Trigeminal somatosensory evoked potentials in trichloroethylene-exposed workers

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ABSTRACT

Objectives: To assess the value of trigeminal somatosensory evoked potentials (TSEP) in evaluating toxicity of trichloroethylene to the trigeminal nerve.

Methods: Trichloroethylene (TCE), an excellent fat solvent largely used in industry, has a neurotoxic effect particularly on the trigeminal nerve. In March 2002, several TCE-exposed workers presented to Habib Bourguiba University Hospital, Sfax, Tunisia, for burned or numbness of the face. Twenty-three workers manipulating TCE underwent clinical, biological and toxicological assessment. Neurophysiologic study consisted of the measurement of the latencies and the amplitudes of TSEP in 23 exposed workers and 23 controls.

Results: Abnormal TSEP were observed in 6 workers with clinical evidence of trigeminal involvement and in 9 asymptomatic workers. We observed a significant positive correlation between duration of exposure and the N2 latency (r=0.5, $p<0.01$) and P2 latency (r=0.6, $p<0.02$). The sensitivity of TSEP was 65% and the specificity was 100%.

Conclusion: Trigeminal somatosensory evoked potentials are a useful tool in the diagnosis and management of chronic neurological complications of TCE intoxication. Since alterations of TSEP precede clinical symptoms, trigeminal nerve involvement can be demonstrated at the infra-clinical stage.

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Trichloroethylene (TCE) is a halogenated hydrocarbon widely used in industry as a degreasing solvent since the first world war. Trichloroethylene is a neurotoxic and can produce selective trigeminal nerve analgesia; hence, it was used as therapy of trigeminal neuralgia (tic douloureux).[1,2] Many cases of cranial neuropathies from industrial exposure to TCE (metal degreasing, dry cleaning and so forth) or intentional inhalation of TCE were reported.[3-8] However, only few electrophysiological reports studied TCE-induced trigeminal neuropathies and no clear conclusions were available.[9-13] The aims of this study were to report our experience with TCE and to evaluate the usefulness of the trigeminal somatosensory evoked potentials (TSEP) in this intoxication. We investigate the effect of TCE on amplitude and latencies of TSEP. We establish the normal control values for our own laboratory. Then, we measured TSEP in persons with occupational TCE exposure.

Methods. In March 2002, several phosphate industry workers presented to our out patient hospital for burned or numbness of the face. These workers were exposed to TCE, they spent up to 6 hours daily within tanks, they have to clean the inside walls of these tanks to be painted. For this task, they used TCE in such confined atmosphere without any ventilation. Protective masks and

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safety advice are provided but not adopted by the workers due to heat and transpiration. All workers of this phosphate industry factory who have an occupational exposure to TCE for at least 2 years were included in the study. Using this unique criterion, we collected in the Department of Neurology a group of 23 males with a mean age of 36.7 ± 9 years (range 20-56 years). They usually manipulate TCE, and the mean duration of exposure to this organic solvent was 12.4 ± 8.3 years (range: 2-27 years). The non-exposed group consisted of 23 healthy males working in the same factory but not exposed to TCE. Their mean age was 36.6 ± 9.8 years (range 24-54 years). None of the control group had a history of solvent exposure. These latter subjects were comparable to TCE exposed group in term of age and gender and were free from potential environmental, morbid or iatrogenic neuropathy. Exclusion criteria for both groups were diabetes mellitus, trigeminal neuralgia, alcohol abuse, multiple sclerosis or significant drug or medication histories.

Baseline clinical evaluation. All subjects were asked for burned or numbness of the face, visual disturbances, restlessness, concentration difficulty and fatigue. We also looked for mood changes such as euphoria and irritability; they are considered if they were concomitant to intensive exposure and were remarkable by patient's family. Neurological examination paid particular attention to the assessment of the cranial nerves. Quality of life and degree of disability was evaluated by the Karnovsky et al[14] performance score.

Laboratory screening. Standard biological investigations included blood cell count, erythrocyte sedimentation rate, fasting blood glucose, blood urea nitrogen, plasma creatinine, plasma cholesterol, plasma triglycerides, plasma bilirubin and hepatic enzymes (transaminase and γ-glutamyl transferase).

