ALCOHOL CONSUMPTION IN THE ELDERLY

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CONTENTS

INTRODUCTION

For over four thousand years, alcohol has been enjoyed in civilized communities, being prescribed by Egyptian physicians before the pyramids were built, and still available on prescription in NHS hospitals. For more than ninety percent of British adults, alcohol is an accepted part of life. The medical and social disadvantages have been understood and discussed throughout this time. Recently, however, the beneficial effects have been minimized, and the hazards exaggerated (Stuttaford, 1989).

Alcohol is popularly promoted as and believed to be everything from an appetite stimulant and pain reliever to a cure for insomnia and tension. Some claims are true, but exaggerated, others false or misleading (Yen, 1983).

The present review is particularly concerned with alcohol intake in the elderly. In 1981, 17% of the UK population was of pensionable age (Redfern, 1986). There are several possible advantages and disadvantages to drinking alcohol, including nutritional and psychological effects. As a result, it is worth exploring the benefits of including alcohol in the diet of older persons and the precautions necessary. It is also necessary to know how alcohol is likely to affect their nutritional status when consumed in moderation or to excess. Alcohol is a nutrient providing energy, and a depressant drug.

The benefits of just one or two drinks a day include increased longevity for moderate drinkers compared with non-drinkers and heavy drinkers (Lopes, 1989). Small amounts of alcohol can help people fall asleep (Mendelson, 1987). Larger amounts are not recommended, because they interfere with deep sleep (Erman, 1986; Mendelson, 1987). Alcohol can stimulate appetite and digestive secretions such as saliva. Wine can also increase the iron storage capacity (Charlton *et al.* 1964; Yen, 1983).

Hill (1983) in his paper on alcohol and brain damage does not imply that the social drinking of ethanol is harmful to the brain, but underscores the theoretical possibility that

some level of consumption may not be harmful to the brain. Even beneficial effects of light-to-moderate social drinking are theoretically possible, although no one has as yet explored that. The now well documented relationship between light drinking and increases in high density lipoprotein, a protective factor in heart disease, illustrates this point (Shaper *et al.* 1985).

The physical and mental effects of alcohol (and thus alcoholism) on older adults are often more severe than in other age groups because of decreased physical and psychological reserves as well as the coexistence of (other) disease conditions (Schuckit & Pastor, 1979).

Most societies tend to ignore or deny the seriousness of alcoholism in older adults. This lack of recognition is related to a tendency to consider the mental health problems of older adults as more acceptable (Schuckit & Pastor, 1979).

Davies (1981) refers to malnutrition in the elderly as being multifactorial in origin. If older persons are already susceptible to nutritional deficiencies, excess alcohol will compound the problems associated with their decreased ability to absorb and use nutrients. A marginally adequate diet becomes inadequate when alcohol replaces food energy and necessary nutrients (Yen, 1983).

Alcohol becomes a problem if people drink it instead of eating nourishing food. Older adults need less energy than younger people, so everything elderly people eat should contribute something to their diet besides just energy. The B-complex vitamins are those most commonly deficient in people with alcoholism. Common deficiencies are those caused by lack of thiamin (vitamin B_1) in the diet or by alcohol's interference with thiamin absorption (Yen, 1983). The reasons for these multiple nutrient deficiencies in alcoholism vary. Alcoholics may not consume enough of the nutrient. They may not absorb or metabolize it properly due to the effect of alcohol on the body, or the need to metabolize large quantities of alcohol may siphon off important nutrients (like thiamin), which are then not available for other essential functions (Yen, 1983).

Alcohol has other effects besides nutritional ones. Cancer risk is increased by drinking, especially cancer of the mouth and throat (Yen, 1983). Payne (1990) noted recent awareness among health workers that consumption of alcoholic beverages, particularly beer, may be causally related to cancers of the descending colon and rectum. Alcohol aggravates ulcers and gout, and potentiates the effects of some drugs, such as tranquillizers (Yen, 1983).

There can be a problem in defining aged populations in research with human subjects (Dye, 1982). Comparisons of older adults may range from those of 60–65 years of age through to the tenth decade. Studies of 'young' or 'mature' subjects may have a range of ten years within each subject grouping whereas the older subjects may range over a period of thirty-five years. This type of grouping implies that individuals over sixty years of age, whether sixty-five or ninety-five, are drawn from a homogeneous population. Recent studies do not, however, support this assumption. Behavioural data show that there are clearly two older developmental groups here – the young-old (defined as being between sixty-five and seventy-five years of age) and the old-old (defined as seventy-five years and older). When these two groups are combined, the variance is increased and treatment effects are masked.

