ATAXIA OF STANCE IN DIFFERENT TYPES OF ALCOHOL DEPENDENCE — A POSTUROGRAPHIC STUDY

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Abstract — The aim of this study was to assess the prevalence of ataxia of stance in different types of alcohol-dependent patients. Posturographic measurements were performed in 82 abstinent alcohol-dependent patients and 54 healthy controls in order to analyse postural control. According to Lesch and co-workers, alcohol dependence was classified as total abstinence (Type I), drinking without loss of control (Type II), fluctuating course (Type III), and persistent severe drinking (Type IV). The mechanisms of alcohol dependence in these subtypes can be summarized as follows: Type I patients drink alcohol to counteract symptoms of alcohol withdrawal; Type II patients use alcohol as an agent for solving conflicts; Type III patients drink alcohol to 'treat' an affective disorder; and Type IV patients have a history of pre-alcoholic neurological and/or psychiatric disorders. The neurological examination showed pathological findings in 39%, whereas posturographic measurements uncovered impaired postural control in 61% (χ² = 8.8, P = 0.003). Comparing the different study groups revealed that ataxia of stance was most common in alcohol-dependent patients classified as Type IV (τ = 0.24, P = 0.005).

In conclusion, posturographic measurements are superior to the clinical examination in detecting postural imbalance in alcohol-dependent patients. The prevalence of postural imbalance is highest in patients classified by Lesch as Type IV. Consequently, this type of alcohol dependence — characterized by pre-alcoholic neurological and/or psychiatric disorders, bears the highest risk of developing ataxia of stance.

INTRODUCTION

Among alcohol-related neurological disorders, impaired postural control occurs not only as the immediate dose-dependent effect of alcohol intake (Schuckit, 1985; Lex et al., 1988; Kubo et al., 1990; Ledin and Ödkvist, 1991a; Monteiro et al., 1991; Bauer and Hesselbrock, 1993; Goebel et al., 1995; Schuckit et al., 1996), but also as a consequence of chronic alcohol intoxication (Diener et al., 1984; Scholz et al., 1986; Ledin and Ödkvist, 1991b). The control of upright posture requires: (1) neuronal pathways which process visual, vestibular and proprioceptive inputs; (2) complex motor programmes involving cerebellar, pyramidal, and extrapyramidal functions; (3) an adequate neuromuscular response. Impairment of postural control in alcohol dependence may be caused by cerebellar dysfunction and/or peripheral neuropathy. The cerebellar dysfunction is morphologically related to an atrophy of the anterior lobe of the cerebellum. Clinically, it is associated with a predominant ataxia of stance and gait, and posturographically, it is characterized by an increased antero-posterior body sway with a frequency of 3/s which can be compensated partially by visual control (Diener et al., 1984; Scholz et al., 1986). Alcoholic peripheral neuropathy is due to axonal nerve damage, and it is associated with increased vibratory perception thresholds, abolished ankle jerks, and hypaesthesia (Behse and Buchthal, 1977). Until now, however, there is a lack of information about factors predicting the risk of postural imbalance in alcohol-dependent patients and none of the earlier posturographic studies distinguished between different types of alcohol dependence. Such differentiation, however, is essential since alcohol dependence does not represent a homogeneous disorder, but a complex development producing various clinical...
pictures with differing symptom constellations requiring different therapeutic approaches.

Multivariate aetiologic models have described alcohol dependence as a developmental interaction between toxic effects of alcohol, personality traits, and social surroundings (Glatt, 1982; Vaillant, 1983; Lesch, 1985). Accordingly, efforts have been made to define subtypes of alcohol dependence in order to estimate the prognosis of each individual patient and to define specific treatment goals. Bowman and Jellinek (1941) distinguished four groups of alcoholics based on drinking behaviour. More recently, Cloninger et al. (1981) identified two types of alcohol dependence: Type I alcoholics were characterized by a late onset of alcohol dependence, provoked by environmental factors in persons with high harm avoidance, low novelty seeking, and high reward dependence, whereas Type II alcoholics were observed to exhibit alcohol-seeking behaviour early in life, based on strong heritable influences independent of the environment and showing an impulsive, risk taking, antisocial behaviour. Morey and Skinner (1986) differentiated three types of alcoholics designated as early stage problem drinkers, affiliative drinkers, and schizoid drinkers. Zucker (1987) described four types of alcohol dependence and distinguished between (1) antisocial, (2) developmentally cumulative, (3) negative affect, and (4) developmentally limited alcoholism. Babor (1992) developed a system of Type A and Type B alcoholism: Type A was characterized by late onset, fewer childhood risks, less severe dependence, fewer alcohol-related physical and social consequences, less previous treatment for alcohol problems, less psychopathological dysfunction, and less distress in the areas of work and family. Type B, on the other hand, was characterized by earlier onset, more childhood and familial risk factors, greater severity of dependence, polydrug use, more serious consequences, a chronic treatment history and more life stress and psychopathological dysfunction.

