LITERATURE REVIEW

The role of polysomnography in OSAS patients

Francesco Peverini[1], Francesco Maria Passali[2], Desiderio Passali[3]

[1]Foundation for Research and Treatment of Sleep Disorders, Mater dei Hospital, Rome, Italy

[2]ENT Department, "Tor Vergata" University of Rome, Italy

[3]ENT Department, Le Scotte University Hospital, University of Siena, Italy

ABSTRACT

Obstructive sleep apnea syndrome (OSAS) is a common sleep-related breathing disorder characterized by recurrent temporary obstructions of airflow during sleep, potentially leading to daytime sleepiness and cardiopulmonary dysfunction. Polysomnographic study is the gold standard diagnostic test for sleep disorders, in particular for OSAS, detecting the occurrence and extent of sleep apnea and identifying the cardiovascular and neurophysiological hallmarks of the disease. Polysomnography permits to detect and classify a number of respiratory (apneas, O2 desaturations) and neurological phenomena (e.g. arousals or periodic movements of the lower limbs), as well as blood pressure and cardiac activity.

KEYWORDS: polysomnography, obstructive apnea, hypopnea, respiratory effort-related arousal

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a common sleep-related breathing disorder, characterized by recurrent temporary obstructions of airflow (mainly apneas and hypopneas) during sleep, potentially leading to daytime sleepiness and cardiopulmonary dysfunction. OSAS patients subject their cardiovascular system to intermittent cycles of hypoxia/reoxygenation, accompanied by markedly negative intrathoracic pressure and awakenings[1].

The majority of OSAS patients also display a constellation of metabolic and non-metabolic cardiovascular risk factors typical of metabolic syndrome ("syndrome X"). Indeed, the suggestion has been put forth that OSAS may be a manifestation of metabolic syndrome ("syndrome Z")[2,3] (Figure 1).

Polysomnographic study is the gold standard diagnostic test for sleep disorders, in particular for OSAS, allowing to detect the occurrence and extent of sleep apnea and identify the cardiovascular and neurophysiological hallmarks of the disease[4,5].

Well-defined national (Italian Association of Sleep Medicine) and international (American Academy of Sleep Medicine) guidelines set out the operating procedures of the aforementioned diagnostic tests[6].

Particular attention has been given recently to ENT (ear, nose and throat) specialists and their possible role in performing polysomnographic studies and overseeing a multidisciplinary team devoted to sleep respiratory disorders.

POLYSOMNOGRAPHY TECHNIQUE

Polysomnography involves monitoring a patient during a night's sleep in the laboratory or at home, for multiple neurophysiological and cardiorespiratory signals:

I. Rapid eye movements
II. Electroencephalogram to detect sleep phases and awakenings
III. Movement of the chest and abdomen for respiratory dynamics
IV. Electrocardiogram
V. Electromyogram of the lower limbs and the masseter
VI. O_2 saturation
VII. Oro-nasal airflow (via thermistor/oro-nasal cannula)

Portable sleep monitoring devices are routinely used at sleep centers, hospitals or the patient's home.

