohol consumption or positively associated with it and so make the observed effect seem less than it really is—for example, smoking, a high fat diet with little fruit and green vegetables, raised blood pressure, and a high body mass index.[14] Socioeconomic factors, which could have confounded the results of some studies, cannot have done so to any material extent in the comparatively homogeneous sample of British doctors and, although physical activity might have done so to some extent, it could account for only a small part of the observed relation.[12]

Some direct beneficial effect is, moreover, physiologically plausible, as is shown later, and the observed relation with vascular disease is most economically explained as one of cause and effect—a conclusion that has been accepted by the independent group of scientists that was commissioned by the European Office of the World Health Organisation to prepare a report on alcohol and public policy.[15]

The relation with total mortality in middle and old age is illustrated by the observations in the British doctors' study in the figure.[12] The increasing mortality with the amount drunk in the ascending limb of the curve after standardisation for smoking is contributed to by many causes that have long been known to be attributable to alcohol or augmented by it–that is, a few deaths from alcoholic psychosis and dependence and many more from hepatic cirrhosis, cancers of the oral cavity, pharynx, oesophagus, larynx, and liver, some cardiomyopathies, haemorrhagic stroke, and accidents and suicide.

The decreasing limb, in contrast, results chiefly from the relation with two diseases that are so common in the latter half of life that any reduction in them has a major effect on total mortality—that is, ischaemic heart disease and cerebral thrombosis. There may, however, be some contribution from other diseases that have not been adequately studied. These include several other vascular diseases, cholelithiasis,[16 17] and non-insulin dependent diabetes mellitus,[18] which Kiechl et al suggest may be reduced in incidence because alcohol improves insulin sensitivity.[19]

Many other questions also remain to be answered, some of which are socially important. One is the level of consumption at which the minimum mortality is obtained; another is the variation in the balance of overall risk with sex and age; and a third is the difference, if any, between the effects of the different types of alcoholic beverage—beer, spirits, and wine.



Mortality standardised for age and smoking according to number of units of alcohol usually drunk per week in British doctors responding to questionnaire in 1978 and followed up to 1991.[12] One unit of alcohol is half a pint of beer, glass of wine, or single measure of spirits. Values are numbers of deaths, vertical lines with 95% confidence intervals of mortality.

Table 1 Relative risks of death in drinkers compared with risk in lifelong abstainers after adjustment for age, sex, smoking, and nine other "risk factors"(after Klatsky et al[9])

Drinking category	Coronary artery disease	Cerebrovascular disease	All causes (2430 deaths)
Lifelong abstainers	1.0	1.0	1.0
Ex-drinkers	1.0	1.0	1.1
Drinkers (No of drinks):			
>1 per month (occasional)	0.8	0.8	0.9
1-2 per day	0.6	0.8	0.9
3-5 per day	0.6	0.7	1.0
⩾6 per day	0.8	1.4	1.4

Optimal consumption

Estimates of the effect of different quantities of alcohol are particularly difficult to make, principally because habits vary and people find it difficult to describe accurately their pattern of drinking and how much they drink over any prolonged period. Heavy drinkers are likely to underestimate the amount, but so are moderate and light drinkers in societies in which the consumption of alcohol may be disapproved of, as in parts of the United States. In some surveys consumption has underestimated sales figures by as much as 30-80%.[20 21] Furthermore, the official definition of a unit of consumption varies from one country to another: from as little as 6.3 g of ethanol in spirits in Austria to as much as 19.75 g in Japan. British units are uniformly defined as 8 g, which corresponds to the amount of ethanol in half a pint of beer (3.5% of alcohol by volume), but this is certainly less than most people would expect to get in a glass of wine and much less than in the tot of spirits that they would pour for themselves at home. The US unit, in contrast, is variously defined as 12 g or 14 g.

It is not surprising, therefore, that the minimum mortality has been found to occur at different levels of consumption in different studies. When Poikolainen reviewed 29 studies he found that the consumption associated with minimum mortality varied from less than one drink a day to five, with one being the most frequently reported.[7] Over half of the studies, however, were American and consequently the amounts drunk were probably underestimated. As, moreover, the American units are some 50% larger than the British, it may be that in terms of British drinks the finding in the study of elderly British doctors that the minimum mortality was associated with two or three drinks a day provides a more accurate picture.