Dye (1982) also says that, in the future, one might suspect that there will be differentiation of those in the group of seventy-five to one hundred year-olds. It is possible that it may become necessary to choose between three or even four groups of elderly persons over sixty-five years of age when comparing subjects over the life span.

THE NUTRITIONAL IMPACT OF ALCOHOL CONSUMPTION IN THE ELDERLY

As part of the ageing process the vitality of most organ systems declines, and there are changes in body composition, especially fluid- and lipid-containing tissues. Ethanol is distributed preferentially into body water compartments (including blood and cerebrospinal fluid), and very little into fat, mineral or cell solids. Ageing decreases the relative contribution of body water to total body weight (and increases the relative contribution of fat to total body weight), so it follows that a given dose of ethanol, on the basis of total body weight, will produce higher peak blood (and presumably brain) concentrations of ethanol in elderly subjects than in young subjects of a similar weight and somatotype (Wood & Elias, 1982).

Elderly adults are therefore potentially more susceptible than younger adults to any effects of alcohol on nutritional status. They tend to have reduced energy intakes resulting in a higher probability of marginal nutritional status. The empty energy (Cerrato, 1986) associated with alcohol could exacerbate this problem in that alcohol may replace foods with higher nutritional content. Alcohol may also interfere with nutritional status through altered absorption, metabolism or excretion of nutrients.

Although there is often an inverse relationship between energy derived from ethanol and from fat or carbohydrate, other surveys have found that alcohol calories are added to the diet rather than replacing other foodstuffs. An American study by Gruchow *et al.* (1985) found that drinkers had significantly higher intakes of energy than non-drinkers. Their intake of non-alcoholic energy decreased as alcohol intake increased, and it was estimated that between 15 and 41 % of alcoholic energy replaced non-alcoholic energy. The most salient difference in nutritional intake between drinkers and non-drinkers was the substantially lower carbohydrate intake of drinkers.

De Castro & Orozco (1990), in their study of moderate alcohol intake and spontaneous eating patterns in humans, suggest that alcohol supplements rather than displaces energy from macronutrients and that alcohol is associated with prolonged meal durations. The patient with a drinking problem is likely to have nutritional problems (Cerrato, 1986) as the empty energy provided by alcohol is not associated with the vitamins and minerals needed to utilize it. The body is likely to draw on its own reserves (which may be already depleted) to use that energy. Since few alcoholics eat a proper diet, the body's micronutrient reserves are never replenished. Lamy (1984) also points out that older people have a diminished metabolic capacity; they metabolize and excrete alcohol more slowly than younger people. Their capacity to drink large amounts of alcohol is reduced, and their sensitivity to the adverse effects of alcohol increased. Subclinical malnutrition, apparently widespread among the elderly, heightens their susceptibility to the adverse effects of alcohol.

In their study of middle-class men, Hillers & Massey (1985) identified a trend towards increased energy intake as alcohol consumption increased. As a result the nutritional quality of the diet declined. The results indicated that alcohol displaced other energy sources in the diet. Many of those with the highest alcohol intake reported that when they drank they ate less food and skipped meals.

These findings are supported and clarified in relation to the differences between the sexes by the following. Three hundred and ninety-three elderly subjects in retirement homes throughout Italy were studied by Ferro-Luzzi *et al.* (1988), who found that alcohol contributed, on average, 12 % of total energy intake in men and 6 % in women. There was a general tendency for women to add alcohol to their habitual diet, as revealed by a positive correlation between total energy intake and alcohol intake. The higher energy intakes of

heavily drinking females were also reflected in their higher body weights. Men tended to displace food energy partly by alcohol.

