Obstructive Sleep Apnea: A Brief Overview For the Primary Care Physician

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Obstructive sleep apnea (OSA) affects 2% to 4% of middle-aged adults.[1] It is even more common in the elderly. Although the primary care physician has the opportunity to play a pivotal role in the detection of this disorder, most physicians have had little or no formal training in OSA; and they frequently underdiagnose the disorder.[2,3] The Walla Walla project[2] demonstrated that with several educational interventions for physicians and patients, the OSA detection rate significantly increased. This article will review the typical presentation of OSA, diagnostic tests, and treatment options as well as follow-up once treatment is initiated.

OSA is characterized by repetitive partial or complete closure of the upper airway during sleep despite continued respiratory drive (Figure 1A4). The patient demonstrates increasingly negative intrathoracic pressures as increasing ventilatory effort is generated to attempt to open the airway. These events are usually associated with a brief arousal and/or an oxygen desaturation and transient hypercapnea. These repetitive respiratory-related arousals result in significant sleep fragmentation, which, in combination with the oxygen desaturation, result in subsequent daytime sleepiness and fatigue

An apnea refers to cessation of airflow for more than 10 seconds. An hypopnea is a reduction of airflow for 10 seconds. Both events are associated with continued respiratory effort. In contrast, central apneas have no airflow and no effort. The average number of apneas and hypopneas per hour of sleep is called the apnea-hypopnea index. The **American Academy of Sleep Medicine (AASM)** consensus statement[5] suggests it is not necessary to distinguish between apneas and hypopneas. Instead, the term "respiratory events" should be used to refer to both because they have similar pathophysiology and consequence. More than five obstructed respiratory events per hour of sleep are considered abnormal.

The AASM consensus statement includes both symptoms and sleep study data in the definition of the **obstructive sleep apnea-hypopnea syndrome (OSAHS)**. OSAHS is defined as criteria A or B plus C. Criterion A: Excessive daytime sleepiness that is not better explained by other factors; Criterion B: two or more of the following that are not better explained by other factors: choking or gasping during sleep, recurrent awakenings from sleep, unrefreshing sleep, daytime fatigue, impaired concentration; Criterion C: overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep.

As these criteria suggest, fatigue and disrupted sleep are frequent symptoms of OSA. (Table 1)

Several recent studies have suggested that certain key symptoms and associations are useful in predicting who will have OSA. Kump, et al[6] found that the three symptoms most predictive of OSA are: Self-reported snoring, witnessed apnea, and sleepy driving. The positive predictive value of their model was enhanced by including **body mass index (BMI)** and gender. Netzer, et al[7] found a simple self-administered patient questionnaire helped identify patients at high risk for OSA. Key symptoms include persistent symptoms (≥3 to 4 times per week) in 2 or more questions regarding snoring, witnessed apnea or daytime sleepiness. Alternatively, persistent symptoms in conjunction with hypertension or obesity were suggestive of OSA. Simply adding questions regarding snoring, pauses, and daytime sleepiness to the primary care physician's review of systems will increase the likelihood of detecting obstructive sleep apnea. If the patient has no reliable bed partner, the lack of a history of snoring, pauses, etc. has less significance. One may then need to rely on other symptoms and associated medical conditions

The medical disorders most commonly associated with OSA include hypertension and upper body obesity. Approximately 50% of patients with obstructive sleep apnea have hypertension. Conversely, 25 to 30% of patients from a hypertension clinic will

Table 1. Symptoms of Obstructive Sleep Apnea

Snoring
Witnessed Apnea/gasping
Choking/shortness of breath arousals
Recurrent awakenings
Nocturia (three times per night)
Morning headache
Excessive daytime somnolence
Automobile accident or near miss
Decreased memory/concentration
Depression/irritability
Enuresis
Sexual dysfunction

Table 2. Risk Factors Associated with Obstructive Sleep Apnea

Hypertension
Upper body obesity
Male sex
Increasing age
Abnormal pharyngeal anatomy
Enlarged tonsils and adenoids
Redundant pharyngeal tissue
Retrognathia
Nasal obstruction
Excessive alcohol use
Untreated hypothyroidism
Acromegaly

have obstructive sleep apnea.[8] A collar size ≥17 in men, ≥16 in women, is associated with an increased risk of OSA. Table 2 lists the predisposing or risk factors commonly associated with OSA.