Lesch (1985), Lesch et al. (1988), and Lesch and Walter (1996) have developed a classification system, or typology (Types I to IV) based on a prospective study in 444 patients, which considered a series of features including parental factors, physical, psychological and social development of the patient, drinking behaviour, biochemical characteristics, psychiatric factors, and neurologi-
All patients underwent in-patient withdrawal therapy at the same institution (Anton Proksch Institute, Vienna, Austria) and had finished organic withdrawal at least 2 weeks prior to this investigation. To classify the type of alcohol dependence cross-sectionally, we used a semi-structured interview, a psychiatric exploration, and a neurological examination (Lesch, 1985; Lesch et al., 1990; Plinius Major Society, 1994). Each patient was assigned to the appropriate type of alcohol dependence following a decision tree published elsewhere (Lesch et al., 1990, 1996). The neurological features in this decision tree included alcohol-related disorders, such as withdrawal symptoms, seizures, peripheral neuropathy, and cognitive impairment, as well as disorders not related to alcohol, e.g. a history of perinatal cerebral damage, febrile convulsions, head injury, meningitis, or encephalitis. The psychiatric features covered psychological withdrawal symptoms, affective disorders, depressive symptoms, sleep disorders, suicidal thoughts, personality traits such as aggressive behaviour, oligophrenia and several others. Assigning an individual patient to the appropriate type of alcohol dependence starts with checking whether he/she fulfils the criteria of Type IV followed by Types III, I, and II. The characteristics of the drinking behaviour, the social, psychological, and somatic features as well as the supposed main pathogenic factor in each of the four types of alcohol dependence were summarized elsewhere (Lesch et al., 1988, 1996).

To quantify and analyse body sway, measurements on a posturographic platform (Kollegger et al., 1992; Wöber et al., 1993) were performed. Patients with uncorrectable severe reduction of visual acuity, double vision, peripheral vestibular lesions, pareses of the lower limbs not related to alcoholic peripheral neuropathy, sequelae of bone features interfering with postural equilibrium, and patients who did not cooperate sufficiently during the examination were excluded. In addition, patients who had experienced delirium tremens within 2 months of the examination were also excluded. Posturography was carried out in a quiet, well-lit, comfortably heated room. The subjects were barefoot and were told to stand on the posturographic platform in an upright position in as stable manner as possible, arms at the side, feet 4 cm apart. They were allowed to compensate for impaired postural control by any strategy, except external support, provided that the position of the feet remained unchanged. One measurement period lasted 20 s. We performed 20 measurements per patient, 10 with eyes open and 10 with eyes closed. In the 'eyes open' condition, the patients viewed a black disc (diameter 10 cm) fixed to the wall 2 m in front of their eyes.

The posturographic equipment consisted of a force measuring platform with piezo-electric strain gauges in all four corners connected to an electronic control unit (Toennies Medical Electronics, Freiburg, Germany) and a personal computer. The strain gauges registered the movements of body sway projected to the centre of foot pressure (CFP). Before the first measurement, a normalizing procedure was completed to compensate for body weight. The signals visualized represented the distance the CFP vector was travelling during 1 s and the area covered by the movement of the vector. The result of the measurement was a sway direction diagram characterized by four sway parameters: sway path, sway area, antero-posterior sway, and lateral sway. To obtain the direction of body sway, it was necessary to divide the circle of possible sway directions into eight segments. The vectors resulting from the displacement of the CFP from the centre of the platform were summed up within the eight segments, according to their directions. Thus, body sway could be visualized, quantified, and analysed sensitively, simply, and accurately.

