An Enquiry into the Role of Cobalt in the Heart Disease of Chronic Beer Drinkers

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SUMMARY
The natural history of a new disease entity in chronic beer drinkers consisting of pericardial effusion, a low cardiac output, and in about half of the cases of polycythemia is presented. Normalization of the heart volume, the hemoglobin value, and the hemodynamic state was obtained in patients who stopped drinking. Further evidence of the importance of cobalt in the development of the disease is presented. All patients drank beer to which cobalt was added and the disease was not seen in chronic alcoholics drinking wine or other alcoholic beverages. In view of the rather small quantity of cobalt consumed, an hypothesis is formulated which attributes the cobalt toxicity in chronic alcoholics, to a dietary deficiency of sulfhydryl-groups containing amino acids and a dietary deficiency of protein.

Additional Indexing Words:
Cardiomyopathy  Anorexia  Pericardial effusion  Cobalt toxicity
Polycythemia  Chronic alcoholism

For many years clinicians have considered alcoholic heart disease as a specific entity.1-5 In the German literature, the cardiac symptoms of chronic alcoholics were described under the heading of "Bierherz." There is still doubt however whether alcoholic heart disease has in fact enough specific pathological and biochemical abnormalities to justify its position as an independent disease.6 It is rather rare among chronic alcoholics, and it is nearly always accompanied by important nutritional deficiencies. In March 1965 at an international meeting in Brussels on the nonobstructive cardiomyopathies, a new heart disease in chronic beer drinkers was described.7 This disease consists essentially of massive pericardial effusion, low cardiac output, and raised venous pressure accompanied in a certain number of cases by polycythemia. This syndrome was thought to be different from the two other forms of alcoholic heart disease already known, namely, the beriberi form2 and the form characterized essentially by the presence of myocardial fibrosis.3 At the end of 1966 a new acute form of myocardial disease with a high mortality was described in chronic alcoholics in Canada and in the U.S.A., whereby cobalt, having been used as an additive in beer, was implicated as a possible cause of the disease.8-10 A comparison of the two materials made a common origin between the cases in North America and our cases likely.

The purpose of this paper is to present supplementary clinical cases and data on the natural history of the disease and further evidence of the importance of cobalt in the genesis of the syndrome. Finally, the importance of nutritional factors and of the remaining problems will be stressed.

Method

This study concerns 16 of the 17 patients who were included in the original publication.6 One of the 17 patients was excluded because in this case myocardial infarction was a complication. This patient was not included in the hemodynamic study and incidentally had the lowest

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hemoglobin value of the whole group. Included in the study also were eight cases that were discovered after the original paper was presented for publication. The average duration of the follow-up was 2.8 years, with a range between 0.5 and 5 years. All of these patients were male and drank an average of 17.6 glasses of beer a day. They all drank brands of beer that contained cobalt at the time that their disease was discovered. Two additional cases have been discovered after Belgian beer no longer contained cobalt. They seem to present the same syndrome however as the other cases, and they will be discussed separately.

To investigate the effects of cobalt on chronic alcoholics without heart complaints, 12 brewery workers were examined. These 12 workers all were heavy drinkers, and they had worked in two different breweries, both of which added cobalt to their beer. One of these breweries produced two brands of beer and a sample of one of the brands had the highest recorded concentration of cobalt in Belgian beer, namely 5 ppm. The other brand of the same brewery, which most of the workers drank, contained 1.2 ppm of cobalt. No cases of heart disease have come to our attention due to the brand of beer containing 5 ppm of cobalt. The concentration of cobalt in beer has been determined as the complex of cuprethol. The photospectrometric measurement has been done at 360 nanometers after correction for the presence of copper at 435 nanometers. In some cases cobalt was determined in the serum and urine by colorimetry using a modification of the method of Sandell. 11

For the calculation of the statistical significance of the differences between the data in the different groups use was made of the $t$-test. For the follow-up study where data obtained on the same patient on different occasions were compared, use was made of the $t$-test for paired samples. In the tables and in the text mean values are given ±1 SD.

