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NONINVASIVE VENTILATION CAN BE USED TO WEAN COPD PATIENTS OFF INVASIVE VENTILATION

A multidisciplinary approach that includes the use of noninvasive ventilation can be successful in weaning most chronic obstructive pulmonary disease (COPD) patients from prolonged invasive mechanical ventilation, researchers found.

Acute ventilatory failure is common in COPD patients requiring hospital admission, the authors note in the January 2006 issue of Chest. Despite the advent of noninvasive ventilation, invasive mechanical ventilation - which has significant mortality and often requires a difficult weaning process - is still needed by a high number of patients.

"We wanted to look at weaning success-the removal of invasive ventilation in the long term in patients that were sent to us because they had trouble weaning," Dr. Timothy G. Quinnell of Papworth Hospital in Cambridge. The researchers found a high rate of success in weaning COPD patients from invasive ventilators, a high rate of hospital survival, and a fairly long-term survival. Out of 67 patients receiving tracheostomy ventilation, 64 (95.5%) were weaned. A total of 62 patients survived to hospital discharge, with a median survival of 2.5 years.

"These results imply that the efforts we made and the resources we used were probably appropriate," Dr. Quinnell said...."We have to remember that this is a retrospective review, not a randomized control trial," Dr. Quinnell warned. "We used noninvasive ventilation as a weaning tool in selected patients, when we considered it appropriate, but we have to be careful."

.....Source: medscape.com

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SEPRACOR-ARFORMOTEROL OFFERS RELIEF

The FDA has filed Sepracor's new drug application for arformoterol inhalation solution, a long-term chronic obstructive pulmonary disease maintenance treatment. If approved, arformoterol will compete in a relatively small market, but possible advantages over rival products could help the drug assume a prominent position in the sector, with peak annual revenues of around $100 million.

Arformoterol, a single isomer of formoterol, is the first long-acting bronchodilator to be developed in an inhalation solution for use with a nebulizer. The drug candidate looks promising as results from phase III studies have shown that patients treated with arformoterol demonstrate a statistically significant improvement in FEV1, a test of lung function, versus placebo.

Bronchodilators are fundamental in symptomatic therapy for chronic obstructive pulmonary disease (COPD) and are given either on an as-needed basis for relief of persistent or worsening symptoms, or on a regular basis to prevent or reduce symptoms. In the US, two long-acting beta2-agonists are currently available, namely GlaxoSmithKline's Serevent (salmeterol) and Novartis' Foradil (formoterol), both of which are delivered using a dry powder inhaler (DPI).

If approved, Sepracor's arformoterol may offer several advantages over Serevent and Foradil. As the first long-acting bronchodilator developed for use with a nebulizer, arformoterol may be preferred over the dry powders in certain COPD patient groups.

.....Source: pharmaceutical-business-review.com

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THE SMOKING GUN: ELASTIN FRAGMENTS DRIVE EMPHYSEMA

Pulmonary emphysema is caused primarily by cigarette smoking, and the underlying cellular mechanisms are thought to involve smoke-induced activation of tissue degrading enzymes known as proteases. Elastases are proteases that specifically degrade the structural protein elastin and include enzymes such as MMP-12 (matrix metalloproteinase -12, also called macrophage metalloelastase), which is secreted by inflammatory cells called macrophages.

Now, researcher A. McGarry Houghton and colleagues at Harvard Medical School in Massachusetts, report that elastases cause emphysema in mice through the generation of pro-inflammatory elastin fragments. The study appears online on February 9 in advance of print publication in the March issue of the Journal of Clinical Investigation. The

authors found that mice that had inhaled pancreatic elastase or who were exposed to cigarette smoke developed elastin fragments in their lungs, macrophage accumulation, and emphysema.

However, when the researchers blocked the activity of elastin fragments using a specific anti-elastin antibody, the macrophage numbers were reduced, and the emphysema was prevented in both models. Using cultured human monocytes (macrophage precursor cells), the authors demonstrate that elastin fragments are chemotactic, meaning that they are able to attract inflammatory cells.

The studies suggest that the degradation products of protease activity, in addition to the proteases themselves, may be promising targets for emphysema therapy.

....Source: Journal of Clinical Investigation www.jci.org

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UVA SCIENTISTS HAVE IDENTIFIED A MOLECULAR TARGET, OR RECEPTOR, FOR POTENTIAL DRUGS TO TREAT DEADLY LUNG FAILURE

Researchers at the University of Virginia Health System have identified a molecular target, or receptor, for potential drugs to treat acute respiratory distress syndrome (ARDS), a sudden and life-threatening failure of the lung. Interestingly, the receptor is in the same class that gives people their sense of sight, smell and taste (G-protein coupled receptors.)