The 1990 consumption of alcoholic beverages per head of population in England and Wales was 7·5 litres, that is, an average of 8·8 units per week (Office of Population Censuses and Surveys, 1991). Mitchell & Herlong (1986) identified alcohol as contributing 4–6 % to the total intake of energy in the USA, not excluding children and abstainers. They quote a national probability sample in 1983 as identifying alcoholic beverages contributing 17 % of energy intake for male drinkers and 21·9 % for female drinkers. However, ethanol *per se* accounted for only 5·3 % of energy in this group after energy from carbohydrate and protein in beer were subtracted. In addition, alcohol itself may be an ineffective source of energy during times of high alcohol consumption (Mitchell & Herlong, 1986; Mahalko *et al*. 1985). The energy value of ethanol as fuel may be dose related. Most evidence suggests that at moderate intake levels of less than 45 g/d (3 drinks) ethanol is efficiently utilized as fuel by the liver. At high intakes, ethanol energy may not be utilized for cellular synthesis of ATP and maintenance of weight. The exact mechanism for this inefficient utilization remains unknown.

Davies & Holdsworth, in their 1985 study of nutrition and health at retirement age in the United Kingdom, state that the 3 % contribution by alcohol to the energy of the diet of the elderly was within National Advisory Committee on Nutrition and Education (NACNE) guidelines.

Lelievre *et al*. (1989) noted a deterioration in taste discrimination in drinkers in comparison with non-drinkers in healthy subjects. This could potentially have an impact on their nutritional status.

Health problems resulting from an excessive alcohol intake include liver disease, muscle wasting (Preedy & Peters, 1990) and demineralization of bone. These tend to be exacerbated by poor nutritional status. A solely nutritional explanation for alcoholic liver disease proved untenable (Mitchell & Herlong, 1986) –direct hepatotoxicity from alcohol alone inadequately explained the clinical, biochemical, and histologic observations in humans with alcohol related liver injury. Nutritional factors may play a role in modulating the hepatotoxicity of alcohol. The prevalence of malnutrition in alcoholics with liver disease is so high that it may be impossible to isolate completely each contributory factor.

Nutritional deficiencies are common in alcoholics even in the absence of liver disease. However the deficits are most severe in those patients with the most clinically severe liver disease. The clinical severity of liver disease is better correlated with nutritional parameters than with morphological changes seen on liver biopsy. Findings imply that patients with active alcoholic liver disease are unable to utilize dietary protein efficiently (Mitchell & Herlong, 1986).

An increased size of the red blood cell is common in alcoholic people. There are varying opinions as to whether this increase in the mean corpuscular volume (MCV) is due to dietary deficiency of folic acid, poor absorption of folic acid or a direct effect of alcohol upon the bone marrow. Hillers & Massey (1985) found an increased MCV to be a reflection of the higher alcohol consumption, but not necessarily indicating a folic acid deficiency. Yen (1983), Jaques *et al*. (1989) and Ferro-Luzzi *et al*. (1988) noted that folic acid status decreased with increasing alcohol intake. Passmore & Eastwood (1986) also noted that folic acid deficiency could be caused by ethanol, though the reduction was seldom large and anaemia was uncommon.

The fact that chronic alcoholism is associated with a decrease in food intake was also identified by Marchini *et al*. (1983). They also found increased excretion of several nutrients and damage to the intestinal tract, liver and pancreas in alcoholics. The end result is an expectation that the nutritional status of the alcoholic patient will be seriously impaired.

There will be riboflavin deficiency, and folic acid deficiency will result in anaemia (Yen, 1983; Jaques *et al.* 1989). A lack of pyridoxine causes convulsions during withdrawal from alcohol (Yen, 1983). On the other hand, Kant & Block (1990) showed in the United States that pyridoxine intake decreased with increasing age, probably due to reduced food intake, and that alcoholic beverages were one of the important sources. The study (Jaques *et al.* 1989) of 586 adequately nourished, elderly non-alcoholic subjects suggested that energy intake and blood concentrations of iron, and high-density lipoprotein cholesterol (HDL-C), increased with increasing alcohol intake. The study of elderly people in retirement homes by Ferro-Luzzi *et al.* (1988) found that the dietary risk of malnutrition, high for retinol and moderate to low for thiamin and riboflavin, did not increase with alcohol consumption. Biochemical evidence of malnutrition indicated a significant deterioration of thiamin status in heavily drinking males. The prevalence of retinol deficiency was lower in heavy drinkers, a finding that needs further study. In conclusion, Ferro-Luzzi *et al.* (1988) found that the effects of habitual alcohol consumption appeared to be gender specific and, for both sexes, to relate more to a direct toxic action of ethanol on folic acid and thiamin absorption and metabolism than to a clearcut alteration of dietary habits.