Corresponding author: Desiderio Passali
e-mail: d.passali@virgilio.it

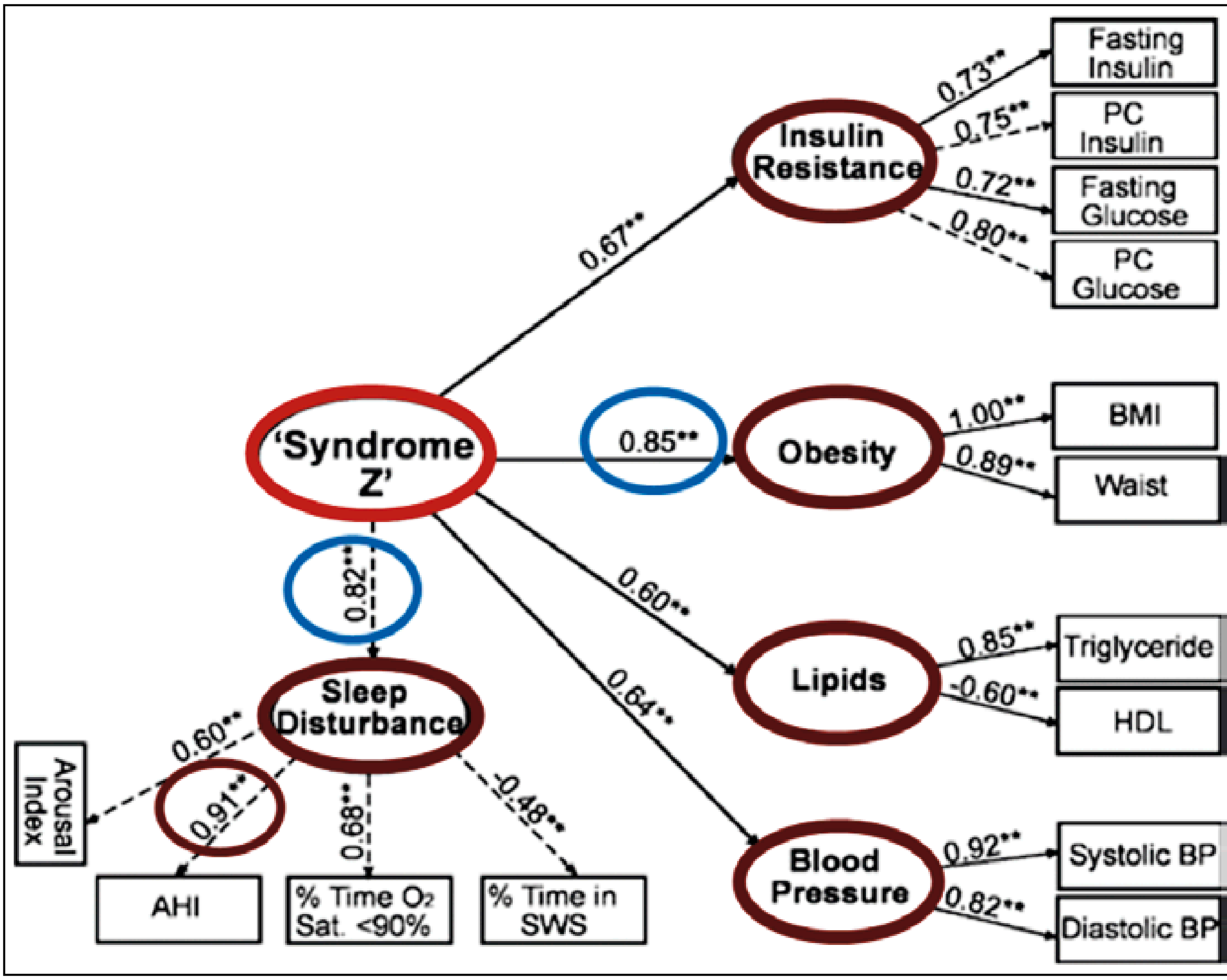

Figure 1 "Hierarchical" hypothesis of the prevalence of metabolic and non-metabolic risk factors of sleep disorders. Amended from: NL Nock et al.: Empirical evidence for "syndrome Z": a hierarchical 5-factor model of metabolic syndrome incorporating measures of sleep disturbance. Sleep 2009, 32:615-622.

Focusing more in detail on sensor placement for a polysomnographic study comprising sleep staging (Figure 2), sensors will need to be placed on the head of the subject for electroencephalogram and electromyogram of the masseter muscle.

This will make it possible to disclose the macro- and micro-structure of sleep.

Other cardiorespiratory sensors used in the polysomnographic study of OSAS patients are shown in Figure 3. These sensors include an oro-nasal cannula, which can be substituted for a thermistor, a snoring sensor, two bands for the movement of the chest and abdomen, sensors for the evaluation of restless legs syndrome, which are arranged at the *tibialis anterior* level of both legs, a position sensor installed on the thorax and an O_2 saturation sensor which also functions as a plethysmographic and heart-rate sensor.

Finally, chest sensors are applied for the recording of a continuous overnight electrocardiogram, which is

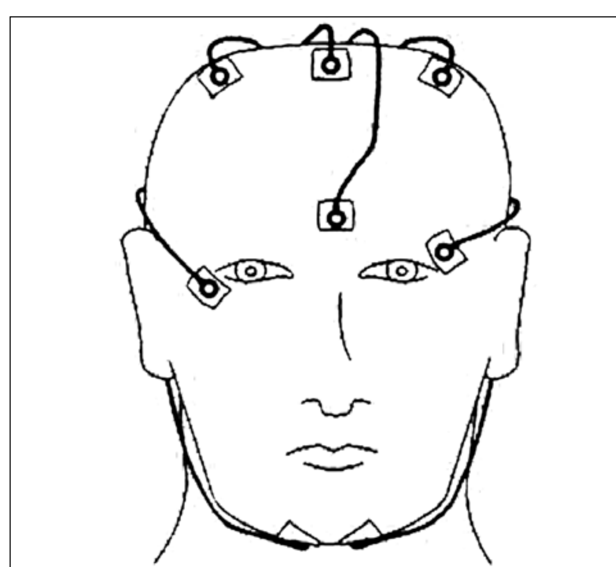

Figure 2 EEG, EOG and EMG sensors for sleep staging during polysomnographic examination.

