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ALCOHOLIC POLYNEUROPATHY: A CLINICAL AND EPIDEMIOLOGICAL STUDY

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Abstract — In the present study, we investigated the frequency of polyneuropathy in a sample of 296 alcoholics who were admitted to the 'S. Maugeri' Medical Centre for detoxification from October 1997 to November 1999. Results revealed a high frequency of polyneuropathy in the sample under study. The disorder was often clinically asymptomatic and demonstrable only on electroneurographic investigation. Significant correlations were found between polyneuropathy, the duration of alcoholism, the type of alcoholic beverage consumed (wine) and the presence of liver disease and macrocytosis.

INTRODUCTION

Alcoholic polyneuropathy is characterized by widespread symmetric axonal involvement in conjunction with sensory and motor symptoms. The disorder corresponds to the pattern of distal axonopathy where axonal changes primarily involve the distal tract of the longest fibres with the largest diameter of the lower limbs, and, to a lesser extent, of the upper limbs (Pinelli, 1985). The reported frequency of sensory and motor polyneuropathy in alcoholics varies from 12.5% (Beghi and Monticelli, 1998) to 29.6% (Wetterling *et al*., 1999). Chronic alcohol misuse (but also vitamin deficiencies and exposure to heavy metals and neurotoxic industrial agents) leads to metabolic alterations in the nerve cells and degeneration of the axial flux. Each axon begins to degenerate from the most distal sections of the cell body whose integrity depends on the consistency of the streams. This explains why the longest axons are the first to be involved. As the underlying disease progresses, the axonal flux becomes less and less efficient, and the degeneration begins to extend to portions of the axons closer to the cell body and is accompanied by destruction of the myelinic sheaths. This is known as the 'dying back' phenomenon or retrograde degeneration (Savoldi, 1995; Manzo and Costa, 1998).

The pathogenesis of alcoholic polyneuropathy is still under debate. While some have argued that it results from a nutritional deficiency, and especially from a deficiency of thiamine, there is both clinical and experimental evidence of a direct toxic effect of alcohol (Pinelli, 1985). The issue is not easily resolved, because there are no direct estimates of nutritional status that are independent of the patient's history. A number of studies have shown that alcoholism can cause both nutritional deficiencies and nervous system disease, and almost invariably ethanol neurotoxicity is encountered in heavy drinkers presenting with nutritional deficits (Martin *et al*., 1986; Charness *et al*., 1989; Charness, 1993). In Victor's (1984) study of hundreds of cases of neuropathy associated with alcoholism, dietary deficiency was always found to be present when a reliable clinical history could be obtained. Behese and Buchtal (1977) made a painstaking attempt to separate out the relative effects of alcohol toxicity and malnutrition. They compared 37 alcoholics with six post-gastrectomy patients, all with polyneuropathy. In the alcoholic group, 23 patients showed no signs of malnutrition and 14 had a history of weight loss (>10 kg). All six of the post-gastrectomy patients reported severe weight loss. The alcoholics drank mainly beer, and these authors pointed out that Danish beer contains 30–40 μg of thiamine and 400 μg of pyridoxine per litre. Deficiency of these vitamins was deemed unlikely in beer drinkers, and measured blood levels of the vitamins, when obtained, were within normal limits. Beer also contains 500–700 calories per litre; thus the beer-drinking alcoholic is rarely calorie-deficient. On clinical and electrophysiological grounds, the post-gastrectomy patients, the malnourished alcoholics and the adequately nourished alcoholics could not be differentiated. However, nerve biopsy studies of the post-gastrectomy patients revealed a greater relative contribution of segmental demyelination to the development of polyneuropathy. This finding, along with the evidence that the alcoholic subjects with alcohol-related polyneuropathy were not malnourished, led Behese and Buchtal (1977) to conclude that polyneuropathy secondary to malnutrition was distinct from that associated with alcohol, and that malnutrition alone does not cause an alcohol-related neuropathy. This study contributes important data regarding the pathogenesis of alcohol-related polyneuropathy and demonstrates that it is probably not caused by a single vitamin B or a calorie deficiency, but by a B complex deficiency associated with the direct neurotoxicity of alcohol (Victor, 1984; Windebank, 1993; Pessione *et al*., 1995). Alcohol-induced vitamin deficiency may occur by a combination of mechanisms such as inadequate dietry intake (since calorie intake is largely constituted by the consumption of alcohol), the relative preponderance of carbohydrates in an alcoholic's diet (a high thiamine intake is required for their metabolism), general malabsorption due to the frequent chronic calcific pancreatitis suffered by alcoholics, and thiamine malabsorption due to the direct effect of alcohol on the gastrointestinal mucosa (Pinelli, 1985).