Although the exact mechanism is still under investigation, sleep-related breathing disorders have been associated not only with hypertension, but also with cardiovascular disease independent of shared risk factors such as obesity, age and gender.[8] **Sleep-related breathing disorders (SRBDs)** have also been associated with an increased risk of stroke. It is not yet clear whether this association is due to the increased stroke risk associated with hypertension or whether SRBD is an independent risk factor.[8] In either case clinicians should have a high index of suspicion for OSA in patients with cardiovascular and cerebrovascular disease.

Although obstructive sleep apnea does not occur more commonly in patients with **chronic obstructive pulmonary disease (COPD)**, patients with both COPD and OSA (termed the overlap syndrome) may present with hypercarbia, polycythemia, and corpulmonale at an earlier point in their disease (i.e. FEV1 > 1 liter) than if they had COPD alone. A patient with significant hypercarbia and an FEV > 1 liter should prompt a search for a concomitant disorder such as OSA or obesity hypoventilation.

Although a complete medical examination is important in the evaluation of patients for sleep apnea, certain key aspects of the examination should get special attention; specifically, weight (or BMI), blood pressure, nose, and oropharynx. It is important to note whether the nasal passages are patent or obstructed by polyps, swollen turbinates, or boggy mucosa. Snoring and obstructive sleep apnea can be created in normal non-apneic patients by plugging the nose. Visualization of the palate, uvula, tonsils, and lateral pharyngeal walls is helpful in understanding what factors may be affecting an individual's breathing during sleep.

Once the clinical history suggestive of obstructive sleep apnea is obtained and physical examination performed, it is appropriate to consider an overnight sleep study. Full polysomnography (16 channels or more) yields the most information regarding sleep architecture, respiratory events, associated arrhythmias, oxygen saturation, and concomitant sleep disorders. It currently remains the gold standard to evaluate sleep disorders. Portable 4-channel studies are helpful in confirming a diagnosis of OSA. However, more subtle respiratory events associated with sleep fragmentation rather than oxygen saturation may be underestimated because sleep is not monitored. Similarly, portable respiratory studies are inadequate to evaluate a general complaint of excessive sleepiness which may be due to other causes such as periodic limb movement disorder. More complicated multichannel home monitors may prove useful in the assessment of OSA. Lastly, night-to-night variability in the frequency of respiratory events has been described in patients with OSA. Therefore, even a single "negative" polysomnogram may not rule out OSA in cases of high clinical suspicion.

Treatment involves behavioral interventions in conjunction with medical, dental or surgical interventions. Obesity, alcohol, tobacco, and sleep deprivation have all been shown to exacerbate OSA. Therefore, behavioral intervention should be aimed at weight loss, reducing evening alcohol consumption, tobacco cessation and avoiding sleep deprivation. Avoiding the supine position in bed may also be helpful for some patients.

Positive airway pressure is the most effective intervention for OSA.[9] This is most often delivered in the form of **continuous positive airway pressure (CPAP)** which applies positive pressure throughout the upper airway preventing collapse (Figure 1B). The patient wears a mask over the nose (or nose and mouth) which is attached via tubing to a "blower" and in-line humidifier. Usually a second polysomogram is performed to titrate CPAP to the optimal pressure which eliminates snoring and obstructive events. Once the best pressure is determined, CPAP is set up at the patient's home by a medical equipment company with whom the patient's insurance company has a contract.

The array of new masks and the development of heated humidification have made CPAP much more user-friendly. If a patient feels uncomfortable exhaling against CPAP, bi-level positive airway pressure may be tried. Patients with severe COPD and hypercarbia may feel more comfortable with an expiratory pressure set 4 to 5 cm lower than the inspiratory pressure

