Coronary artery occlusion and alcohol intake

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A group of over 900 patients who underwent coronary arteriography were asked about their alcohol intake. The patients who were abstainers or consumed less than an equivalent of 180 ml absolute alcohol per week had higher coronary artery occlusion scores than the group consuming more than that amount, though the latter group smoked more and had higher plasma triglyceride levels.

Studies dealing with a possible association between alcohol intake and the development of coronary artery disease, or relating alcohol intake to changes in some variables associated with coronary artery disease, have produced conflicting results. Most reports have shown that moderate to excessive alcohol consumption leads to increased blood lipid levels (especially triglyceride levels), in chronic alcoholics (Albrink and Klatskin, 1957; Zieve, 1958; Lieber et al., 1963), in patients with type IV hyperlipoproteinaemia (Mendelson and Mello, 1973; Ginsberg et al., 1974), and even in presumably normal individuals (Ostrander et al., 1974; Wilson et al., 1970; Barboriak and Meade, 1968). Since high plasma lipid levels are usually associated with a higher incidence of cardiovascular disease (Albrink et al., 1961; Kannel et al., 1971; Carlson and Bottinger, 1972), one might have expected a similar tendency to higher incidence of heart disease in regular imbibers of alcoholic beverages. However, studies relating alcohol intake directly to development of coronary artery disease have failed to show a consistent relation. Most necropsy data suggest either no clear relation to coronary artery disease (Sackett et al., 1968) or less extensive atherosclerotic lesions in coronary arteries of patients with chronic alcoholism than in abstainers (Rissanen, 1974; Wilens, 1947a).

The apparent discrepancy between the effect of alcohol on plasma lipids and the absence of correlation between alcohol consumption and atherosclerosis may be partially the result of differences in study populations. While the blood lipid studies were usually carried out in populations consisting mainly of abstainers and moderate drinkers, most of the necropsies were on patients with advanced alcoholism, many of whom had suffered from other debilitating diseases usually associated with excessive chronic alcohol intake (Wilens, 1947b). The availability of data from a follow-up study of patients who had had coronary arteriograms, who were also repeatedly questioned as to their alcohol intake, gave us the opportunity to study in the same group of patients, both the association between alcohol intake and plasma lipid levels, and the association between alcohol intake and coronary arterial obstruction as shown by angiography.

Methods

A group of 909 male nondiabetic patients had selective coronary angiograms in two Milwaukee hospitals over a 4-year-period. The indications for coronary angiography were angina pectoris, previous myocardial infarction, chest pain on stress test, or chest pain associated with strong family history of heart disease. Each patient answered a self-administered life quality questionnaire which contained questions on the current alcohol intake, including the frequency, amount, and type of alcoholic beverage consumed. The alcohol intake, in ml per week of absolute alcohol, was then calculated using the usual alcohol concentration values (4% for beer, 12% for wine, and 43% for 'hard' liquor). In order to find out whether the information on alcohol intake reflected a regular drinking habit, 413 patients were asked to answer the questionnaire again one year later. An equivalent of 60 ml absolute alcohol per week was taken as a dividing line between...
regular and occasional drinking. Over 80% of the group continued to consume more than this amount, indicating a relative stability of the habit.

The extent of coronary artery disease was recorded as a 'coronary artery occlusion score', the method of calculation being based on that of Rowe et al. (1969). Each of the three major coronary arteries (right, left anterior descending, and left circumflex) is given a score from 0 (normal) to 100 (complete occlusion). Thus, 50 per cent occlusion of the left anterior descending artery would score 50; 50 per cent occlusion of a branch estimated to be 50 per cent of the size of the parent vessel would score 25; the left main coronary artery is scored from 0 to 200, so that 50 per cent occlusion of this vessel would score 100.

Plasma cholesterol and triglyceride levels were determined on fasting blood samples obtained shortly before the cardiac catheterisation. Information about hypertension, smoking habits, and previous myocardial infarction was obtained by direct questioning of the patients and from their medical records. Smoking habit, past and present, was graded on a 5-point scale:

1. Not currently smoking and no history of smoking.
2. Less than $\frac{1}{2}$ package per day for less than 20 years.
3. One package per day for less than 20 years or $\frac{1}{2}$ package per day for more than 20 years.
4. One and one-half packages per day for 10 to 30 years, 2 packages per day for less than 20 years, or 1 package per day for more than 20 years.
5. Two packages or more per day for 20 years or 1$\frac{1}{2}$ packages or more per day for 30 years.

The procedure used for these determinations in our laboratory (Kessler and Lederer, 1965; Block et al., 1965) passed the certification requirements of the Cooperative Cholesterol and Triglyceride Standardization Program, Center for Communicable Diseases, Atlanta, Georgia.

