

Short report

Alcoholic vagal neuropathy: recovery following prolonged abstinence

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SUMMARY Cardiac vagal reflexes were studied in 11 alcoholic subjects, 1 to 6 weeks after withdrawal and again after up to 27 months of continued abstinence. On initial investigation six subjects had vagal neuropathy. On the second occasion only two subjects had vagal neuropathy and significant improvement was seen in the total patient group with regard to heart rate responses to standing, Valsalva's manoeuvre and atropine infusion. Vagal neuropathy in alcoholics may be reversible with abstinence and/or improved nutrition.

Chronic alcohol abuse may cause parasympathetic nerve damage. Novak and Victor¹ described four alcoholics who had dysphonia and dysphagia and confirmed the destruction of the vagus nerve in three of these patients at post-mortem examinations. In previous studies we showed by physiological testing that alcoholics may show abnormal parasympathetic control of the heart² and of the pupil.³ This may be of clinical importance. Autonomic neuropathy is associated with high mortality in patients with diabetes.⁴ Also, autonomic neuropathy may be a factor in impotence in alcoholic patients.⁵ This report describes a sequential study on a group of alcoholic patients to assess the course of vagal neuropathy with continued abstinence.

Subjects

Eleven alcoholics were studied. They had a mean age of 55.2 yr (range 47-62 yr) and, when first studied, a mean period of abstinence of 3.4 weeks (range 1.4-6 weeks). They had previously been drinking 100-300 g ethanol daily over a period of 10 years or more. Seven healthy male control subjects with a mean age of 45.9 yr (range 38-50 yr) also participated in the study.

Methods

Physiological testing of vagal control of the heart was carried out as follows:

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(a) Heart rate responses to standing ("30/15" ratio)

The subject lay supine and basal heart rate was recorded on an electrocardiogram (ECG). The subject was asked to stand up quickly. The longest R-R interval, around the 30th beat after standing and the shortest R-R interval around the 15th beat after standing were expressed as the 30:15 ratio. The normal range for this ratio was taken to be ≥ 1.04 .⁶

(b) Heart rate response to Valsalva's manoeuvre

The subject was seated, and blew for 20 seconds into a mouthpiece attached to an aneroid manometer, a pressure of 40 mmHg being maintained. The longest R-R interval after the manoeuvre and the shortest R-R interval during the manoeuvre were measured from the ECG and expressed as the Valsalva ratio. A Valsalva ratio of ≥ 1.21 was taken as normal.⁷

(c) Heart rate responses to deep breathing

The subject breathed deeply at a rate of 6-8 breaths/min for one min, while sitting. The variation in heart rate was calculated from the average maximum R-R interval and the average minimum R-R interval during expiration and inspiration respectively. Interpretation of the results was based on an age-dependent normal range described by other workers.⁸

(d) Heart rate response to atropine

The subject lay supine. Atropine sulphate (1.8 mg) was administered intravenously at a rate of 0.6 mg/min, with a one minute interval between each 0.6 mg dose. The maximum change in heart rate from basal was measured. A change of >20 beats/min was taken as normal. The tests of vagal function were carried out in all alcoholic subjects at the beginning of the study and again after a period (4-27 months) of continued abstinence. For the control subjects, the vagal function tests were similarly repeated 9-30

months after first assessment. Statistical comparisons of results between the first and the second study were made using Wilcoxon's signed ranks test for related samples.

Results

Six alcoholic subjects had evidence of vagal neuropathy during the first assessment, as determined by abnormalities in two or more tests (patients 6–11, table). The other five alcoholics (patients 1–5) had no significant evidence of vagal neuropathy. The results of both studies for all the patients are shown in the table. Significant difference were found between the first and the second study for the Valsalva ratio ($p < 0.01$), 30:15 ratio ($p < 0.01$) and heart rate response to atropine ($p < 0.02$) for the eleven alcoholic subjects. The mean resting heart rate fell from 79.4 ± 13.7 (SD) at the time of first study to 64.5 ± 6.1 beats/min at the time of the second study ($p < 0.01$).

The results of the vagal function tests for the seven control subjects on the first and second occasions are presented in the table. There were no significant differences between the first and the second study for any test.