Several studies have explored these mechanisms. It has been shown that, during binge drinking, chronic alcoholics may markedly reduce their essential nutrient intake due to

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decreased appetite. Additionally, ethanol is a rich source of non-nutritive calories and an alcoholic may consume more than a third of the body's daily energy requirements as ethanol, and thus have a significantly reduced demand for food to meet his caloric needs (Charness *et al*., 1989; Lieber, 1990). Nutritional deficiencies ranging from undernutrition to severe malnutrition may also occur as complications of alcoholism. Both chronic and acute ethanol consumption have direct negative effects on the gastrointestinal mucosa and the pancreas. As a result, absorption of such nutrients as amino acids and vitamins may be hindered (Persson, 1991). Additionally, alcohol misuse may alter metabolism, transport, use, activation, and storage of many essential nutrients. Such alterations are partially attributable to the direct toxic effect of ethanol on the liver (Lieber, 1991). Chronic alcoholics frequently suffer from illnesses such as infection, anaemia, and bleeding related to peptic ulcers, which may exacerbate the nutritional deficiency and increase the patient's overall metabolic demands. Most of the neurological syndromes related to alcohol misuse arise from a complex interaction of events involving direct ethanol neurotoxicity, nutritional deficiencies due to heavy drinking, and possibly genetic predisposition (Martin *et al*., 1986; Charness *et al*., 1989; Manzo *et al*., 1994; Pessione *et al*., 1995). Despite the complex interactions between alcohol and diet, there is a consistent clinical pattern of polyneuropathy in chronic alcoholics (Victor, 1984). Alcoholics with polyneuropathy commonly show the typical features of alcoholic misuse including disordered social relationships, skin changes, memory disorders and ataxia. Neuropathy may be a minor aspect of the neurological presentation of Wernicke's encephalopathy or Korsakoff's syndrome. When polyneuropathy is the main complaint, the early symptoms are usually distal and symmetric, dysaesthetic sensory disorders in the feet. If present, the pain tends to be described as cramp-like, burning or stabbing. Sensory symptoms progress from distal to proximal regions in the lower limbs and in severe cases may involve the hands. Symptoms in the hands, characterized by sensory loss or electric tingling, are typically less painful than those in the feet. Walking becomes increasingly problematic because of the weakness of the distal muscles and manual dexterity is progressively reduced due to the combination of muscle weakness, sensory loss and ataxia. Clinical examination reveals signs of symmetric and distal sensory motor and autonomic loss. Sensory loss tends to begin with a loss of superficial pain sensation, which in the severest cases progresses to the loss of all types of sensation in a glove-and-stocking distribution. Often, such sensory symptoms are accompanied by increased painful sensitivity of the limbs initially to superficial light touch, and, as the neuropathy evolves, to deep palpation of muscles and tendons (Victor, 1984). When present, autonomic skin changes give rise to reddening, atrophy, and hair loss in the same distribution as the sensory damage.

From a diagnostic viewpoint, the clinical manifestations of polyneuropathy in isolation are not distinctive. Hence the importance of obtaining an accurate clinical history of alcohol misuse, and of nutritional deficits, because many cases of polyneuropathy present asymptomatically (Victor, 1984; Wetterling *et al*., 1999). Behse and Buchtal (1977) have proposed that 100 ml of ethyl alcohol (3 l of beer or 300 ml of spirits) per day for 3 years represents the minimal amount of alcohol consumed by patients with polyneuropathy. The aim of the present study was to evaluate the occurrence of symptomatic and asymptomatic polyneuropathy in a group of alcoholic subjects. Polyneuropathy is in fact a frequent complication of chronic alcoholism and may involve symmetric paraesthesia and/or dysaesthesia associated with sensory, motor, and sometimes vegetative deficits. A further aim of this study was to correlate the presence and severity of polyneuropathy with other clinical and laboratory manifestations of chronic alcoholism, such as liver disease, macrocytosis and changes in hepatic enzymes. The type of alcohol consumed, the duration of alcohol misuse, and other current or past dependencies, such as psychoactive drug dependence, were also taken into account.