### Results

To correct for the known association between age and coronary artery disease, the patients were divided into four different age groups (Table 1). The average coronary artery occlusion score for combined groups (all ages) of the abstainers and moderate drinkers was 145, and this was significantly higher ($P < 0.01$) than the combined scores of 123 for the patients with high and very high alcohol intake. This negative association between occlusive disease and alcohol consumption was evident for all but the youngest age groups. It is interesting to note that a weekly alcohol intake of 180 ml seemed to be a dividing point in alcohol consumption associated with high and low coronary artery occlusion scores. The patients who were abstainers or ingested less than 180 ml per week had similar relatively high scores. The coronary artery occlusion scores of the two groups of patients who drank more than 180 ml per week were also similar but lower than in the two other groups. As expected, there was a significant increase in the coronary artery occlusion score with ageing of the patients.

To determine whether this negative correlation between drinking and coronary artery occlusion score could be attributed to some change in other risk factors which may be associated with drinking, plasma cholesterol and triglyceride levels, history of smoking, the presence of hypertension, and history of previous myocardial infarction were also considered. Data in Table 2 show that there was a statistically significant difference between the individual alcohol intake groups and average smoking scale, higher alcohol intake being associated with higher smoking scale. There was also a statistically significant difference in plasma triglyceride between the four alcohol intake groups, higher alcohol intake being associated with higher triglyceride level. Prevalence of hypertension,
Coronary occlusion and alcohol

Table 2. Plasma cholesterol and triglyceride levels, extent of smoking experience, and prevalence of hypertension and previous myocardial infarction in patients with angiographic examination

<table>
<thead>
<tr>
<th>Alcohol intake per week (ml)</th>
<th>0</th>
<th>&lt;180</th>
<th>180–360</th>
<th>&gt;360</th>
<th>All intake of alcohol combined</th>
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<tbody>
<tr>
<td></td>
<td>Age &lt;40</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Plasma cholesterol (mg/100 ml)</td>
<td>(C)</td>
<td>219</td>
<td>229</td>
<td>251</td>
<td>229</td>
</tr>
<tr>
<td></td>
<td>(T)</td>
<td>172</td>
<td>150</td>
<td>178</td>
<td>205</td>
</tr>
<tr>
<td>Smoking scale</td>
<td>(S)</td>
<td>2.5</td>
<td>2.8</td>
<td>3.1</td>
<td>3.6</td>
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<tr>
<td>Hypertension (%)</td>
<td>(H)</td>
<td>36</td>
<td>36</td>
<td>41</td>
<td>37</td>
</tr>
<tr>
<td>Previous myocardial infarctions (%)</td>
<td>(MI)</td>
<td>29</td>
<td>50</td>
<td>41</td>
<td>42</td>
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<tr>
<td></td>
<td>Age 40-49</td>
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<td></td>
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<tr>
<td>Plasma cholesterol (mg/100 ml)</td>
<td>(C)</td>
<td>258</td>
<td>250</td>
<td>246</td>
<td>267</td>
</tr>
<tr>
<td></td>
<td>(T)</td>
<td>178</td>
<td>178</td>
<td>217</td>
<td>207</td>
</tr>
<tr>
<td>Smoking scale</td>
<td>(S)</td>
<td>3.8</td>
<td>3.4</td>
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<tr>
<td>Hypertension (%)</td>
<td>(H)</td>
<td>32</td>
<td>26</td>
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<tr>
<td>Previous myocardial infarctions (%)</td>
<td>(MI)</td>
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<td>56</td>
<td>63</td>
<td>44</td>
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<tr>
<td></td>
<td>Age 50-59</td>
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<td>Plasma cholesterol (mg/100 ml)</td>
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<td>242</td>
<td>246</td>
<td>252</td>
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<tr>
<td></td>
<td>(T)</td>
<td>160</td>
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<td>181</td>
<td>212</td>
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<td>Smoking scale</td>
<td>(S)</td>
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<td>3.3</td>
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<tr>
<td>Hypertension (%)</td>
<td>(H)</td>
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<td>41</td>
<td>28</td>
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<tr>
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<tr>
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<td>234</td>
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<td>245</td>
</tr>
<tr>
<td></td>
<td>(T)</td>
<td>160</td>
<td>174</td>
<td>150</td>
<td>182</td>
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<tr>
<td>Smoking scale</td>
<td>(S)</td>
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</tr>
<tr>
<td>Hypertension (%)</td>
<td>(H)</td>
<td>44</td>
<td>19</td>
<td>32</td>
<td>39</td>
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<tr>
<td>Previous myocardial infarctions (%)</td>
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<td>51</td>
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<td></td>
<td>All ages combined</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma cholesterol (mg/100 ml)</td>
<td>(C)</td>
<td>247</td>
<td>244</td>
<td>248</td>
<td>251</td>
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<tr>
<td></td>
<td>(T)</td>
<td>166</td>
<td>178</td>
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<tr>
<td>Smoking scale</td>
<td>(S)</td>
<td>3.4</td>
<td>3.5</td>
<td>3.6</td>
<td>3.8</td>
</tr>
<tr>
<td>Hypertension (%)</td>
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<td>32</td>
<td>29</td>
<td>42</td>
</tr>
<tr>
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<td>(MI)</td>
<td>50</td>
<td>58</td>
<td>49</td>
<td>47</td>
</tr>
</tbody>
</table>

*Standard deviation.
†Significant correlation with alcohol consumption: P < 0.01 (calculated for all ages combined only).
Conversion factor from Traditional Units to SI Units: cholesterol, 1 mg/100 ml≈0.0259 mmol/l; triglycerides, 1 mg/100 ml≈0.0113 mmol/l.

previous myocardial infarction, and plasma cholesterol did not show any consistent correlation with alcohol intake.