Variation with age and sex

Some of the variation reported could, however, result from differences in the age distribution of the populations studied, for the ratio of the mortality from conditions that are prevented by alcohol and that from conditions that are made more common by it varies greatly with age. This is illustrated by the death rates from ischaemic heart disease and external injury and poisoning recorded in the developed countries with market economies in 1990. The ratios of these rates are shown for four age groups in table 2.[22] Alcohol, it would seem, is unlikely to produce any reduction in total mortality under about 45 years of age. The contrast is illustrated by the results of two studies, one of a cohort of young conscripts in the Swedish army,

Table 2 Ratios of deaths from ischaemic heart disease to deaths from violence in developed countries with market economies by sex and age in 1990 (after Murray and Lopez[22])

Age (years)	Men	Women
30-44	0.24	0.16
45-59	1.8	1.3
60-69	5.7	5.2
≥70	9.0	9.9

which showed a steadily increasing mortality with amount drunk at all levels of consumption[23] and one of Massachusetts men aged 65 years and over,[24] which showed a progressive reduction in mortality up to about three American or four British units a day.

Plato, it seems, knew something that was subsequently forgotten for 2000 years. According to Montaigne,[25] he forbad the use of wine in people under 18 years of age and intoxication under 40 years. "But," I quote, "after they have passed that age he orders them to take a pleasure in it ... intoxication being, he says ... calculated to put heart into the elderly."

Sex, too, affects the pattern, but quantitatively and not qualitatively as age does. For women in middle and old age, the optimum level of consumption may be less than that for men, not only because of their smaller size but also because of their lesser risk of heart disease, greater susceptibility to liver damage, and high risk of breast cancer, which is increased by about 10% for each additional unit drunk on average per day.[26] In compensation, however, women are less prone to death from external causes, so that they continue to have overall benefit with moderate consumption.

Variation with type of drink

There remains the question of the type of drink that produces benefit. The idea that it might differ with different types arose from an attempt by Cochrane and his colleagues of the Medical Research Council's epidemiology unit to dissect out the social and environmental factors that determined mortality in 18 developed countries.[27 28] Their findings for ischaemic heart disease at ages 55 to 64 are shown in table 3. The strong negative correlation with the consumption of alcohol was independent of, and almost as strong as, the positive correlation with the predictive index for the effect of different combinations of dietary fat given by Keys's equation.[29] The correlation with alcohol was not surprising, but it was surprising that it should have been entirely explained by the correlation with the consumption of wine ($r = -0.70$ for men and -0.61 for women).

Table 3 Correlations between certain variables and mortality from ischaemic heart disease in men and women aged 55-64 years (after St Leger et al[28])

Variable	Men	Women
Gross national product	–0.17	–0.26
Cigarettes	0.28	0.44
Saturated fat	0.64	0.62
Keys's predictive fat index[29]	0.70	0.69
Alcohol	–0.70	–0.58
Wine	–0.70	–0.61
Beer	0.23	0.31
Spirits	–0.26	–0.32

Cochrane and his colleagues were well aware of the potential pitfalls of ecological studies and were restrained in their conclusions. If, however, wine had a specific protective effect it might, they thought, be due to constituents other than alcohol, and they suggested that the next step would be "to examine the effect of alcohol and, in particular, wine on blood lipids, platelet aggregation, and such other blood constituents as may plausibly be involved in the pathogenesis of atheroma."[28] Then, if the results were sufficiently promising they would hope to see the conduct of randomised controlled trials of the preventive or therapeutic effects of moderate wine consumption. Even such a staunch advocate of randomisation as Cochrane recognised, however, that the conduct of such trials would (I quote) "have severe ethical and practical difficulties" and they have never been carried out.[28] If research led to the conclusion that wine contained a specific constituent protective against ischaemic heart disease Cochrane thought that it would be "almost a sacrilege" to isolate the effective constituent, for the medicine was already in a highly palatable form.[28]

In the event, many subsequent studies produced similar results. A few, however, showed the greatest effect with beer or spirits, while others showed similar effects with all three types.[30 31] Experiments have shown that ethanol increases the blood concentration of both high density lipoprotein cholesterol and apolipoproteins A1 and A2 and slightly reduces the level of low density lipoprotein cholesterol,[7 32] reduces the aggregability of blood platelets,[33] decreases the concentration of fibrinogen,[34] and increases the activity of the fibrinolytic system,[35] all of which would tend to reduce the risk of myocardial infarction and vascular disease in general. In contrast with these benefits, large amounts of ethanol increase blood pressure, but there seems to be a threshold for this harmful effect at about three British units a day.[14] There is no experimental evidence to show that any particular alcoholic beverage has a more beneficial effect on blood constituents or blood pressure than the equivalent amount of ethanol. And the idea that antioxidants or flavenoids or any other specific constituent of wine provides an additional benefit is entirely speculative.