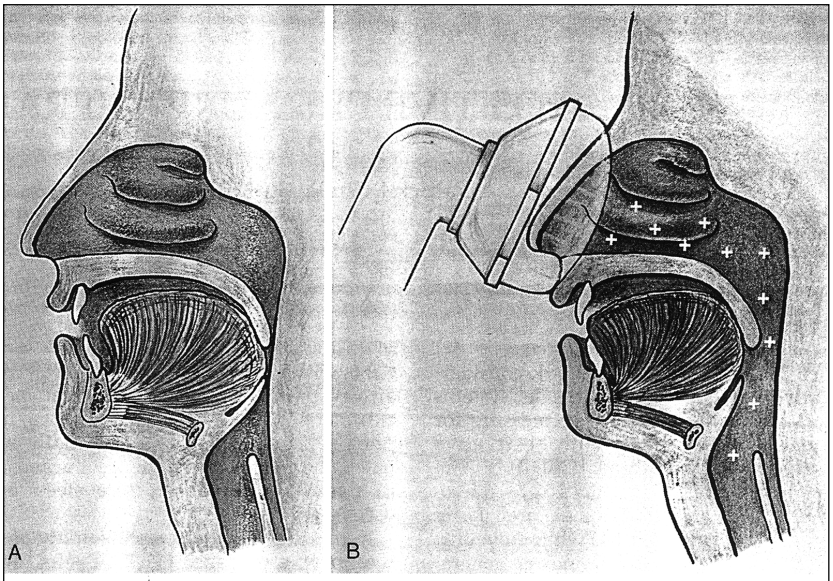

Figure 1. *A,* The sites of airway closure during sleep. Note that the soft palate and the base of the tongue are involved. *B,* Pressurization of the upper airway with continuous positive airway pressure (CPAP). Note that the soft palate moves against the base of the tongue to seal the upper airway and as a result reduce the possibility of a mouth leak. (©1997, American Sleep Disorders Association, reprinted by permission.)

rather than having a continuous pressure. CPAP is extremely effective for most patients. However, compliance is in the 50% to 70% range. This is not significantly different from compliance with other pulmonary treatments.[10] With the addition of new masks, new pressure settings, and heated humidification, compliance will hopefully improve.

Dental appliances work by moving the lower jaw and hence the tongue forward away from the palate and posterior wall of the pharynx. Eveloff et al[11] found that it also elevates the palate. The overall efficacy of a dental appliance for mild to moderate OSA is approximately 60%. The better appliances are adjustable so that the position of the jaw may be adjusted according to tolerance and symptoms. A follow-up sleep study with the dental appliance in place is necessary to document adequate control of OSA. Severe OSA is not likely to be controlled with a dental appliance alone.

Surgical options for OSA include traditional **uvulopalato-pharyngoplasty (UPPP)** alone or in conjunction with procedures to move the lower jaw forward.[12] Tracheostomy is extremely effective but rarely offered now because of its cosmetic effects and associated complications and because CPAP is so effective. The overall efficacy of UPPP is approximately 50%. Laser uvuloplasty (which removes less tissue) should be considered only for snoring not OSA. For patients with OSA who demonstrate narrowing posterior to the tongue, other procedures such as the inferior sagittal mandibular osteotomy and genioglossal advancement with hyoid myotomy and suspension (GAHM) may be considered. Bi-maxillary mandibular advancement (LeForte 1 procedure) has also been done for obstructive sleep apnea. The Stanford group[12] has studied this extensively and has found a high success rate. However, lower success rates have been published from other centers. This more invasive procedure is usually reserved for those who fail UPPP or GAHM or have significant craniofacial abnormalities. It should be performed in centers experienced with this operation.

Because the overall efficacy of the standard uvulopalatopharyngoplasty is not high, all OSA patients who undergo surgery should have a follow-up sleep study approximately three months after surgery to reevaluate the degree of residual sleep apnea. They may show some improvement in symptoms and snoring with continued underlying significant sleep apnea.

The reason to treat OSA is to alleviate symptoms and to decrease the associated morbidity and mortality. Excessive sleepiness, impaired concentration, neurocognitive function and mood have all been shown to improve with CPAP treatment of obstructive sleep apnea.[13] Similarly, Findlay et al[14] has shown that performance on driving simulator tests significantly improves after CPAP is initiated.

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Reduction in blood pressure has been demonstrated in hypertensive patients following treatment of OSA with CPAP. Similarly, mortality data from He, et al[15] showed that for patients with severe OSA, both CPAP and tracheostomy, but not UPPP were associated with improved survival compared to no treatment. Partinen, et al[16] demonstrated that patients with OSA successfully treated (by tracheostomy) had fewer cardiovascular events than those who were conservatively treated (weight loss recommendation).

Office follow-up for patients with sleep apnea following treatment should include questions again regarding residual snoring, witnessed pauses, excessive daytime sleepiness, sleepy driving, mood and neurocognitive function. If the patient is using CPAP or a dental appliance, it is important to ascertain how many nights per week and how many hours per night they are using it. Nasal symptoms may limit CPAP use. Therefore, specific questions regarding nasal congestion and coryza need to be asked. Symptoms may improve with use of topical nasal steroids or oral antihistamines. The use of an in-line heated humidifier with CPAP significantly increases moisture delivery to the upper airway and decreases nasal irritation and symptoms. This is especially important in New England where indoor heating dries out the air.

