JOURNAL REVIEW

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Researchers in Japan have looked at whether OSA impedes the recovery of Left ventricular function after Acute myocardial infarction. In consequestively taken 86 patients with first time AMI they found presence of OSA to be 43% (37/86) (AHI>15). Left ventricular ejection fraction, Left end diastolic volume index and regional wall motion abnormalities were evaluated with left ventriculograms at time of PCI and 21 days post PCI. Increases in the above mentioned indices after 21 days were significantly lower in group with OSA then without.

The above study again reiterates the high prevalence of sleep disorderered breathing in patients with acute MI . The fact that it may impede the recovery of the cardiac function has profound implications on patient care and long term prognosis.

Nakashima H.Eur Heart J. 2006 Sep 6

In a similar finding Sharma et al at the University of South Carolina, USA looked at the prevalence of undetected OSA in a patient population undergoing cardiac rehabilitation.118 consequetive patients were screened with Berlin questionnaire.

Out of 118 2o patients had pre-existing OSA. Of the remaining 98 patients 43 paitients (44%) were found to have undiagnosed OSA. Surprisingly there was no difference in prevalence between male and females as seen in general population. BMI in the two groups was also not significantly different. Clinical implications of above findings are that gender and BMI may not matter in patients with cardiac diseases and that screening of Sleep disordered Breathing may be of value in a cardiac rehab setting where there is ample opportunity to educate the patients which may lead to improved compliance. Further studies are being conducted at University of south Carolina to look if cytoline levels (markers of inflammation) are raised in patients with OSA in cardiac rehab and if concomitant treatment with positive airway pressure therapy would improve outcome of rehab programs.

Sharma S, et al Prevalence of undetected OSA I n population undergoing cardiac rehab. Abstract . Accepted for presentation at World Sleep Apnea Conference , Montreal sept. 2006.

Other than history physicians also rely on host of physical findings when screening for Obstructive Sleep Apnea.Commonly used physical cues are increased BMI, increased neck circumference and airway anatomy. Researchers at university of California looked at usefulness of Mallampati score as one of the tools for screening of OSA. Adjustment for confounding factors like BMI, neck circumference ,ethnicity (total of 30 variables taken in consideration).

Researhers found Mallampati score to be independently associated with both presence and severity of the disease.With every 1-point increase in Mallampati score odds of having OSA increased 2 fold and AHI increased by 5 events per hour.

These results suggest that mallampati score can be an important tool for physicians in evaluation of patients with suspected OSA. Mallampati score though has not been rigoursly evaluated for different ethnicities and hence studies with local population to validate its effectiveness need to be considered.

Nuckton et al.Sleep. 2006 Jul 1;29(7):903-8.

Javaheri and other investigators have shown in the past that there is more ventricular irritability associated with patient with OSA than without. Though this appears intuitive as OSA causes hypoxemia and increased sympathetic tone which predispose to arrythmias there is not much literature published to further define the association between the two. Recently researchers at Case Western Reserve University took two samples of the original Sleep Heart Health Study and looked at the prevalence of atrial fibrillation and clinically significant ventricular arrythmias between patients with OSA and non-OSA. After matching for confounding factors

individuals with severe Sleep –disorered breathing were found to have 2-4 fold higher odds of complex arrythmias than those without sleep disordered breathing.More rigourous trials are needed to find if treatment with positive airway pressure would reduce the occurrence of arythmias on a sustained basis.

Mehra et al. AJRCCM 2006:173;910-916

REM behavior disorders though not common have a profound adverse impact on the patient. Traditionally patients have been treated with Clonazepam which is considered first line therapy for the disease. However in the geriatric population which is mainly affected by REM behavior disorder clonazepam may not be well tolerated due to several adverse effects. Recent research suggests that the pathophysiology underlying RBD may involve a dopaminergic deficiency, given its association with Parkinson syndromes and restless legs syndrome (RLS).

In a case series of 10 patients researchers used pramipaxole , a dopaminergic D2,3 agonist to treat REM behavior disorder for mean period of 13 months.Pramipexole was found to significantly reduce the frequency and severity of RBD symptoms. Due to our experience with pramipexole in restless leg syndrome and limited side effect profile physicians may consider this therapy in certain subset of patients who cannot toerlate the sedation from clonezepam (increased risk of falls) or have significant sleep disordered breathing. Larger randomized trials would be helpful in confirming the role of dopamine agonists and will also provide into the pathophysiology of the disease.

