PHOTOSENSITIVITY – Photophobia

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An unusual sensitivity to light is rampant: photophobia, irritating and distracting, a safety risk in road traffic: "A potentially debilitating symptom." Bright light can act as a trigger, for example in connection with dysfunctions of neural networks.

Search engines don't have the answer. Among other things, they deliver information on regional sympatholysis or botulinum toxin treatments up to "beta-blockers, calcium channel blockers, anticonvulsants, and CGRP inhibitors". After all, "avoiding intense light" is also mentioned – an almost prophetic final sentence, provided that a small correction is made: "preventing intense (shortwave-dominated) light".

Glaring bluish-white light triggers physiological protective processes, such as reflexive palpebral constriction and pupil contractions, as well as avoidance of overdosed dazzling light (light aversion), even in newborns, still blind mice (without electrophysiologically measurable cone and rod function). Intrinsic photosensitive retinal ganglion cells (ipRGC) take over this warning, even – surprisingly, in blind patients - with intact ipRGC systems. The evolutionarily grown "avoiding intense light-habit" is increasingly ignored since early childhood - under the undesirable influence of glaring bright monitors in smartphones and tablets etc.

Conditioning and epigenetic imprinting

Even small children are often ‘sedated’ with funny moving images from smartphones and "child-friendly" tablets. Not unexpectedly, attention deficit/hyperactivity disorder (ADHD) is quoted in this context. The high integral brightness of these monitors induces – cave temporal summations – conditioning and epigenetic imprinting, à la longue.

Retinal light exposure does not decrease during adolescence, on the contrary. An impairment of light sensitivity (ICD 11) develops unnoticed; Children with their crystal-free media and adolescents force themselves to stare at bright bluish bright screens for extended periods of time – into the old age – perhaps with macular degeneration.

The widespread ailment of dry eye is also involved, together with chronic headaches and dysphorias occasionally. Some pathophysiological processes triggered by melanopsin ipRGC reach the posterior thalamic nuclei via trigeminothalamic pathways. Concentric cortical depolarizations can cause the release of neuropeptides, such as the inflammatory mediator calcitonin gene-related peptide (CGRP), and excessive vascular responses in the dura and meninges.

Bright light as a trigger

Reports of blind patients complaining of discomfort under the influence of bluish-bright artificial light make people sit up and take notice. The answer is – the sensitivity of intact ipRGC-system. Blue - (not yellow -) light can cause inflammation of trigeminal ganglia – especially comorbid with blunt brain trauma - whereby secondary sympathetic and parasympathetic processes are involved with exaggerated reactions to short-wavelength dominated light. CGRP activates protein kinases and transcription factors, ultimately inflammatory cascades (interleukins and cytokines). CGRP administration causes photophobia in both animal experiments and human clinical trials: photophobia can be accompanied by headache, occasionally by corneal symptoms – seemingly unclear..

Dysfunctions of neural networks

In addition to the symptoms of dry eye, outgoing trigeminal dysfunction provokes pain sensations. Neuronal and glial depolarizations (spreading depolarization) are the pathophysiological substrate of migraine aura. CGRP-levels of migraine patients are increased significantly. Blunt traumatic brain injury (TBI) after sports or traffic accidents can result in various undesired sequelae. Post-traumatic headaches (PTH) can flare up again and again for months and years. Diffuse axonal lesions (DAI), inflammation and impaired healing processes up to collapse of the blood-brain barrier cause chronic trigeminal hypersensitivity.

The symptom photophobia runs like a red thread through the dysfunctions of neural networks. Many unsuccessful therapeutic efforts are made understandable by trigeminothalamic pathophysiology. Photophobia can be exacerbated by emotional reactions, and depressive moods can join already existing symptoms. For a long time the outpatient clinic for dry eyes paid attention to the emotional factor; accompanying dysphorias were not dismissed as meaningless or harmless.

Conclusion:

Bright bluish light makes no significant contribution to central vision (Brindley 1954) and has to be reduced or prevented – especially in road traffic.

Photophobia, ICD 11: "Impairment of light sensitivity"

Literature:

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