PATIENTS AND METHODS

The study involved 296 chronic alcoholics aged between 20 and 77 years (mean ± SD: 45.2 ± 11.2) who were admitted to the Alcohol Unit of the Medical Centre of Rehabilitation, 'S. Maugeri' Clinica del Lavoro Foundation, Pavia, from October 1997 to November 1999. There were 209 (70.6%) men (mean age ± SD: 44.3 ± 11.5 years) and 87 (29.4%) women (mean age ± SD: 47.2 ± 10.3 years). Patients with diabetes, current or past neuropathies, osteoarthrosis of the spine with nerve root involvement, cancer, dysparaproteinaemia, dysthyroidism, or who were HIV-positive were excluded. In addition to a complete clinical interview with particular attention to the history of problem drinking, all patients underwent routine laboratory tests, hepatitis B and C virus markers and an electroneurographic investigation (ENG). Furthermore, all subjects were submitted to the common diagnostic procedure for liver disease; from medical history to the assessment of enzymes and biochemical blood parameters (aspartate aminotransferase, γ-glutamyltransferase, alkaline phosphatase and prothrombin time), abdominal ultrasonography and 63 patients, with uncertain diagnosis of cirrhosis, to liver biopsy. ENG involved the assessment of mixed or sensory nerves, with a particular emphasis on the motor and sensory compartments of the lower limbs. When the latter were involved, the investigation was extended to include the upper limbs. The motor compartment of the lower limbs was assessed by studying the sciatic popliteus externus nerve bilaterally; and the sensory compartment by studying the sural nerve bilaterally. When the investigation was extended to the upper limbs, both the motor and sensory components of the median nerve were studied. To examine the motor compartment, we considered the maximal conduction velocity and the amplitude of the motor potential obtained from the most distal stimulation. Investigation of the sensory compartment (sural and median nerves) was based on sensory conduction velocity (antidromic for the sural nerve, orthodromic for the median nerve) and the amplitude of the sensory potential. The resultant data were then compared with the normal reference values used in the Centre's Neurophysiology Unit. Polyneuropathy was classified on the basis of the ENG findings described in Table 1. Statistical analysis was performed using analysis of variance, Kruskal–Wallis test and χ^2-test. $P < 5\%$ was considered significant. Frequency distributions were compared using the χ^2-test (Figs 1–4, Table 6). Age was compared over types of alcohol consumed, of symptoms and of severity of

Table 1. Classification of the seriousness of polyneuropathy based on electroneurographic alterations

Class	Electroneurographic signs
Absence of polyneuropathy	Results within normal limits
Borderline polyneuropathy	At least two values symmetrically at the limits of the norm. Conduction velocity values (CV) and/or potential amplitude within 1.5 and 2 SD of the average normality value
Mild polyneuropathy	At least two symmetrically and slightly altered values. Fall in CV to between 1 and 10% of the normality value limits or fall in the potential amplitude to between 1 and 20% of the normality value limits
Moderate polyneuropathy	At least two symmetrically altered values. Fall in CV to between 10 and 30% of the normality value limits and fall in the potential range/amplitude to between 20 and 50% of the normality value limits
Severe polyneuropathy	At least two symmetrically and severely altered values. Fall in CV to >30% of the normality value limits and fall in the potential amplitude to >50% of the normality value limits or absence of potential
Neurophysiological tests[a]	**Normal values**
Median nerve	
Motor conduction velocity	>50 m/s
Compound muscle action potential	>8 mV
Sensory conduction velocity	>36 m/s
Sensory action potential	>7 μV
Peroneal nerve	
Motor conduction velocity	>43 m/s
Compound muscle action potential	>7 mV
Sural nerve	
Sensory conduction velocity	>34 m/s
Sensory action potential	>5 μV

[a]Sensory conduction velocities were calculated at the negative peak. Amplitudes were calculated peak-to-peak.

Table 2. Relationship between the type of alcohol consumed and age

Alcoholic beverage(s)	No. of subjects	Frequency (%)	Age (years; mean ± SD)
Wine	111	37.5	50.9 ± 10.2
Wine and spirits	59	19.9	45.6 ± 10.7
Spirits	35	11.8	41.6 ± 10.7
Wine, spirits and beer	63	21.3	40.0 ± 10.0
Beer	20	6.8	38.8 ± 8.5
Beer and spirits	8	2.7	37.4 ± 9.7

$P < 0.001$.

neuropathy using Kruskal–Wallis test, whereas analysis of variance was applied to investigate the relationship between severity of neuropathy and mean cell volume (MCV).