Other questions that are important in follow-up for patients on CPAP regard comfort with their mask and skin integrity. Pressure points may be alleviated with small pads or cushions. A dry mouth in the morning may point to air leaking through the mouth, which a chin strap may ameliorate. Mask and head straps do wear out and need to be replaced periodically. Patients' use or tolerance of the machine may decrease as the materials wear. They may be more comfortable with new equipment. Lastly, if the patient redevelops symptoms of excessive sleepiness or snoring or has a significant weight change while on CPAP, it would be reasonable to reevaluate the optimal pressure with a repeat sleep study.

In summary, OSA is a common disorder with significant morbidity and mortality. The morbidity relates to the sleepiness and associated automobile accidents, associated cardiovascular diseases, and neurocognitive and personality changes. In the review of systems, the primary care physician can easily screen for this treatable, but often overlooked, disorder. In addition, if a patient complains of fatigue or snoring, a more detailed history regarding other sleep symptoms is appropriate.

If symptoms and/or associated disorders suggest OSA, it is reasonable to refer the patient to a sleep center for further evaluation. Once treatment is initiated and the patient stabilized, the primary care physician can join the subspecialist in screening for recurrence of OSA symptoms and assessing compliance with treatment.

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End of Life Issues In the Critically Ill

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The process of dying has changed drastically in the last century. In the past, doctors simply did all they could for a patient. When their treatments failed, their patients died, almost always in their homes. Currently, in the United States about 80% of people die in a healthcare facility (60% in acute care facilities),[1] despite the fact that about 90% of Americans polled say they would wish to die at home.[2] This disparity is caused by two factors. First, many people die while undergoing treatments meant to postpone death. Second, many families feel they are unable to care for a dying person or are uncomfortable having a loved one die at home. The net result is that most people will die in a hospital, or other healthcare facility, and most likely undergo high levels of medical care. The Robert Wood Johnson Foundation estimates that about 20% of Americans will die in an intensive care unit or be treated in an intensive care unit just prior to death.

Two conclusions can be drawn. First, a tremendous amount of healthcare is being delivered to dying patients. This has been reflected in several studies like that of Cher and coworkers in 1997 showing that a relatively large percentage of Medicare expenditures goes to treat patients in the last weeks of their lives.[4] Second, doctors practicing in America today must learn skills not necessary in the past. Doctors need to recognize patients who are going to die despite medical care and help decide which of the medical therapies are appropriate and which are not. They need to guide their patients through a maze of medical options in an attempt to balance preservation of life with quality of life, a daunting task. This paper will review some of the major medical, ethical, and legal issues involved in these end-of-life decisions. This paper will focus only on patients who become critically ill acutely, not those with long-term, progressive, terminal illness.

How are critically ill patients dying in hospitals?

Most Americans today are dying in healthcare facilities. Furthermore, studies have shown that the vast majority of these deaths, about 75% or more, occur only after the patient, or family, has decided to limit care.[5-7] In two landmark studies, Predergast and coworkers helped define just how patients die in ICUs. In their first study, they compared deaths in their ICU from two time periods, 1987-88 and 1992-93, to determine how often CPR was performed prior to death and how often limits were placed on care prior to death. Their data showed that the incidence of CPR in their ICU had declined from 49% to 10% and that the incidence of limiting care by withholding or withdrawing some therapy had increased from 51% to 90% of all ICU deaths.[8]

To compare their data with the rest of the country, the same investigators did a large follow-up study, a year later. They collected data from over 6,000 patient deaths occurring in 131 ICUs in 38 states over a 6 month period and analyzed the data for the incidence of various limits of care. They found that on the average only 25% of patients dying in ICUs got CPR prior to death. About 70% of patients had some restriction on care prior to death and almost 50% of patients actually had some medical therapy withheld or withdrawn prior to death.[6] It is important to note that these were deaths occurring in an ICU, a place established for the most aggressive care.

The other data to emerge from this study was the variability among ICUs. The incidence of patients dying with full aggressive measures ranged from 4% in one ICU to 79% in another. Likewise, the incidence of withdrawing medical support ranged from 0% to 79%, depending on the ICU. While the overall practice of limiting care in ICUs is common, there is tremendous variability from place to place in end-of-life care.

Who decides?

Surrogate Decision making

The vast majority of people will die with some limit of care in place, whether in or out of an ICU. Unfortunately, the patient rarely participates in these decisions. Someone else generally decides to limit a dying patient's care 60 to 70% of the time.[7,9] Only about 15 to 20% of patients have an advance directive at admission to hospital; and those advance directives are often inadequate to handle anything but the most obvious treatment decisions. Therefore, the burden of difficult decisions falls to a proxy (a legal delegation) or surrogate (a non-legal delegation). Most often, this is a family member.