Discussion

The results of the present study indicate that patients with angina pectoris and/or a previous myocardial infarction, who also consumed large amounts of alcohol, showed less arteriographically demonstrable occlusive coronary artery disease than the abstainers or moderate drinkers. This reduced extent of coronary artery disease was especially remarkable in patients consuming more than 360 ml alcohol weekly, since they also had increased plasma triglyceride levels and a high proportion of heavy smokers, and were thus a group which could be considered to be at a higher risk of developing coronary artery disease (Albrink et al., 1961; Sackett and Winkelstein, 1967).

Other investigators observed a similar reduced tendency to develop certain features of coronary heart disease in imbibers of alcoholic beverages. Klatsky et al. (1974), investigating the prevalence of myocardial infarction in a group of 464 patients, found this to be higher in teetotallers than in regular drinkers. Rissanen (1974) compared the extent of coronary artery lesions found at necropsy in age-matched chronic alcoholics and presumably non-alcoholic victims of accidents. He found significantly smaller areas of fatty streaks in the former group; however, the severity of raised lesions was about equal. Goto et al. (1974) found a delay and reduction in the development of atherosclerosis in rabbits receiving alcohol along with their atherogenic diet. The findings in this study and other published data thus indicate the possibility that ingestion of alcohol may have some retarding effect on the development and progression of occlusive coronary artery disease.

In other studies, however, there was little or no association between alcohol intake and the development of heart disease. Sackett et al. (1968) attempted to correlate the consumption of alcohol with the extent of coronary artery lesions determined at necropsy in 1010 cancer patients but found no significant association. Alcohol intake in their patients was, however, much lower than in this and
other studies (Klatsky et al., 1974). It is possible that some patients with malignant disease consume less alcohol (Bichel, 1959) or that the presence of malignant disease may have affected the course of coronary artery disease.

Hirst et al. (1965) found fewer coronary artery lesions in patients with hepatic cirrhosis than in chronic alcoholics who did not have cirrhosis. Unfortunately, no definite information is available on the possible presence of cirrhosis in our patients. Since most of the patients continued in gainful employment, it is likely that the prevalence of serious hepatic cirrhosis was relatively low. In a study of excessive alcohol consumption in ischaemic heart disease, in twins with differing alcohol intake, Myrhem (1974) failed to find any significant differences in the major indicators of ischaemic heart disease; however, the studied populations were quite young and there were no cases of myocardial infarction in either group.

The increase in plasma triglyceride levels in parallel with alcohol intake confirms the previously reported findings of Ostrander et al. (1974), showing a lipaemic effect of regular alcohol intake even in a non-alcoholic population. Our results further suggest that, under certain circumstances, a substantial increase in fasting plasma triglyceride may not be associated with increased coronary artery obstruction. It is possible that an increase in both plasma cholesterol and triglyceride is needed to accelerate progression of coronary atherosclerosis. In our previous study (Barboriak et al., 1974), the extent of angiographically demonstrable coronary artery disease correlated better with increased plasma cholesterol than plasma triglyceride levels. The absence of any consistent relation between increasing amounts of ingested alcohol and plasma cholesterol levels in this study may thus be one of the reasons for the observed lower coronary artery occlusion scores of patients with high alcohol intake. However, it is also possible that these lower coronary artery occlusion scores are the result of the known cardiotoxic effects of alcohol, not directly related to the extent of coronary artery disease. It has been shown that alcohol depresses myocardial function in patients with heart disease (Conway, 1968), reduces myocardial contractility (Ahmed et al., 1973), leads to other myocardial abnormalities (Wong, 1974; Alexander, 1975), and aggravates exercise-induced angina (Orlando et al., 1976). It is possible that a chronic intake of excessive amounts of alcohol has produced more severe symptoms than would be expected on the basis of the extent of the coronary artery disease.

It should also be stressed that the present study was carried out in patients with symptoms of coronary artery disease. It remains unclear whether, and to what extent, this group may differ from the general population with respect to alcohol intake, susceptibility to the effects of alcohol, possible genetic factors, and other variables affecting development of coronary artery disease.

The authors wish to acknowledge the assistance and interest of the members of the Milwaukee Cardiovascular Data Registry. The work was supported in part by a grant from the National Heart and Lung Institute (HL 14378), and by the Medical Research Service of the Veterans Administration.

References


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Coronary artery occlusion and alcohol intake.


Br Heart J 1977 39: 289-293
doi: 10.1136/hrt.39.3.289

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