RESULTS

The prevalence of misuse of different alcoholic beverages in the age-divided sample is shown in Table 2. On ENG grounds, the presence of polyneuropathy was documented in 144 subjects (48.6%), out of whom 121 (57.4% of total male subjects) are men and 23 are women (26.4% of total female subjects).

Tables 3 and 4 report respectively the presence of neurological symptoms, and the correlation between the severity of polyneuropathy (defined by the amplitude of the electric potential and motor and sensory conduction velocity) and Table 5 presents a comparison between the severity of polyneuropathy and MCV, the increase of which is one of the most valid markers of alcoholic misuse (Schuckit, 1995). Table 6 presents the relationship between ENG diagnosis of polyneuropathy, alcohol misuse and the misuse of other substances (tobacco smoking, heroin, cocaine and cannabinols).

Table 3. Relationship between the symptoms of polyneuropathy and age

Symptoms	No. of cases	Frequency (%)	Age (years; mean ± SD)
Present	48	16.2	50.9 ± 9.5
Absent	248	83.8	44.0 ± 11.2

$P < 0.001$.

Table 4. Relationship between the severity of alcoholic polyneuropathy and age

Polyneuropathy	No. of cases	Frequency (%)	Age (years; mean ± SD)
Severe	9	3.0	50.5 ± 13.6
Moderate	52	17.6	49.1 ± 11.1
Mild	41	13.9	47.6 ± 10.2
Borderline	42	14.2	46.7 ± 10.9
No evidence of polyneuropathy	152	51.4	42.5 ± 10.9

$P < 0.001$.

HCV antibodies were found in 70 patients (23.7%), of whom 37 (52.9%) had normal ENG signs, whereas the remaining 47.1% showed abnormal ENG parameters suggestive of severe (1.4%), moderate (18.6%), mild (11.4%) and borderline (15.7%) degrees of polyneuropathy. The presence of HbsAg was found only in nine subjects, the combination HCV-Ab and HbsAg only in five; these last two findings represent a very small portion of the sample. Figure 1 shows the frequency distribution of liver disease in patients with or without ENG evidence of polyneuropathy. Figure 2 presents the frequency distributions of polyneuropathy severity (based on ENG findings) in relation to the duration of alcohol misuse. The presence of subjective symptoms was found after a relatively short period of misuse (19.8% of alcoholics had neuropathy symptoms in the first 5 years of misuse, 40.4% after 10 years). Figure 3 illustrates the frequency of the symptoms reported by the patients in the various polyneuropathy groups (based on ENG findings) and Figure 4 shows the frequency of the different degrees of polyneuropathy (assessed using ENG) in relation to the type of alcoholic beverage consumed.

DISCUSSION

The present study sample comprised subjects who volunteered to be admitted to our department for detoxification. Prior to admission, all subjects attended a preliminary interview with a physician and/or psychologist aimed at evaluating not only physical status, but also their determination to break an often long-standing dependency. Thus, the subjects were selected on the basis of their having made the decision to 'stop drinking', more frequently as a result of social and personal problems (job loss, threat of marital breakdown, road accidents, clashes with the law, exasperation of family members) than of medical disorders. Because of the known characteristics of alcoholics, we were unable to ascertain their exact daily alcohol intake. However, reliable information indicated that all subjects consumed at least 100 g of alcohol per day. Most of our subjects were aged from 35 to 55 years and therefore of working age. The marked prevalence of males in our sample (70.6 vs 29.4%) agrees with epidemiological studies indicating that alcoholism prevails in men (Indagine Nazionale DOXA, 1998; GESIA, 1999). There has, however, been a recent increase in alcohol misuse in women (Hammer and Vaglum, 1989; Indagine Nazionale DOXA, 1998). Also noteworthy is that in Italy heavy drinking in men is often regarded as an expression of virility or strength, whereas in women it is openly condemned and so tends to be more surreptitious and kept behind closed doors. In short, women tend to drink alone at home whereas men drink publicly and in company (Biscaldi *et al*., 1986). True to Italian tradition, our sample mostly drank wine. Recently however, there has been a decline in the consumption of wine in favour of beer (particularly among young people) and spirits (Indagine Nazionale DOXA, 1998). In fact, wine alone was consumed by 37% of our subjects, while only 21% drank other alcoholic beverages alone or in combination but without wine (Table 2). The use of wine in our sample does not appear to be of secondary importance since the subjective neurological symptoms and ENG findings were more serious in the subjects who misused wine alone or in combination with other alcoholic beverages compared with those who obtained their alcohol from beer and/or spirits (Fig. 4). This phenomenon is not easy to interpret. One possible explanation may be that a greater amount of wine was drunk (and therefore a greater daily alcohol intake) to achieve the alcoholic's desired effect, or possibly the presence of neurotoxic impurities such as lead in the wine itself (Minoia *et al*., 1994; Roggi *et al*., 1994; Schuckit, 1995). A number of authors have drawn attention to the existence of a relationship between alcohol consumption and blood lead concentration (Ikeda *et al*., 1989; Staessen *et al*., 1990; Cezard *et al*., 1992). There is widespread agreement that wine, whose mean lead concentration is 130 ± 256 μg/1, is a potentially 'important' source of dietary intake of lead, and, particularly in chronic alcoholics who drink mostly wine, a direct toxic action on the somatic load (Eschnauer, 1986; Roggi *et al*., 1995). With regard to non-professional saturnism, a previous study (Biscaldi *et al*., 1992) on the