Toxicologic investigations: Measurement of urinary metabolites of TCE (trichloroethanol, trichloroacetic acid) was performed 3 times for each worker and usually on Friday, which is the last day of the week. Only the highest value is considered. Repeated careful measurements of TCE air contents were made.[15,16] In air atmosphere, the maximum tolerated concentration of trichloroethylene was 50 ppm and in urine, the maximum tolerated concentration trichloroacetic acid 100 mg/g of creatinine.[17,18]

Neurophysiological screening. Trigeminal somatosensory evoked potentials were recorded using Nihon-Kohden EMG-evoked potential system (Tokyo, Japan) as described by Stöhr and Petrüch.[19] Every subject underwent consecutive bilateral recording of somatosensory evoked potentials following trigeminal stimulation. A surface electrode, applied 1 cm under the corner of the mouth, delivered square wave pulses of 0.1 ms duration at the rate of 3/seconds. Stimulus intensity was adjusted to produce moderate twitching of the inferior lip. The recording electrode is placed over the contralateral parietal area using a non-cephalic reference. Evoked potentials were amplified and filtered between 20-2000 Hz. Averaging was performed on at least 500 responses over an analysis time of 100 ms. Two averages were superimposed to confirm reproductivity. Latency and amplitude were determined for each wave: first negative potential (N1), first positive potential (P1), second negative potential (N2), second positive potential (P2) and third negative potential (N3). Trigeminal somatosensory evoked potentials latencies higher than our control mean plus 2.5 SD are considered abnormal. Therefore, subjects with asymmetrical amplitudes of TSEP were considered as pathologic (difference between the 2 sides was >50%). Motor nerve conduction velocities were measured for median, ulnar, medial and lateral popliteal nerves. For sensory nerve conduction velocities, lateral sural cutaneous nerve and median nerve were assessed.

Statistical analyses. Statistical methods included paired or unpaired Student's t-test as appropriate. Results are expressed as mean ± 1 standard deviation; p values <0.05 were considered significant. Spearman rank-correlation procedure was used for correlation analysis.

Results.

Clinical results (Table 1). Among the 23 workers, only 6 presented sensory disturbance of the face. It consisted of numbness, burned, or both, of the cheeks and hypoesthesia in the territory of the first and second branch of the trigeminal nerve including corneal hypesthesia. Corneal and blink reflexes were absent in 2 (case 11-12) and weak in one (case 7). Moreover, general symptoms were observed usually during episodes of intensive exposure including fatigue (19 cases), consciousness disturbance (18 cases), stance and gait instability (15 cases) and disturbed behavior such as restlessness, irritability, concentration difficulty with euphoria (12 cases). These disturbances were transient, workers had a quiet normal activity and the Karnovsky performance score was high (80-100, mean of 86 ± 8).

Results of laboratory screening. Blood cell count was normal. Fasting blood glucose was at 4.8 ± 0.2 mmol/l (range 4-6), blood urea nitrogen at 5 ± 0.3 mmol/l (range 4-7.2), plasma creatinine level 123.8 ± 12.7 µmol/l (range 70-251), plasma cholesterol 5 ± 0.3 mmol/l (range 3.9-6.9), plasma triglyceride 1.7 ± 0.1 mmol/l (range 1.1-2.7), plasma bilirubin 10.2 ± 1.1 µmol/l (range 6-17), alanine amino-transferase at 24.1 ± 2.8 UI/l (range 13-45), asparagine amino-transferase at 26.9 ± 1.9 UI/l (range 16-42) and γ-glutamyl transferase 19.8 ± 4.8 UI/l (range 11-75). Mean urinary

trichloroethanol expressed as mg/g of urinary creatinine was 79.3 ± 8.8, trichloroacetic acid was 32.6 ± 4.6 and total trichlorides was 111.9 ± 11.5 (**Table 2**). Only one subject had urinary TCE metabolites levels over tolerated limits (case 2). Trichloroethylene air contents were over tolerated limits of 50 ppm and ranged from 50-150 ppm.