In ARDS, patients cannot breathe on their own because fluid gets into the lungs. Essentially, the body's immune system causes lung inflammation and accumulation of fluid in the air sacs, or alveoli, leading to low blood-oxygen levels. Up to 30 percent of patients in intensive care units can die from ARDS. There is no current therapy other than general life support and putting patients on a breathing machine. If they survive, many people face long-term lung problems. Common causes of ARDS are pneumonia, septic shock, trauma, or inhaling chemicals.

The receptor identified by UVa doctors is called CXCR2. It's expressed on the endothelial cells that line the blood vessels of the lung and on inflammatory leukocytes. Using animal models, UVa doctors have found that CXCR2 attracts white blood cells called neutrophils into the lung, a key event in the early development of ARDS. CXCR2 has been characterized in the past, but the endothelial cell effects define a new role for this receptor in the body's physiology.

"We can't say yet that if you target this receptor you will stop ARDS," said Klaus Ley, M.D., Ph.D., director of the cardiovascular research center at UVa. "But it is reasonable to be hopeful and to pursue this type of research that might one day translate into clinical application." Ley is senior author on a paper describing the receptor CXCR2 in the Feb. 16, 2006 "Online First Articles" of The Journal of Clinical Investigation found on the web at http://www.jci.org.

Dr. Jörg Reutershan, M.D., an anesthesiologist from Germany doing research in Ley's lab, discovered that CXCR2 expressed on endothelial cells is involved in acute respiratory syndrome. "Our finding is that expression of this receptor is in the lung itself," Reutershan said. "Our hope is that drug companies might be able to target the lung with an aerosol, which would have the advantage of hitting receptor without compromising the entire immune system, which is always a problem. Aerosol treatment would be a great advantage."

.....Source: University of Virginia Health System

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ANTICHOLINERGIC DRUGS LINKED TO MENTAL IMPAIRMENT IN ELDERLY PEOPLE

Anticholinergic drugs may lead to mild cognitive (mental) impairment in elderly people, finds a study published online by the BMJ. These drugs are commonly used in elderly patients to treat illnesses such as irritable bowel syndrome, urinary incontinence, and Parkinson's disease, so it is important that doctors are aware of this effect, say the researchers.

They interviewed 372 elderly people without dementia about current and past illnesses and drug use. Cognitive performance was assessed and participants were monitored for up to eight years. About 10% of the people in the sample took anticholinergic drugs over an extended period. Drug users showed poorer cognitive performance compared with non-users and 80% met the criteria for mild cognitive impairment compared with 35% of non-users. However, drug users were not at increased risk of developing dementia.

Even after taking account of other known risk factors for cognitive impairment, anticholinergic drugs remained the most highly significant predictor of this condition, say the authors. Given the aim of identifying mild cognitive impairment is the early treatment of dementia, people with mild cognitive impairment due to anticholinergic drugs could be in the absurd situation of receiving pro-cholinergic drugs to counteract the effects of anticholinergic agents, say the authors. They suggest doctors assess current use of anticholinergic drugs in elderly people with mild cognitive impairment before considering treatment for dementia.

.....Source: BMJ-British Medical Journal

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OXYGEN SHORTAGE FEAR AS NHS SUPPLY GOES PRIVATE

The health of thousands of NHS patients could be at risk and one woman is reported to have died as a result of problems with the privatisation earlier this month of the NHS home oxygen supply service. The Department of Health admitted last night that the transfer to four private companies of the service which delivers supplies to patients' homes has resulted in a shortage of oxygen. Around 60,000 patients in the UK rely on deliveries of oxygen to their homes, which have been hit by the transfer of supplies to the new privately managed Home Oxygen Service at the start of this month.

The North Cumbria primary care trust could not confirm last night that the death was linked to the oxygen episode but said it was launching an investigation.

The DoH last night ascribed the shortage of oxygen to an unnecessary rush of orders from GPs and healthcare professionals apparently concerned about the reliability of supply for their patients under the new arrangements. That appears to have become a self-fulfilling prophecy. A rush to order oxygen for patients who do not yet need it led to a shortage just two weeks after the privatisation began. The DoH said it has secured assurances from the four private firms involved - Air Products, Allied Oxycare/Medigas, BOC and Linde - that those patients who need an urgent supply of oxygen will continue to receive it.