Schmidt et al .Sleep Med. 2006 Aug;7(5):418-23. Epub 2006 Jul 3

Controversies on Auto-CPAP devices fail to die. First despite a decade of experience and research it is not clear as to what is the sub-set of patients who would benefit from this device. Secondly disturbing reports have come up with regular frequency about the inability of different devices to respond to different flow patterns. Recently researchers in france performed a bench study of five commercially available auto-CPAP devices using a breath waveform simulator to evaluate the sensitivity for detecting flattened wave forms. This was done because flattened inspiratory flow contours (FIFC's) are believed to predict subtle upper airway obstruction and should be eliminated. Studies looking at elimination of FIFC's with appropriate CPAP pressures were associated with better daytime alertness. In the current study major differences were found in the sensitivity and response across the five auto-CPAP devices when subjected to different waveform protocols. Some devices were found to be too sensitive and one device was not able to detect even simulated normal breathing.

Auto- CPAP devices these days are based on complex algorithms. Furthermore most manufacturers will not reveal the algorithm information as it is proprietary in nature.

Physicians typically do not have good understanding of the complex physics principles behind these devices and hence tend to take them at face value.

This opens up a Pandora box where physicians need to determine if they trust the particular device for there patients. It raises questions whether auto- CPAP device should be tested under laboratory conditions for each patient? Should manufacturers disseminate information regarding algorithms to physicians for them to be able to make a decision in the best interest of the patient? It is possible that future generation auto- CPAP devices will be able to detect specific inspiratory flow patterns, but until then should we put the current one's in closet?

Lofaso et al. Chest 2006; 130:343-349

Emergence of Central events or Cheyenne stroke respiration on initiation of positive airway pressure therapy has been labeled as complex sleep apnea syndrome. Limited studies have suggested that these patients may not tolerate CPAP as well as patients as patients with OSAS. Recent study by Mayo Clinics looks at this phenomenon closely in a retrospective analysis of patients who had underwent overnight polysomnography. 133 patients with OSAS were compared to 33 patients identified as having complex sleep apnea syndrome. Researchers found that interface difficulty occurred more common in patients with complex sleep Apnea syndrome. These included air hunger or dyspnea and inadvertent mask removal. No other differences were observed. Final pressures prescribed was similar, follow up on daytime sleepiness by Epworth Score was also similar.These finding are different from previous smaller trials which recorded higher residual symptoms.

This is a new sleep disorder pattern being increasingly

recognized. Therapeutic and prognostic implications are not clear. Physicians should be cognizant of this Abnormal sleep pattern as they will frequently encounter it in their practice.

Morganthaler et al. Sleep Medicine Volume 7, Issue 6 , September 2006, Pages 474-479

In a recent study researchers looked at sleep disorders breathing in patients admitted for Acute coronary syndrome (ACS). Portable sleep studies were performed within 72 hours. Researchers found high prevalence of sleep disordered breathing in this population. Of 104 patients with complete and adequate sleep studies, 66.4% had an apnea–hypopnea index (AHI) >10/h, and 26.0%, an AHI>30 with the prevalent apnea pattern being obstructive (72.1%).

This high prevalence in patients with cardiovascular diseases has been shown in several studies, however it is to be noted that neither pre-test probability nor degree of daytime sleepiness predicted sleep disorder breathing in this group. Although symptoms of dyspnea and paroxysmal nocturnal dyspnea were significantly higher in SDB (AHIe"10) compared to non-SDB (AHI<10) 6 months after admission for ACS, odds of readmission were not significantly different, and this lack of association persisted after covariate adjustment. Hence the authors concluded that there is no short term adverse consequences of OSAS in patients with ACS.

However it is well documented that untreated OSAS has long term cardiovascular consequences. Many short term markers of morbidity were not evaluated like nocturnal chest pain , exercise capacity etc.

Study does highlights the point that traditional screening tools for OSAS may not be valid in patient population with cardiovascular diseases and high index of suspicion and vigilance has to maintained in this population.

Mehra et al. Sleep Medicine Volume 7, Issue 6 , September 2006, Pages 521-528