Table 5. Relationship between the severity of polyneuropathy and mean cellular volume (MCV)

Polyneuropathy	MCV (fl; mean ± SD)
Severe	100.0 ± 7.1
Moderate	101.3 ± 8.0
Mild	100.9 ± 7.8
Borderline	99.6 ± 7.0
Negative ENG	97.6 ± 8.4
Study population	98.7 ± 8.2

$P < 0.05$.
ENG, electroneurographic investigation.

Table 6. Relationship between substance abuse other than alcohol and severity of polyneuropathy

	Polyneuropathy (severe, moderate, mild, borderline) *n* (%)	Negative ENG *n* (%)	Total subjects (sample of 296 alcoholics) *n* (%)
Other addiction			
Heroin and/or cocaine and/or cannabinols	16 (11.1)	28 (18.4)	44 (14.9)
No other addiction	128 (88.9)	124 (81.6)	252 (81.1)
Total	144 (48.6)	152 (51.4)	296
Smoking			
Smokers	113 (78.5)	127 (83.6)	240 (81.1)
Ex- and non-smokers	31 (21.5)	25 (16.4)	56 (18.9)
Total	144 (48.6)	152 (51.4)	296

ENG, electroneurographic investigation.

behaviour of blood lead (Pb), urine Pb, 5-aminolaevulinate dehydratase (5-ALA-DH) and zinc protoporphyrin in a group of alcoholics documented the presence of values within the upper limits of the reference ranges for blood Pb (26.5 μg/dl ± 14.7) and urine Pb (28.4 μg/dl ± 13.5). Other authors do not rule out the possibility of an indirect action correlated with the effect of alcohol on the toxico-kinetics of lead. Such an effect could result in increased absorption of the metal, greater movement from the deposit organs and reduced elimination (Roggi *et al*., 1995).

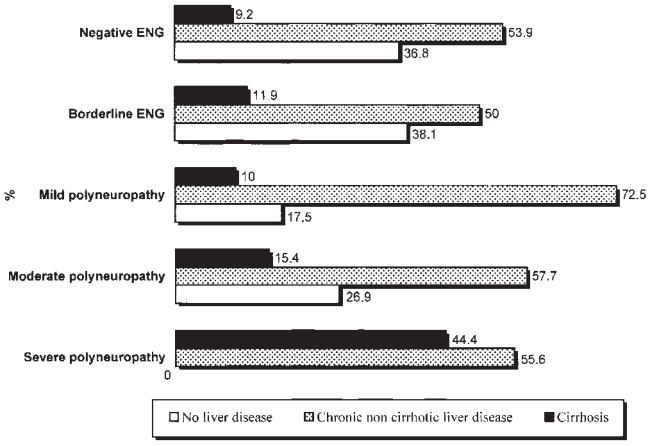
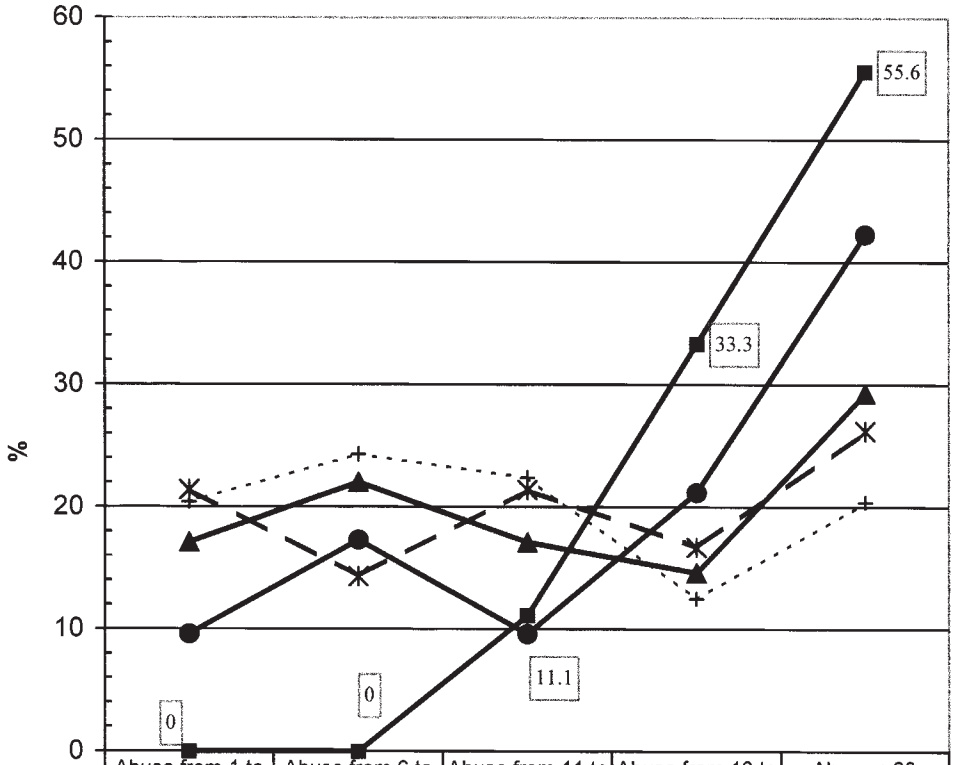