"Suppliers have taken urgent steps to remedy the situation," the spokeswoman said, adding that local pharmacy contractors have been told they can supply oxygen to patients' homes and will receive payments from the NHS. "We are aware that some difficulties have been experienced," she said. "The service has been oversubscribed. All primary care trusts have been contacted about this and asked to be aware of the situation."

Oxygen services are vital in supporting adults and children with breathing difficulties, including those with long-term medical conditions such as cystic fibrosis and emphysema. The service helps them manage their symptoms so that they can live at home rather than in a hospital. The privatisation plan was announced last summer by health minister Jane Kennedy who said patients would enjoy "round the clock access to expert advice and support in making the best use of the latest equipment".

The old system saw GPs order gas for patients through local pharmacists who would then procure gas from suppliers. The new system allows GPs to go directly to a network of gas manufacturers. Ms Kennedy said the new system would help reduce the £600m annual cost of emergency admissions for patients suffering chronic obstructive pulmonary disease which represents more than 10% of all acute admissions every year.

.....Source: guardian.co.uk

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PULMONARY REHAB IN THE UK

"The British Lung Foundation has long been campaigning for improvements in all areas of lung health and in particular for improvements in the provision of pulmonary rehabilitation and oxygen therapy. The general public, people
living with lung disease and Breathe Easy supporters have all played a valuable role in ensuring that these issues remain at the forefront of political debate at Westminster - and in the political chambers of Scotland, Wales, and N Ireland.

PULMONARY REHABILITATION CAMPAIGN

Pulmonary rehabilitation is a course of tailored exercise that helps those with a lung condition make better use of their lungs. It really helps to increase activity levels, cope with breathlessness and live a more independent life. Yet only 1.7% of COPD patients have access to pulmonary rehabilitation. The British Lung Foundation and its supporters have been fighting for pulmonary rehabilitation to be made more widely available by lobbying the NHS and Primary Care Trusts/Health Boards.

In 2002 the British Lung Foundation and British Thoracic Society conducted a survey into the availability of pulmonary rehabilitation in the UK. The results highlighted a woefully inadequate service. The Pulmonary Rehabilitation
Survey is available for download.

We are encouraging as many people as possible to write to their local MP, (MSP) Assembly member, Primary Care Trust or Health Board asking that pulmonary rehabilitation be made available - as a matter of urgency. So far the response that we have received from the public has been overwhelming - and their hard work has resulted in pulmonary rehabilitation being started in many different parts of the UK.

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The website provides 'three ways you can get involved in our campaigns right now:'

- by sending a campaign email
- by signing an on-line petition
- by voting in an on-line poll

.....Source: http://www.lunguk.org/campaigning.asp

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INHALED CORTICOSTEROIDS COPD

The use of inhaled corticosteroids for the treatment of COPD is controversial, and results of major trials are mixed.

The term chronic obstructive pulmonary disease (COPD) refers to a spectrum of chronic pulmonary diseases characterized by shortness of breath, coughing, sputum production, airflow limitation, and impaired gas exchange. COPD affects as many as 30 million people in the United States, and represents the fourth leading cause of death and our most rapidly growing public health problem. The death rate from COPD has increased by more than 20% in the past decade. The mortality rate 10 years after diagnosis is greater than 50%, and this percentage is on the rise. Cigarette smoking is by far the most important etiologic factor. Although we have made strides in the United States to decrease smoking rates, COPD prevalence and mortality are still increasing in most of the world and likely will continue to rise in response to increases in smoking, particularly by women and adolescents.

Clinical Manifestations

The preclinical course of patients with COPD is highly variable. Patients with a history of lung disease early in life may exhibit reduced pulmonary function in adulthood. Smoking in patients with a history of childhood lung disease may lead to a progressive decline in lung function. Other patients who smoke probably begin adulthood with normal lung function. Forced expiratory volume in 1 second (FEV1) usually remains within normal limits until middle age, when a rapid decline in pulmonary function ensues. Generally, this decline stops in patients who quit smoking. The clinical symptoms of COPD usually appear in patients who continue to smoke.