The process of surrogate decision making is fraught with problems. While most would agree that family or friends are the best people to decide for the patient, several studies have shown that patients rarely discuss specific treatment options with their proxies; and surrogate decisions correlate poorly with what the patient would actually want done.[10,11] Furthermore, a study by Hare et al. showed that surrogates often valued different aspects of dying, such as pain and suffering, than the patients, who were more concerned with burdening families and amount of time left to live.[10]

Legal Issues

All fifty states recognize the legality of a patient's right to refuse medical care although there remains some controversy and confusion about specific issues. The legal issues involved in proxy decision-making can be confusing. Perhaps because it is impossible to account for the many family and social relationships that may be the source of medical surrogates, most states have few laws dealing with this issue and have purposely kept the codes vague and malleable.[12] Most states, including Rhode Island, will accept a

properly drafted written advance directive as sufficient legal guidance to limit care. Unfortunately, most advance directives or living wills are too vague, using phrases such as "terminal illness" and "little chance of recovery" that are subject to interpretation. COPD and congestive heart failure may be considered terminal illnesses by some people and not by others. In contrast, some people may consider diseases such as early stage lung cancer not eminently terminal.

Nevertheless, these directives can help prevent futile or unwanted care when no other surrogate is available. More often they are useful in family decision-making when an unconscious patient faces potentially futile care. The previously stated wishes of the patient in an advance directive can assuage guilt or uncertainty regarding end-of-life decisions. They can also be helpful when surrogates disagree as to a course of action. Since a surrogate, by definition, represents what the patient would decide if able, the advance directive can be a helpful guide.

Sometimes advance directives can spur discord - for instance, when the written directive differs from a surrogate's decision. In most states including Rhode Island, the law recognizes a properly drafted and witnessed directive as the legal opinion that should be followed; however, many physicians would be wary of ignoring the requests of a living surrogate, especially if it is a spouse or other close family member. In such situations, attempts should be made to build consensus among all parties prior to making any decision. Most state laws regarding written advance directives also allow for some flexibility in the physician's obligation to follow them. For example, if a physician questions the validity of the directive or feels ethically unable to follow the directive, in most states the directive will not be binding.

Predicting Outcomes

A central problem complicating end-of-life decisions is the difficulty of predicting outcomes in critically ill patients. The combination of multiple coinciding medical problems and rapidly changing clinical status can make this a very difficult task. Essentially the physician has three tools: published outcomes, severity scores, and personal experience. All can be helpful yet all have limitations.

Perhaps the most glaring problem of severity scores is that they say nothing about morbidity, disability, or survival after hospitalization.

Severity Scores

Severity Scores have been available for almost three decades. In most severity score algorithms data are collected during the first twenty-four hours of admission and used to compile a score that, theoretically, predicts risk of death during hospitalization. These scoring systems were developed by reviewing data from thousand of ICU patients and employing logistical regression models to choose some important input variables. Other variables were simply chosen based on presumed clinical value. These scores were then validated prospectively on patients.

Unfortunately, there are several problems with these systems. First, these scoring systems make predictions based on hospital outcomes at the time of their creation. As medical treatments improve, the scores need to be updated. In the 1970s, for example, ARDS had a mortality approaching 80%; thus the diagnosis might justifiably increase a patient's severity score. Today ARDS has about a 40% mortality; thus a severity scoring system employing the diagnosis of ARDS, or even components of the diagnosis such as hypoxemia, would need to be adjusted. Some commercially available proprietary severity scoring systems such as APACHE III® are updated and revalidated on a regular basis to avoid this problem but many widely in use today, such as APACHE II, are based on patient data collected as long as two decades ago.

Also, most models derive their predictions from factors present at or shortly after admission to the ICU, and do not provide updated mortality estimates as the patient's condition changes. Furthermore, severity scores often give intermediate mortality estimates such as 60% instead of clear yes or no answers. Even these numbers are subject to confidence intervals. Perhaps the most glaring problem of severity scores is that they say nothing about morbidity, disability, or survival after hospitalization. These factors are often just as important as risk of death in making end-of-life decisions. A patient may accept a 30% chance of survival if it were followed by a high quality of life, while not accepting a 70% chance of survival if it were likely to entail a poor quality of life.

Outcomes Research

Many of the same problems encountered with severity scores apply to outcomes data. While published outcomes studies remain an essential tool for helping clinicians predict a course of illness, they suffer from two major problems.