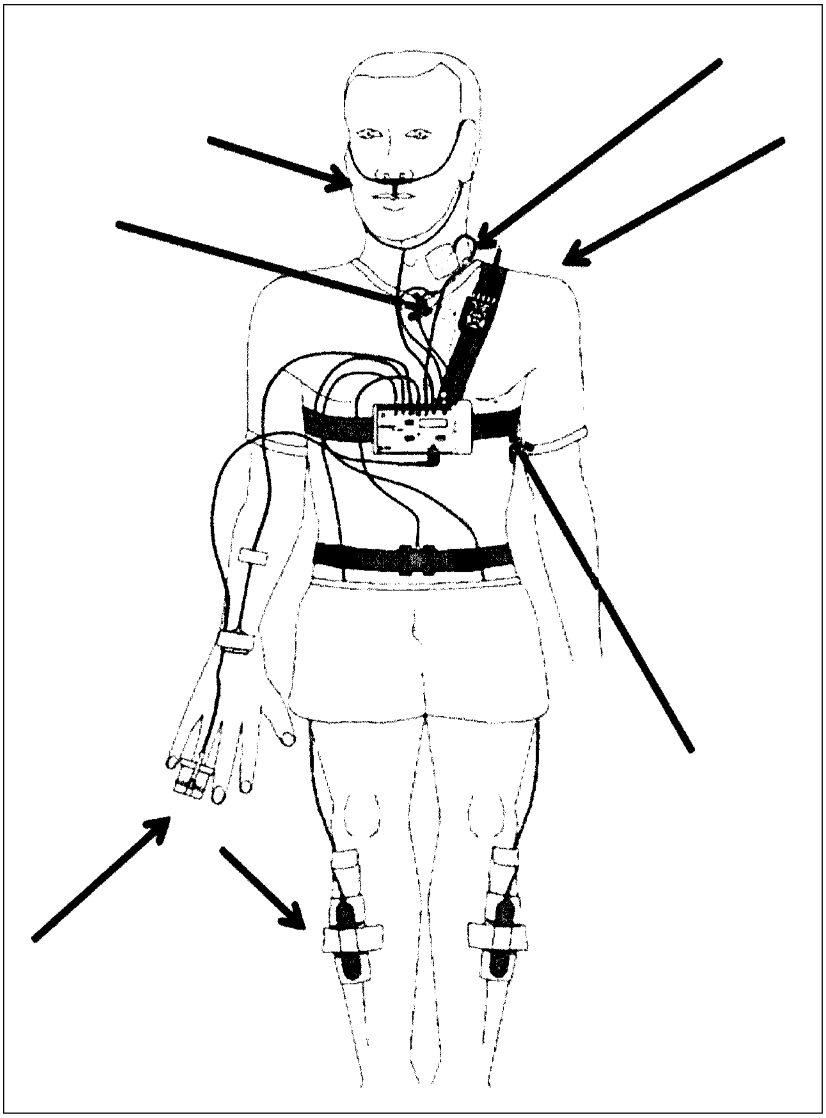

Figure 3 Sensors for the detection of cardiorespiratory events and the study of restless legs syndrome (RLS).

to be compared with the finger heart rate; the time interval elapsing between actual heartbeat and its detection by the pulse oximeter is an interesting investigative technique of intrathoracic pressure ("Pulse Transit Time").

METHODS

The instrumental methods for the definitive diagnosis of adult OSAS include four investigative techniques[7]:

1. Overnight cardiorespiratory system monitoring via portable scaled-down system
2. Overnight cardiorespiratory system monitoring via portable complete system
3. Overnight polysomnography via portable system
4. Overnight polysomnography in the laboratory

The overnight cardiorespiratory monitoring via portable scaled-down system provides recording of at least four parameters: respiratory noise or airflow, heart rate, body position, oxygen saturation. It allows the indirect identification of respiratory events, but does not allow the identification of sleep periods, i.e., sleep staging.

The overnight cardiorespiratory system monitoring via portable complete system provides information about the thoracic-abdominal movements and allows the direct identification of respiratory events, but it does not allow the identification of sleep periods. On the other hand, it permits to evaluate respiratory noise, airflow, thoracic-abdominal movements, heart rate, body position and oxygen saturation.

The overnight polysomnography via portable system allows the identification of respiratory noise, airflow, thoracic-abdominal movements, heart rate, body

Figure 4 Example of a polysomnographic track: the page display covers a 5-minute period. A record is shown here of the following signals: EEG, EOG, EKG, the EMG of the right and left tibial muscles, the snoring signal. The main cardiorespiratory alterations typical of OSAS can be easily identified in the graphical traces including oronasal airflow, thoracic and abdominal respiratory movements, oxygen saturation, heart rate and plethysmography. In this example, a mixed-type apnea is evident, involving a consensual decrease in oxygen saturation reaching the trough a few seconds after the end of the respiratory phenomenon.

Figure 5 Page display covering a 1-minute period, allowing for a more detailed examination of the phenomena occurring at the end of an apneic event. An electroencephalographic alteration (arousal) is evident at the end of the obstructive apnea, which can lead to an unconscious micro-awakening of the patient. Respiratory recovery is heralded by chest and abdomen movements of increased intensity, renewed respiratory noise (snoring) and a drop in oxygen levels whose trough, remarkably, befalls at the beginning of the subsequent apnea. At the same time, a heart rate increase is detected and plethysmography reveals an increase in blood pressure levels.

position, oxygen saturation and electroencephalography with at least two bipolar leads, the electrooculogram, submental electromyography, the study of lower limbs movements for the assessment of restless legs syndrome (RLS). Hence, this method allows the analysis of sleep staging, the elucidation of its micro-structural elements and the direct identification of respiratory events. It does not, however, provide a real-time assessment of track quality or patient oversight throughout the night (Figures 4 and 5).

