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CEREBROVASCULAR SYMPATHETIC NERVOUS ACTIVITY DURING CLUSTER HEADACHES <u>Hisaka Igarashi</u>, Fumihiko Sakai, Syuichi Suzuki, Yoshiaki Tazaki Department of Medicine, Kitasato University, Sagamihara, Kanagawa, Japan

To investigate the role of cerebrovascular sympathetic nervous system in the pathogenesis of cluster headache (CH), arterial and jugular venous norepinephrine (NE) was measured serially during attacks of CH.

Nine patients were studied during CH: 5 during nitroglycerin (NG) induced CH, 1 during alcohol induced CH and 3 during spontaneous CH. Six were studied during remission periods. Blood were sampled through catheters in the brachial artery (A) and in the internal-jugular vein (J). NE was measured by high performance liquid chromatography. Relative changes in cerebral blood flow (%CBF change) were measured by A-J differences of oxygen contents. Cerebrovascular release index (CRI) for NE was calculated by (J-A difference of NE)* (%CBF change).

During the headache-free period NE was 149.8± 13.0pg/ml(m+SEM) in (A) and 143.8±15.2 in (J). Following sublingual NG (0.9mg), NE was increased significantly at 5, 10 min by 38-89%(A) and 115-130%(J), but thereafter became reduced. CH occured at 37±3 min (N=5/5) following NG when NE was reduced to the steady state level. During CH, NE was increased by 84% (A) and 172% (J). CBF was increased immediately after NG but only for 3-4 min, then became reduced and dependent on paCo_. CRI was -5.3±12.5("minus" means release < metabolism) before NG, +38.8±22.5 at 10 min after NG, -8.7±14.1 at the beginning of CH and +6.1±16.3 when headache was maximum. Oxygen inhalation relieved headache and reduced CRI for NE. During remission period the increase in CRI following NG was lower than during cluster period.

The present results indicated that NG caused the release of NE from the cerebral vessels and CH occured after the release of NE had subsided. Possible role of cerebrovascular sympathetic nervous activity in the pathogenesis of CH was suggested.

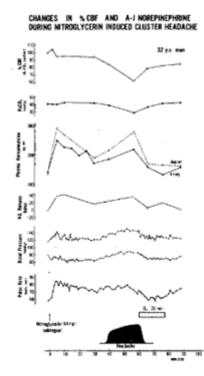


Fig 2: Following sublingual nitroglycerin (0.9mg) %CBF increased for 3-4 min, then became dependent on paCO₂. Norepinephrine (NE) was increased significantly (Jugular>Arterial) after nitroglycerin and then became reduced. Headache occured when NE was reduced to the steady state level.

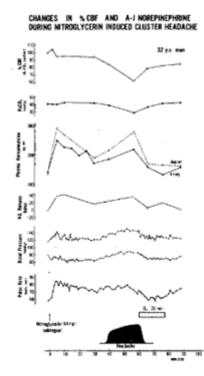


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