First, the population studied for a particular illness may not share the same characteristics as your particular patient. In a recent large multicenter clinical trial of a new therapy for sepsis, the mortality in the control (untreated) population was 31%.[13] It is important to note, however, that this trial excluded patients with renal failure, liver failure, pancreatitis, AIDS, and variety of other co-morbid conditions, thus limiting the usefulness of these data for prognostic purposes.

Second, therapies can change and improve rapidly. In a series of four published studies by different authors between 1981 to 2000 examining the mortality of *pneumocystis carinii* pneumonia in ICU patients, the mortality decreased from 86% to approximately 50%.[14-17] Similar changes in outcome over time have been reported with a variety of other illnesses such as ARDS as treatments have improved.

Patient Autonomy vs. Medical Paternalism

A central problem to the end-of-life decision making-process is defining the role of the physician. Usually the physician is a combination of educator and advisor, but this is not always the case. In the past physicians were more likely to dictate courses of action or treatment plans for their patients, a concept referred to as medical paternalism. In many parts of the world to this day, medical decisions are made this way, with little input from the patient or family. In these cultures patients are comfortable with this kind of decision-making. More recently in the United States, the concept of patient autonomy has dictated medical decision-making. In its extreme form, patient autonomy holds that the physician's role is to educate the patient about the problem and offer plausible treatment plans, with their risks and benefits. The patient would then independently choose a course of action. Many physicians use this model of practice today, or a variant of it, feeling that it empowers patients, freeing them from physician bias.

In contrast to this philosophy, many physicians and patients feel the physician is obliged to offer a recommend course of action. While the discrepancies outlined here may not be of great significance in deciding whether to choose one medication over another, they take on tremendous significance when the decision is life or death. Ultimately, each physician must determine the degree of involvement he or she feels is warranted in end-of-life decisions.

Conclusion

In summary, the process of dying in America is changing rapidly. While the physician has always had an important role in the dying process, that role has now changed. Today's physician must not only be adept at administering comfort measures, he or she must decide when to initiate those measures over other therapies aimed at restoring health. Because the dying process now involves the healthcare system more and more, physicians need to have good end-of-life skills more than ever. Failure to address these issues will result in patients getting more potentially futile care at the expense of their own comfort and increasing costs to the healthcare system.

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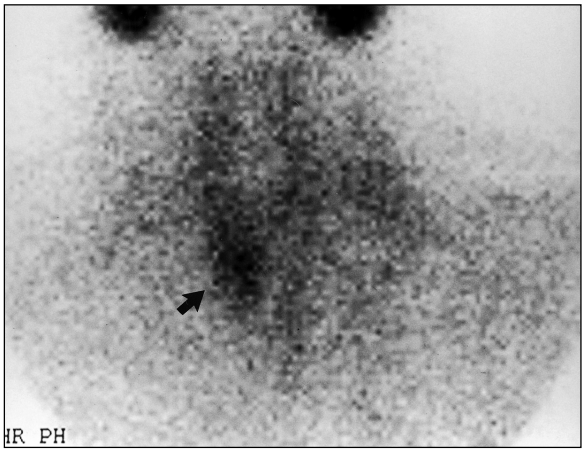

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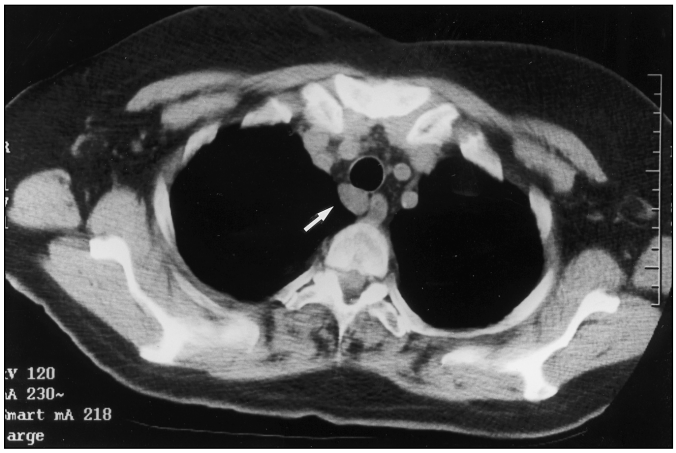

Figure 2.