The overnight polysomnography in the laboratory allows the observation of the electroencephalographic parameters (with at least two bipolar derivations), the electrooculogram, submental electromyogram, respiratory noise, oro-nasal airflow, thoracic-abdominal movements, heart rate, oximetry, body position and the electromyography of the left and right tibialis muscles for the study of RLS.

This method allows sleep staging and recognition of its micro-structural elements, besides direct identification of respiratory events. Likewise, it provides for the overnight monitoring of the patient in a dedicated room and the real-time analysis of the track quality.

CLASSIFICATION OF RESPIRATORY EVENTS IN SLEEP

Apnea

Respiratory event characterized by a greater than 90% drop in airflow, a duration of at least 90% of the overall event, for a longer time than 10 seconds. Desaturation and/or arousal are not necessarily observed features.

Apneas can be distinguished based on respiratory effort into:

- **Obstructive apneas:** sustained by thoracic-abdominal respiratory effort (Figure 6).

- **Central apneas:** respiratory effort is absent; cessation of airflow despite continued patency of the upper airways. This type of apnea is mainly associated with periodic Cheyne-Stokes respiration in chronic heart failure (Figure 7).

- **Mixed apneas:** Respiratory effort is initially absent, but occurs at the end of the event (Figure 8).

Hypopnea

Hypopnea - first classification criterion

This is a respiratory event characterized by reduced airflow to less than 30% of baseline and an oxygen desaturation greater than 4%, which must persist for more than 90% of the duration of the overall event, or for a time not shorter than 10 seconds. Arousal is not a necessarily observed feature (Figure 9).

Hypopnea - second classification criterion

It is necessary to provide a second classification criterion for hypopneas, since this phenomenon can be detected in polysomnographic studies for the assessment of sleep. In this case, breathing must be associated with a greater than 50% reduction in airflow, in addition to a desaturation exceeding 3% for at least 90% of the overall duration of the event (or a time greater than or equal to 10 seconds), or alternatively an EEG-detected arousal.