Neurophysiological findings. In the non-exposed group, N1 and P1 potentials were inconstant with low amplitude and frequently masqued by the stimulation artifact. Whereas N2 potential was constant ample and was considered as the best component for analysis. On the other hand, P2 and N3 potentials were relatively constant but their latencies showed a large variability.

The latencies of N1 were 12.7 ± 1.1 ms, P1 were 18.2 ± 1 ms, N2 were 27 ± 1.1 ms, P2 were 35 ± 2.7 ms and N3 were 45.9 ± 3.8 ms (**Figure 1a**). The amplitudes of the different components showed large variations in this group, indeed mean values of N1 were 0.9 ± 0.3 µv, P1 were 1.1 ± 0.4 µv, N2 were 2.2 ± 1.1 µv, P2 were 1.1 ± 0.4 µv and N3 were 1.4 ± 0.7 µv. No difference was observed between the 2 sides regarding latencies. Amplitude may vary from one side to other, but the difference did not exceed 50%. In the exposed group, mean latencies of the different potentials were significantly delayed. However, a significant decrease of the amplitude was noted only for P1 and N2 potentials (**Table 3**). Delayed latencies were observed in 12 cases, they were unilateral in 9 cases and bilateral in 3 cases (**Figure 1b**). Asymmetrical amplitude was the only abnormality in 4 patients. The limb motor and sensory nerve conduction velocities were normal in all exposed workers. A significant positive correlation was found between duration of exposure and both N2 latency (r=0.5, $p<0.01$) and P2 latency (r=0.6, $p<0.02$) (**Figure 2**). No correlation was noted between the TSEP and urinary TCE metabolites.

Discussion. Industrial surveys have revealed a number of examples of isolated and otherwise, unexplained facial numbness in workers exposed to TCE. Selective damage of the fifth cranial nerve has been documented by autopsy.[3] The most striking changes were seen in the brainstem particularly the fifth cranial nerve nuclei, spinal tract and nerve roots. Paradoxically, the structures involved in TCE intoxication have no feature in common such as axon length and size, energy consumption rates,

Table 1 - Clinical data of trichloroethylene-exposed workers.

Case	Age*	Duration of exposure (years)†	General symptoms				Karnovsky score‡	Trigeminal nerve involvement symptoms				
			Fatigue	Disturbed behavior	Consciousness disturbance	Gait instability		Numbness of the face	Hypesthesia of the face	Corneal hypesthesia	Corneal reflex	Blink reflex
1	30	9	+	+	+	+	80	-	-	-	+	+
2	20	3	-	-	+	+	90	-	-	-	+	+
3	27	5	+	+	+	+	80	-	-	-	+	+
4	47	30	+	+	+	-	80	+	-	-	+	+
5	34	5	+	-	+	-	90	-	-	-	+	+
6	34	15	+	+	+	+	80	+	+	+	+	+
7	42	17	+	+	+	-	80	+	+	+	+/-	+/-
8	51	25	+	-	+	+	90	-	+	+	+	+
9	48	16	+	+	+	+	80	+	+	+	+	+
10	35	12	+	+	+	+	80	-	-	-	+	+
11	46	27	+	+	+	+	80	+	+	+	-	-
12	41	21	+	+	+	+	80	+	+	+	-	-
13	31	13	-	-	-	-	100	-	-	-	+	+
14	56	22	+	-	+	-	90	-	-	-	+	+
15	29	4	+	+	+	+	80	-	-	-	+	+
16	37	6	+	-	+	+	80	-	-	-	+	+
17	28	6	+	-	+	+	80	-	-	-	+	+
18	33	13	+	+	+	+	80	-	-	-	+	+
19	40	5	+	+	+	+	80	-	-	-	+	+
20	29	6	+	-	-	-	100	-	-	-	+	+
21	30	2	+	-	-	+	90	-	-	-	+	+
22	26	7	-	-	-	-	100	-	-	-	+	+
23	39	17	-	-	-	-	100	-	-	-	+	+

*mean ± SD 36.7 ± 9, range 20 - 56 years, †mean ± SD 12.4 ± 8 , range 2 - 27 years, ‡mean ± SD 86 ± 8, range 80 - 100 years,
+ = presence, - = absence, +/- = present but weak

Table 2 - Trichloroethylene urinary metabolites expressed as mg per gram of excreted creatinine.

