

# Brainstem auditory evoked potentials in traffic policemen occupationally exposed to vehicular emission

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## Abstract

**Background and Aim:** It is a well-known fact that traffic policemen in Delhi are exposed to immense vehicular pollution particularly at crowded intersections. These pollutants affect various systems of the body including nervous system in due course of time. Realizing these facts, we recorded absolute and inter peak latencies (IPLs) of brainstem auditory evoked potentials (BAEPs) in traffic policemen exposed to enormous vehicular emission and compared with the normal controls living in the residential areas in Delhi.

**Methods:** Totally, 60 subjects all males and nonsmokers between 30 and 50 years of age were studied, 30 belonged to exposed group comprised of traffic policemen and 30 nonexposed controls. Their BAEPs were recorded using MEB 5200 Neuropack II plus evoked potential recorder (Nihon Kohden, Japan) and evaluation of anthropometric and neuro-psychiatric parameters was done. To compare BAEP results, Student's *t*-test was used.

**Results:** Significantly prolonged absolute peak latencies I, II, III, IV, V and IPL I-III in exposed group were observed as compared to controls.

**Conclusion:** Findings suggest prolongation of peripheral and central auditory conduction time in exposed subjects. The prolonged latencies in exposed group may be attributed to occupational exposure to vehicular emission.

**Key words:** Brainstem auditory evoked potentials, polycyclic aromatic hydrocarbon, particulate matter, traffic policemen, vehicular emission

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## INTRODUCTION

Delhi is one of the most polluted cities, and large number of vehicles producing enormous pollutants can have detrimental effect on developing central nervous system (CNS).<sup>[1-3]</sup> Air quality is deteriorated by release of poisonous gases (NO<sub>2</sub>, SO<sub>2</sub>) suspended particulate matter (SPM), respirable SPM (RSPM), lead, CO and volatile organic compounds (VOC) that is, benzene, polycyclic aromatic hydrocarbons (PAH), toluene etc., and these compounds are present in excess of the recommended levels.<sup>[4]</sup> These have shown an increasing trend during 2000–2010 in which two wheelers are

contributing maximum emissions followed by passenger cars.<sup>[5]</sup> Various pollutants, e.g. PAH compounds and lead (Pb) can cross blood brain barrier and are neurotoxic because of their lipid solubility.<sup>[6,7]</sup> Prolonged exposure to these can lead to failure of memory, difficulty in concentration, learning difficulty and organic brain damage in some persons.<sup>[8,9]</sup> Children exposed to vehicular emission exhibited abnormalities of brainstem auditory evoked potential (BAEP) waves and latencies.<sup>[10]</sup> Current and chronic lead exposure have shown altered conduction in the auditory pathway.<sup>[11]</sup> Association of long-term air pollution with neuroinflammation and disruption of blood brain barrier is a known fact in adults and children.<sup>[12]</sup> Exposure to SPM and PAH compounds in rubber factory workers have shown abnormality in auditory and visual conduction pathways.<sup>[13,14]</sup> Lead and diesel exhaust exposure can produce neurobehavioral changes, visual and CNS impairments.<sup>[15,16]</sup>

We hypothesized that exposure to immense vehicular pollution involving the CNS can be detected at an earlier

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subclinical stage by recording BAEPs and tested the hypothesis that traffic policemen who are exposed such pollution would have an altered BAEPs as compared to normal controls.

Primary objective of our study was to measure absolute and inter-peak latencies (IPLs) of BAEPs in traffic policemen exposed to vehicular emission at traffic intersections and normal controls living in residential areas in Delhi.

## MATERIALS AND METHODS

### Material

The study was carried out in 60 subjects in the electrophysiology laboratory, department of Physiology. These subjects were divided into two groups. Group I included 30 clinically healthy traffic policemen (exposed group), and Group II included 30 healthy controls (nonexposed group). All the subjects in our study were males, nonsmokers and age-matched. They were selected after meeting exclusion criteria's. Subjects with any history of smoking, alcoholism, drug addiction, neurological illness, psychiatric illness, ear pathology or any other medical illness affecting CNS were excluded from the study. Traffic policemen posted at various crowded traffic intersections (e.g. ITO, ISBT etc.) in Delhi with a minimum exposure of 5 years were included in the study and healthy controls were taken from medical college and hospital (staff and security persons) who reside in the residential campus which is surrounded by residential colonies from all sides. All the subjects were instructed not to apply hair oil on the day of recording and recording was done in a sound proof room.

