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# Mechanisms of Postural Ataxia After Intake of Alcohol *

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**Summary.** The effect of an acute intoxication with alcohol on the stability of stance was examined in 12 healthy subjects. They drank 1 l of wine within 1 h. The resulting blood alcohol concentrations ranged between 0.9 and 1.67 mg/ml. Static posturography at the end of drinking and 1 h later revealed a significant increase in body sway which was mainly due to an increase in antero-posterior sway only present with closed eyes. A comparison of the results of posturography after acute intoxication with the data of patients with permanent lesions confined to the different functional subunits of the cerebellum shows that the acute effect of alcohol largely resembles that of a chronic lesion of the cerebellar anterior lobe (the spinocerebellum).

**Key words:** Alcohol, effect on the spinocerebellum – Posturography

**Zusammenfassung.** Die Alkoholwirkung auf die Standsicherheit wurde in einem Akutexperiment bei 12 gesunden Versuchspersonen untersucht. Sie tranken innerhalb 1 h 1 l Wein. Die danach gemessenen Blutalkoholspiegel lagen zwischen 0,9 und 1,67 mg/ml. Die Standmessung am Ende der Trinkphase und 1 h danach ergab bei geschlossenen Augen eine signifikante Zunahme der Körperunruhe, vor allem durch vermehrtes antero-posteriores Schwanken. Der Vergleich der Ergebnisse der Posturographie im Akutexperiment und bei Patienten mit umschriebenen cerebellären Läsionen verschiedener Lokalisation zeigt, daß auch im Akutexperiment, wie durch den chronischen Abusus, vor allem das Spinocerebellum durch den Alkohol in seiner Funktion beeinträchtigt wird.

**Schlüsselwörter:** Alkohol, Wirkung auf Spinocerebellum – Posturographie

## Introduction

Postural imbalance after alcohol intake is a well-known phenomenon. Static and dynamic posturography is a method which allows to relate specific sway patterns

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**Table 1.** Means/SD of sway parameters at the beginning and 60 respectively 120 min after alcohol intake. Analysis of variance ($\chi^2_R$)

| Parameter | Eyes | 0 min | 60 min | 120 min | $\chi^2_R$ | Sign. |
|---|---|---|---|---|---|---|
| Sway path (cm) | Open | 16.5 / 4.2 | 22.2 / 9.9 | 21.9 / 6.5 | 3.6 | – |
| Sway path (cm) | Closed | 26.1 / 8.6 | 32.9 / 14.9 | 50.4 / 26.7 | 12.0 | + ◀ |
| Sway area ($cm^2$) | Open | 1.9 / 1.1 | 5.2 / 5.1 | 4.3 / 3.7 | 3.9 | – |
| Sway area ($cm^2$) | Closed | 3.6 / 1.6 | 6.9 / 5.8 | 13.5 / 10.6 | 8.9 | + ◀ |
| Antero-post. sway | Open | 2.6 / 0.8 | 3.9 / 2.7 | 4.3 / 1.5 | 1.5 | – |
| Antero-post. sway | Closed | 4.8 / 2.5 | 6.6 / 3.1 | 13.3 / 11.2 | 9.9 | + ◀ |
| Lateral sway | Open | 1.9 / 1.1 | 2.4 / 1.4 | 1.9 / 0.8 | 0.9 | – |
| Lateral sway | Closed | 2.4 / 1.4 | 2.9 / 1.9 | 3.1 / 1.6 | 0.5 | – |
| Romberg Q. sway path | – | 1.7 / 0.7 | 1.6 / 0.6 | 2.3 / 1.0 | 5.6 | – |
| Romberg Q. sway area | – | 2.4 / 1.4 | 2.9 / 1.9 | 3.1 / 1.6 | 4.1 | – |
| Q AP: LAT | Open | 2.0 / 1.6 | 2.7 / 3.5 | 2.8 / 1.9 | 3.9 | – |
| Q AP: LAT | Closed | 2.8 / 2.1 | 3.1 / 1.9 | 5.3 / 5.0 | 2.9 | – |
| rms CFP AP | Open | 20.1 / 4.3 | 28.9 / 24.3 | 27.8 / 6.7 | 5.4 | – |
| rms CFP AP | Closed | 30.5 / 16.5 | 36.2 / 14.3 | 51.9 / 27.1 | 9.9 | + ◀ |
| rms sinus CFP | Open | 151.8 / 63.2 | 195.4 / 116 | 165.4 / 58.2 | 2.5 | – |
| Amplitude, peak CFP (mV) | Open | 40.6 / 23.5 | 54.2 / 41.4 | 42.0 / 21.3 | 1.5 | – |
| Amplitude, head (mV) | Open | 17.4 / 15.0 | 14.9 / 8.4 | 12.5 / 6.6 | 0.1 | – |
| Amplitude, hip (mV) | Open | 15.2 / 9.2 | 20.4 / 15.8 | 15.4 / 9.6 | 4.6 | – |
| Latency TS 1 (ms) | Open | 42.5 / 5.8 | 48.3 / 5.7 | 49.0 / 7.1 | 3.9 | – |
| Latency TS 2 (ms) | Open | 91.5 / 12.5 | 93.0 / 11.5 | 100.9 / 14.0 | 3.0 | – |
| Latency TA 1 (ms) | Open | 123.2 / 19.7 | 156.5 / 24.5 | 164.3 / 27.9 | 12.5 | + ◀ |