**Results**

**Evolution of Drinking Habits and Nutritional Status**

At the time of their illness the patients of the total group had drunk an admitted mean of 17.6 glasses of beer a day for a mean period of 17.8 years. They drank beer brewed in nine different breweries. At the time of the follow-up study the 18 patients available for it admitted a mean consumption of only 3.4 glasses of beer a day against an original mean of 18 glasses a day. Of these 18 patients nine stopped drinking alcohol, six admitted a consumption of between 1 and 5 glasses of beer a day, and two a consumption of between 6 and 10 glasses a day. One patient still drank 20 glasses of beer a day at the time of the follow-up study.

As noted in the original publication, anorexia was one of the predominant symptoms at the time of the initial illness, and the caloric intake from sources other than alcohol was of poor quality and quantity. At the time of the follow-up study all of the patients consumed a more diversified and appropriate quantity of food than at the time of their illness. However, we do not possess objective data on this matter.

**Pericardial Effusion**

This was found in 13 of the 16 published cases and in each of the eight additional cases. In all cases the pericardial effusion was an exudate. It was extremely poor in cells on microscopical examination, and aerobic, anaerobic, and Löwenstein cultures were always negative. The mean cardiothoracic ratio in the total group was 64.1 ± 7.8%. In two cases the cholesterol values of the effusion have been determined and were 135 mg% and 145 mg%, respectively. In all patients that were available for the follow-up study, the heart volume diminished, and in most of them it became completely normal (fig. 1). The mean cardiothoracic ratio in the follow-up group diminished from 65 ± 7.5% to 48 ± 5.7% ($P < 0.001$). In most of the cases the effusion cleared spontaneously and rapidly after some holes were made in the pericardium with an ordinary puncture needle. It cleared spontaneously over a period of 1 year in two patients, who changed their drinking habits drastically.

In one case the pericardial effusion did not clear after two punctures, and this patient was operated upon 3 months after he stopped drinking. On microscopic examination the pericardium was only moderately thickened and did not present any sign of inflammation. A small myocardial biopsy was taken, and the heart muscle appeared normal on microscopic examination. A higher than normal number of lymph vessels was thought by our
pathologist to be present in both the subepicardial and subpericardial tissue. At operation it was found that the reason why the pericardial effusion did not clear through the puncture openings was the presence of a large quantity of fat over the pericardium, which presumably sealed off the small openings made in the pericardium by the puncturing.

**Hemoglobin and Hematocrit Values**

For the total group the mean hemoglobin value (Hgb) was 18.4 ± 2 g% and the mean hematocrit value (hct) 52.5 ± 5.7%. Of the 24 patients, 11 had an Hgb value of more than 19 g% (range, 19.1 g% to 21.7 g%) and eight of more than 20 g%. At the time of the follow-up study the mean Hgb value had diminished in the follow-up group from a mean of 18.6 ± 1.9 g% to 15.15 ± 1.3 g% (P < 0.001) and the mean hct value from a mean value of 53 ± 5.6% to 44.8 ± 4.3% (P < 0.0025).

**Hemodynamic Study**

All of the eight additional patients underwent right heart catheterization. The findings corroborated the results obtained in the 13 published cases. In the total group, at the time of the initial illness, the circulation time was 20.6 ± 6.9 sec, and the left ventricular ejection time, determined from the indirect carotid artery tracing and corrected for heart rate from the tables of Willems and Kesteloot, was 81.6 ± 6.9% of normal. In the follow-up group the circulation time fell from a mean of 20.1 ± 6.8 sec to 12.3 ± 3.7 sec (P < 0.0025) and the ejection time increased from a mean of 80 ± 6.4% of normal to 98 ± 7.1% of normal. The correlation has been calculated between the hemoglobin value and the circulation time (r = 0.33, P < 0.1). The venous pressure, determined clinically, was normal, and none of the patients showed any signs of congestive heart failure at the time of the control study.