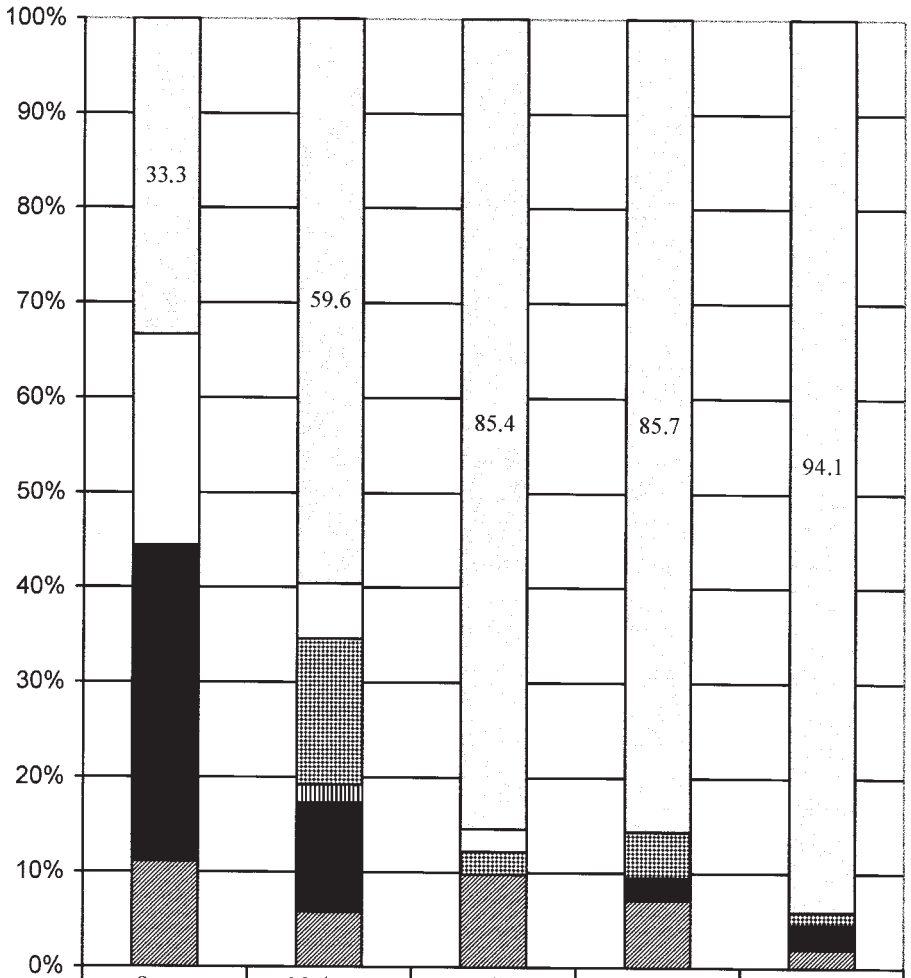
Fig. 1. Relationship between polyneuropathy and liver disease. $P < 0.05$. ENG, electroneurographic investigation.