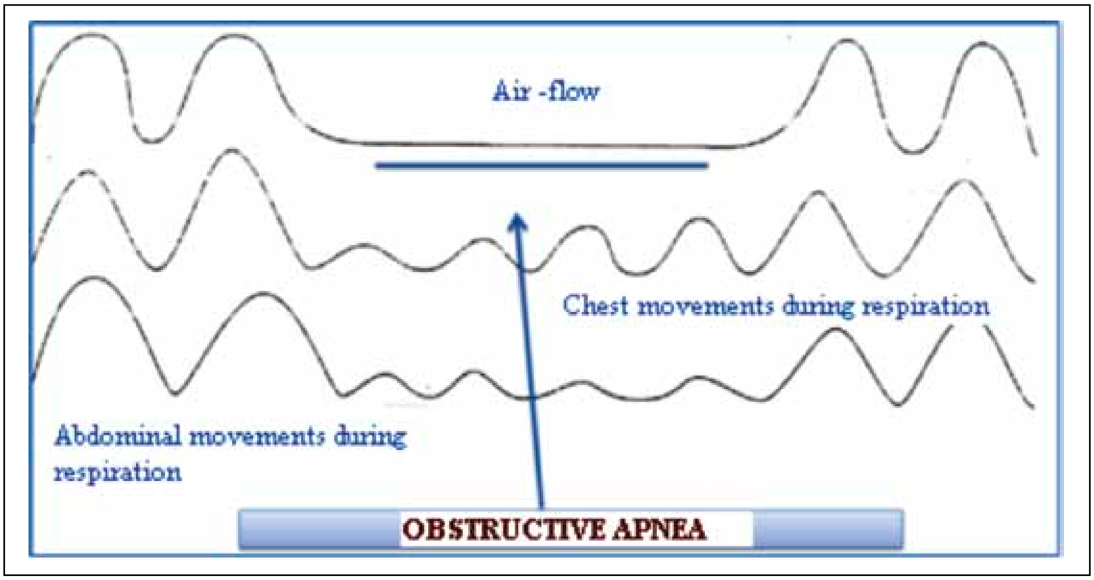

Figure 6 Obstructive apneas

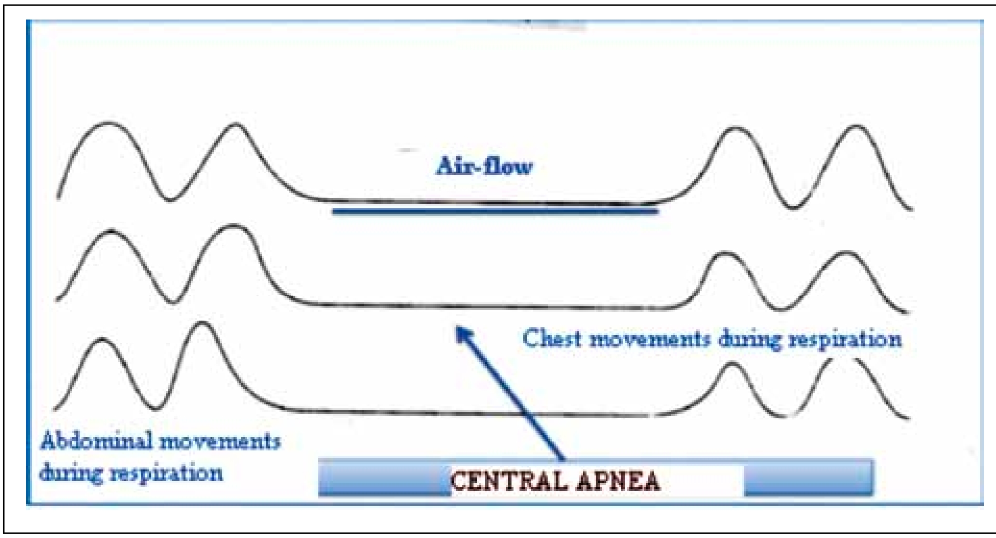

Figure 7 Central apneas

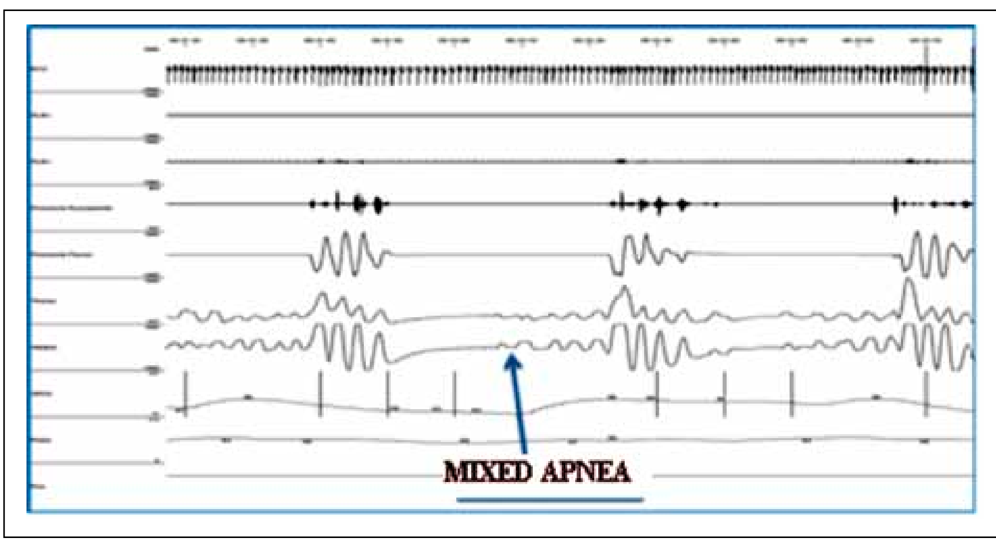

Figure 8 Mixed apneas

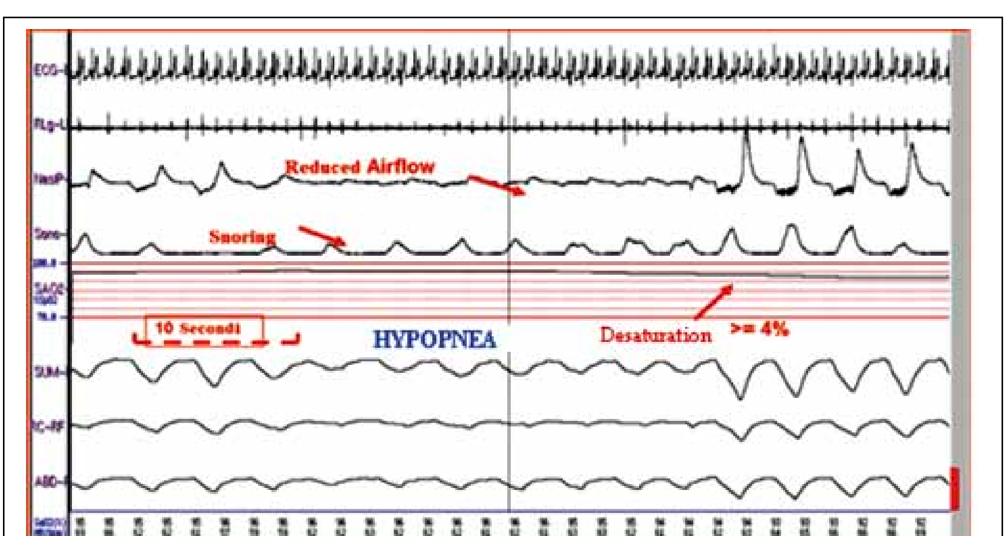

Figure 9 Hypopnea (the first classification criterion)

