**Abstract 35 Table 2** Investigation and management cAHI - 1-5 vs ≥5

| cAHI | No of patients | Investigations | | | | Management | | |
|---|---|---|---|---|---|---|---|---|
| | | ENT referral | EEG | Neuroimaging | Genetics | O2 | CPAP/NIV | Follow-up study |
| 1-5.0 | 148 | 30 | 0 | 0 | 0 | 0 | 4 | 51 |
| ≥5.0 | 14 | 4 | 2 | 3 | 2 | 4 | 2 | 7 |

Patients with isolated cAHI 1-5 had no specific clinical features except adenotonsillar hypertrophy. Isolated severe CSA was identified in ex-preterm or complex neurodisability (table 1). Only severe CSA patients had further investigations to exclude a central/genetic cause (table 2).

45 patients with a cAHI of 1-5 had a follow-up study. of these, 41/45 had a comparable CSA severity with cAHI <5. 4 patients had cAHI 1-5 but newly identified OSA on follow-up.

**Discussion** Our analysis shows that children with an isolated cAHI of 1-5 had reproducible results on follow-up study. There was an association between CSA≥5 and OSA which has not been reported in the literature suggesting the central component may resolve after treating OSA.

## REFERENCE

1. Ghirardo S, Amaddeo A, Griffon L, et al. Central apnea and periodic breathing in children with underlying conditions. J Sleep Res 2021:e13388.

## 36 LITERATURE REVIEW ON THE EFFECTS OF ACUTE AND CHRONIC ALCOHOL USE ON THE GLYMPHATIC TRANSPORT SYSTEM

Anne Marie Bonnici-Mallia*, Vidya Girishankar. *South London and Maudsley NHS Foundation Trust, London, UK*

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**Introduction** Alcohol is known to disrupt various brain processes, including accumulation of βamyloid involved in the pathology of Alzheimer's disease. The glymphatic system clears toxins including βamyloid. It is mainly active during Non-REM slow wave sleep [1]. We wanted to research effects of acute and chronic use of alcohol on glymphatic clearance.

**Method** Peer reviewed articles identified in PubMed from inception until 5 September 2021. Search terms included: 'glymphatic', 'glial-lymphatic', 'alcohol' and 'ethanol'. This resulted in thirteen publications of which four were relevant and included.

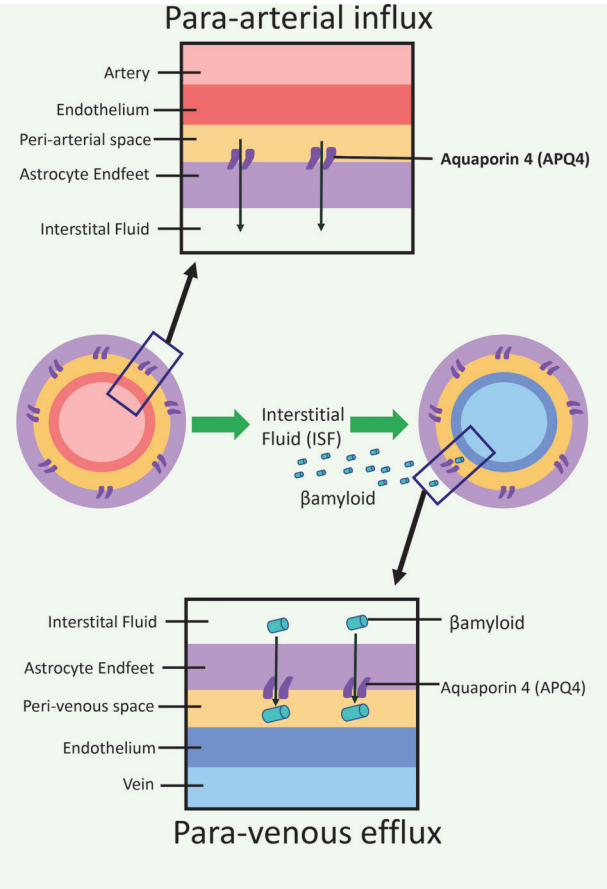
**Results** The glymphatic system is a perivascular transport system (figure 1).

Low dose alcohol improved glymphatic clearance, increasing influx and efflux of CSF also influenced by increased cerebral blood flow [2]. This promoted clearance of βamyloid [7]. There was a decline in GFAP with reduced AQP4 loss in chronic low use [2] as well as improving endothelial tone [7]. After stopping chronic low alcohol an increase in CSF influx and glymphatic function was observed [2].

Acute medium-high alcohol causes reversible decline in glymphatic transport, worse with higher dose [2,4]. The decreased influx may be mediated by release of endogenous opioids. Alcohol decreases cardiac output [6], with decline in heart rate [1] decreasing pulsatility of vessels and CSF influx [4]. This process may also be influenced by reduced glucose metabolism [2].

Chronic high alcohol caused decreased glymphatic clearance [1,2,6]. This process is partially irreversible due to astrogliosis causing increased GFAP with depolarization of APQ4 [6] and compromise of the blood brain barrier [3]. Although there is evidence of some reversible changes following abstinence [2].

**Discussion** Low dose alcohol appears to promotes glymphatic function. Medium-high alcohol disruption is initially reversible, becoming at least partially irreversible with chronic use. These changes impact the role of the glymphatic system, inhibiting its function during sleep and increasing risk of Alzheimer disease.




**Abstract 36 Figure 1** Representation of CSF flow from subarachnoid space into interstitial fluid and parenchyma. Aquaporin 4 (AQP4) water channels are polarised toward the perivascular space allowing flow of soluble molecules, including efflux of βamyloid from the interstitial fluid