Retrotracheal Parathyroid Adenoma

A 70 year-old female had elevated serum calcium on routine biochemistry profile. Her parathyroid hormone level was subsequently found to be in the 200-300 mg % range (normal up to 72mg %). As part of the diagnostic work-up, a parathyroid scan was performed using Technetium-99m sestamibi, which showed a persistent focus of abnormal increased activity posterior, inferior, and medial to the right lobe of the thyroid gland [Figure 1]. The neck ultrasound was normal. Computed tomography of the neck was performed [Figure 2], which demonstrated a 1 cm mass (arrow) posterior to the right aspect of the trachea, corresponding to the finding on the parathyroid scan. The mass was resected and proved to be a parathyroid adenoma.

Approximately 3% of parathyroid adenomas are located ectopically within the mediastinum. Preoperative localization reduces the morbidity and surgical exploration time when the adenoma is in an ectopic location. Most studies show a sensitivity for detection of parathyroid adenomas of 90% using technetium 99m-sestamibi, with less sensitivity for MRI, CT scan, or ultrasound. In this case, the retrotracheal position of the adenoma obscured its visualization on ultrasound.

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Images in Medicine: We encourage submission to the Images in Medicine section from all medical disciplines. Image(s) should capture the essence of how a diagnosis is established, and include a brief discussion of the disease process. The manuscript should be less than 250 words and include one reference. The manuscript and one or two cropped 5 by 7 inch prints should be submitted with the author's name, degree, institution and e-mail address to: John Pezzullo, MD, Department of Radiology, Rhode Island Hospital, 593 Eddy St., Providence, RI 02903. An electronic version of the text should be sent to the editor at jpezzullo@lifespan.org.

Rhode Island Quality Partners, Inc.

Update On Treatment For Congestive Heart Failure

Andrew Sucov, MD

Background

Congestive Heart Failure (CHF) is a common diagnosis in the United States, with approximately 1 million hospital admissions and 40,000 deaths yearly attributable to it.[1] In Rhode Island, the impact of CHF is also large - approximately 3,500 admissions and 80 deaths annually (personal communication from RI Department of Health). In late 1994, the **Agency for Health Care Policy and Research (AHCPR - now AHRQ)** released a guideline for management of CHF, which was updated in 1999. This review will predominantly focus on two treatment modalities - the use of ACE inhibitors and spironolactone. Other common treatments will be summarized at the conclusion of the review.

Pathophysiology

CHF is the end result of myocardial damage or overload, usually as a result of atherosclerotic cardiovascular disease. The heart is not capable of keeping up with the body's demand for oxygenated blood, leading to neurohumoral activation throughout the body, most notably an increase in adrenergic tone and stimulation of the **renin-angiotensin-aldosterone (RAA)** system. These responses, when kept in balance, enable the heart to function further along the pressure-volume (Starling) curve and maintain cardiac output; but when they become out of balance, serve to put additional stress on the heart and overload the body with fluid. Chronic management seeks to rebalance the physiologic changes and enable the heart to perform, without producing systemic side-effects, along with preservation or even improvement of cardiac function.

ACE inhibitors

ACE inhibitors have reproducibly been shown to reduce mortality and reduce progression of disease, especially in patients with higher **New York Heart Association (NYHA)** levels of disease severity.[1,2] As a result of their efficacy and safety, the guideline and major textbooks recommend them as standard treatment for virtually all patients with CHF, especially those with systolic dysfunction (LVEF < 40%). ACE inhibitors function via two different pathways - vasodilation and blocking renin-angiotensin-aldosterone. In acute management, ACE inhibitors function primarily as vasodilators, improving cardiac output.[3] On a chronic basis, their role is more attributable to local moderation of renin-angiotensin-aldosterone levels.[2] These help limit vasoconstriction and water retention. Given their generally well tolerated status and clear impact on mortality, current recommendations would suggest that these should be first line agents, used ahead of diuretics, in patients of any functional class. While patients may symptomatically improve at low doses, higher doses have been shown to reduce mortality and patients should be titrated to these levels when possible (captopril 150 mg/d, enalapril and lisinopril 20 mg/d).[2]

Up to 10% of patients may have contraindications to ACE inhibitors. A new class of agents, the **angiotensin receptor blockers (ARB)**, seems to avoid the angioedema and cough side effects. While it would appear that these agents should have similar impacts on CHF morbidity and mortality as ACE inhibitors, the literature to date does not support a mortality benefit in CHF patients.[4] Until literature supports a mortality benefit, the ARB should remain second line. Another second-line alternative for patients with ACE inhibitor contraindications is the combination of hydralizine and nitrates.[2]