That wine should have been associated with greater benefit than beer or spirits in ecological studies and in many case control and cohort studies can be explained by differences in the pattern of drinking. The effects of ethanol on blood constituents that affect the risk of thrombosis last less than 24 hours, and Jackson et al have found that this is reflected clinically in an association between the risk of myocardial infarction and the consumption of alcohol within 24 hours, irrespective of the number of drinks a week.[37] A similar finding was obtained in New South Wales by McElduff and Dobson, who also observed that greater benefit was obtained when comparatively small amounts were taken regularly, which is the way wine is drunk in many countries, than when the same total amount was taken one or two days a week, in the way that beer and spirits often tend to be drunk.[38]

There is no specific benefit associated with one type of beverage, but the benefit derives from the content of ethanol and the extra benefit associated with wine in some studies can be accounted for by differences in the pattern of drinking.

Implications for public health

The formulation of public policy is complicated by the conclusion that a certain amount of alcohol can have a beneficial effect on personal health, decreasing mortality from some major conditions to such an extent that in middle and old age it more than compensates for an increased mortality from others. Previously, policy could, and generally did, aim to discourage drinking altogether. Policies aimed solely at reducing heavy drinking had little success, and the most effective means of reducing the social and medical effects of alcohol misuse was to reduce the average amount consumed by the population as a whole, something that could be readily achieved by increasing taxation.

Precisely what public health policy should now be will not be the same everywhere, for the importance of vascular thrombosis, which may be alleviated by alcohol, and of trauma, which may be increased by it, varies enormously not only with age and sex but also from one country to another. This is shown in table 4, which gives for different parts of the world and for each sex the variation in the ratio of the number of deaths attributed to ischaemic heart disease and the number attributed to violence, omitting only deaths attributed to the operation of war. The ratio varies eightfold from the market economies of the developed world and the Middle East to China and the countries of Africa south of the Sahara, from 4.1:1 to 0.5:1 in men and from 11.5:1 to 1.7:1 in women. The balance of benefit and harm from the consumption of alcohol must therefore be different in different countries, and policies that might be good in one country could be disastrous in another. The harm that may be done by the relaxation of controls in unstable social circumstances is illustrated by the increase in total mortality in Russia between 1987 and 1994, which is most plausibly explained by the increase in the consumption of alcohol, aggravated, perhaps, by the more toxic effects associated with alcohol that is produced illicitly[39].

In Britain, where the effects on overall mortality for a person who drinks alcohol is in later life strongly in favour of benefit, other factors also have to be taken into account, such as the social effects of intoxication and chronic alcoholism and the effect of drinking even quite small amounts on the handling of vehicles and machinery. In our study of British doctors we examined the effect of the consumption of alcohol only on their own mortality and not on that of their patients. I have not attempted to quantify such other effects and it would not, therefore, be appropriate to suggest how national policy in this country should now be formulated in its entirety.

Table 4 Ratios of deaths from ischaemic heart disease to deaths from violence (excluding war) in different populations by sex in 1990 (after Murray and Lopez[22])

Population	Men	Women
Developed countries:		
Market economies	4.0	7.1
Former socialist economies	2.4	7.9
India	3.0	4.1
China	1.2	1.7
Other Asia and islands	1.5	4.1
Middle East	4.1	11.5
Latin America	1.3	4.4
Sub-Saharan Africa	0.5	2.6
World	2.1	4.4

ZOE FIGG

According to Montaigne, intoxication is "calculated to put heart into the elderly"

People should be treated as adults and should be told the facts. These still need to be refined in detail, but in broad outline they are quite clear: in middle and old age some small amount of alcohol within the range of one to four drinks each day reduces the risk of premature death, irrespective of the medium in which it is taken.

I thank Green College for having stimulated me to prepare a lecture in memory of Archie Cochrane, on which this article is largely based; a version of the lecture was also included in one chapter of *Non-Random Reflections on Health Services Research*,[31] and I also thank Professor Richard Peto for comments on the first draft.