The control group consisted of 54 healthy subjects similar in age and gender. All control subjects showed normal neurological findings. They consumed less than 40 g of alcohol/day and none had a history of alcohol abuse or dependence. The protocol of the posturographic measurements was the same as in the patient group and normal values were defined as the mean ± 2 SD.

For calculations and statistical analysis, SAS software (SAS Institute Inc, 1985) was used. To analyse the demographic and clinical data, Kendall r correlation coefficients and $\chi^2$ tests were applied as appropriate. In each of the eight posturographic parameters, i.e. sway path, sway area, antero-posterior and lateral sway during stance with eyes open and eyes closed, the mean of the 10 measurements in each patient was used for further calculations. To examine the hypotheses that
Table 1. Demographic findings, duration of alcohol dependence, alcohol intake during the 6 months before admission for withdrawal therapy and estimated lifetime alcohol consumption in four types of alcohol dependence

<table>
<thead>
<tr>
<th>Demography</th>
<th>Type I (n = 17)</th>
<th>Type II (n = 21)</th>
<th>Type III (n = 26)</th>
<th>Type IV (n = 18)</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>39.8 ± 8.5</td>
<td>41.0 ± 9.2</td>
<td>41.6 ± 10.0</td>
<td>41.1 ± 9.7</td>
<td>( \tau = 0.02 ) n.s.</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>16/1</td>
<td>17/4</td>
<td>24/3</td>
<td>17/0</td>
<td>( \chi^2 = 4.40 ) n.s.</td>
</tr>
<tr>
<td>Education (years, mean ± SD)</td>
<td>9.3 ± 1.2</td>
<td>9.3 ± 1.2</td>
<td>9.1 ± 0.8</td>
<td>9.3 ± 1.0</td>
<td>( \tau = 0.02 ) n.s.</td>
</tr>
<tr>
<td>Profession</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skilled</td>
<td>11</td>
<td>11</td>
<td>14</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Unskilled</td>
<td>6</td>
<td>5</td>
<td>7</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Retired</td>
<td>0</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Duration of alcohol dependence (years, mean ± SD)</td>
<td>14.8 ± 10.2</td>
<td>10.5 ± 6.1</td>
<td>12.4 ± 6.8</td>
<td>17.7 ± 9.8</td>
<td>( \tau = 0.12 ) n.s.</td>
</tr>
<tr>
<td>Pre-admission alcohol consum-</td>
<td>64.8 ± 19.3</td>
<td>55.4 ± 22.2</td>
<td>59.2 ± 22.1</td>
<td>70.2 ± 19.8</td>
<td>( \tau = 0.04 ) n.s.</td>
</tr>
<tr>
<td>lifetime alcohol consumption (kg, mean ± SD)</td>
<td>1684 ± 1271</td>
<td>1095 ± 783</td>
<td>1377 ± 868</td>
<td>2395 ± 1271</td>
<td>( \tau = 0.22 ) ( P = 0.009 )</td>
</tr>
</tbody>
</table>

For statistical analysis, Kendall \( \tau \) correlation coefficients and \( \chi^2 \) tests were calculated. n.s. Denotes not significant.

postural imbalance is related to the type of alcohol dependence and that this imbalance increases from healthy subjects to patients classified as Type IV, Kendall \( \tau \) correlation coefficients were calculated. Kendall \( \tau \) correlation coefficients were also calculated to analyse the relations between alcohol consumption, clinical, and posturographic findings. For these calculations, the pathological posturographic values were summed. Accordingly, each patient had a score between zero (all posturographic parameters < mean + 2 SD) and eight (all parameters > mean + 2 SD). The level of statistical significance was fixed at \( P < 0.05 \).

RESULTS

Classification of alcohol dependence

According to the criteria of Lesch (1985), Lesch et al. (1988), and Lesch and Walter (1996), 17 patients were classified as Type I, 21 as Type II, 26 as Type III, and 18 patients as Type IV. The durations of alcohol dependence and daily alcohol consumption prior to admission for alcohol withdrawal therapy were similar in all four types. The estimated lifetime alcohol consumption, however, was highest in Type IV and much lower in the other types and this finding was statistically significant (Table 1).