This research proposal was approved by institutional research protocol and ethics committee, and written consent was obtained from all the subjects involved in the study.

### Methods

Anthropometric parameters were measured, and a standard questionnaire was worked out in each subject particularly related to neurological and psychiatric illnesses.

### Brainstem auditory evoked potentials

Brainstem auditory evoked potentials were recorded on MEB 5200 Neuropack II plus evoked potential recorder (Nihon Kohden, Japan). The active electrode was placed over the vertex area and reference electrode at the left and right ear lobule (A1, A2) after cleaning the part thoroughly. A ground electrode was placed on the forehead. The waves of BAEPs were recorded using a standardized technique.<sup>[17,18]</sup> Click stimuli having intensity 70 dB above normal hearing

threshold were presented independently to each ear at a rate of 10/s and 0.1 ms duration. During testing of one ear, another ear was masked by a white noise – 40 dB Hz. These clicks were generated by passing 0.1 ms square pulses through shielded headphones with alternating polarity. The signals picked up by these electrodes were displayed on the screen in the form of waves after filtration, amplification and averaging. Waves in first 10 ms of latency were recorded. Absolute peak latencies (APL) of all the waves, IPLs I-III, III-V, I-V and amplitude of I and V were determined with each ear separately.

The data obtained from 60 subjects were statistically analyzed. Tabulation and computation of various statistical measures such as mean standard deviation and analysis of variance were done. Student's *t*-test was done to compare BAEPs among traffic policemen (Group I) and controls (Group II). *P* < 0.05 was considered as statistically significant.

## RESULTS

Mean age (years), height (cm), weight (kg) and body surface area (m<sup>2</sup>) in traffic policemen (Group I) and control (Group II) are shown in Table 1. Anthropometric parameters in both the groups studied by us indicate the homogeneity with slightly higher values in traffic policemen (Group I). All the subjects in both the groups were nonsmokers.

The APLs, IPL and amplitude of different wave of BAEP are shown in Tables 2-4 respectively. The BAEPs values obtained from normal controls (Group II) are in close agreement with data from earlier studies,<sup>[18]</sup> using similar stimulus and recording parameters. In traffic policemen (Group I), a significant prolongation of all the waves of BAEP that is, wave I, II, III, IV and V was observed. All the IPLs that is, I-III, III-V and I-V were prolonged in traffic policemen, but significant prolongation was seen for IPL I-III only. Figures 1 and 2 depicts comparative pattern of values of APL and IPL respectively in the form of bar diagrams amongst traffic policemen Group I) and normal controls (Group II). In our study, no morphological or amplitude anomalies were found. Our analysis mainly focused on commonly reproducible

**Table 1:** Result of anthropometric variables in traffic policemen (Group I) and normal control (Group II)

Variable	Mean±SD		P
	Group I (n=30) (traffic policemen)	Group II (n=30) (normal controls)	
Age (year)	36.09±5.09	36.07±4.49	0.982
Height (cm)	178.56±3.26	174.73±4.49	0.000*
Weight (kg)	70.78±8.20	69.63±8.85	0.598
BSA (m <sup>2</sup> )	1.88±0.10	1.83±0.13	0.174

\**P* < 0.05. SD: Standard deviation, BSA: Body surface area

BAEP components in our subjects. Figure 3 depicts BAEP tracings in traffic policemen and normal control for better understanding of its different waves.

In Delhi, mean annual average levels of various pollutants in the year 2003 (when this study was carried out) is given in Table 5. These values of various pollutants are based on the data published by Central Pollution Control Board (CPCB).<sup>[4]</sup> These data compare the levels of different

pollutants in residential areas and traffic intersection (ITO crossing) and shows that concentrations of SPM, RSPM, SO<sub>2</sub>, benzo (a) pyrene, PAH, benzene and toluene are much higher at traffic intersection than the residential areas. We could not get the data on levels of PAH and benzo (a) pyrene in the year 2003 and included their available levels in December 2004 and January 2005. We could not find data on the levels of various pollutants at other traffic intersections.