in patients to different lesions of functional subunits within the cerebellum (Diener 1982). To investigate whether alcohol has a specific affinity to one or several of the structures involved in postural stabilization we compared the results of posturography and electromyography after an acute alcohol intoxication with those obtained from patients.

## Material and Methods

Subjects stood on a force measuring platform by means of which the displacement of the center of foot pressure (CFP) was recorded in the antero-posterior and lateral direction. Displacements of the head and the hip were recorded by means of goniometers. An on-line computer program calculated the sway path, the sway area, and the antero-posterior, as well as the lateral sway components within a time interval of 25 s (for details see Diener 1982). Romberg's quotient compares the parameters of sway with closed and open eyes and indicates the amount of visual stabilization. A histogram of sway direction was also calculated. Additionally, the displacements of the CFP were fed into a Fourier analyzer (0.01–10 Hz, 0.025 Hz steps) calculating the power spectrum and its root mean square. Dynamic posturography used sinusoidal movements of the platform with 1 Hz and measured the displacements of the head, the hip, and the CFP which then were further processed by Fourier analysis (sample interval 40 s). EMGs of the anterior tibial and triceps surae muscles were recorded with surface electrodes after sudden tilting of the platform toe-up. EMG signals were amplified, rectified, and averaged over eight runs. Latencies from the beginning of the platform movement were measured by visual identification on single runs.

### *Procedure*

First we recorded two 1-min periods of sway on the stable platform with open and closed eyes, respectively. Afterwards, the platform was tilted sinusoidally in pitch with a frequency of 1 Hz and an amplitude of +/− 4° for an identical time interval. Then eight ramp movements were performed with an amplitude of 4° and a velocity of 50°/s. The time interval between the single platform movements toe-up varied randomly between 3 and 10 s. Experiments with the platform moving were invariably performed under visual control.

Following the initial reference testing, subjects drank 1 l of wine containing 9% alcohol (vol.) within 1 h. The whole test sequence with the stable and moving platform was then repeated twice, first immediately after alcohol intake and second, 1 h thereafter. A blood sample for the spectrographic determination of blood alcohol concentration was finally taken. We also interrogated our subjects about their subjective estimation of postural stability.

### *Subjects*

Eleven males and one woman were investigated. All were physically in good health and were paid for their participation. Their mean age was 23.7 ($s = 2.7$), their mean weight 77.6 ($s = 8.4$) kg.

## Results

The blood level of alcohol roughly 1 h after the end of ingestion ranged between 0.91 and 1.67 mg/ml with a mean of 1.30‰. With the platform stable, sway parameters increased only little with increasing blood levels of alcohol, as long as visual control allowed for postural stabilization. After eye closure sway path and sway area increased significantly (Table 1). This increase in sway was mainly due