Case	Trichloro-ethanol (mg/g of UC)	Trichloro-acetic acid (mg/g of UC)	Total trichlorides (mg/g of UC)	UC (g/l)
1	145	46	191	1.47
2	211	58	269	0.93
3	61	97	158	0.95
4	49	12	61	1.47
5	135	65	200	0.89
6	75	45	120	1.32
7	9.5	3	12.5	2.42
8	100	20	120	1.56
9	93	45	138	1.30
10	50	55	105	1.72
11	63	20	83	1.83
12	75	30	105	1.34
13	86	22	108	0.86
14	66	3	69	1.02
15	90	32	122	1.71
16	72	38	110	0.93
17	58	18	76	1.10
18	75	27	102	2.26
19	59	12	71	1.09
20	67	19	86	1.43
21	10	20	30	2.15
22	83	35	118	1.93
23	91	29	120	1.42
Mean ± SD	79.3 ± 42	32.6 ± 22	111.9 ± 55	1.4 ± 0.5
Range	9.5 - 211	3 - 97	12.5 - 269	0.9 - 2.4

UC - urinary creatinine

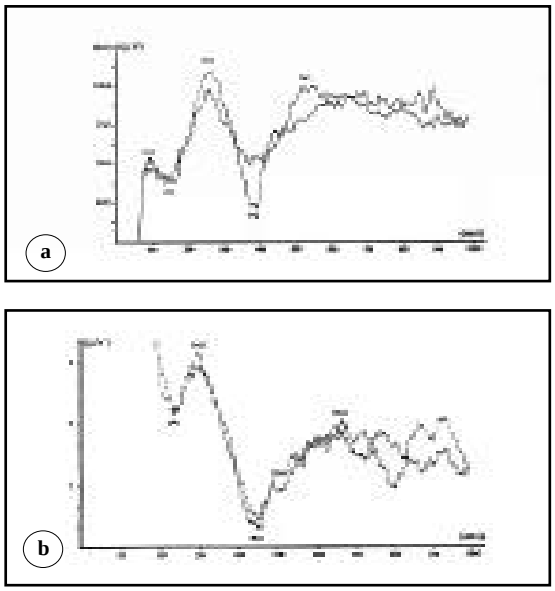

Figure 1 - Graphs of trigeminal somatosensory evoked potentials in **a)** controls and **b)** trichloroethylene-exposed workers.

specific transmitter, metabolism and so forth. However, vascular permeability of the region accounts for the vulnerability of the trigeminal nerve nuclei and may explain specific damage.[20] On the other hand, the occurrence of orofacial herpes simplex lesions associated with TCE-induced trigeminal neuropathy suggests that TCE may act as a reactivating factor of the neurotropic virus.[21] The recording of TSEP following trigeminal stimulation is rarely used in current practice. Trigeminal somatosensory evoked potentials were first reported in humans by Stöhr and Petruch[19] and followed by Bennett and Jannetta,[22] Findler and Feinsod,[23] and by Garrel et al.[24] Clinical application was emphasized in trigeminal neuralgia[25,26] and TCE intoxication.[9,10] Stimulation can be applied on the lips[10,19,22-24] or on the tongue.[26]

In our outpatient hospital, several workers presented with trigeminal sensory disturbances. Anamnesis confirmed chronic TCE exposure. A cross-sectional clinical, toxicological and neurophysiological investigation was performed. In the studied group, only 6 of 23 had clinical evidence of trigeminal involvement. However, neurophysiological study disclosed abnormal TSEP in 15 subjects (65%). The abnormalities observed were similar to those reported by Garrel and Barret[9] and Dogui et al,[10] and it consists of delayed latencies, asymmetrical amplitude, or both. Unlike previous reports, we found a high percentage of abnormal TSEP in our exposed group (65%),

Table 3 - Mean latencies and amplitudes of the trigeminal somatosensory evoked potentials in trichloroethylene (TCE) exposed workers and controls.