The elderly population is at risk from B_1 deficiency irrespective of alcohol intake. Thiamin deficiency in the poorly nourished, elderly alcoholic can produce confusion, weakness, and neuropathy, as noted by Pentney (1982), Young (1984) and Cerrato (1986). This is characterized by Wernicke's encephalopathy, which is an acute confusional state with double vision and unsteadiness on the feet, and may in turn lead to the permanent memory loss of Korsakoff's psychosis. This is relatively rare and can be prevented by intravenous thiamin administration. Passmore & Eastwood (1986) also concluded that thiamin deficiency (in countries where rice is not the staple food) occurs mostly in people whose diet is greatly restricted, usually as a result of chronic alcoholism. They continue to suggest that awareness of the risks of thiamin deficiency in alcoholics should be higher, as it is treatable if not left too late.

A relationship between excessive consumption of alcohol and the development of iron overload was identified by Charlton *et al.* (1964). On other hand, Chapman *et al.* (1983) found that iron absorption in alcoholics did not differ significantly from normal, though increased liver iron concentrations were seen in approximately one third of alcoholics. The latter could not be attributed to an increase in iron absorption as a result of chronic alcohol ingestion.

Passmore & Eastwood (1986) stated that iron absorption may be promoted by alcohol. Further studies were needed to elucidate the effects of alcohol on iron absorption.

Hajnal *et al.* (1990) demonstrated that adding alcohol to meals inhibits for 3 hours the secretion of pancreatic trypsin in response to the meal. This may explain some nutritional effects of alcohol.

ALCOHOL AND THE CARDIOVASCULAR SYSTEM

The identified link between alcohol and HDL-C leads logically to a consideration of the effects upon the cardiovascular system.

High-density lipoprotein cholesterol (HDL-C) showing substantial correlation with alcohol intake was demonstrated by Shaper *et al.* (1985). The effect of alcohol consumption on HDL-C is now established and has aroused considerable interest because of the possible protective effect against coronary heart disease, also identified by Ernst *et al.* (1980). In this study it was noted that those that never drank had lower mean HDL-C levels than those who did drink but had not done so in the past week. It is also interesting to note that they

found weaker inverse relationships between HDL-C levels and intakes of total carbohydrate, sucrose and starch (percent energy and grammes). The association of alcohol with HDL-C was strongest at ages sixty and older. This makes it of interest to those studying the elderly and alcohol. The New Zealand study of Jackson *et al.* (1991) also supports the hypothesis that light and moderate alcohol consumption reduces the risk of coronary heart disease, but their cohort was under sixty-five years old. It is, however, almost certain that the risks/benefits of different levels of consumption of alcohol follow a J-shaped curve relationship, with the lowest risks of cardiovascular disease among light drinkers and higher risks among non-drinkers and heavy drinkers (Moore & Pearson, 1986). The margin between safe and dangerous drinking is likely to be a very narrow one. Shaper (1990) has argued that the reference group of alcohol abstainers or low consumers is typically contaminated with subjects who have reduced alcohol consumption because of pre-existing disease, and recommends (Wannamethee & Shafer 1988) the use of men who drink fewer than fifteen drinks a week as a reference group. The USA Health Professionals Follow-up Study (Rimm *et al.* 1991), adjusted for dietary intake of cholesterol, fat, and dietary fibre, showed an inverse relationship between increasing alcohol intake and coronary disease incidence, despite excluding current non-drinkers. They also found that the inverse association between alcohol and coronary artery disease did not appreciably differ by age or smoking status. Lazarus *et al.* (1991) in their California study attempted to differentiate between long term abstainers and more recent non-drinkers. They classified their cohort as 35 and over, with no top age given, and identified an increased risk of death from all causes and from ischaemic heart disease associated with women who had given up drinking, rather than long term abstainers. In men, long term abstainers were possibly at increased risk of death, when compared with those who continued to drink.

Alcohol, above certain levels in blood, becomes a depressor of myocardial fibre (Lopes, 1989). Excessive intake of alcoholic drinks is responsible for dysrhythmias, congestive heart failure, thromboembolitic phenomena and, sometimes, sudden death. Dilated cardio-myopathy is the usual clinical presentation of these patients. Consumption of more than 60 g/d of ethanol may be an important factor in the aetiology of high blood pressure.