**Liver Function Tests**

No significant change was found in the follow-up group in the total mean protein value of 7 g% which remained unchanged, nor in the mean serum globulin value which changed from 1.06 g% to 0.91 g%. The mean serum glutamic oxalacetic transaminase (SGOT) value fell from 29.3 ± 12.4 IU to 19.7
± 8.9 IU (P < 0.05) and the mean serum glutamic pyruvic transaminase (SGPT) value from 22 ± 10 IU to 18.9 ± 2.6 IU. (This change was not statistically significant.) The serum iron, copper, and iron-binding capacity was determined in five patients at the time of their initial observation, and the results were normal in all.

Table 1

<table>
<thead>
<tr>
<th>Electrocardiographic Voltage Changes*</th>
<th>Total mean voltage</th>
<th>L11</th>
<th>R wave</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>L1+ L11+ L11+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Patients at initial admission</td>
<td>1.23 ± 0.47 mv</td>
<td>0.38 ± 0.22 mv</td>
<td>0.68 ± 0.29 mv</td>
</tr>
<tr>
<td>2. Patients after treatment</td>
<td>1.67 ± 0.67 mv</td>
<td>0.50 ± 0.32 mv</td>
<td>0.81 ± 0.46 mv</td>
</tr>
<tr>
<td>3. Normals†</td>
<td>1.98 mv</td>
<td>0.75 ± 0.33 mv</td>
<td>1.05 ± 0.35 mv</td>
</tr>
<tr>
<td>Difference between 1 and 3</td>
<td></td>
<td>P &lt; 0.0005</td>
<td></td>
</tr>
<tr>
<td>Difference between 2 and 3</td>
<td></td>
<td>P &lt; 0.005</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

*In tables 1 and 2 mean values ± 1 sd are given.
†According to Simonson.13

Figure 2

Important electrocardiographic changes and especially the microvoltage persist, although clinically the patient was well.
Evolution of Electrocardiographic Changes

As noted in our original publication, important electrocardiographic changes were present in the patients. The most important changes were the presence of microvoltage and of important ST-T changes in lead I and in the left precordial leads. The ST-T changes consisted of negative T waves and ST-depressions. Moreover in three patients a QS pattern suggestive of myocardial infarction existed; in two patients in the right precordial leads and in one patient in standard leads II and III. In the 18 patients available for follow-up the mean total voltage of the three standard leads taken together containing both the R and the S waves, increased from 1.23 ± 0.47 to 1.67 ± 0.67 mv. The R wave in lead II augmented from a mean of 0.38 ± 0.22 to 0.50 ± 0.32 mv. In lead V₆ the R wave increased from a mean value of 0.68 ± 0.29 to 0.81 ± 0.46 mv. In normals between 40 and 59 years of age the mean total voltage of the standard leads is 1.98 mv, and the mean voltage of the R wave in lead II is 0.75 ± 0.33 mv and in lead V₆ 1.05 ± 0.35 mv. These findings together with their statistical significance are summarized in table 1. In eight of the patients the ST-T changes did not change on follow-up; in one patient they became worse; and in eight patients they became less pronounced. Although 11 patients were taking digitalis at the time of the follow-up, in only three of them were the changes thought to be due to digitalis alone. In two of the patients with QS changes

![Figure 3](http://circ.ahajournals.org/)

*Normalization of the electrocardiogram more than 4 years after the beginning of the disease.*

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Table 2
Summary of Hemodynamic and Other Studies