**Table 2:** Observed results of APL of BAEP waves in traffic policemen (Group I) and normal controls (Group II)

APL	Mean±SD (msec)		P
	Group I (n=30) (traffic policemen)	Group II (n=30) (normal controls)	
I	1.60±0.17	1.51±0.07	0.000*
II	2.67±0.14	2.60±0.10	0.037*
III	3.73±0.31	3.57±0.19	0.016*
IV	4.81±0.23	4.62±0.11	0.001*
V	5.69±0.21	5.51±0.08	0.000*

\*P<0.05. APL: Absolute peak latencies, SD: Standard deviation, BAEP: Brainstem auditory evoked potential

**Table 3:** Observed results of IPL of BAEP waves in traffic policemen (Group I) and controls (Group II)

IPL	Mean±SD (msec)		P
	Group I (n=30) (traffic policemen)	Group II (n=30) (normal controls)	
I-III	2.13±0.21	2.05±0.06	0.032*
III-V	1.96±0.25	1.89±0.13	0.200
I-V	4.08±0.27	3.93±0.33	0.051

\*P<0.05. IPL: Inter peak latencies, BAEP: Brainstem auditory evoked potential, SD: Standard deviation

**Table 4:** Observed results of amplitude of BAEP waves in traffic policemen (Group I) and normal controls (Group II)

Amplitude	Mean±SD (µv)		P
	Group I (n=30) (traffic policemen)	Group II (n=30) (normal controls)	
I	0.33±0.12	0.30±0.09	0.420
V	0.37±0.11	0.30±0.14	0.097

BAEP: Brainstem auditory evoked potential, SD: Standard deviation

**Table 5:** Mean annual average levels of various pollutants in residential areas and traffic intersection in Delhi\*

Pollutant	Year	Mean annual average concentration in residential area	Mean annual average concentration at traffic intersection (ITO)
SPM (µg/m <sup>3</sup> )	2003	346	509
RSPM (µg/m <sup>3</sup> )	2003	144	244
NO <sub>2</sub> (µg/m <sup>3</sup> )	2003	43	94
SO <sub>2</sub> (µg/m <sup>3</sup> )	2003	10	09
PAH (ng/m <sup>3</sup> )	December 2004 and January 2005	23.8	54.4
SPM laden-Benzo (a) pyrene (ng/m <sup>3</sup> )	December 2004 and January 2005	2.77	7.31
Benzene (VOC) (µg/m <sup>3</sup> )	2003	11.2	14
Toluene (VOC) (µg/m <sup>3</sup> )	2003	09	24

\*Source for levels of pollutants is CPCB [4]. SPM: Suspended particulate matter, VOC: Volatile organic compounds, RSPM: Respirable suspended particulate matter, PAH: Polycyclic aromatic hydrocarbons, CPCB: Central Pollution Control Board, ITO: Income tax office

## DISCUSSION

Auditory evoked responses serve as a noninvasive clinical tool in characterizing the electrophysiological phenomenon of neural excitation, conduction and transmission across auditory pathways from cochlea to cortex.<sup>[18]</sup> In the present study, auditory evoked potentials in normal controls (Group II) and traffic policemen (Group I) were recorded and compared. The morphology of BAEPs was normal in both the groups with clear demarcation of all the waves in all the subjects. Our study on clinically healthy traffic policemen posted at various traffic intersection in Delhi with long exposure to severe vehicular emission, exhibited significant prolongation of APLs of all components of BAEPs and IPL I-III along with nonsignificant prolongation of IPL III-V and I-V as compared to normal healthy controls residing in the residential areas with low pollution levels.