In a review of the epidemiological evidence linking moderate alcohol consumption and the risk of stroke, Camargo (1989) showed that moderate drinking (less than 60 g ethanol/d) and ischaemic stroke have some association in predominantly white populations, noting the J-shaped association. It was also noted that moderate drinking increases the risk of intracerebral and sub-arachnoid haemorrhage in diverse populations.

The list of cardiovascular effects of alcohol reviewed by Davidson (1989) includes modification of the risk of coronary artery disease. He quotes cross-sectional studies in which people who drank fewer than six units a day of alcohol were shown to be less likely to have coronary artery disease than heavier drinkers and abstainers. The list continued with development of alcoholic cardiomyopathy, exacerbation of conduction disorders, atrial and ventricular dysrhythmias, and an increased risk of hypertension, haemorrhagic stroke and infectious endocarditis.

It thus appears that the cardiovascular risks of alcohol consumption may well prove, over time, to outweigh the advantages.

ALCOHOL AND SLEEP

A popular use of alcohol is as a hypnotic drug, to relax the subject and induce sleep. Alcohol appears to reduce sleep latency, even if consumed some hours beforehand, so that there is rapid onset of sleep, but the onset of rapid eye movement (REM) sleep is delayed,

and there is a reduction in REM sleep in the latter part of the night. The drinker wakes up early and cannot fall asleep again. The REM suppression is dose related; that is, the higher the dose, and the less they normally drink, the longer the REM suppression experienced (Mendelson, 1987). As elderly people may have unrealistic expectations of the amount of sleep they can hope to gain, they may use alcohol as a strategy.

These findings are supported by Erman (1986). Consuming two or more units of alcohol may 'knock out' the weary insomniac, but the nightcap may damage the quality of sleep for the rest of the night. In some cases, alcohol can trigger life-threatening problems. There is evidence that even one unit of alcohol taken less than an hour before going to bed disturbs the pattern of sleep, making it light, unsettled and less than refreshing. The person initially lulled to sleep by drink may sleep soundly for the first few hours, but then awaken or sleep poorly. The sleeper may then experience nightmares and feel unusually tired the next day. People who drink regularly may also experience nightmares on nights when they refrain from alcohol. The potential for abuse of this home cure is high because the amount of alcohol needed to induce drowsiness increases rapidly in a short space of time.

Alcohol, states Erman (1986), is a major cause of insomnia because it disturbs the REM cycle, which appears about 80–90 minutes after the onset of sleep (Borbely, 1987). The first REM phase lasts about 10 min. For the sleeper who has had a few drinks of alcohol before retiring, alcohol in the bloodstream acts as an enemy to REM, fighting off the cycle. About five hours after sleep occurs, as the body eliminates the alcohol, sleep becomes disturbed for the rest of the night. One reason for this disturbance is that REM sleep 'rebounds' and can return with a vengeance to intrude on the other cycles and deprive the body of deep, restful sleep.

For several nights after a habitual drinker stops drinking, the person may suffer from nightmares stemming from REM sleep rebound. An alcoholic on a drinking binge may suppress REM sleep for so long that it will rebound when the individual is awake. Researchers have theorized that delirium tremens (hallucinations and muscle jerks) are actually portions of the REM cycle in action while the alcoholic is awake (Erman 1986). The elderly person consuming alcohol to help them get to sleep may find it an unhelpful strategy.

To develop Erman's (1986) point about life-threatening disorders, alcohol is also believed to trigger or aggravate sleep apnoea (SA) in many people. This is a disorder characterized by snoring and periods of not breathing during sleep. This problem is common, potentially life-threatening, and is seen more frequently in older people. SA is especially dangerous to those with a history of pulmonary or cardiac arrest. Potentially fatal conditions, including an erratic heartbeat, severe drops in oxygen levels in the blood and a rise in blood pressure can be triggered by SA. For those already suffering from cardiac and pulmonary disease, even moderate drinking before bedtime presents a risk. These effects of alcohol may be even greater in men than in women (Erman, 1986).

The conclusion may be drawn that alcohol has an extremely limited place in the promotion of sleep in elderly patients. Only those with onset difficulties are likely to find it helpful.