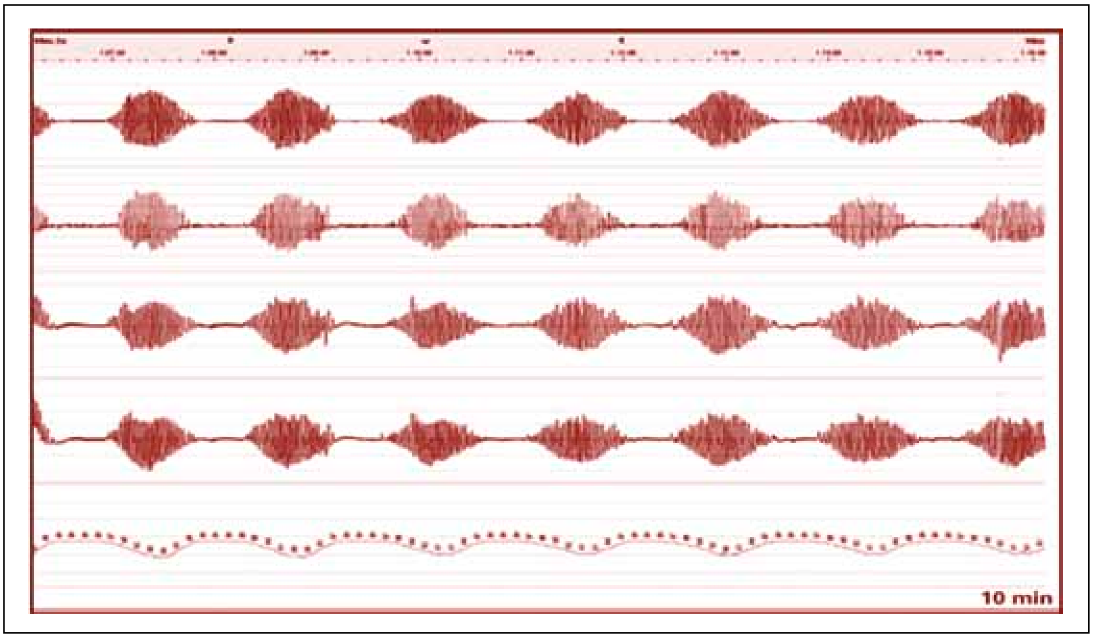

Figure 10 Cheyne-Stokes respiration

RERA (Respiratory Effort-Related Arousal)

RERA is a sleep-related respiratory phenomenon identifiable exclusively by sleep study equipment, with the potential to cause cardiovascular risk.

So defined is a sequence of breaths associated with reduced air flow and a progressively increasing inspiration effort leading to arousal and an airflow reduction less than 30%; it is unnecessary to detect oxygen saturation, and the overall duration of the event is greater than or equal to 10 seconds.

Cheyne-Stokes respiration

This is a cyclical breathing fluctuation with "crescendo-decrescendo" features. It must persist for at least three consecutive cycles, or for a longer duration than 10 minutes, or when more than five central apneas are detected per hour of sleep (Figure 10).

Complex apnea

Complex apnea is a peculiar respiratory disorder, detectable in subjects undergoing CPAP or BIPAP therapy. In complex apnea, a central respiratory phenomenon persists or emerges during the titration reading of the polysomnography used for the ventilation therapy.

DIAGNOSTIC GUIDELINES

In order to make a diagnosis of OSAS, at least one of the following symptoms must be present[7]:

I. Daytime sleepiness, fatigue, non-restful sleep or insomnia

II. Awakenings with sensations of blocked breathing, cough or air hunger

III. Loud snoring and/or respiratory pauses in sleep as reported by the partner

or: **R.D.I.** (apneas + hypopneas + RERAs/hour of sleep)> 5

or: a respiratory disorder in sleep that is not otherwise explained by other sleep disorders, medical or neurological diseases, or drugs.

An instrumental evaluation throughout the whole night is essential for the diagnosis of OSAS.

Evidence A *Consensus A*

Before sending a patient with suspected OSAS to instrumental evaluation, the following signs and symptoms must be searched for[7,8]:

Symptoms

I. Habitual snoring (every night), persisting for more than 6 months

II. Respiratory pauses during sleep as reported by the partner

III. Awakenings accompanied by a choking sensation in a (not necessarily habitual) snorer

IV. Daytime sleepiness

Signs:

I. BMI > 29

II. Neck circumference > 43 cm for men, or > 41 cm for women.

III. Craniofacial dysmorphisms and oro-pharyngeal abnormalities (any anatomical condition causing a reduced airways caliber).

Evidence B *Consensus A*

I. Habitual persistent snoring alone or with other symptoms or signs
II. At least two more symptoms, other than habitual persistent snoring: respiratory pauses +waking up with choking respiratory pauses + daytime sleepiness waking up with choking + daytime sleepiness
III. Presence of a symptom other than habitual persistent snoring plus at least two signs
IV. Presence of a symptom other than habitual persistent snoring plus at least one sign in subjects for whom snoring cannot be ascertained (e.g. Patients sleeping alone).