Patients with COPD generally present with shortness of breath, cough, and/or wheezing. Coughing usually indicates excess mucus production. The shortness of breath is a result of increased work of breathing through obstructed airways, and is usually initially associated with increasing levels of exertion. Over time, the dyspnea worsens to the point of occurring at rest. Wheezing occurs as a result of airway narrowing, mucosal edema, and retained secretions. Classically, patients with chronic pulmonary disease have been described as either “blue bloaters” or “pink puffers.” Blue bloaters have central cyanosis with secondary polycythemia and edema. Arterial blood gas (ABG) evaluation usually reveals evidence of hypoxemia (Po2, 45-55 mm Hg), carbon dioxide retention (Pco2, 50-60 mm Hg), and compensated respiratory acidosis (pH, 7.38-7.42).7 Pink puffers do not have secondary polycythemia, and edema is not present. They have less hypoxemia (Po2, 60-80 mm Hg) and no carbon dioxide retention (Pco2, 30-40 mm Hg). Many patients have features of both conditions.

General Principles of Management

There are four general principles of management of the patient with COPD:

(1) slow disease progression;

(2) prevent infection;

(3) treat reversible symptoms; and

(4) educate patients. The respiratory care practitioner is intimately involved in all four cornerstones of care.

Slow Disease Progression

If the extent of disease is not completely irreversible (end-stage), progression of disease can be slowed by smoking cessation, reduction of exposure to environmental or occupational irritants, and therapy with supplemental oxygen.

Prevent Infection

Infection is usually considered the main cause of acute exacerbation in COPD, although many COPD patients have evidence of lower respiratory tract bacterial colonization even during periods of remission. In fact, potentially pathogenic organisms can be recovered from the respiratory tract secretions of virtually all patients with COPD at some time during the course of their disease. Many bacterial and other organisms have been found in the sputum of patients with chronic pulmonary disease. Nonetheless, the role of antibiotic therapy in acute exacerbations of COPD remains controversial. Broad-spectrum antibiotic prophylaxis has not been shown to decrease the frequency of infections, but may decrease the severity and duration of symptoms. Recent evidence has shown that bacterial colonization is associated with enhanced airway inflammation and that resolution of bronchial inflammation following acute exacerbations of chronic bronchitis may be related to bacterial eradication.

Patients with COPD should be encouraged to have annual influenza vaccinations. Some authorities also recommend vaccination against pneumococcal disease.

Treat Reversible Symptoms

Pharmacotherapy with inhaled ß2-agonists and long-acting theophylline preparations may reverse the symptoms of airway obstruction. The use of inhaled corticosteroids (ICS) is reviewed later in this article.

Educate Patients

Education should be an integral part of chronic pulmonary disease managemet. Patients and their families should be given basic facts about the disease process and offered a list of resources in the event they desire additional information. Medication issues should be addressed. If appropriate, the subject of intubation and resuscitative intervention should be reviewed and the

patient's desires should be delineated.

Surgery

Surgical procedures for COPD are very rare. They are expensive and often not covered by insurance. The great majority of patients cannot be helped by surgery, and no single procedure is ideal for those who can be helped.

Lung transplantation has been successfully employed in some patients with end-stage COPD. After transplantation, pulmonary function is usually markedly improved. Postoperatively, dyspnea at rest is abolished in most patients, and dyspnea on exertion is significantly diminished. Supplemental oxygen is usually no longer required, and exercise tolerance is markedly improved. Unfortunately, lung transplantation is associated with potentially severe complications, including pulmonary edema, ventilation-perfusion abnormalities, and organ rejection. In the hands of an experienced team, the 3-year survival rate is approximately 50%.

Lung volume reduction surgery (LVRS) removes 20% to 30% of severely diseased lung tissue; the remaining parts of the lung are joined together. Mortality rates can be as high as 15% and complication rates are even higher. When the operation is successful, patients report significant improvement in symptoms.

Pulmonary Rehabilitation

A structured, outpatient pulmonary rehabilitation program improves functional capacity in certain patients with COPD. Services may include general exercise training, administration of oxygen and nutritional supplements, intermittent mechanical ventilatory support, continuous positive airway pressure (CPAP), relaxation techniques, breathing exercises and techniques (such as pursed lip breathing), and methods for mobilizing and removing secretions.

Use of Inhaled Corticosteroids

ICS are used for long-term maintenance treatment in COPD, but the efficacy of these agents is controversial. There are at least two possible reasons why COPD patients might respond to anti-inflammatory treatment, despite the general acknowledgment that the loss of lung tissue elasticity is relatively fixed. Some asthma patients may be misdiagnosed as COPD. The differentiation of severe COPD from chronic severe asthma can be difficult because some degree of reversibility (shown as improvement in FEV1) can be achieved in the majority of patients. The pathological changes of bronchial asthma in the large airways can coexist with those of COPD, which predominantly affects the small airways. Patients with COPD who respond to corticosteroids may have a degree of inflammation, which may be component specific to the disease.