ALCOHOL, MOOD AND ANXIETY REDUCTION

Other popular effects ascribed to alcohol by the lay person include alterations in mood to induce relaxation, increased sociability and reduction in anxiety.

In an attempt to discover why people drink normal amounts in natural settings, Kalin *et al.* (1965) found that alcohol had no effect on a fear-anxiety measure in discussion

sessions, but a significant reduction in fear-anxiety was observed in an experimental party setting after alcohol ingestion. Alcohol generally produced a decrease in 'inhibitory thoughts', after four or five units. Williams' (1966) similar study tested the hypothesis that alcohol decreases anxiety and depression. This was found to be so after 4 ounces of a commercial alcoholic beverage, but there were significant increases to previous levels after more beverage was consumed.

Cloninger (1987) identified two groups of alcoholics, with a range of characteristics associated with each. These groups were: type II (juvenile onset) and, likely to include the elderly patient, Type 1 (mature onset). At rest, the latter are hypervigilant and apprehensive, with much anticipatory worrying; their resting electroencephalogram (EEG) shows minimal brain wave activity on the slow alpha frequency, excessive beta activity and poor synchrony. In response to alcohol these individuals show a marked increase in alpha activity and subjectively report a sense of calm alertness that they regard as a pleasant relief of tension (Cloninger, 1987).

Much of the evidence for the tension reducing effects of alcohol has been identified as being negative, equivocal and often contradictory (Cappell & Herman, 1972). They quote Conger's classic 1956 reinforcement theory of alcoholism, containing two distinct hypotheses: (1) alcohol reduces tension, and (2) organisms drink alcohol for its tension reducing effect.

In a cross-sectional study, comparing the psychological profile of light social drinkers with that of heavy social drinkers, and alcohol dependent patients showing no evidence of clinical depression, Robson (1989) found that the dependent patients demonstrated significantly greater distortion of cognitive style than heavy social drinkers, who in turn rated significantly higher than light social drinkers. Heavy social drinkers occupied a middle ground with regard to psychological profile between light drinkers and 'alcoholics', sharing some attributes with each.

There can be no clear conclusions drawn from these studies. Individuals apparently will develop their own perceptions of what they feel alcohol 'does' for them, and the elderly drinker may be very fixed in this belief, despite evidence to the contrary.

THE SOCIAL AND PERSONAL IMPACT OF ALCOHOL CONSUMPTION IN THE ELDERLY

Excessive alcohol consumption in the elderly can be said to have a social and a personal impact in a number of ways. The physical damage to sight, hearing, kinaesthetic sense, muscle, bone, and other body systems that may result can lead to a higher rate of accidents, falls, and hospitalization for a range of health problems, caused or exacerbated by the alcohol and increased physical vulnerability. The mental damage, especially the effects on short term memory and cognitive loss, may lead the elderly person to require a higher level of support from the community and an earlier onset of some form of institutionalized care. It also increases the risk of suicide. Both physical and mental effects are potentially very costly to the nation, as well as distressing to the individuals concerned.

Alcohol abuse may increase in some types of elderly people (Wood & Elias, 1982), e.g. those stressed by the ageing process, retirement and widowhood, leading to anxiety, depression and loneliness. Drinking among the elderly in the general population is a deeply rooted behaviour, developed over the course of a lifetime in response to social norms and numerous other psychosocial and biological factors. It is less likely that the elderly will suddenly begin problematical drinking in response to the stresses of ageing if they have not previously used this coping mechanism in younger years to deal with life crises (Wood & Elias, 1982). Late onset alcoholism is more often associated with the loss of social support

systems such as the loss of a spouse; the presence of other environmental stress, such as a change in living arrangements, is also identified by Simon (1980). This could be reasonably applied to an elderly person entering residential care, for example.

PHYSICAL DAMAGE

Sensory alterations due to old age are well documented (e.g. Redfern, 1986). When that person also abuses alcohol, the sensory losses are likely to be more severe, with resulting social costs of isolation, vulnerability, lack of awareness of dangers in the environment, diminished quality of life and so on.

The effects of alcohol upon sight, including visual field restriction, impaired peripheral vision, diminished depth perception, decreased reaction speed of the eye, visual blurring, restricted night vision, and decreased ability to distinguish colour are identified by Squires *et al.* (1985). They also note the impact of alcohol upon hearing, which includes shifting of the acoustic reflex threshold, and suggest that retinol deficiency in alcoholics leads to poor hearing. Decreased kinaesthetic sensitivity has also been observed in patients with alcoholic polyneuropathy. The latter may well render an old person more likely to falls.

