The American comedian Henny Youngman (1906–1998) once said, “When I read about the evils of drinking, I gave up reading.” Ironic, but interestingly as though with a sense of foresight, he did not speak of giving up drinking! It is despite the fact that alcohol is responsible for increased illness, being causally related to more than 60 different medical conditions (Rehm et al., 2003). Around 4% of the global disease burden is also thought to be alcohol related, which is comparable with that attributed to the effects of tobacco (4.1%) and high blood pressure (4.4%) (Ezzati et al., 2002; WHO, 2002).

For most diseases related to alcohol consumption, a dose–response relationship exists with risk of the disease increasing with greater amounts of alcohol intake, with cardiovascular disease being a possible exception. Apart from being linked to disease, alcohol is also associated with violent crime and aggression under certain circumstances (Room and Rossow, 2001).

The relationship between alcohol and cardiovascular disease, especially hypertension and coronary heart disease, is not as clear-cut. In France, the prevalence of coronary artery disease is lower—although their diets and their dietary fat content remain the similar—compared with many other Western countries. This was attributed to their tradition of wine drinking and these observations led to the so-called French paradox (Renaud and de Lorgeril, 1992) and an often-used excuse to enjoy a nice Bordeaux with every meal.

However, both cross-sectional and prospective epidemiological studies have established a relationship between hypertension and alcohol consumption (Marmot et al., 1994; Klatsky, 1995). Initial interests in the relationship between alcohol and hypertension dates back to 1915 when Lian, a French physician, described his findings amongst wine-drinking French men (Lian, 1915). We now know that the relationship between hypertension and alcohol appears to be independent of sex, race, the type of alcoholic beverage, education, smoking, and salt intake (Marmot et al., 1994; Klatsky, 1995). More recently, a substudy of the LIFE trial reported that moderate alcohol consumption did not alter the stroke risk reduction with losartan compared with atenolol in high-risk hypertensives with left ventricular hypertrophy, but reduced the risk of myocardial infarction, however, the risk of stroke tended to increase with higher alcohol intake. Interestingly though, a J-shaped association may exist between alcohol consumption and blood pressure (Criqui et al., 1982; Corrao et al., 2000), and total mortality (Marmot et al., 1981; Gordon and Doyle, 1987). A closer examination of drinking patterns also reveals that men consuming two or more drinks per day show little or no association for increased total and CVD mortality, and the detrimental effects of heavy drinking may begin to appear with three or more drinks a day (Malinski et al., 2004). This is in keeping with the benefits of light to moderate alcohol consumption on cardiovascular mortality. However, many of the results were based on epidemiological population studies, and hence some caveats may be necessary with respect to recording actual alcohol exposure and patterns of drinking, and the relationship to CVD at the individual level.

Nonetheless, prospective studies have indicated increasing blood pressure over time and an elevated risk of developing overt hypertension with consumption of alcohol (Saunders et al., 1981). Some studies suggest the blood pressure raising effects associated with alcohol consumption are seen with ethanol consumption >30 g. The INTERSALT study also emphasized that the relationship between alcohol intake and blood pressure was probably even stronger than the relationship between salt and blood pressure (Marmot et al., 1994). As mentioned previously, sometimes a J-shaped relationship is found, where lower blood pressure levels are associated with low levels of alcohol intake, when compared with teetotalers or those drinking three or more drinks per day (Room and Rossow, 2001). Indeed, teetotalers have higher average blood pressure than those who drink alcohol in small quantities (Corrao et al., 2000; Reims et al., 2004). Conversely, systolic blood pressure falls by 2–4 mmHg with reduction in alcohol intake (Aguilera et al., 1999; Xin et al., 2001). Interestingly, the hypertensive effect of alcohol disappears within days of abstinence, with the relationship between alcohol and blood pressure being most evident in the first 24 h (Moreira et al., 1998). Alcohol consumed outside meal times appears to have an effect on blood pressure risk which was independent of the amount (Strange et al., 2004).

In this respect, the study by Ceccanti et al. (2005), in this issue of Alcohol and Alcoholism makes interesting reading. They studied blood pressure patterns over an 18 day period of 147 chronic alcoholics, who were assessed for alcohol abstinence by measuring blood alcohol levels. About 50% of these patients had high blood pressure at baseline, which was followed by a steady and sharp decline in blood pressure over the ensuing days, broadly similar to previous observations (Aguilera et al., 1999). There were neither sustained correlations between the severity of alcohol withdrawal symptoms and blood pressure, nor any relation to recent drinking patterns. After 18 days, ~20% of the study subjects were still hypertensive, although not enough is known to help rule out existence of hypertension prior to alcohol excess or enrollment.

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in to the study. Also, the study was voluntary, with ‘walk-in’ participation, and thus, ensuring total abstinence may have been difficult. Perhaps 24 h ambulatory blood pressure monitoring at intervals during the study could have yielded much more information about the hypertension burden in this population.

The variability of blood pressure can only be partly attributable to the effects of alcohol, suggesting a role for alcohol independent or dependent mechanisms, as yet unclear to influencing the blood pressure changes. Mechanisms underlying the relationship between alcohol and blood pressure remain ambiguous, though several have been proposed (Randin et al., 1995; Yamada et al., 2004). Some suggested mechanisms include stimulation of the sympathetic nervous system and the renin–angiotensin–aldosterone system, raised cortisol levels, inhibition of nitric oxide, depletion of ions, increased intracellular calcium especially in vascular smooth muscle, mediated by changes in electrolyte transport and alteration of insulin resistance. These are apart from a suggested genetic predisposition (Klatsky, 1995), effects on insulin resistance (Yamada et al., 2004), and a hyperdynamic circulation. Indeed, all or some of these may well explain the findings seen in the study by Ceccanti et al. (2005).

Despite these limitations, the study by Ceccanti et al. (2005) leaves us with a few interesting snippets. On the one hand, this cohort had a significant raised blood pressure burden which responded to abstinence. However, we already know that light to moderate drinking could have cardio-protective effects mediated through modifications in lipid and coagulation profiles, as well as lowering of blood pressures and vasodilatation (Langer et al., 1992). Hence, would they benefit from drinking in moderation rather than 100% abstinence per se? The cynics would argue that in chronic alcoholics, there is no such thing as ‘moderation’—as longstanding alcohol excess would have led to higher blood pressures, alcohol-related liver damage or cardiovascular disease—but the benefits of total abstinence also remain unclear, especially with the lack of sufficiently long-term follow-up studies to adequately address the question. Ceccanti et al. (2005) do conclude that complete alcohol abstinence ‘must be recommended to all hypertensive alcoholics’, although alcohol withdrawal-induced transient hypertension was harmless, and abstinence led to a complete recovery from hypertension in most cases. Clearly, this is relevant to the great burden of hypertension on cardiovascular disease (He and MacGregor, 2003), and abstinence of alcohol features as part of non-pharmacological management in current hypertension treatment guidelines (Williams et al., 2004). Sometimes, old aetiological and pathophysiological relationships still remain relevant in modern hypertension management.

REFERENCES