Absolute peak latencies of BAEP reflect the neural conduction velocity in the corresponding segment of auditory pathways. A delay in time of APL of wave I to wave V in traffic policemen suggest decreased conduction velocity both at the level of the auditory nerve and central auditory pathways in the brainstem. This is further evidenced by significant prolongation of IPL I-III and longer IPL III-V and I-V in traffic policemen. IPL I-III is a measure of conduction in the most caudal segment of brainstem auditory pathway – acoustic nerve and pontomedullary portion. Prolongation of peripheral and central conduction time could be related to pathological changes in neural

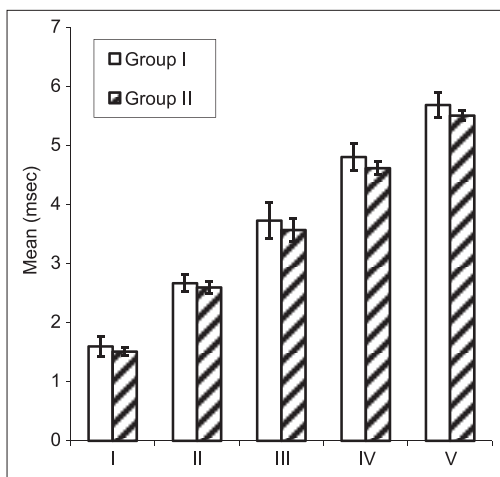
pathways that may be induced by RSPM and lipid soluble pollutants (e.g. Benzo[a] pyrene and PAH) present in vehicular emission, as concentration of these pollutants at crowded intersections exceeds to much higher levels than their levels in residential areas in Delhi.

Study on clinically healthy children with lifelong exposure to severe urban air pollution exhibited significant delays in wave III and V of BAEP when compared to controls residing in low pollution area. Significantly longer IPLs I-III, III-V and I-V among exposed children are consistent with delayed central conduction time of neural transmission through the brainstem which indicate transmission through cochlear nuclei (wave III), superior olivary complex (wave IV), and lateral lemniscus (wave V) pathways are abnormal in exposed children<sup>[10]</sup> and these changes are consistent with the changes seen in our study as far as involvement of central conduction pathway is concerned.

Workplace environment in a rubber factory contains various lipid soluble compounds (e.g. PAH and benzo[a] pyrene) which are also present in vehicular emission. A study on these workers has shown prolonged APLs of wave I, II, IV, V and prolonged IPL I-III,<sup>[13]</sup> which are similar to the results as shown in our study. Pattern reversal visual evoked potentials studied in rubber factory workers revealed that rubber factory workers had abnormal latencies of P1 (dominant component of PEVP) suggesting rubber factory environment affect conduction processes in optical pathways from the origin in retina to striate cortex.<sup>[14]</sup>

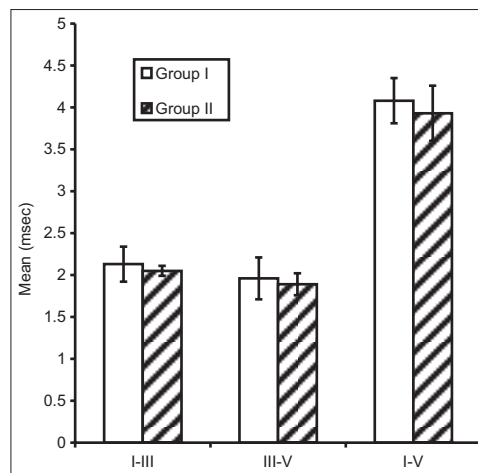
Traffic policemen posted at crowded intersection are exposed to severe vehicular emission containing particulate matter (PM), RSPM, PAH, VOC that is, benzo (a) pyrene and toluene in concentration much higher than their concentration in residential area in Delhi.<sup>[4]</sup> PM could reach brainstem by uptake through olfactory neuron (nasal cavity) and cranial nerves that is, trigeminal nerve (oral cavity), vagus nerve (respiratory and gastrointestinal tracts) and by trafficking of macrophage-like cells loaded with PM from lung capillary bed to systemic circulation<sup>[12,19-21]</sup> and may causes inflammatory changes in the brainstem.<sup>[10,12]</sup> Further PAH, VOC and lead present in vehicular emission, being lipid soluble these may cross blood brain barrier and can affect neural tissues as reported in rubber factory workers are probably also responsible for conduction processes abnormalities recorded in our study. Respirable PM along with lipid soluble pollutants may induce brainstem inflammatory changes. Peripheral and central conduction delay in auditory pathways in traffic policemen may perhaps be attributed to immense vehicular emission to which they are exposed particularly at busy traffic intersections in Delhi.