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Mean age (yr)</th>
<th>Hemoglobin (g/L)</th>
<th>Hematocrit (%)</th>
<th>Cardio-thoracic ratio</th>
<th>Serum album (g/L)</th>
<th>Total serum prot. (g/L)</th>
<th>Circ. time (sec)</th>
<th>LVET (% of normal)</th>
<th>Mean beer consumpt. per day (glasses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Total group at first admission</td>
<td>24</td>
<td>45</td>
<td>18.4 ± 2.0</td>
<td>52.5 ± 5.7</td>
<td>64.1 ± 7.8</td>
<td>4.1</td>
<td>7.0</td>
<td>20.6 ± 6.9</td>
<td>81.6 ± 6.9</td>
<td>17.6</td>
</tr>
<tr>
<td>2. Follow-up group at first admission</td>
<td>18</td>
<td>45</td>
<td>18.6 ± 1.9</td>
<td>52.9 ± 5.6</td>
<td>65.0 ± 7.5</td>
<td>3.9</td>
<td>6.8</td>
<td>20.1 ± 6.8</td>
<td>80.0 ± 6.4</td>
<td>18.0</td>
</tr>
<tr>
<td>3. Control study of follow-up group*</td>
<td>18</td>
<td>48</td>
<td>15.1 ± 1.3</td>
<td>44.8 ± 4.3</td>
<td>48.3 ± 5.7</td>
<td>4.3</td>
<td>7.0</td>
<td>12.3 ± 3.7</td>
<td>98.0 ± 7.1</td>
<td>3.4</td>
</tr>
<tr>
<td>4. Control group†</td>
<td>12</td>
<td>51</td>
<td>15.9 ± 1.1</td>
<td>46.0 ± 2.7</td>
<td>46.2 ± 3.3</td>
<td>4.5</td>
<td>7.2</td>
<td>13.3 ± 4.0</td>
<td>99.2 ± 5.6</td>
<td>15.9</td>
</tr>
<tr>
<td>P value between groups 2 and 3</td>
<td>&lt; 0.001</td>
<td>&lt; 0.0025</td>
<td>&lt; 0.001</td>
<td>&lt; 0.005</td>
<td>&lt; 0.0025</td>
<td>&lt; 0.001</td>
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<tr>
<td>P value between groups 1 and 4</td>
<td>&lt; 0.001</td>
<td>&lt; 0.01</td>
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</tbody>
</table>

*Mean follow-up time was 2.8 yr (limits, 0.5 to 5 yr).
†Brewery workers without heart complaints drinking cobalt containing beer.
Use was made of the t-test for paired samples for the statistical calculations between groups 2 and 3.
LVET = left ventricular ejection time corrected for heart rate.

Twelve workers have been examined who did not have any complaints and were working normally in two different breweries. All of them were heavy beer drinkers. They admitted a mean consumption of 16 glasses of beer a day.
At the time of the examination six of them still drank beer containing cobalt, and six were examined 14 days after the brewery stopped adding cobalt to beer. In our experience the clinical manifestations of the disease have never changed spontaneously during such an interval. In 11 of these 12 controls the SGOT, SGPT, and LDH values and the electrophoretic patterns were normal and abnormal in one whose values were nearly normal on control 1 month later. The clinical cardiac examination was normal except in one patient who was 60 years old and who had tachycardia and a gallop sound. This persisted during a control examination 6 weeks later; at that time he drank beer no longer containing cobalt. The serum iron, copper, and iron-binding capacity values were also normal. Their mean Hgb value was 15.9 ± 1.1 g% and the mean hct value 46.0 ± 2.7%. These values are very similar to those of the follow-up group during the control study at a time when they no longer were drinking large quantities of beer. The same applies to the mean circulation time of 13.3 ± 4.0 sec. The cardiothoracic ratio in this group was 46.2 ± 3.3% and the left ventricular ejection time (LVET) was 99.2 ± 5.6% of normal. Both values are statistically different from the values obtained in the patient group (P < 0.01).