Spironolactone

Spironolactone is an aldosterone antagonist and a weak diuretic on its own. In combination with either other diuretics or ACE inhibitors its effect on volume status may be quite significant. The benefits of spironolactone are two-fold - it does not have the same negative effects on electrolytes as the most commonly used diuretics, and as aldosterone is an essential component of the neurohumoral response to CHF, use of spironolactone makes mechanistic sense to combat the deterioration in function and mortality.[5] The major concern for increased use of spironolactone is on potassium levels, as both ACE inhibitors and spironolactone may elevate the levels. Close monitoring should accompany any switch in diuretic medication. A recent report suggests that addition of spironolactone to standard treatment (ACE inhibitors, beta blockers and diuretics) led to reduced mortality and hospitalization in patients with NYHA class III or IV CHF.[6] As this is only a single well-performed study, its results can't be seen as conclusive for all patients. Regardless, the original guidelines suggest using spironolactone in patients with NYHA class IV CHF, further supported by this study. Additional studies may extend these results to patients of less severe dysfunction.

Other treatment options

For patients in NYHA class II or III failure, beta blocker (beta-1 selective or mixed alpha and beta blocker) use is considered to be first line, along with ACE inhibitors.[2] They appear to have a greater effect on mortality than ACE inhibitors, likely because of effects on neurohumoral status, arrhythmia supression and reversal of pathophysiologic cardiac remodeling (carvedilol may also increase LVEF).[7] Contraindications include advanced AV block, MNA class IV failure and significant reactive airway disease.

Traditional diuretics have powerful effects on volume status, but no evidence suggests a mortality benefit. There is a significant negative effect on potassium and magnesium, which may predispose patients with CHF to arrhythmias. Routine use should be

limited to patients with fluid retention, and typically in combination with ACE inhibitors and beta blockers.[2]

Digoxin use has declined in the past few decades. No evidence suggests a mortality benefit in CHF. Electrolyte imbalances and toxicity are significant concerns, especially when combined with diuretics. Digoxin may be useful in patients who are unresponsive to ACE inhibitors and beta blockers or those with atrial fibrillation requiring rate control.[2]

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– A Physician's Lexicon –

A Radical Perspective on Words

The radiologist, enjoying the radiant sunshine of a glorious spring morning, interrupted his lunch of irradiated radishes to examine the forearm of a political radical thought to have sustained a limb fracture. One look at the X-rays, however, eradicated any doubt that the radius had indeed been fractured.

This contrived paragraph contains eight words, of widely different meaning, each a descendant of the Latin word, *radix*, meaning root.

Mathematicians preceded physicians in exploiting the word, *radix* [as well as its plural, *radices*, and its diminutive, *radicle*]. They defined *radix* [or, in English, radius] as any straight line connecting two points; more specifically, as a measurement of any linear spoke between the center and its surrounding circle. A radius then came to mean any extension from some central point spreading [or radiating] out in all directions. Early anatomists perceived the principal forearm bone, the radius, as a spoke extending from the trunk of the body to its periphery. The neuroanatomists were also not shy in expropriating *radix*. The proximal nerve roots of the spinal cord are named the radicles; and inflammatory disease of these structures, radiculitis.

Language usage over the centuries corrupted the word radix to the word, ray, confining its meaning to a beam of light extending outward from a solitary source of illumination. [But when physicists then demonstrated that there were rays other than those within the range of visible light, the meaing broadened to embrace such entities as X-rays and gamma rays.] Physicians trained in the diagnostic and therapeutic uses of these rays were called radiologists, and these emanations came to be known as radiations [and when intentionally generated, the process was called irradiation].

A shiny new fabric was synthesized by chemists during the last century. Because it glistened, they called it rayon.

As science contrives new technologies, the belabored word, *radix,* was repeatedly incorporated into many new words such as radio, radium, radioactive, radiobiology, radiopelvimetry, radectomy [the extraction of dental roots] and even radar [an acronym of Radio Detecting and Ranging].

Botanists, perhaps because they are more grounded in earthy reality, retained the original Latin meaning of *radix* ; and thus small plant roots are called radicles and a particularly pungent root-derived vegetable is called a radish.

To a mathematician, a radical is a numeral which modifies a numeric root. But to an earlier historian a radical was a person who sought out the fundamental or root meaning of things. Gradually, though, a radical came to mean an extremist, someone favoring extreme solutions to social problems. The word has now taken on a negative connotation as when Robert Frost said: "I never dared be radical when young for fear it would make me conservative when old."

– Stanley M. Aronson, MD