Discussion

Alcoholics may suffer central damage to autonomic pathways resulting in hypothermia, and peripheral autonomic involvement may result in sweating loss or disorder of parasympathetic control of the heart and pupil,³ although orthostatic hypotension is rarely a feature of chronic alcoholism.^{9,10} Six of eleven alcoholic subjects in this study had evidence of vagal neuropathy on initial investigation. On follow-up investigation, after 4–27 months of continued abstinence, only two patients (7, 97) had definite evidence of vagal neuropathy (abnormalities in two or more tests) and for the total group of alcoholic patients there were significant increases in the heart rate responses to standing, Valsalva's manoeuvre and atropine. Thus there is evidence of recovery of vagal function with prolonged abstinence and improved nutrition at least in some alcoholic patients. This possibility of reversibility of autonomic neuropathy is in line with pathological evidence. Histological examination of myelinated fibres of the sympathetic chains of alcoholic patients showed short internodes which were consistent with either Wallerian or "dying back" degeneration followed by partial regeneration.¹¹

Nutritional factors rather than a direct toxic effect

Table Results of tests of vagal function in alcoholic and control patients on initial (1) and follow-up (2) investigation. Alcoholic patients were initially studied 1–6 weeks after withdrawal, and then again after 4–27 months of continued abstinence

Patient	Age (yr)	Time between studies (months)	Resting HR		Valsalva ratio		30:15 ratio		HR deep Δ breathing		HR Δ atropine		
			1	2	1	2	1	2	1	2	1	2	
Alcoholics													
1	60	20	† 75	68	† 1.36	1.38	† 0.97	1.11	14	10	*	29	32
2	57	11	72	67	1.31	1.86	1.08	1.12	34	35		21	30
3	54	24	80	65	1.47	2.14	1.06	1.13	16	15		30	26
4	54	4	65	61	1.36	1.44	1.06	1.15	8	14		28	33
5	57	6	76	68	1.43	1.77	1.05	1.13	26	23		28	32
6	51	13	63	57	1.77	1.60	1.03	1.19	11	15		28	26
7	62	27	72	56	1.15	1.14	0.90	1.00	4	9		20	31
8	58	6	108	57	1.04	1.33	1.00	1.09	3	18		8	31
9	48	16	79	75	1.20	1.43	1.00	1.00	6	5		15	17
10	47	5	100	70	1.08	1.33	1.00	1.18	12	21		15	23
11	59	4	83	85	1.20	1.55	1.03	1.24	15	17		15	14
Mean	55	11	79	64	1.31	1.54	1.02	1.12	16	17		22	27
±SD	±5	±9	±14	±6	±0.21	±0.29	±0.05	±0.29	±9	±9		±7	±6
Controls													
12	50	30	70	78	1.85	1.77	1.17	1.21	18	18		—	—
13	48	10	57	56	1.67	1.44	1.18	1.15	26	17		—	—
14	45	9	52	64	1.62	2.11	1.25	1.29	21	26		—	—
15	47	9	42	54	1.57	2.00	1.17	1.37	25	29		—	—
16	47	26	74	60	2.10	1.67	1.14	1.09	23	22		—	—
17	46	29	78	77	1.62	1.57	1.20	1.13	38	40		—	—
18	38	28	69	63	1.55	1.89	1.25	1.22	18	20		—	—
Mean	46	20	63	65	1.71	1.78	1.19	1.21	24	25		—	—
±SD	±4	±10	±13	±9	±0.20	±0.24	±0.04	±0.10	±7	±8		—	—

*significant difference between first and second studies $p < 0.02$

†significant difference between first and second studies $p < 0.01$

of alcohol are generally regarded as most important in the pathogenesis of alcoholic peripheral neuropathy.^{12,13} The patients in this study were under institutional care for several weeks after withdrawal and received vitamin and mineral supplements under supervision. Recovery of vagal function has previously been described in new diabetic patients, initially depressed heart rate responses to deep breathing becoming normal within a few weeks of the start of insulin therapy.^{14,15} Also, reversibility of abnormalities of autonomic innervation of the pupil has been described in diabetics with improved metabolic control.¹⁶ A good diet is likely to protect against the development of vagal neuropathy in alcoholics, as has previously been described for peripheral neuropathy.¹¹

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