While the presence of inflammatory changes in the airways of patients with COPD provides a rationale for the use of corticosteroids, the association between these changes, lung function, and the therapeutic response to corticosteroids has not yet been established clearly. Several uncontrolled retrospective studies performed in the 1980s suggested that long-term treatment with oral corticosteroids might slow the decline in FEV1 in patients with COPD. Long-term use of oral corticosteroids would not generally be recommended because of the risk of systemic adverse events, however. Inhaled corticosteroids offer an option for achieving similar benefits with fewer systemic side effects. More recent investigations evaluating the efficacy of corticosteroids in COPD have focused on inhaled agents.

Trials Have Mixed Outcomes

An early trial of ICS in COPD suggested an improvement in FEV1 and reduction in the decline in FEV1 over 1 year of treatment with inhaled beclomethasone. A 2-year study with inhaled budesonide showed significant reduction in respiratory symptoms, with a halving of the median decline in FEV1 in a group of nonallergic patients with COPD. The number of patients withdrawing due to pulmonary problems was significantly higher in the placebo group.

The ISOLDE (Inhaled Steroids in Obstructive Lung Disease in Europe) study was a UK-based, multicenter, double-blind, placebo-controlled study of fluticasone in 753 patients with moderate-to-severe COPD, with the main outcome measure being the rate of decline of postbronchodilator FEV1 over 3 years. Secondary endpoints were the frequencies of exacerbations, changes in health status, withdrawals because of respiratory disease, morning serum cortisol concentrations, and adverse events. Patients recruited had a diagnosis of COPD, were aged 40 to 75 years, and had a postbronchodilator ratio of FEV1 to FVC less than 70%. The average prebronchodilator FEV1 was 1.24 L, suggesting that the participants had severe COPD. There was no difference in the decline of respiratory function, as measured by FEV1, over the 3 years of the study in the fluticasone or placebo groups. The yearly exacerbation rate was lower in the fluticasone group than in the placebo group. Health status, measured by the increase in questionnaire score, declined at a slower rate in the fluticasone group than in the placebo group. Adverse effects were similar in each group. The only clinical benefit seen in this trial was a decrease in the frequency of exacerbations requiring oral steroid or antibiotic treatment.

The EUROSCOP (European Respiratory Society Study on Chronic Obstructive Pulmonary Disease) study compared budesonide 400 g bid, to placebo in actively

smoking subjects with mild COPD over a period of 3 years. The primary outcome was the rate of postbronchodilator decline in the FEV1. Over the first 6 months of the study, the FEV1 improved by about 10 mL in the budesonide group while it declined by about 40 mL in the placebo group. Thereafter, the rates of FEV1 decline were nearly similar in the two treatment groups. Patients receiving active treatment experienced significantly more skin bruising and adverse upper airway effects. Thus, relatively large doses of ICS given for 3 years to smokers with mild COPD were associated with some side effects and limited benefit.

A meta-analysis of five randomized, placebo-controlled trials of ICS in patients with COPD was performed to evaluate the long-term effects of these agents on the rate of FEV1 decline in patients with COPD. The use of ICS did not change the rate of FEV1 decline in 3,571 patients followed for 24 to 54 months.

Investigators have evaluated, in a randomized, double-blind, parallel-group, placebo-controlled study, whether the combination of inhaled ß2-agonists and ICS provides better pulmonary outcomes than treatment with either agent alone in patients with COPD. Subjects were treated with either salmeterol 50 g bid, fluticasone 500 mg bid, the combination of both , or placebo for 12 months. All of the active treatments improved lung function, pulmonary symptoms, and health status and reduced use of rescue medication and frequency of exacerbations. Combination therapy improved pretreatment FEV1 significantly better than did placebo, salmeterol alone, or fluticasone alone. Combination treatment also produced a clinically significant improvement in health status and the greatest reduction in daily symptoms. Based on these results, the investigators suggested that the combination of inhaled long-acting ß2-agonists and corticosteroids be considered for patients with COPD.

Since the ISOLDE results were published, a number of investigators have reported improvements in health-related quality of life in patients with moderate to advanced COPD treated with inhaled corticosteroids.The Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines recommend a trial of inhaled corticosteroids for patients with an FEV1 <50% of predicted and a clinical course characterized by significant symptoms or repeated exacerbations.

