Letter to the Editor

Hyperbaric oxygen therapy in multiple sclerosis

The treatment of multiple sclerosis (MS) with hyperbaric oxygen (HO) has been suggested by experimental studies (1). Results and methodological questions concerning HO therapy have been recently reviewed by Mertin and McDonald (2). The actual effect of HO on demyelinating process is unknown and several hypotheses have been formulated: a possible role in the immune system (3), in vascular changes (2), in nervous conduction (4). The first controlled study by Fischer (5) reported transient improvement after HO in chronic progressive MS but more recent papers do not agree with these results: no improvement on disease progression but a slight, statistically insignificant improvement of visual evoked response was found by Neiman (6), while Barnes (7) failed to observe benefits after HO in a large series of MS patients. In order to assess the effect of HO on MS course and to elucidate the possible mechanism on the immune system, we studied 13 clinically definite MS patients: mean age 35.4 years, mean duration of disease 9.5 years. No patient had had a relapse for at least 6 months before the trial, none had received immunosuppressants or corticosteroids for at least 6 months. The mean disease progression index (disability/disease duration) was 0.41. The treatment regimen consisted of 15 exposures (once daily for 5 days per week), followed by 5 monthly exposures (on 5 consecutive days) for 5 months. Oxygen was given at 2 ATA pressure for 60 min.

Evoked potentials, blood lymphocyte H/S ratio and Kurtzke's evaluation scale were assessed before the treatment, after one month, and after 6 months, according to the protocol described elsewhere (8).

Clinical and laboratory results are summarized in the table.

Three of 13 cases worsened after the short-term treatment, the others remained unchanged. After 6 months, 2 cases ameliorated, another 2 worsened, the remaining were unchanged. Three patients refused further HO treatment, so that we decided to conclude this pilot study at this point.

Our clinical results after 6 months of HO treatment do not seem to confirm the beneficial effect described by Fischer (5). Cerebral evoked responses and H/S ratios did not change in a significant way after the treatment, nor did their changes correlate with clinical evolution. This suggests that HO has no effect on nervous conduction and on immune regulation. EPs have been claimed to be an objective method to quantify neurological improvement after HO (4). Our results do not demonstrate such an improvement, in agreement with more recent and conclusive papers (6, 7), and indicate that HO is ineffective in the clinical form of MS we considered, and with the schedule we adopted.

Table 1

	Normal values	Pre-treatment values	Post-treatment values
VEP P_2			
Latency (msec)	99.6 ± 4.5	128.5 ± 27.7	128.3 ± 27.2
BAEP (msec)			
I-III interpeak latency	2.14 ± 0.15	2.14 ± 0.19	2.29 ± 0.39*
I-V interpeak latency	3.94 ± 0.17	4.33 ± 0.32	4.30 ± 0.37**
SEP (msec)			
N9-N13 interpeak latency	3.7 ± 0.5	5.11 ± 1.41	4.68 ± 1.08*
N13-N19 interpeak latency	5.6 ± 0.6	10.19 ± 6.35	8.65 ± 4.38*
H/S Ratio	2.7 ± 1.0	3.5 ± 2.1	3.2 ± 1.3

Results of hyperbaric oxygen in our MS series. VEP = visual evoked potential, BAEP = brainstem auditory evoked potential, SEP = somatosensory evoked potential. H/S ratio = OKT4+/OKT8+ lymphocyte ratio.

(*) Number of patients whose responses were undetectable. Post-treatment mean values did not differ significantly from pre-treatment ones (paired Student's test).

References

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