As shown in Table 3, 48 of our subjects (16.2%) showed symptoms of polyneuropathy. These were predominantly muscle cramps and paraesthesiae (Fig. 3). On ENG grounds, the absence of neuropathy was documented in 51.4% of the subjects, whereas the remaining 48.6% had abnormal ENG signs suggestive of varying degrees of polyneuropathy: 3% severe, 17.6% moderate, 13.9% mild, and 14.2% borderline (Table 4). The rate of polyneuropathy was significantly higher in men than in women, as has recently been reported in the literature (Wetterling *et al*., 1999). The frequency of both subjective and objective symptoms increased significantly with age in our sample (Tables 3 and 4). These results clearly show that some of our asymptomatic subjects had signs of polyneuropathy on ENG, whereas some of the symptomatic subjects had none (Fig. 3). The former finding may be related to the fact that alcoholics tend to treat negative symptoms such



	Abuse from 1 to 5 years	Abuse from 6 to 10 years	Abuse from 11 to 15 years	Abuse from 16 to 20 years	Abuse >20 years
Severe polyneuropathy	0	0	11.1	33.3	55.6
Moderate polyneuropathy	9.6	17.3	9.6	21.2	42.3
Mild polyneuropathy	17.1	22	17.1	14.6	29.3
Borderline ENG	21.4	14.3	21.4	16.7	26.2
Negative ENG findings	20.4	24.3	22.4	12.5	20.4

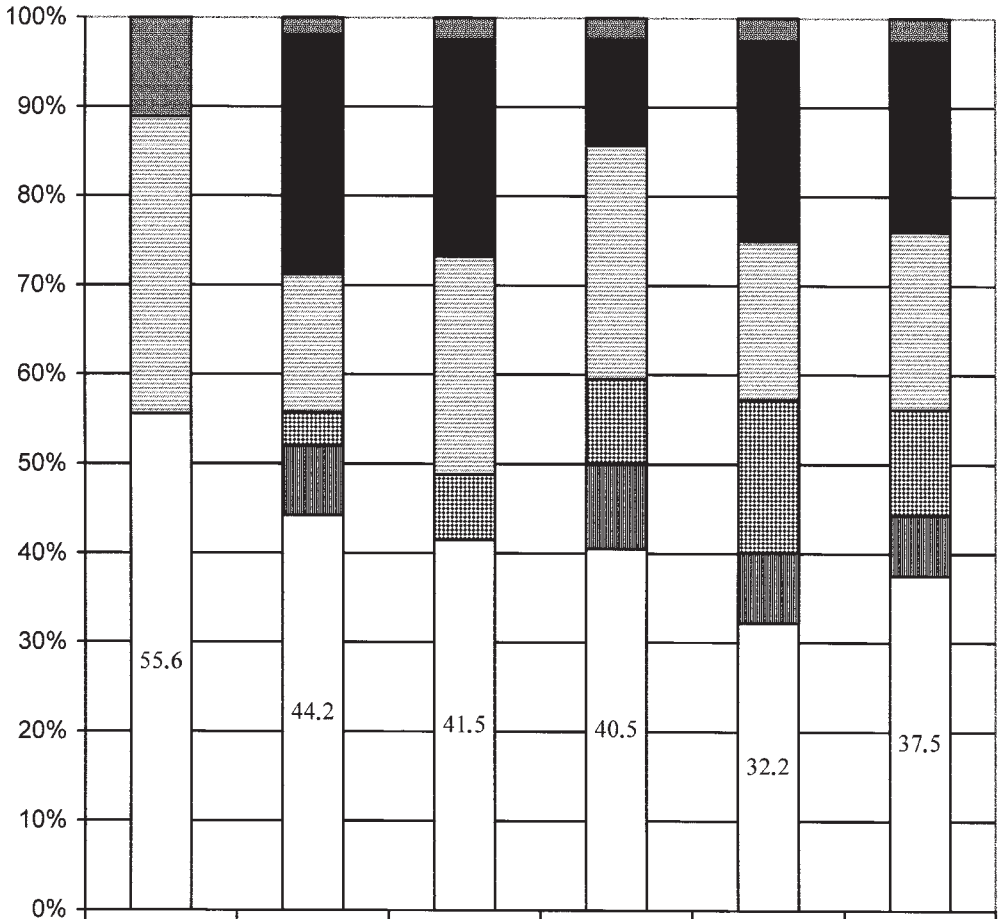
Fig. 2. Relationship between severity of polyneuropathy and the duration of alcohol misuse. $P < 0.05$. ENG, electroneurographic investigation.



