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P257 SUSTAINED ASPIRIN EFFECTS ON PLATELETS FUNCTION OVER 24 HOURS IN PATIENTS WITH UNTREATED OBSTRUCTIVE SLEEP APNOEA SYNDROME (OSAS)

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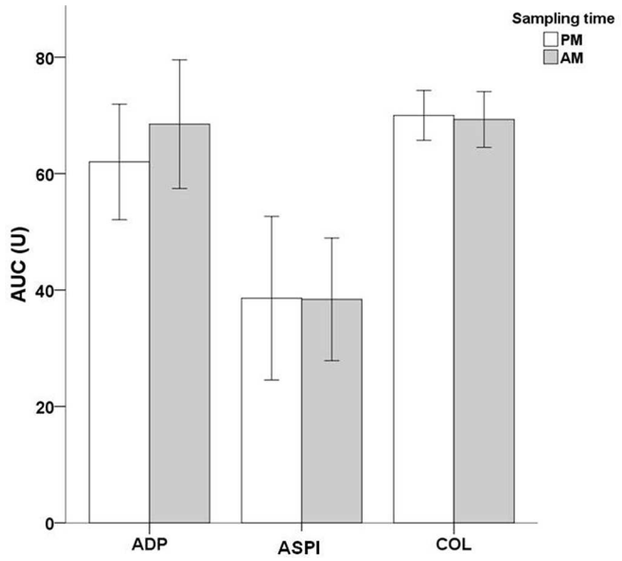
Introduction Prevalence of OSAS in people with cardiovascular disease is much higher than in the general population. Many OSAS patients are prescribed Aspirin for secondary prevention of cardiovascular events but the effects of morning Aspirin might be attenuated by night of recurrent apnoeas and intermittent hypoxia. Therefore evening dosing might be more appropriate.
Aim To assess platelets function in the afternoon and immediately post-sleep in untreated OSAS subjects who are on long term once daily 75mg Aspirin (am).
Methods 11 subjects with newly diagnosed and untreated severe OSAS prescribed Aspirin by their physicians: 10 males, (mean + SD) BMI 40.8 ± 7.2 kg/m^2, age 60.8 ± 10.02 years, 4% Desaturation rate (4% DR) 58.4 ± 40.8 events/hour.

Platelet aggregation was induced *in vitro* by collagen (COL), adenosine-diphoshate (ADP), and arachidonic acid (ASPI). Platelet activation was measured by multiple electrode platelet aggregometry (Multiplate™). Blood samples were collected at 4pm then 7:30am the following morning, prior to the Aspirin dose.
Results Platelet aggregation in response to ASPI was reduced in the presence of Aspirin as expected and the effects were the same in the afternoon (38.6 ± 44.4 units) versus morning (39.2 ± 36.2 units).



Abstract P257 Figure 1. Platelets aggregation in vitro in response to agonists stimulation: collagen (COL). adenosine-diphoshate (ADP). and arachidonic acid (ASPI) in subjects with severe OSAS on regular morning Aspirin. Results are presented as mean ± SEM.

There was no difference between afternoon and morning platelet function ($p > 0.05$ for all measures of platelet aggregation using three agonists) (Figure 1).
Conclusions 75 mg of once daily (morning) Aspirin was sufficient to block *in vitro* platelet aggregation in untreated severe OSAS over a 24 hour period and was not influenced by recurrent apnoeas occurring just prior to blood sampling.

P258 A CASE-CONTROL STUDY OF ASSOCIATIONS BETWEEN HEREDITARY HAEMORRHAGIC TELANGIECTASIA AND COMMON RESPIRATORY CONDITIONS

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Background Hereditary haemorrhagic telangiectasia (HHT) is an inherited disease of the vasculature presenting with nosebleeds, telangiectasia and arteriovenous malformations (AVMs). HHT is caused by gene mutations affecting the transforming growth factor beta (TGF-) superfamily signalling pathway. As TGF- signalling is crucial for embryonic lung maturation, respiratory homeostasis and plays a pivotal role in the pathogenesis of several respiratory diseases, we hypothesised that HHT could alter the respiratory environment and respiratory disease susceptibility. As a first step, we aimed to examine associations between HHT and other respiratory conditions common in the British population.
Methods An online questionnaire at www.imperial.ac.uk/medicine/HHTsurvey2012 was used to collect data. Questions regarding HHT were used to assign HHT status. Participants were categorised based on self-reported status, positive/negative family history and presence of Curaçao criteria (nosebleeds, telangiectasia in specific patterns, and AVMs) Participants' demographics and coexisting respiratory conditions were analysed with Spearman's, Mann-Whitney, and logistic regression analyses using STATA (version 12, Texas USA).
Results By data download on 5 April 2013, 1,465 participants had completed the questionnaire. 1,080 were classified as HHT patients and 179 as controls. Ages ranged from 17–92 (53)years. A history of smoking was provided by 31.5% of the HHT population and 33.1% of the controls. In univariate analyses, COPD, venous thromboemboli, pulmonary hypertension and sleep apnoea were more common in the HHT population in comparison to the controls (respectively, $p = 0.038$; $p = 0.019$; $p = 0.004$; $p = 0.015$). When multiple regression analyses were used to correct for age and smoking, the association with COPD was lost. However, the positive associations with pulmonary hypertension ($p = 0.034$), venous thromboemboli ($p = 0.036$) and sleep apnoea ($p = 0.033$) remained. Sleep apnoea demonstrated a 2.6 fold higher prevalence in the study HHT population (69/100,000) in comparison to published figures for the general population (27/100,000 from the Hospital Episodes System (HES).
Conclusions The study identified two known associations (HHT and pulmonary hypertension; HHT and venous thromboemboli), supporting the validity of the study methodology. Further investigation is needed to validate and understand the rationale for a possible novel association between HHT and sleep apnoea.

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