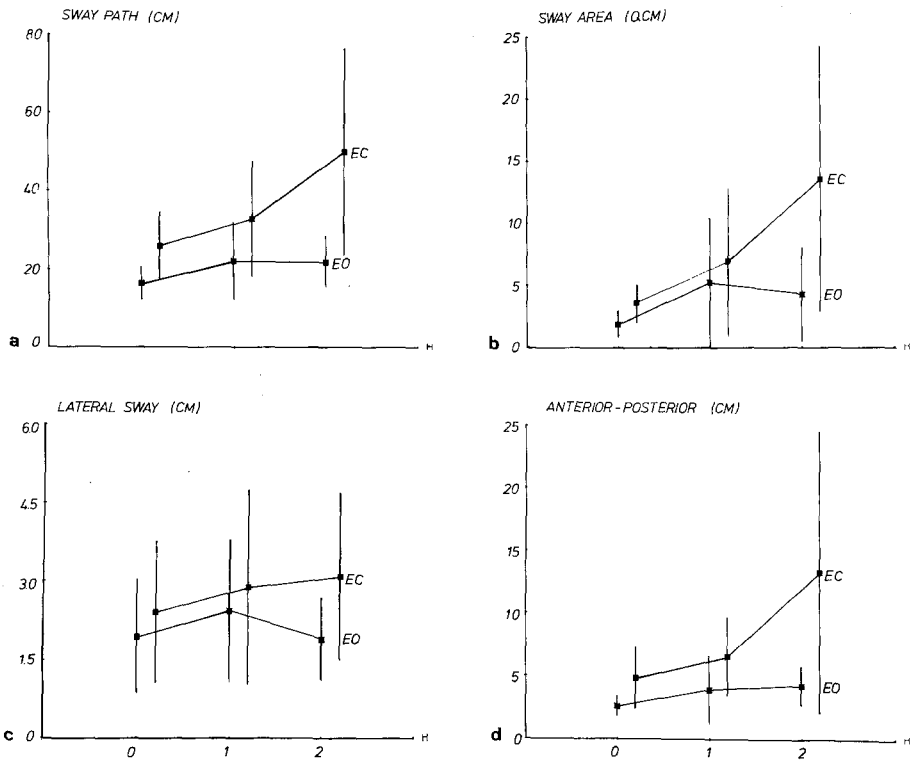


**Fig. 1a–d.** Means and SD ($n=12$) for the calculated sway path (**a**) and sway area (**b**) and the lateral and antero-posterior sway (**c, d**) on the stable platform before (0), at the end of drinking (1) and 1 h later (2). EO = eyes open; EC = eyes closed

to an increase of sway in the antero-posterior direction (Fig. 1). The root mean squares (rms) of the power spectra, which give a measurement of the global instability, also increased with alcohol when visual stabilization was excluded. Despite the measured increase in body sway subjects did not perceive their instability.

The postural sway during sinusoidal platform movements was measured in terms of the amplitude of the dominant peak within the Fourier power spectrum at 1 Hz. With eyes open, the displacement of CFP, hip, and head was independent of alcohol compared to initial testing. This means that at the level of intoxication tested, visual stabilization is uninfluenced by alcohol even with dynamic disturbances. Testing with eyes closed was not performed since without visual stabilization alcohol already destabilized posture under static conditions.

After a sudden platform tilt toe-up a stretch reflex can be observed in the triceps surae muscle with a latency of 40–50 ms. After 90–100 ms a second EMG response is evoked. This corresponds to a long loop reflex (M 2). Under our experimental conditions both EMG responses further destabilize upright posture, since they support the passive backward displacement of the body induced by the platform tilt. Upright posture is guaranteed by a later EMG response in the antagonistic anterior tibial muscle (mean latency 123 ms). After