	Severe polyneuropathy	Moderate polyneuropathy	Mild polyneuropathy	Borderline ENG findings	Negative ENG findings
Asymptomatic	33.3	59.6	85.4	85.7	94.1
Muscle weakness	22.2	5.8	2.4	0	0
Paraesthesiae and muscle cramps	0	15.4	2.4	4.8	1.3
Hyperalgesia	0	1.9	0	0	0
Muscle cramps	33.3	11.5	0	2.4	2.6
Paraesthesiae	11.1	5.8	9.8	7.1	2

Fig. 3. Distribution of symptoms in the different polyneuropathy groups: relationship between polyneuropathy and symptoms. $P < 0.001$.

as hypostenia and hypoaesthesia as of no account, whereas the latter result may be explained by the presence of generally hyperalgic forms of polyneuropathy that selectively involve small nerve fibres whose alterations are not revealed by routine ENG investigations. There was, however, a highly significant correlation between the referred symptoms and the polyneuropathy documented with the use of ENG. As demonstrated in previous studies (Behese and Buchtal, 1977; Wetterling *et al*., 1999) the duration of alcohol misuse was one of the most important contributing factors to the occurrence of polyneuropathies. Subjective symptoms were found to occur after relatively short durations of misuse (1–5 years), whereas the development of severe polyneuropathy required 10 or more years of excessive drinking (Fig. 2). As shown, the longer the duration of excessive alcohol consumption, the greater the number of subjects with neuropathy. The task of the physician, therefore, is carefully to evaluate both the symptoms referred by the patient and the ENG findings, even when there is a relatively short history of alcohol misuse. Early observation of subtle signs of the disease will allow prompt therapeutic intervention, of course in association with the subject's abstinence from alcohol. The cessation of drinking remains the principal if not only means of preventing the progression of the disease. Moreover, it should be recalled that early forms of neuropathy may be completely reversed by stopping drinking (Savoldi, 1995). In our alcoholic subjects, the presence of concomitant



	Severe polyneuropathy	Moderate polyneuropathy	Mild polyneuropathy	Borderline ENG findings	Negative ENG findings	Total consumption
Spirits and beer	11.1	1.9	2.4	2.4	2.6	2.7
Wine, spirits and beer	0	26.9	24.4	11.9	22.4	21.3
Wine and spirits	33.3	15.4	24.4	26.2	17.8	19.9
Spirits	0	3.8	7.3	9.5	17.1	11.8
Beer	0	7.7	0	9.5	7.9	6.8
Wine	55.6	44.2	41.5	40.5	32.2	37.5

Fig. 4. Distribution of the severity of polyneuropathy according to the different types of alcohol consumed. $P < 0.05$.

substance misuse (tobacco smoking, heroin, cocaine, cannabinols) was not significantly related to the presence of polyneuropathy (Table 6). The severity of polyneuropathy showed a significant correlation with the presence of macrocytosis (Table 3) and hepatic disease (Fig. 1), both of which are well-known features of chronic alcoholism. Some authors attribute the concomitant presence of macrocytosis, liver disease and polyneuropathy in chronic alcoholism to vitamin B deficiency (pyridoxine and thiamine), due to the malabsorption and malnutrition occurring in chronic alcoholics (Victor, 1984; Windebank, 1993; Manzo *et al*., 1994). From our data, the contemporaneous presence of HCV infection doesn't seem to be an important element for the genesis or aggravation of polyneuropathy in alcoholics. In conclusion, the present results confirm that polyneuropathy occurs in a high percentage of chronic alcoholics and that many cases may be clinically asymptomatic. Both the duration of alcohol misuse and the type of alcoholic beverage consumed are particularly relevant. In fact, wine appears to be the main factor for the development of polyneuropathy, perhaps not only in relation to the amount consumed but also the possible toxicants it contains (lead in particular). However, we were unable to examine correlations between the presence of polyneuropathy and daily alcohol intake, due to the lack of sufficiently reliable information provided by the patients under examination. However, we were able to establish that their daily intake was always >100 ml alcohol. The finding of a strong correlation between polyneuropathy and the presence of liver disease and macrocytosis confirms yet again the plethora of diseases related to alcohol misuse.

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