Our study demonstrating changes in BAEPs suggest that vehicular emission to which these traffic policemen are exposed causes delayed conduction in auditory neural tissues. Such exposure for long duration if continued may affect other brain tissues as well. So comprehensive steps should be taken to improve air quality using advanced techniques of monitoring air quality, reducing numbers of vehicles promoting use of public transport and better traffic management system. Good quality air filter mask should be provided to these policemen. Steps should



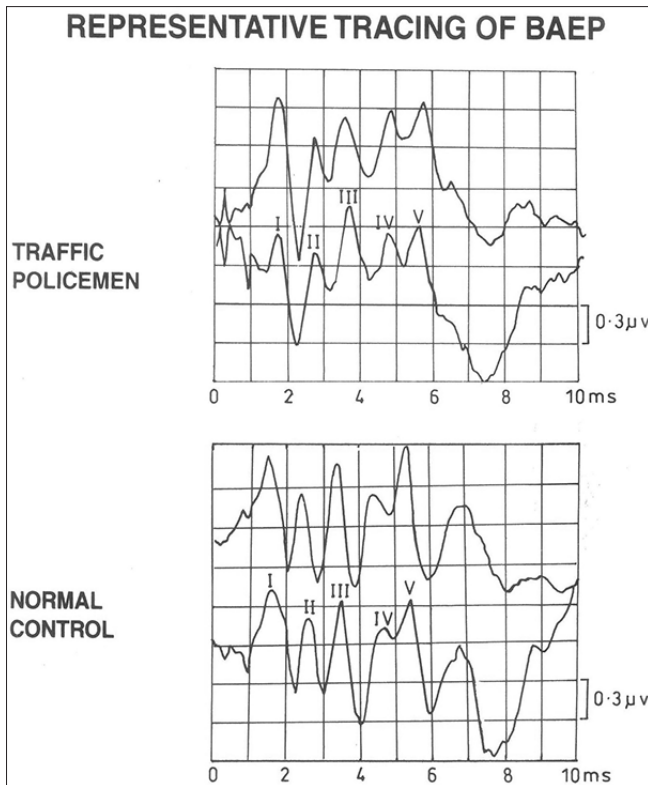
Variable	Group I	Group II
I	1.6	1.51
II	2.67	2.6
III	3.73	3.57
IV	4.81	4.62
V	5.69	5.51

**Figure 1:** Absolute peak latencies in traffic policemen (Group I) and controls (Group II). Absolute peak latencies



Variable	Group I	Group II
I-III	2.13	2.05
III-V	1.96	1.89
I-V	4.08	3.93

**Figure 2:** Inter peak latencies in traffic policemen (Group I) and controls (Group II). Inter peak latencies



**Figure 3:** Brainstem auditory evoked potential tracing in traffic policemen and control

also be taken to make technologically advanced traffic signals so as to minimize the number of traffic policemen at traffic signals.

### Limitations of the study

In our study, we could not measure the level of pollutants at traffic intersections and residential areas because of resource crunch and were mainly dependent on the data that we got from CPCB sources. Inflammatory markers (interleukin-6, tumor necrosis factor alpha etc.,) measurement could not be estimated due to limited availability of resources. Levels of these pro-inflammatory markers do increase under the influence of various pollutants and are responsible for inflammatory reactions of brainstem structures and other neural structure.

### CONCLUSION

In our study significantly prolonged APLs, IPL I-III and nonsignificant prolongation of IPL (III-V, I-V) are seen in traffic policemen posted at crowded traffic intersections as compared to the normal controls in residential areas in Delhi. Since exposure to pollution caused due to vehicular emission is the only variable in the two groups, in conclusion we may state that data on BAEPs reported by us show an association between exposure to immense vehicular emission at traffic intersections

and neural dysfunction of auditory pathway in exposed group.