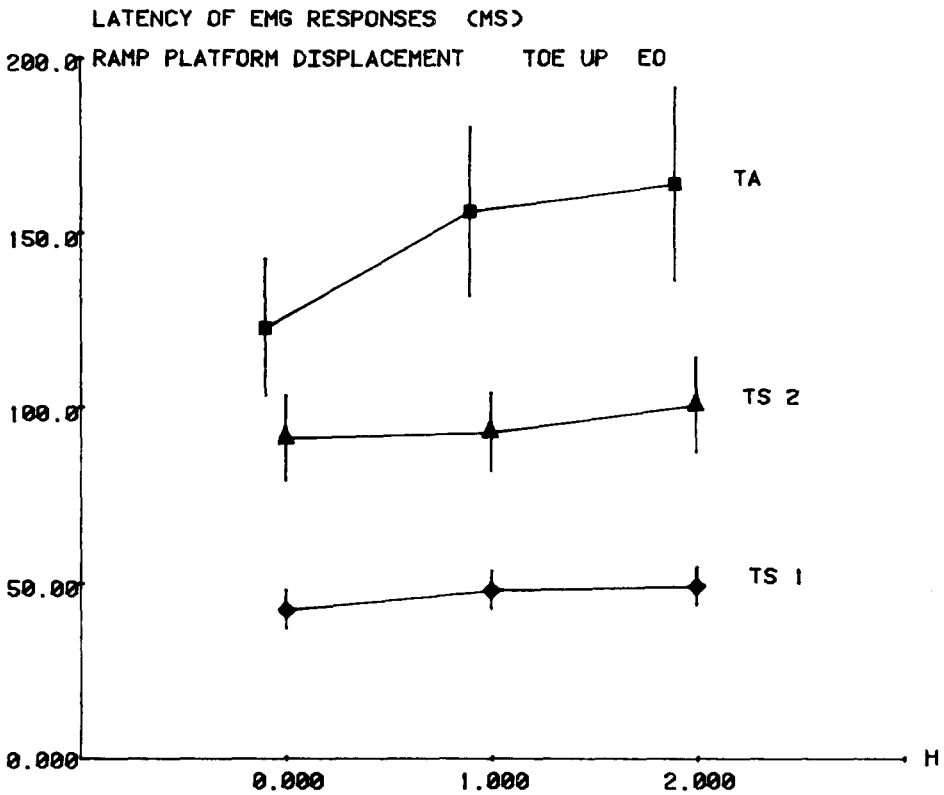


**Fig. 2.** Means and SD ($n=12$) for the latency of EMG responses after sudden platform displacements toe-up with eyes open. TS 1 = stretch reflex in the triceps surae; TS 2 = medium latency response in the triceps surae; TA = late antagonistic response in the anterior tibial muscle; 0 = before drinking; 1 = at the end of drinking; 2 = 1 h later

alcohol intake, the latency of both reflex responses in the triceps surae is unchanged (Table 1). The latency of the late stabilizing response in the anterior tibial muscle, however, increases significantly by an amount of 30–40 ms (Fig. 2). Regression coefficients for the relationship between alcohol level within the blood and the difference of the initial sway parameters and their values 2 h thereafter were also calculated. All of them were positive, but none reached the level of significance.

## Discussion

Sway quantifications in normals after the intake of alcohol confirmed the global increase of postural sway earlier observed by others (Begbie 1966; Goldberg 1966; Fregly and Graybiel 1968; Soulairac et al. 1970; Baron et al. 1972; Baron and Soulairac 1972). The critical alcohol blood level for a significant increase ranges between 0.6 and 0.9 mg/ml (Thyssen et al. 1981). New is the finding that within the range of intoxication tested, the alcohol-induced ataxia can be compensated

totally by visual stabilization. The increase in sway with eye closure is nearly exclusively due to an increase in antero-posterior sway. The same two features are also found in patients with an atrophy of the anterior lobe of the cerebellum due to chronic alcohol abuse (Marie et al. 1922; Victor et al. 1959). The mostly preserved visual compensation and the vast predominance of antero-posterior sway are pathognomic for lesions of the spinocerebellum (Dichgans et al. 1976; Mauritz et al. 1979), while patients with an archicerebellar (vestibulo-cerebellar) lesion show an omnidirectional sway and no visual stabilization (Mauritz et al. 1976; Hufschmidt et al. 1980). Patients with hemispheric lesions of the cerebellum show no or only little postural instability (Mauritz et al. 1979). The fact that sinusoidal movements of the measuring platform are similarly well compensated by vision was also seen in patients with anterior lobe atrophy (Diener and Dichgans 1983). It may be concluded, therefore, that the acute intoxication with alcohol also primarily affects the spinocerebellum just as it may occur with the chronic intoxication. The additional interaction of alcohol with vestibular functions, as demonstrated by Fregly et al. (1966), cannot be excluded, but so far there has been no evidence of a predominance of a.p. sway in patients with acute vestibular lesions.

The latencies of the short and medium latency reflexes in the stretched triceps surae muscle were not influenced by alcohol. The changes in reflex amplitude in response to a sudden displacement reported in voluntary ramp finger movements after ingestion of alcohol (Marsden et al. 1977) could not be seen in leg muscles in our experiments. The explanation for the significant delay in the latency of the late antagonistic response in the anterior tibial muscle is difficult, since we, at the moment, do not exactly know, how this response is generated. In analogy with results from patients with Friedreich's ataxia (Diener and Dichgans 1983) and patients with spinal lesions (unpublished observations) we suppose that spinocerebellar and proprioceptive afferences are involved. Patients with a spinocerebellar lesion due to chronic alcohol abuse do not show this phenomenon.

Summarizing the above results for the use of everyday practice we conclude that the action of alcohol on postural stability is most easily seen without visual stabilization when the eyes are closed. The resulting increase in sway is predominantly antero-posterior. The delay of reflex responses of the stabilizing leg muscles after sudden disturbances of upright stance shows the risk of alcohol consumption in highly demanding situations.

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