1 Pearl R. *Alcohol and longevity*. New York: Alfred A Knopf, 1926.
2 Hall EM, Olsen AY, Davis FE. Clinical and pathologic review of 782 cases from 16,600 necropsies. *Am J Pathol* 1953;29:993-1027.
3 Moore RD, Pearson TA. Moderate alcohol consumption and coronary heart disease. *Medicine* 1986;65:242-67.
4 Klatsky AL, Friedman DG, Siegelaub AD. Alcohol consumption before myocardial infarction. Results from the Kaiser-Permanente epidemiologic study of myocardial infarction. *Ann Intern Med* 1974;81:294-301.
5 Stason WB, Neff RK, Miettinen OS, Jick H. Alcohol consumption and non-fatal myocardial infarction. *Am J Epidemiol* 1976;104:603-8.
6 Marmot M, Brunner E. Alcohol and cardiovascular disease: the status of the U-shaped curve. *BMJ* 1991;303:565-8.
7 Poikolainen K. Alcohol and mortality: a review. *J Clin Epidemiol* 1995;488:445-65.
8 Shaper AG, Wannamethee G, Walker M. Alcohol and mortality: explaining the U-shaped curve. *Lancet* 1988;ii:1268-73.
9 Klatsky AL, Armstrong MA, Friedman GD. Risk of cardiovascular mortality in alcohol drinkers, ex-drinkers and non-drinkers. *Am J Cardiol* 1990;66:1237-43.
10 Boffeta P, Garfinkel L. Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology* 1990;1:342-8.
11 Thun MJ, Peto R, Lopez AD, Monaco JH, Henley SJ, Heath CW, Doll R. Alcohol consumption and mortality in middle-aged and elderly US adults. *N Engl J Med* 1997;337:1705-14.
12 Doll R, Peto R, Hall E, Wheatley K, Gray R. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. *BMJ* 1994;309:911-8.
13 Shaper AG. Alcohol and coronary heart disease. *Eur Heart J* 1995;16:1760-4.
14 Doll R, Peto R, Hall E, Wheatley K, Gray R. Alcohol and coronary heart disease reduction among British doctors: confounding or causality? *Eur Heart J* 1997;18:23-5.

15 Edwards G, Anderson P, Babor TF, Casswell S, Ferrence R, Giesbrecht N, et al. *Alcohol policy and the public good*. Oxford: Oxford University Press, 1994.
16 Thornton J, Heaton K, Syme S. Moderate alcohol intake reduces bile cholesterol saturation and raises HDL cholesterol. *Lancet* 1986;ii:819-21.
17 English DR, Holman CDJ, Milne E, Winter MG, Hulse GK, Lewin GF, et al. *The quantification of drug caused morbidity and mortality in Australia 1995*. Canberra:Australian Government Publishing Service, 1995.
18 Rimm EB, Stampfer MJ, Colditz GA, Willett WC. Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. *BMJ* 1995;310:555-9.
19 Kiechl S, Willett J, Poewe W, Eggar G, Oberhollenzer F, Muggeo M, et al. Insulin sensitivity and regular alcohol consumption: large prospective, cross sectional population study (Bruneck study). *BMJ* 1996;313:1040-4.
20 Polich JM. Epidemiology of alcohol abuse in military and civilian populations. *Am J Public Health* 1981;1:1125-32.
21 Simpura J. *Finnish drinking habits: results from interview surveys held in 1968, 1976 and 1985*. Helsinki: Finnish Foundation for Alcohol Studies, 1987.
22 Murray CJL, Lopez AD. In: *The global burden of disease*. Geneva: World Health Organisation, 1996.
23 Andréasson S, Romelsjo A, Allebeck P. Alcohol and mortality among young men: longitudinal study of Swedish conscripts. *BMJ* 1988;296:1021-5.
24 Colditz GA, Branch LG, Lipnick RJ, Willett WC, Rosner B, Posner B, et al. Moderate alcohol and decreased cardiovascular mortality in an elderly cohort. *Am Heart J* 1985;198:886-9.
25 Montaigne M. *Essais de Messire Michel Seigneur de Montaigne Chevalier de l'Ordre du Roy et Gentilhomme Ordinaire de sa chambre*. Translated by EJ Trechmann as *The Essays of Montaigne*. Oxford: Oxford University Press, 1995.
26 Longnecker MP. Alcoholic beverage consumption in relation to risk of breast cancer: meta-analysis and review. *Cancer Causes Control* 1994;5: 73-82.
27 Cochrane AL, St Leger AS, Moore F. Health service "input" and mortality "output" in developed countries. *J Epidemiol Community Health* 1978;32:200-5.
28 St Leger AS, Cochrane AL, Moore F. Factors associated with cardiac mortality in developed countries with particular reference to the consumption of wine. *Lancet* 1979;i:1017-20.
29 Keys A, Anderson JT, Grande F. Prediction of serum-cholesterol responses of man to changes in fats in the diet. *Lancet* 1957;ii:959-66.
30 Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirit? *BMJ* 1996;312:731-6.
31 Doll R. Cochrane and the benefits of wine. In: Maynard A, Chalmers I, eds. *Non-random reflections on health services research*. London: BMJ Publishing Group, 1997.
32 Haskell WL, Camargo C, Williams PT, Vranizan KM, Krauss RM, Lindgren FT, et al. The effect of cessation and resumption of moderate alcohol intake on serum high density lipoprotein sub-fractions: a controlled study. *N Engl J Med* 1984;310:805-10.
33 Renaud SC, Beswick AD, Fehily AM, Sharp PS, Elwood PC. Alcohol and platelet aggregation: the Caerphilly prospective heart disease study. *Am J Clin Nutr* 1992;55:1012-7.
34 Meade JW, Imeson J, Stirling Y. Effects of changes in smoking and other characteristics on clotting factors and the risk of ischaemic heart disease. *Lancet* 1987;ii:986-8.
35 Hendriks HF, Veenstra J, Wierik EJMV, Schaafsma G, Kluft C. Effect of moderate dose of alcohol with evening meal on fibrinolytic factors. *BMJ* 1994;308:1003-6.
36 Criqui MH. Alcohol and hypertension: new insights from population studies. *Eur Heart J* 1987;8 (suppl B):73-85.
37 Jackson R, Scragg R, Beaglehole R. Does recent alcohol consumption reduce the risk of acute myocardial infarction and coronary death in regular drinkers? *Am J Epidemiol* 1992;136:819-24.
38 McElduff P, Dobson AJ. How much alcohol and how often? Population based case-control study of alcohol consumption and risk of a major coronary event. *BMJ*1997;314:1159-64.
39 Leon DA, Chenet L, Shkolnikov VM, Zakharov S, Shapiro J, Rakhmanova G, et al. Huge variation in Russian mortality rates 1984-94: an artefact, alcohol, or what? *Lancet* 1997;350:383-8.