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### REFERENCES

1. CPCB (Central Pollution Control Board). Status of the Vehicular Pollution Control Programme in India; 2010. Available from: [http://www.cpcb.nic.in/upload/NewItems/NewItem\\_156\\_VPC\\_REPORT.pdf](http://www.cpcb.nic.in/upload/NewItems/NewItem_156_VPC_REPORT.pdf). [Last accessed on 2015 Jan 27].
2. Danzer SC. Postnatal and adult neurogenesis in the development of human disease. *Neuroscientist* 2008;14:446-58.
3. Goyal SK, Ghatge SV, Nema P, M Tamhane S. Understanding urban vehicular pollution problem vis-a-vis ambient air quality – Case study of a megacity (Delhi, India). *Environ Monit Assess* 2006;119:557-69.
4. CPCB (Central Pollution Control Board). Epidemiological study on effect of air pollution on human health (Adults) in Delhi. New Delhi: Environmental Health Series; 2008. p. 317.
5. Sindhvani R, Goyal P. Assessment of traffic generated gases and particulate matter emission and trends over Delhi (2000-2010). *Atmos Pollut Res* 2014;5:438-46.
6. Mutti A, Cavatorta A, Lommi G, Lotta S, Franchini I. Neurophysiological effects of long-term exposure to hydrocarbon mixtures. *Arch Toxicol Suppl* 1982;5:120-4.
7. Grasso P, Sharratt M, Davies DM, Irvine D. Neurophysiological and psychological disorders and occupational exposure to organic solvents. *Food Chem Toxicol* 1984;22:819-52.
8. Cone JE, Bowler R, So Y. Medical surveillance for neurologic endpoints. *Occup Med* 1990;5:547-62.
9. Jensen LK, Klausen H, Elsnab C. Organic brain damage in garage workers after long-term exposure to diesel exhaust fumes. *Ugeskr Laeger* 1989;151:2255-8.
10. Calderón-Garcidueñas L, D'Angiulli A, Kulesza RJ, Torres-Jardón R, Osnaya N, Romero L, *et al.* Air pollution is associated with brainstem auditory nuclei pathology and delayed brainstem auditory evoked potentials. *Int J Dev Neurosci* 2011;29:365-75.
11. Bleeker ML, Ford DP, Lindgren KN, Scheetz K, Tiburzi MJ. Association of chronic and current measures of lead exposure with different components of brainstem auditory evoked potentials. *Neurotoxicology* 2003;24:625-31.
12. Calderon GL, Solt AC, Henriquez RC, Torres JR, Nuse B, Herriott L, *et al.* Long term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of blood brain-barrier, ultrafine particle deposition and accumulation of amyloid beta 42 and alpha synuclein in children and young adults. *Toxicol Pathol* 2008;36:289-310.
13. Tandon OP, Gupta P, Bhargava SK, Chaswal M. Brainstem auditory evoked potentials among rubber factory workers. *Indian J Physiol Pharmacol* 1999;43:205-10.
14. Tandon OP, Kumar V. Visual evoked potentials in rubber factory workers. *Occup Med (Lond)* 1997;47:11-4.
15. Shukla GS, Singhal RL. The present status of biological effects of toxic metals in the environment: Lead, cadmium, and manganese. *Can J Physiol Pharmacol* 1984;62:1015-31.

16. Kilburn KH. Effect of diesel exhaust on neurobehavioural and pulmonary functions. *Arch Environ Health* 2000;55:11-7.
17. Stockard JJ, Sharbrough FW. Unique contribution of short latency sensory evoked potentials to neurological diagnosis. *Prog Clin Neurophysiol* 1980;7:231-63.
18. Tandon OP. Auditory brainstem evoked responses in healthy north Indians. *Indian J Med Res* 1990;92:252-6.
19. Calderón-Garcidueñas L, Mora-Tiscareño A, Fordham LA, Chung CJ, García R, Osnaya N, *et al.* Canines as sentinel species for assessing chronic exposures to air pollutants: Part 1. Respiratory pathology. *Toxicol Sci* 2001;61:342-55.
20. Calderón-Garcidueñas L, Vincent R, Mora-Tiscareño A, Franco-Lira M, Henríquez-Roldán C, Barragán-Mejía G, *et al.* Elevated plasma endothelin-1 and pulmonary arterial pressure in children exposed to air pollution. *Environ Health Perspect* 2007;115:1248-53.
21. Calderón-Garcidueñas L, Franco-Lira M, Torres-Jardón R, Henríquez-Roldán C, Barragán-Mejía G, Valencia-Salazar G, *et al.* Pediatric respiratory and systemic effects of chronic air pollution exposure: Nose, lung, heart, and brain pathology. *Toxicol Pathol* 2007;35:154-62.

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