

Sawaguchi T, Kato I, Franco P, Sottiaux M, Kadhim H, Shimizu S, Groswasser J, Togari H, Kobayashi M, Nishida H, Sawaguchi A, Kahn A.

Apnea, glial apoptosis and neuronal plasticity in the arousal pathway of victims of SIDS.

Forensic Sci Int. 2005 May 10;149(2-3):205-17.

Department of Legal Medicine, Tokyo Women's Medical University, 8-1 Kawada-cho, Shinjuku, 162-8666 Tokyo, Japan.

Of 27,000 infants whose sleep-wake characteristics were studied under the age of 6 months, 38 died unexpectedly 2-12 weeks after the sleep recording in a pediatric sleep laboratory. Of these infants, 26 died of sudden infant death syndrome (SIDS), and 12 of definitely identified causes. The frequency and duration of sleep apneas were analysed. Sleep recordings and brainstem histopathology were studied to elucidate the possible relationship between sleep apnea and neuropathological changes within the arousal system. Immunohistochemical analyses were conducted using tryptophan hydroxylase (TrypH), a serotonin synthesizing enzyme, and growth-associated phosphoprotein 43 (GAP43), a marker of synaptic plasticity. The terminal-deoxynucleotidyl transferase-mediated dUTP nick end labeling (TUNEL) method was used for apoptosis. The pathological and physiological data were correlated for each infant. In the SIDS victims, statistically significant positive correlations were seen between the number of TrypH-positive neurons in the dorsal raphe nucleus of the midbrain and the

duration of central apneas ($p=0.03$), between the number of TUNEL-positive glial cells in the pedunculo pontine tegmental nucleus (PPTN) and the average number of spines in GAP43-positive neurons in the PPTN ($p=0.04$). These findings in the dorsal raphe nucleus of the midbrain and PPTN, that play important roles in the arousal pathway suggest a possible link between changes in arousal and SIDS.

For full-text: <http://www.sciencedirect.com>