Demographic findings

The mean age of the patients was 41 ± 9.3 years (range 20–67 years), which did not differ between the four subtypes. Only a small minority of patients (8 out of 82) were females. The level of education was similar in all subtypes, only eight patients had completed more than 9 years of school. The majority of patients were skilled (n = 42) or unskilled workers (n = 24). Unemployment or retirement (because of alcohol dependence) was found most frequently among patients classified as Type IV and least frequently among those classified as Type I, but this difference was not statistically significant (Table 1).

Neurological findings

The clinical neurological examination revealed normal findings in 52 patients (63.4%), symptoms and/or signs of alcoholic neuropathy in 29 (35.4%) and cerebellar signs in nine patients (11%). Eight of these patients had both neuropathic and cerebellar signs. The prevalence of neuropathy showed a statistically significant increase from Type I to Type IV. The prevalence of cerebellar signs and other neurological abnormalities was also highest in Type IV, but these differences did not reach the level of statistical significance.
antero-posterior sway increased significantly from 35.3% in Type I to 100% in Type IV (T = 0.32, P = 0.0003). Sway path, sway area, lateral sway, and antero-posterior sway increased significantly from healthy subjects to patients classified as Type IV, whereas lateral sway was similar in the five study groups (Tables 2–5, Figs 1–4). In addition, postural imbalance was consistently more pro-
Sway path

Fig. 1. Median, upper, and lower quartile of sway path during upright stance with eyes open and eyes closed in healthy controls and four types of alcohol dependence. **P < 0.001.

Sway area

Fig. 2. Median, upper, and lower quartile of sway area during upright stance with eyes open and eyes closed in healthy controls and four types of alcohol dependence. **P < 0.01; ***P < 0.001.

nounced during stance with eyes closed than with eyes open (Figs 1–4).

Relation of clinical and posturographic findings

Impaired postural control was uncovered by posturographic measurements significantly more often than by purely clinical assessment (χ² = 8.8, P = 0.003). Pathological posturographic findings were found in 46.2% of patients with normal neurological findings, in 82.8% of patients with signs of alcoholic neuropathy, and in 88.9% of subjects with cerebellar signs. Postural imbalance was strongly related to the presence of peripheral neuropathy (τ = 0.38, P = 0.0001) as well as to the presence of cerebellar signs (τ = 0.30, P = 0.001).

Relation of clinical findings and alcohol consumption

The alcohol consumption during the 6 months before admission for withdrawal therapy was related neither to the prevalence of peripheral
Antero-posterior sway

![Antero-posterior sway graph](image)

Fig. 3. Median, upper, and lower quartile of antero-posterior sway during upright stance with eyes open and eyes closed in healthy controls and four types of alcohol dependence.

* ***P < 0.001.

Lateral sway

![Lateral sway graph](image)

Fig. 4. Median, upper, and lower quartile of lateral sway during upright stance with eyes open and eyes closed in healthy controls and four types of alcohol dependence.

neuropathy (τ = −0.06, P = 0.5) nor to the prevalence of cerebellar signs (τ = 0.03, P = 0.7). By contrast, the estimated lifetime alcohol consumption was related to peripheral neuropathy (τ = 0.24; P = 0.007), but not to cerebellar signs (τ = 0.10; P = 0.3).

Relation of posturographic findings and alcohol consumption

Similar to the clinical findings, the pre-admission alcohol consumption level was not related to the number of pathological posturographic values (τ = 0.07; P = 0.4). Increased lifetime alcohol consumption, however, was associated with increased postural imbalance (τ = 0.26; P = 0.001).

DISCUSSION

This study on postural control in abstinent alcohol-dependent patients, showed pathological posturographic findings in more than 60% of all patients and in almost 50% of those with normal...
clinical neurological findings. Impaired postural control was associated with the type of alcohol dependence and occurred most frequently in Lesch's Type IV (Lesch, 1985; Lesch et al., 1988; Lesch and Walter, 1996), and less often in Types I, II, and III, even though the alcohol consumption prior to admission for alcohol withdrawal therapy was similar in all four types. This discrepancy may be explained by differences in the underlying pathogenic mechanisms. Type I patients are characterized by a primary organic weakness, Type II by a developmental disturbance, and Type III by sociopathic development. Type IV patients have a history of neurological and psychiatric disorders (which precede the development of alcohol dependence) as well as a developmental disturbance. However, Type IV patients also had the highest lifetime alcohol consumption. Accordingly, impaired postural control in Type IV may be the consequence of both pre-alcoholic neurological damage and a certain type of alcohol dependence.