A summary of all these findings is presented in table 2. In seven of these 12 controls a dietitian made a study of the caloric intake. The nonalcoholic caloric intake was over 2,000 kcal in all of them (range, 2,180 to 3,290 kcal). In the remaining five the history also permitted us to accept an adequate nonalcoholic caloric intake.

Perimyocardiopathy in Drinkers of Beer Without Cobalt

Two cases of perimyocardiopathy have been discovered at a time that cobalt was no longer added to beer. This was confirmed by determining the absence of cobalt in the beer they were drinking. Both patients however had drunk beer that contained cobalt in the past. In both patients the disease was diagnosed at a time of grossly inadequate nonalcoholic caloric intake, while they were still performing relatively heavy physical work. Both had an Hgb value superior to 20 g% and pericardial effusion. One of these was the only woman of the series. She drank only 6 glasses of beer a day, but she ate practically nothing during the month before the diagnosis was made. In the other patient the serum cobalt concentration was 0.15 ppm against 0.07 ppm in controls who never drank beer containing cobalt. It rose to 0.75 ppm 2 hours after the administration of 2 g of calcium versenate intravenously but only to 0.10 ppm in two controls.

Discussion

Although in the beginning there were difficulties in differentiating the syndrome that was described from the classical forms of alcoholic heart disease, it rapidly became apparent that this was a new disease entity. The disease was called “alcoholic perimyocardiopathy” to emphasize the massive pericardial effusion and the hemodynamical changes that persisted for some time after the disappearance of the pericardial effusion. It was thought to be due to a direct influence of alcohol, although another toxic origin was not excluded. In the beginning of 1967 we became aware through correspondence with Professor Morin of the Laval University of Quebec that a similarity existed between our published cases and the cases he had observed in Quebec. The prevailing opinion in Canada was that the syndrome could be due to the addition of cobalt to beer. Cases similar to those of Quebec have been described in Omaha. Until that time we were unaware of the fact that cobalt was added to beer.

At the time of our first publication the whole processing of beer was followed in one of the biggest Belgian breweries in the presence of the chief biochemist. This brewery however had never added cobalt to its production, and it certainly was a bad choice because none of the patients drank beer produced by that brewery. This particular point escaped us because so many different brands were involved. As all the patients drank beer containing cobalt, it was possible to confirm

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the role played by this agent in the genesis of the syndrome. This finding is further enhanced by the fact that 80% of the beer market in the city of Leuven is dominated by the biggest Belgian brewery, which never added cobalt to its beer, and none of the patients exclusively or predominantly drank beer of this brewery. Moreover the first cases were discovered 2 years after some Belgian breweries began adding cobalt to beer, and no cases have been seen in chronic alcoholics drinking wine or other alcoholic beverages. If one accepts that in the region from which the patients were drawn 50% of the quantity of beer consumed contained cobalt, and this is probably too high an estimation, then the probability of all patients drinking beer containing cobalt by change only is 0.524 which is extremely small.

That cobalt administration causes polycythemia is well known.14-17 Of equally great importance is the fact that cobalt has been shown experimentally to produce massive pericardial effusions in animals.18 The two most important signs of the disease can thus be attributed to the addition of cobalt to beer, especially as these two signs have not been described in the classical forms of alcoholic cardiomyopathy. The etiological role of cobalt can thus be accepted as proven.