The main indicators of respiratory events in sleep are:

- **AHI** = Index of apnea + hypopnea or Apnea Hypopnea Index
  - Number of apneas + hypopneas per hour of sleep
  - There exists also the possibility to denote:
  - AHIc (central respiratory phenomena)
  - AHIo (obstructive respiratory phenomena)
  - AHIs (respiratory phenomena exclusive to the supine position)
- **RDI** = Index of Respiratory Disorder in Sleep or Respiratory Disorder Index
  - Number of apneas + hypopneas + RERAs per hour of sleep
- **ODI** = Index of O_2 desaturation event or Oxygen Desaturation Index
  - Number of O_2 desaturation events per hour of sleep, being mindful to indicate the cut-off value (-4% / - 3%)

PREDICTIVE TEST

A great help in the screening of OSAS is provided by predictive tests, which can indicate a condition of increased daytime sleepiness or OSAS-associated disorders.

When interviewing a patient with suspected DRS, listening to the bed partner is especially relevant, because of the important information he/she can provide from the direct observation of the respiratory phenomena at night. A different perspective may also be provided on daytime symptoms, with respect to sleepiness or neurocognitive impairment.

The very high percentage of the population unaware of having a Sleep Apnea Syndrome and who will probably never be directed to undertake a sleep study is a real challenge for sleep health care professionals and a serious problem for cardiologists, neurologists, anesthesiologists, intensivists and finally insurance companies, given the high rate of accidents from falling asleep and the high incidence of complications of the disease.

To that end, several screening tests have been developed over time, in order to identify by low-cost and high-throughput procedures, as many at-risk patients for OSAS as possible; such procedures would have the obvious advantage of directing available resources to a population selected in accordance to tests (as well as guidelines), reducing as much as possible the number of negative tests.

Some important data on sleepiness and associated disorders can be obtained from the quick administration to the patient of questionnaires; our clinical experience and literature data have allowed us to prefer three in particular[9]:

1. Epworth Sleepiness Scale
2. Berlin Test
3. STOP BANG Test

CONCLUSIONS

Among sleep disorders, those with the greatest impact on health and highest healthcare-related costs are the respiratory disorders in sleep (mainly night apneas - OSAS).

Their incidence in the Italian population is estimated at about 180000 people, 95% of which probably undiagnosed.

There has been barely any increase in newly-diagnosed patients over the last 20 years, a greatly concerning figure from a public health and social point of view[10,11]. Unfortunately, this problem is still inadequately addressed, in all its complexity and also at the educational (university), preventative or patient-care level.

Daytime sleepiness, a paramount symptom of OSAS, results from non restful sleep at night and can lead to very serious consequences (traffic accidents, workplace accidents, etc.). Furthermore, traffic accidents by excessive daytime sleepiness carry high costs, both public and private, are generally more severe, and result in a mortality rate almost twice that of accidents due to other causes[5,10,12].

Different, but equally relevant for public health, is the impact of sleep apnea syndromes on cardiovascular risk: few disorders like sleep apnea display such a strong and important association with hypertension, arrhythmias and heart failure[4].

Several scientific studies have reported that 45-50% of all hypertensive subjects are actually suffering from a hidden, undiagnosed sleep apnea and the same applies to 25-50% of patients with heart failure[13-15].

The most impressive data concern patients who have had a Transient Ischemic Attack (TIA) or a stroke: a sleep apnea syndrome may be present in 60% of these cases and the figure rises consistently once the presence of a relatively common heart defect, such as patent foramen ovale, is factored in[16].

Polysomnography permits to detect and classify a number of respiratory (apneas, O2 desaturations) and neurological phenomena (e.g. arousals or periodic movements of the lower limbs), as well as blood pressure and cardiac activity; it is therefore essential for the interpretation of such data to have some basic knowledge of specific technical standards.

In order to set up an adequate treatment[17,18], OSAS medical specialists need to make the best use of the available severity criteria provided by the international guidelines.

However, it is often the case that the indexes resulting from the scoring parameters of sleep-related respiratory events (RDI, AHI, ODI) prove inadequate to classify and confidently deal with all clinical situations.

Indeed, patterns of sleep-related events frequently occur that hardly fit a patient on the exclusive basis of strict taxonomic criteria[19], providing an interesting interpretative and therapeutic challenge.

This consideration, together with the high incidence of OSAS in the general population and the vast array of disease-associated cardiovascular and neurological complications, further highlights the need to assess OSAS patients in an eminently clinical and multidisciplinary framework, reserving instrumentation exclusively to the task of confirming the physician's diagnostic hypotheses, developed on the basis of the patient's signs and symptoms.

It is now necessary to address the question: who is to be tested by polysomnography? In Italy, are the current AIMS – AIPO guidelines the most appropriate to deal with the problem? Who are the best specialists for expanding diagnostic opportunities?

The Otolaryngologist has always been closely linked to the diagnosis and surgical treatment of OSAS. Is it possible to broaden their role in the diagnosis and treatment of OSAS patients?

There are some rationales in this regard:

I. higher frequency of OSAS patients' first visit
II. early identification of obstruction sites
III. widespread availability of ENT specialists

and some challenges to be met:

I. management of diagnostic polysomnography
II. management of the multidisciplinary team
III. carrying out prospective studies to evaluate the efficacy of surgical approach.