As the degree of alcohol abuse in the elderly increases, the more neurophysiological systems are vulnerable to damage, such as the vestibular sense (Blusewicz *et al.* 1982; Squires *et al.* 1985). Duthie (1989) looked at falls in the elderly, and cited alcohol use as one of the causes, increasing morbidity and mortality. A moderate intake of less than two units a day can be a predictor of accidental death, as noted by Ross *et al.* (1990) in their study of risk factors in a Californian retirement community. Age Concern's UK (1977) profiles of the elderly show a sharp increase in deaths from accidents, falls, fire and accidental poisoning in the over-75s compared to the rest of the population. There is also an increased incidence of deaths from burns and scalds, and road traffic accidents, especially as pedestrians. Logically, alcohol is likely to be implicated in many of these. Stewart's (1989) findings have a bearing here, in that elevated blood alcohol was consistently found to be associated with shorter survival times after severe injury for those attending an Accident and Emergency Unit. One in four emergency general medical admissions can be directly or indirectly related to alcohol (Lockhart, 1986).

MENTAL IMPAIRMENT

Physical damage is not the only effect of excess alcohol intake in the elderly. The effects of mental impairment may be the first to be noticed by carers.

It is clear that the ageing central nervous system is more sensitive to the deleterious effects of alcohol. For example, verbal short term memory is extremely and dramatically vulnerable to any degree of chronic alcohol abuse (Blusewicz *et al.* 1982). Wernicke-Korsakoff syndrome, outlined in the section on nutritional consequences, is the most marked manifestation of mental impairment due to alcohol (Pentney, 1982; Young, 1984; Cerrato, 1986).

Suicide risk is also increased by alcohol intake. For example, Ross *et al.* (1990), in a study of 11 888 Southern Californian retirement community residents, showed that drinking more than three alcoholic beverages a day was a significant predictor of suicide risk.

One view of alcohol misuse and abuse among the elderly included the conclusion that alcohol is most likely to be consumed by socially active older people who consider themselves to be in good health (Lamy, 1984). He focuses attention on the fact that damaging effects of alcohol occur also with moderate intake, particularly if it is over a long time. It is known that the amount of alcohol intake is negatively associated with sober

cognitive performance. Age, itself, is significantly associated with reduced abstraction scores. An increase of one drink per drinking session can lead to cognitive loss equivalent to an increase of three years of age (Lamy, 1984). Goodwin (1989) reviewed the conflicting evidence for and against the amount of alcohol that produces significant and permanent impairments in cognitive function. His own study of alcohol use by 270 healthy elderly people (Goodwin *et al.* 1987) found no association between alcohol intake and psychological status or cognition. The nutritional consequences of cognitive impairment as a result of even a moderate intake of alcohol may be profound, leading to a descending spiral of malnutrition in the elderly drinker.

INCIDENCE OF ALCOHOLISM

Planning for the social cost of alcoholism demands some idea of the scale of the problem. Identifying the incidence and quantity of alcohol consumption in the elderly is not an exact art. Figures were unavailable on the subject until relatively recently. Prior to this the subject was deemed unworthy of study. This may be related to a tendency to regard the mental health problems of older adults as more acceptable, and drinking to excess a choice that is of no concern to others. More recent work seeks to determine the causes of these behaviours, with clearer evidence of the damage that can be done by alcohol. Theories on the development of alcoholism are legion. Cloninger (1987), to quote just one, divides alcoholism into two types. The one that is of concern here is Type 1, characterized by onset after the age of twenty-five, psychological dependence, and guilt and fear about alcohol dependence. The relevant personality traits are high harm avoidance, high reward dependence and low novelty seeking. These could be said to characterize some patterns of behaviour in the elderly. The importance of distinguishing subgroups of alcoholics is shown by findings that they also differ in neurophysiological and neurochemical characteristics. The harm avoidance personality dimension is influenced by the behavioural inhibition brain system, the principal monoamine neuromodulator of which is serotonin. The dietary precursor of serotonin is tryptophan, an amino acid common in protein. Another relevant theory on the development of alcoholism is that a segment of the population has a defect in the metabolism of tryptophan that causes a lowered concentration of serotonin in the brain (Thomson & McMillen, 1987), and indicates a predisposition towards chronic alcoholism.