Summary

The evidence base supporting efficacy for ICS in COPD is growing, but the results of major trials are still mixed, and the use of ICS in COPD is still considered controversial. The therapy of COPD should be tailored to the individual patient. The GOLD guidelines suggest using ICS in selected patients with moderate to severe disease. Some clinicians suggest targeting use of ICS to those patients who respond to a 2-week trial of these agents. .....Source: RT Magazine

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OXYGEN SHOWS PROMISE AS STROKE TREATMENT

When someone suffers a stroke, time is of the essence: There's only about a three-hour window to administer the most effective drug treatment. But now, an analysis of previous research provides more evidence that paramedics and doctors can stop the clock -- at least temporarily -- with heavy doses of oxygen. "It [the initial finding] still holds up," said study senior author Dr. Aneesh Singhal, an assistant professor of neurology at Harvard Medical School. "And theoretically, the risks are very minimal as compared to the potential benefit."

Strokes occur when blood flow through the brain is disrupted. About 700,000 Americans have strokes each year, and nearly 157,000 die, according to the American Stroke Association. Over the past decade, doctors have made major strides in stroke care, thanks to a drug treatment called tissue plasminogen activator, or tPA. If given within three hours of a stroke, the drug can break up clots that prevent proper blood flow. But it's not always possible to get a stroke victim to a hospital and run the appropriate tests in time for that deadline. Enter oxygen treatment, which could provide enough valuable oxygen to the brain to keep conditions stable for a while.

One idea was to put stroke patients in hyperbaric chambers, which force oxygen into the body through high pressure; the chambers are perhaps best known for their use as a treatment for scuba divers struggling with the bends. But the hyperbaric approach didn't work in stroke patients, Singhal said. So, he and other researchers tried a different approach, using simple facemasks to deliver heavy doses of oxygen. They used MRI brain scans to track 19 stroke patients, 11 of whom received special treatment with oxygen and eight who only breathed room air.

The researchers found that oxygen treatment appeared to significantly reduce damage from stroke both four hours and 24 hours after an attack. The findings were first reported in the journal Stroke in October 2005. Singhal and his colleagues re-examined the existing research using a different statistical analysis and released their latest findings Friday at the American Stroke Association's annual conference, in Kissimmee, Fla.

Their conclusion: Even when viewed in a different light, the oxygen treatment seems to work. Oxygen therapy for stroke patients might serve to protect them as they wait for full treatment in a hospital, Singhal said. And

since the treatment is simple, paramedics could administer it in an ambulance. Why does oxygen work? Singhal said researchers aren't sure about the exact process, but it likely has something to do with bringing oxygen to brain tissues that aren't getting enough of it because of disruption in blood flow.

Dr. Argye Hillis, associate professor of neurology at Johns Hopkins University, said the findings are promising, although the number of participants was small. "It certainly is a cheap intervention," Hillis said, adding that there's "very little risk" from getting oxygen for a short period of time. If further research confirms that the treatment works, there could be major benefits by extending the time window for treating stroke, she said. "If we could extend the time window to four hours instead of three hours, we might be able to treat a lot more patients." .....Source: 2006 ScoutNews LLC

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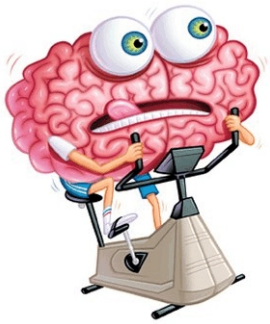
BUFF AND BRAINY

Exercising the body can benefit the mind

Anyone who frequents the local gym has probably noticed a cyclical pattern to attendance. Workout kings and queens exercise religiously throughout the year, but as swimsuit season approaches, a rash of new faces flocks to the facility. Every treadmill is taken, each elliptical machine is engaged, and without fail, there's a waiting line for a weight machine. Just as physical exercise primes the body, researchers are finding that it also primes the mind. Exercise prompts brain cells to multiply, strengthens their connections, and boosts their resilience against damage and disease.



While exercise may be the path to looking gr in a two-piece, everyone knows that it's also heal for the body. It strengthens the heart and lungs, shores up thinning bones, and wards off a host of evils, including diabetes, heart disease, and stroke. But what these newly inaugurated gym rats probably don't know is that besides buffing up their bodies for summer, they're also buffing up their brains. New research suggests that physical exercise encourages healthy brains to function at their optimum levels. Fitness prompts nerve cells to multiply, strengthens their connections, and protects them from harm. Benefits seem to extend to brains and nerves that are diseased or damaged. These findings could suggest new treatments for people with Alzheimer's disease, Parkinson's disease, and spinal cord injuries.