Potential	TCE-exposed	*p* value	Control
N1			
Latency (ms)	15.9 ± 1.7	<0.001	12.6 ± 0.9
Amplitude (µv)	1.0 ± 0.4	NS	1.0 ± 0.2
P1			
Latency (ms)	20.2 ± 2.5	<0.01	18.4 ± 1
Amplitude (µv)	0.8 ± 0.5	<0.02	1.1 ± 0.3
N2			
Latency (ms)	29.0 ± 3.5	<0.01	27.0 ± 0.9
Amplitude (µv)	1.6 ± 0.7	<0.05	2.3 ± 0.7
P2			
Latency (ms)	38.0 ± 4.6	0.01	35.0 ± 2.1
Amplitude (µv)	1.3 ± 0.6	NS	1.4 ± 1
N3			
Latency (ms)	52.4 ± 8.2	0.001	45.5 ± 3
Amplitude (µv)	1.7 ± 0.7	NS	1.4 ± 0.6

NS - not significant

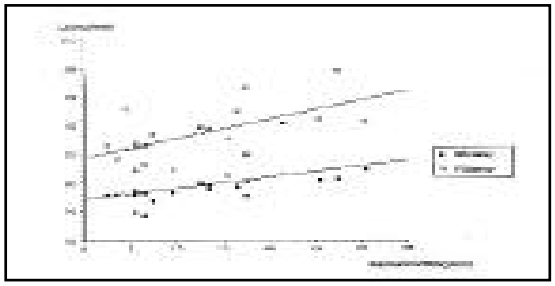

Figure 2 - Positive correlation between duration of exposure and the N2 and P2 latencies.

Table 4 - Matrice 2 by 2 of validity indicators regarding the trigeminal somatosensory evoked potentials (TSEP).

TSEP	Trichloroethylene exposed	Control
Normal TSEP	8	18
Pathologic TSEP	15	0

whereas the percentage of positivity found by Garrel and Barret[9] was 38% and by Dogui et al[10] 45%. This difference may be explained by the long exposure duration in our series (12.4 ± 8 years). The most striking finding was a positive correlation between TSEP latencies and the exposure duration (for N2 latency r=0.5 and $p<0.01$ and for P2 latency r=0.6 and $p<0.02$), which may suggest a cumulative dose-response relationship. No correlation was found between neurophysiological alteration and urinary TCE metabolite concentration and TCE air-content. To assess the general validity of the TSEP measurement as an indicator of exposure to TCE, a 2 x 2 table was constructed (**Table 4)**. The sensitivity was 65% and specificity was 100%.[27] The sensitivity of TSEP may be improved if the 3 branches of the trigeminal nerve are explored. Early evoked potentials can be recorded on the scalp after stimulation of the infra-orbital nerve. The waves have been named W_1 with mean latency of 0.98 ms, W_2 with mean latency of 1.92 ms and W_3 with mean latency of 2.57 ms.[28,29] These early components are probably generated by the proximal part of the maxillary nerve or from the gasserian ganglion (W_1) and from the trigeminal root fibres running through the brainstem before (W_2) and after the dichotomisation (W_3).[30] They are interesting to evaluate the trigeminal system in its peripheral parts, but it needs a very short pulse duration (0.05 ms) and alternating polarity essential for artifact suppression.[31] Middle latency components of TSEP (N1, P1, N2, P2 and N3) were considered to arise from suprathalamic generators. Since, the most neuropathological changes induced by TCE were seen in the brainstem especially the fifth nerve nuclei, it is preferable to study middle than early latency trigeminal evoked potentials. The blink reflex was reported as an alternative approach in the management of TCE exposed workers.[11] Although this technique seems to be easier than TSEP, it is less sensitive (50%) and specific (90%). The lack of sensitivity may be attributed to the fact that the test does not investigate trigeminal involvement specifically but facial and trigeminal nerve involvement.

In conclusion, our study demonstrates that TSEP, a safe and non invasive investigation, is a useful tool in diagnosis and the management of trigeminal nerve involvement secondary to chronic TCE intoxication. Trigeminal somatosensory evoked potential abnormalities precede clinical symptoms and are related to the exposure duration. They should be performed periodically in all patients exposed to this organic solvent to detect neurological effects early.

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