The underidentification of alcoholism in the elderly is discussed by Willenbring *et al.* (1987) who suggest that it occurs because of differences in the presentation and symptom patterns compared to younger individuals. The different ways that an alcohol problem may present are almost endless, with virtually all body systems potentially affected. 'Alcohol may have replaced syphilis as the great masquerader' (Willenbring, 1987, p. 869).

Although few studies have been made on alcoholism in the elderly, Simon's (1980) estimates are that 10% of those who are over age 60 in the USA have drinking problems. Schuckit & Pastor's (1979) American figures agree with Simon's when they suggest that anyone in clinical practice with older adults will encounter an alcoholism rate of between 10 and 20%. Johnson (1989) quotes a range of between 2 and 70% of adults over sixty in the USA who have illnesses or other serious consequences as a result of alcohol abuse.

Assumptions are made in the UK, and mentioned by Watts (1987), that in a typical general practice of 2000 patients, at least twenty (1%) will have some damage through alcohol. Other sources (unspecified by Watts) use a formula which arrives at the number of problem drinkers through a comparison of hidden to known problem drinkers; this is calculated at 9:1 for men and 4:1 for women. Wattis (1983) stated that doctors do not expect to find alcohol abuse in the elderly.

In women in the UK the figures for admission to hospital for alcoholism were 10·0 % for all ages and 4·8 % for women over sixty-five (Age Concern, 1977). The study of elderly patients and alcohol consumption and dependence in a UK urban community by Bridgewater *et al.* (1987) came up with the figure of 27 % male heavy drinkers and 9 % females. The Office of Population Censuses & Surveys (1980) would predict 16 % male and 10 % female. Patients under seventy-five drank more than those over seventy-five.

Many elderly alcoholics remain unidentified and untreated (Gulino & Kadin, 1986). They quote a community survey (1965) in Baltimore where 12 % of the elderly were problem drinkers. The alcoholism sex ratio was five men to one woman. A study in New York (1965) reported that elderly widowers were the most likely to abuse alcohol. The latter study is also quoted by Zimberg (1974), showing a second peak prevalence of 2 % alcoholism in the sixty-five to seventy-four age group, and a 50 % drop for the seventy-five and older age group. Ferro-Luzzi *et al.* (1988) found, in their Italian cohort of 393 sixty-five- to ninety-year-olds, 48 % of males and 39 % of females could be classified as heavy drinkers. Ticehurst (1990) identified alcohol abuse and dependence as the third most common psychiatric diagnosis of elderly men in New South Wales, occurring in 3 % over the age of sixty-five.

General population surveys from 1970 to 1980, quoted by Wood & Elias (1982), show that over sixty-fives have higher rates of abstention, and lower rates of heavy drinking than do younger adults in the same populations. Adams *et al.* (1990) identified a true age-related decline in alcohol intake with increasing age rather than a cohort effect. Longitudinal analysis showed a statistically significant decline in the percentage of subjects consuming any alcohol over time. The slope was -2 % per year. Iber (1990) also refers to alcohol use in the United States as diminishing with age, with the number of users declining with a ten to twenty percent decrease in each decade. Research suggests an average intake of four drinks per day, spread across six out of seven days, for practising alcoholics over sixty-five. This rate is considerably lower than that for younger alcoholics found by Schuckit & Pastor (1979).

The studies indicate that, although there is a decline in drinking across time in the elderly, and the percentages identified as alcohol abusers are smaller than in 'younger' populations, those at risk are still underidentified.

CONCLUSIONS

Taking the findings reviewed above into account, there appears to be more weight of evidence and volume of studies on the damaging effects of alcohol; less clearcut information is available on the possible positive use of alcohol in reasonable amounts as a social lubricant, as a reducer of anxiety, a route for increasing energy intake, or the therapeutic effects of alcohol in moderation upon the lifestyle and coping mechanisms of an elderly person. Because by the year 2000 the number of people over the age of seventy-five is expected to increase by 25 % (Bernard, 1985), there appears to be a need for more studies to identify clearly and with confidence what level of alcohol consumption is safe in the elderly population.

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Printed in Great Britain