Sweating to the oldies

The cliché about a healthy mind residing in a healthy body has ancient roots. The famous quote of the same meaning, "mens sana in corpore sano" came from the Roman writer Juvenal in the early 100s A.D. And a century earlier, the philosopher Seneca was prescribing exercise as a way to achieve both physical and mental health. But it wasn't until the early 1950s that reports that exercise conveys neurological benefits appeared in the scientific literature. These articles usually described what doctors had witnessed in their own practices, says neurobiologist Fernando Gómez-Pinilla of the University of California, Los Angeles. "This clinical literature described that exercise could be good for many different things," he says. The studies cited benefits ranging from alleviating depression and pain to regaining mobility in paralyzed limbs to maintaining good memory in old age.

However, for scientists who research how nerve cells work at a molecular level, such reports raise a bevy of questions. Gómez-Pinilla and other neurobiologists have aimed to fill this information gap by working with lab animals such as mice and rats—creatures that can be easily manipulated to sort out each one of an experiment's variables and that, unlike people, can be dissected in the end to get an insider's view of the brain. By the mid-1990s, researchers began to get answers. Preliminary studies indicated that when lab animals exercise, their nerve cells release chemicals called neurotrophic factors. These proteins buffer nerve cells against illness or injury, prompt them to grow and multiply, and strengthen each neuron's connection with other nerve cells.

Out of the variety of neurotrophic factors released during exercise, however, scientists found that one in particular stood out: brain-derived neurotrophic factor, or BDNF. This protein seems to act as a ringleader, both prompting brain benefits on its own and triggering a cascade of other neural health–promoting chemicals to spring into action. "I think of BDNF as brain fertilizer. It's thrilling to see what it does to cells in culture," says Carl Cotman, a neuroscientist at the University of California, Irvine. Sprinkling a dilute solution of BDNF onto neurons in a lab dish makes the cells "grow like crazy," he adds. The cells sprout branches prolifically and extend them rapidly.

Let's get physical

Knowing what BDNF can do to neurons in the lab, researchers wondered whether the BDNF that exercising animals produce has similar effects on neurons in their brains. If so, could these physical effects translate into behavioral ones, making the animals learn quicker and better? In 1999, Fred H. Gage of the Salk Institute in La

Jolla, Calif., and his colleagues, including Salk's Henriette Van Praag, began exploring these questions. They studied two groups of healthy mice housed individually in cages that were identical except for one detail: One group of mice had running wheels. "The mice just love [the wheel]. They run on it as soon as you put it in their cages," says Van Praag. "If you let them run as much as they want, they run all night long." Over the next several weeks, the researchers kept track as the runners voluntarily racked up an average of 4 to 5 kilometers on their wheels every night. The scientists then tested whether the groups differed in how quickly each mouse solved a popular learning test known as the Morris water maze. Although both groups of mice swam at about the same speed, Gage and his colleagues noticed that the runners learned the location of a platform hidden under the maze's opaque water significantly sooner than their less-fit counterparts did.

Dissections showed that the runners had about twice as many new brain neurons as the sedentary mice did. When the researchers tested individual neurons isolated from both groups, they discovered that neurons taken from the runners showed greater signs of strengthened connections and cellular learning.

In a related study published in 2004, Gage's team teased out the molecular factors responsible for the behavioral effects that come with exercise. The researchers provided a group of rats with running wheels and compared them with rats without access to the wheels. On average, the runners voluntarily racked up an astounding 48 km per day over the next several weeks. When they dissected the rats' brains, Gage's team found changes similar to those that they'd seen in the previous study's mice: The runners had more new neurons and stronger connectivity, which is evidence of learning, than did the rats that didn't have running wheels. After examining the messenger RNA of both groups, an indicator of gene expression, the researchers found that the running rats had consistently higher activity in the gene that codes for BDNF than the nonrunners did.

Gómez-Pinilla and his colleagues added more evidence that BDNF is a primary source for the behavioral benefits of exercise. Like Gage's group, Gómez-Pinilla's team worked with rats that were either sedentary or had access to a running wheel. After a week, some members of each group began receiving daily injections of a drug that blocked the action of BDNF. The rest of the animals were injected daily for several days with a chemical called cytochrome-C, which isn't known to cause any physical or behavioral effects. The researchers then tested all the animals on the Morris water maze. While runners receiving cytochrome-C excelled at the test, runners that received the chemical that blocked BDNF performed only as well as the sedentary mice did. Performance by the nonrunners was about the same, regardless of which injection they received. "If we block the action of BDNF, we block learning and memory," concludes Gómez-Pinilla.