Several important problems however remain. Cobalt has been added to beer in the form of cobaltous chloride at a dosage of about 1 ppm in most breweries. It may be assumed that beer drinkers consumed an average of 6 mg of cobalt a day. Much larger doses of cobalt however have been given in the treatment of anemias without cardiotoxicity's being reported as a result of the therapy. Doses of between 20 and 35 mg of cobalt a day have been given during several months and no heart disease has been described although hypothyroidism and thyroid hyperplasia have developed.19 Cobalt has been given in a dosage of 75 mg a day for 6 weeks in cases of sickle cell anemia, nerve deafness, and tinnitus, but no cardiac toxicity was reported.20 Cobalt has also been given in a dosage of about 20 mg a day for 3 months to 78 preg-
nant women. The anemia of pregnancy was prevented in this group, and no toxic manifestations were observed.15 Cardiac toxicity has not been described in a study of cases of professional intoxication of humans working in cobalt mines or in industries engaged in the purification of cobalt.21 Cobalt has even been used for therapy in cases of hypertension in a dosage of 6 to 8 mg a day, and no toxic effects have been noted.22 Doses of cobalt of 2.5 mg/kg/day have been given to experimental animals for a period of 6 months. At autopsy, nothing special was found. In a dosage of 5 mg of cobalt/kg/day, no animal died after 5 months. At autopsy some myocardial edema without degeneration was noted. In a dosage of cobalt of 25 mg/kg/day the animals died after 1 month and massive pericardial effusion was noted. The myocardium was edematous; degeneration and swelling of the myocardial fibers were present with clarification and vacuolization of the cytoplasm.18 Thus, very high concentrations of cobalt seem to be necessary to produce toxicity both in humans and in experimental animals. Cardiac toxicity is known however to exist during cobalt therapy.23 Cardiotoxicity developed in a case of aplastic anemia treated at our clinic and apparently cured by cobalt in a dosage of 10 mg a day during 4 months. Cardiac decompensation, cardiomegaly, and possibly pericardial effusion developed and disappeared after the treatment was stopped. The patient died 3 years later of lung cancer.

Only a small fraction of the population drinking beer containing cobalt developed manifest heart disease. This could be due to an abnormal sensitivity of certain subjects to cobalt or to a synergetic action by chance of different other factors in certain individuals. One factor however seems to differentiate the patients with heart disease from the other drinkers, and this is the profound anorexia that was present in almost all of them. This anorexia was also present in most of the patients who developed the disease in Quebec24 where only 16 of 48 patients were considered to follow a relatively adequate diet.25 Prior to this period of anorexia the protein content
of the diet seems to have been relatively normal. Nearly all of our patients who developed heart disease did this at a time that their diet was grossly inadequate and while most of them continued to do rather heavy physical work. One of the patients who worked in a brewery, when asked whether other workers, who drank as much as he, did not develop heart disease, answered in the negative, and added, ‘but the others still continued to eat whereas I could not.’ The diet of the 12 brewery employees who did not develop manifest heart disease was still adequate, especially in proteins. Both patients who had drunk beer containing cobalt in the past, although no longer drinking it at the time of their disease, developed heart disease at a time that their diet had become grossly inadequate. Presumably the quantity of cobalt still present in the organism became toxic at that time. That excess cobalt was present is probable in view of the important rise of the serum cobalt value in one patient after calcium versenate injection.

Experimental evidence exists that protein binds cobalt and that a diet rich in proteins and in certain amino acids containing SH groups protects against cobalt toxicity. The binding capacity by cobalt of -SH groups is well known, and it has been suggested that cobalt effects its influence on cellular respiration by inhibition of the utilization of keto acids through the formation of a complex with the dithiol form of lipoic acid, which results in a blocking of the Krebs cycle. Cobalt also inhibits the α-oxoglutarate dehydrogenase, but both these effects are largely overcome when excess cysteine is present. Cysteine forms with cobalt a practically irreversible chelate which effectively eliminates cobalt as well as the amino acid; part of the toxicity of cobalt could in this manner be due to the elimination of sulphydryl groups containing amino acids. The addition of cysteine, methionine, and even of histidine to the diet containing cobalt has been shown to prevent the development of polycythemia. In rats who fail to grow, if cobalt in a dosage of 50 mg/kg of body weight/day is added to their diet, this effect is largely overcome when cysteine, cystine, or methionine is added. The inhibition of growth and respiration of animal tissues by cobalt may be overcome by the addition of histidine. The deficiency in certain amino acids in the grossly inadequate diet of chronic alcoholics associated with the presence of cobalt is probably an important factor in the genesis of the syndrome. This is further confirmed by the fact that many patients had drunk beer containing cobalt during several years, but only developed their illness after a period of anorexia. Whether this anorexia could be due to cobalt is still unknown. This finding however would also tend to eliminate an abnormal sensitivity to cobalt as the cause of the disease.