REFERENCES

1. Linz D., Schotten U., Neuberger H.R., Bohm M. et al. - Negative tracheal pressure during obstructive respiratory events promotes atrial fibrillation by vagal activation. Heart Rhythm, 2011 sept.;8(9):1436-1443.
2. Wilcox I., McNamara S.G., Collins F.L., Grunstein R.R., Sullivan C.E. - "Syndrome Z": the interaction of sleep apnoea, vascular risk factors and heart disease. Thorax, 1998;53(Suppl 3):S25–28.
3. Nock N.L., Li L., Larkin E.K., Patel S.R., Redline S. - Empirical evidence for "syndrome Z": a hierarchical 5-factor model of the metabolic syndrome incorporating sleep disturbance measures. Sleep, 2009;32(5):615–622.
4. Bradley T.D., Floras J.S. - Obstructive sleep apnoea and its cardiovascular consequences. Lancet, 2009;373:82–93.
5. Brown W.D. - The psychosocial aspects of obstructive sleep apnea. Semin Respir Crit Care Med., 2005;26:33–43.
6. Collop N.A. et al. - Clinical Guidelines for the Use of Unattended Portable Monitors in the Diagnosis of Obstructive Sleep Apnea in Adult Patients. J Clin Sleep Med., 2007 Dec 15;3(7):737-47.
7. Commissione Paritetica Associazione Italiana Medicina del Sonno (AIMS) e Associazione Italiana Pneumologi Ospedalieri (AIPO): Linee Guida di Procedura Diagnostica nella Sindrome delle Apnee Ostruttive nel Sonno dell'Adulto. 2008.
8. Aurora N.R., Zak R.S., Karippot A., Lamm C.I., Morgenthaler T.I., Auerbach S.H., Bista S.R., Casey K.R., Chowdhuri S., Kristo D.A., Ramar K.. - Practice Parameters for the Respiratory Indications for Polysomnography in Children. Sleep, 2011;34(3):379-88.
9. Proimos E., Kiagiadaki D., Kaprana A., Chimona T.S., Maroudias N.J., Papadakis C.E. - Clinical Application of Subjective Measurements for OSAS Assessment: Predictive Factors of Syndrome Severity. ORL, 2012;74:240-245.
10. Young T., Peppard P.E., Gottlieb D.J. - Epidemiology of obstructive sleep apnea: a population health perspective. Am J Respir Crit Care Med., 2002;165(9):1217–1239.
11. Bixler E.O., Vgontzas A.N., Lin H.M., Liao D., Calhoun S., Vela – Bueno A., Fedok F., Vlasic V., Graff G.. - Sleep disordered breathing in children in a general population sample: prevalence and risk factors. Sleep, 2009;32(6):731-6.
12. Punjabi N.M., Caffo B.S., Goodwin J.L., Gottlieb D.J., Newman A.B., O'Connor G., Rapoport D.M., Redline S., Resnick H.R., Robbins J.A., Shahar E., Unruh M.L., Samet J.M. - Sleep-disordered breathing and mortality: a prospective cohort study. PLoS Med., 2009;6:e1000132.
13. Yumino D., Wang H., Floras J.S., Newton G.E., Mak S., Ruttanaumpawan P., Parker J.D., Bradley T.D. - Prevalence and physiological predictors of sleep apnea in patients with heart failure and systolic dysfunction. J Card Fail, 2009;15:279–85.
14. McNicholas W.T., Bonsigore M.R. - Sleep apnoea as an independent risk factor for cardiovascular disease: current evidence, basic mechanisms and research priorities. Eur Respir J., 2007;29:156 –78.
15. Garvey J.F., Taylor C.T., McNicholas W.T. - Cardiovascular disease in obstructive sleep apnoea syndrome: the role of intermittent hypoxia and inflammation. Eur Respir J., 2009;33:1195–1205.

16. Shaikh Z.F., Jaye J., Ward N., et al. - Patent foramen ovale in severe obstructive sleep apnea: clinical features and effects of closure. Chest, 2013 Jan;143(1):56-63.
17. Vicini C., Dallan I., Canzi P., Frassineti S., Nacci A., Seccia V., Panicuccci E., La Pietra M.G., Montevecchi F., Tschabitscher M.. - Transoral robotic surgery of the tongue base in Obstructive sleep apnea-hypopnea syndrome: Anatomic considerations and clinical experience. Head Neck, 2012;34:15–22.
18. Mantovani M., Minetti A., Torretta S., Pincherle A., Tassone G., Pignataro L. - The velo-uvulo-pharyngeal lift or "roman blinds" technique for treatment of snoring: a preliminary report. Acta Otorhinolaryngologica Italica, 2012;32(1):48-53.
19. Tatti P., Passali D., Passali L. - The undisclosed role of anoxia/hypoxia and disturbed sleep on glucose metabolism. Journal of Diabetes Mellitus, 2012;2:186-190.