Concerning the importance of demographic data, the small number of women included did not allow us to examine gender differences. Age and onset of alcohol dependence were similar in all four types. This finding is in contrast to other typologies (Cloninger et al., 1981; Babor et al., 1992), which use these parameters as diagnostic features.

The pattern of postural imbalance was similar to that reported by Diener et al. (1984) and Scholz et al. (1986). The percentage of pathological posturographic findings was also comparable to earlier reports (Scholz et al., 1986). However, our study is the first to demonstrate that the prevalence of postural imbalance is not evenly distributed among patients fulfilling the criteria of alcohol dependence, but that a well-defined group characterized by pre-alcoholic neurological and/or psychiatric disorders (Lesch's Type IV) is predominantly afflicted.

The prevalence of alcohol-related neuropathy was also related to the type of alcohol dependence, showing the highest prevalence in Type IV. The overall percentage of patients with neuropathy was within the range reported in the literature. In our study, 29 out of 82 patients (35.4%) had signs of peripheral neuropathy consisting of reduced vibration sense and absent ankle jerks in 75.9%, more severe sensory deficits in 13.8% and pareses in 10.3%. Scholz et al. (1986) found clinical signs of peripheral neuropathy in 45% and Tuck and Jackson (1991) reported a prevalence of 34%. Investigating the vibration sense in alcohol-dependent patients and healthy controls, Sosenko et al. (1991) found increased thresholds in 18% of younger and 47% of older alcoholic subjects. Palliyath and Schwartz (1993) reported findings which suggest that peripheral nerve functions improve in chronic alcoholic patients with abstinence. In agreement with this report, Monforte et al. (1995) have shown that peripheral neuropathy in alcohol-dependent patients is due to a dose-related toxic effect of alcohol. Similarly, we found an association between the lifetime consumption of alcohol and peripheral neuropathy.

The lack of an association between cerebellar signs and type of alcohol dependence might be explained by the small number of patients, since the raw data showed at least a trend towards an increase of cerebellar signs from Type I to Type IV. Concerning the pre-admission and lifetime alcohol consumption, however, there was not even a trend associated with the prevalence of cerebellar signs. Similarly, a comparison between ataxic and non-ataxic alcohol-dependent patients (Estrin, 1987) revealed no relation between alcohol consumption and ataxia. On the contrary, the ataxic patients showed even less alcohol consumption than the non-ataxic controls, suggesting that alcoholic cerebellar degeneration is not a dose-dependent phenomenon. This hypothesis, however, seems to be contradicted by reports that ataxia in alcoholic cerebellar atrophy may improve through abstinence (Diener et al., 1984; Leblhuber et al., 1985).

Examining the relation between clinical and posturographic findings demonstrated clearly that posturographic measurements are superior to the clinical assessment in detecting postural imbalance. The issue of the causal relation between peripheral neuropathy, cerebellar signs, and postural imbalance remains controversial, however. Melgaard and Ahlgren (1986) suspected that peripheral neuropathy contributes to the ataxia in alcohol-dependent patients. Scholz et al. (1986), however, found no correlation between the severity of peripheral neuropathy and the severity of cerebellar dysfunction, and concluded that cerebellar damage and peripheral neuropathy result from different mechanisms of alcohol.
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toxicity. In our study, there was a strong correlation between postural imbalance and both clinical signs of peripheral neuropathy and cerebellar signs. In addition, all but one patient with cerebellar signs had signs of peripheral neuropathy. Accordingly, there is a considerable overlap between cerebellar and peripheral dysfunction. The (subclinical) postural imbalance as measured by posturography might be based on either cerebellar or peripheral dysfunction or a combination of both. The posturographic pattern characterized by antero-posterior body sway, which can be compensated partially by visual control, seems to favour a cerebellar origin, since postural sway in peripheral neuropathy is usually found to be omnidirectional (Ucciofi et al., 1995). Further studies, however, are required to clarify this issue.

In conclusion, our study shows that posturographic measurements are superior to clinical examination in detecting postural imbalance in abstinent alcohol-dependent patients. The prevalence of postural imbalance is highest in patients classified as Type IV according to Lesch. Consequently, this type of alcohol dependence—characterized by pre-alcoholic neurological and/or psychiatric disorders, bears the highest risk of ataxia of stance.

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REFERENCES