It is to be noted that beer contains a certain amount of protein. This protein is of vegetal origin, and plant proteins contain less cysteine and especially methionine than animal proteins. Belgian beer contains approximately 2.5 g of protein per liter (range, 2.2 to 3.1 g), and this protein is especially poor in cysteine and methionine. Whether alcohol itself plays a role in the development of cobalt toxicity is still unknown. Such an effect can certainly not be excluded at this time, as alcohol itself has profound effects on the metabolism of the myocardium. As a rapid response to vitamin therapy, with one possible exception, could not be obtained, a deficiency in these substances probably only plays a minor role in the causation of the disease.

In Quebec, of a total of 48 patients studied, 20 died and in Omaha 11 of a total of 28 patients died. Most patients died in profound shock and multiple arterial emboli were seen. Shock is a known complication of the intravenous administration of cobalt. It has been found in the dog that the intravenous administration of 3 mg of cobalt/kg lowers the blood pressure to between 50 and 70% of the initial value. This is not due to a direct action of cobalt on the heart, as its action in this concentration has been shown to be positively inotropic. It is explained by the vasodilating
effects of cobalt, due to a diminution in sympathetic tone. Shock is also a complication of the acute form of beri-beri, called shoshin. The massive pericardial effusion could also contribute to the development of shock.

Only one of our 24 patients died, 1½ years after the original discovery of this disease. He became prostrated and died within 24 hours. He was known to have continued to drink beer containing cobalt in large quantities. One patient was in shock during his admission to the hospital. He survived following the administration of thiamine, norepinephrine, nalorphine, epinephrine, digitalis (Digitali), and fluid. This difference in mortality between the American and our series remains unexplained. Difference in diet between the two groups and a slightly higher alcoholic intake could be a factor in explaining the more acute toxicity in Canada. The possible role of a nutritional deficiency was also accepted by the American authors. The Hgb value of our group was 18.4 ± 2.0 g% and this is distinctly higher than the values observed in Omaha and Quebec, where they were 16.3 ± 2.1 g% and 16.4 ± 1.6 g%, respectively (personal communication from Y. Morin). This difference could be due to the fact that cobalt has been present in Belgian beer for a much longer time than in the American beer.

The prognosis in our series was good. At the time of the follow-up all the patients took a more adequate diet. Their hemoglobin value, heart volume, LVET, circulation time, and venous pressure became normal. The electrocardiographic tracing, however, only rarely normalized, and in several patients important ST-T changes and the presence of microvolt- age persisted. Liver function was within normal limits at the stage of the acute illness and remained so at the time of the follow-up study. In one patient a myocardial biopsy has been taken during the receding state of the disease, and the myocardium appeared normal by microscopic examination. In the American series cellular degeneration with vascular degeneration and edema with absence of inflammatory changes or fibrosis was present in the myocardium of the patients who died.

The origin of the pericardial effusion remains unknown. There is no doubt that the important cardiomegaly was nearly exclusively due to the pericardial effusion. In all the cases in which the pericardial fluid disappeared within 24 hours after the making of some holes in the pericardium with a pericardial puncture needle, the heart volume was normal or near normal. Fluid did not again accumulate except in one patient, who continued drinking beer containing cobalt; it disappeared however after he finally stopped drinking. Pericardial effusion has also been described in chronic alcoholics drinking beer without cobalt. As cobalt was not quantitatively measured however in the beer consumed, this should be accepted with some reserve.

This syndrome is a typical example of the challenge that modern food processing presents to public health. A continuous watch will be necessary to prevent the recurrence of such events in the future.

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