What's in a Gnome?

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The Gnomes were conceived in Gothenburg in 1967 at the second meeting of the European Association for the Study of the Liver (EASL) because of a need to regularise the then confused nomenclature of chronic hepatitis. A small group of interested hepatologists met and, after animated discussion, agreed to circulate slides of relevant liver biopsy specimens and to reconvene the following year.

The second meeting in Zürich led to the publication of the classification of chronic hepatitis used for the next 25 years.[1] Professor (now Dame) Sheila Sherlock commented that the members were manipulating the nomenclature of liver disease just as the "Gnomes of Zürich" were then said to be manipulating Europe's finances.

The name stuck. Attempts to call the group by more formal titles failed. After 1968 the Gnomes perceived a need to discuss other hepatological topics, and have since written eight more guideline papers. Each year relevant slides are circulated and discussed. Presentations are frequently interrupted by one or other Gnome jumping up excitedly to voice a point of view or to express a new idea. Most importantly, each member has over the years felt free to express ignorance as well as knowledge: reputations are not at stake. The meetings provide an opportunity to share research data and to provide an update on developments in hepatology.

A few years ago one of my students, hearing about the group during an undergraduate tutorial, persuaded his mother to construct a hat suitable for gnomes to wear. This has since been passed from chairman to chairman (figure).

So much for Gnomes. What about Elves? A young American pathologist working with me at the time of the third London meeting of the Gnomes in 1989 was impressed by what he saw as a productive and enjoyable way of working. He and two European colleagues decided to start a new group along broadly similar lines. Once again, more cumbersome titles faded and the Elves were born.

Professor Roderick MacSween, currently president of the Royal College of Pathologists, wearing the ceremonial headgear (note bell on top). Next to him is Professor Amelia Baptista from Lisbon

Perhaps there are similar working groups out there discussing other organs and disciplines.

I thank my fellow Gnomes for facts and encouragement.

1 De Groote J, Desmet VJ, Gedigk P, Korb G, Popper H, Poulsen H, et al. A classification of chronic hepatitis. *Lancet* 1968;ii:626-8.