Keep on moving

With mounting evidence of what exercise and its associated BDNF can do for healthy animals, researchers speculated that a similar mechanism could benefit animals and people stricken with neurological disease or injury. For example, in the April 27, 2005 Journal of Neuroscience, Cotman and his colleagues suggested that exercise could slow the progression of Alzheimer's disease. In the study, Cotman's team worked with mice that were genetically predisposed to develop an Alzheimer's-like disease. When they're a few weeks old—that's young adulthood in mice—the rodents' brains start accumulating a protein known as beta-amyloid. In the brains of people with Alzheimer's, this protein surges to form thick plaques that are one of the hallmarks of the disease. As in other exercise-related studies, Cotman housed Alzheimer's-prone mice individually in cages, some of which were equipped with running wheels. At the start of the experiment, the animals were around 1 month old. Alzheimer's-like symptoms "had barely started by then," says Cotman.

After 5 months, the researchers tested the animals in the Morris water maze. As in the earlier studies, the exercisers fared significantly better on that memory test than the sedimentary mice did. However, in the "really exciting" part of the study, says Cotman, he and his colleagues dissected the animals' brains at 6 months of age to measure the beta-amyloid. They were surprised to find about half as much accumulation of the substance in the runners as in the nonrunners. Cotman says that his team hasn't figured out how exercise reduces the buildup of amyloid-beta. But regardless of the mechanism, he notes that his results suggest that physical activity could eventually fight early Alzheimer's disease.

Exercise also shows promise in preventing Parkinson's-like symptoms from developing in animal models of that disease. Surveys of lifestyle and health have suggested that people who exercise moderately, such as walking an hour each day, are less likely than others to develop Parkinson's disease. For the past 5 years, Michael Zigmond of the University of Pittsburgh and his colleagues have been experimenting with rats to explain this preventive effect.

In one study, the researchers forced healthy rats to exercise on a treadmill daily for a week. They then injected the animals with a chemical called 6-hydroxydopamine, which selectively kills dopamine-producing neurons. These cells also die in Parkinson's disease patients. After several days, Zigmond's team examined the animals' brains. Compared with rats that received 6-hydroxydopamine but hadn't worked out on the treadmill, the exercisers lost fewer dopamine-producing neurons. Earlier studies had suggested that a protein called glial cell–derived neurotrophic factor (GDNF) protects dopamine-producing neurons in patients with Parkinson's disease and that neurons produce GDNF, just as they do BDNF, in response to exercise. So, Zigmond proposes that GDNF protected brain cells in the rats that exercised. He described his team's findings at the Society for Neuroscience meeting in October 2005 in Washington, D.C.

Researchers are also exploring physical activity as a way to treat neurological injuries, such as spinal cord damage. Although physical therapists have long helped patients regain some function by moving individual limbs affected by neurological damage, they have typically considered a patient with paralysis from the waist or neck down too damaged to rehabilitate.
"When you're told to go home and sit in a chair, your body doesn't get the feedback that comes from physical activity," says John McDonald, director of the International Center for Spinal Cord Injury at the Kennedy Krieger Institute in Baltimore. He developed exercise programs for Christopher Reeve as part of the late actor's therapy after his paralysis. Without the neurotrophic factors produced in response to physical activity, McDonald hypothesizes, the nervous system fails to establish connections between damaged neurons and grow new ones.

To alleviate this problem, McDonald and his colleagues came up with a way for people with extensive paralysis to exercise. The researchers started with exercise bikes that had been equipped with electrodes that stimulate a patient's muscles to pedal. Heavy versions of these electrical-stimulation bikes had been used in physical therapy years ago, but their expense and inconvenience had made them fall out of favor. McDonald worked with the bikes' manufacturer to design models light enough for patients to use at home. In a recent study, 24 people who had been paralyzed for an average of 5 years used the special bikes three times a week. Another 24 participants only stretched. After 2 years, 40 percent of the exercisers had regained some motor function, compared with only 4 percent of the other patients. More research could eventually boost the bikes and similar assisted-movement equipment to become standard therapy for spinal cord injuries, says McDonald.

Long may you run

While evidence is soaring for exercise's brain benefits, physical fitness in the United States is plummeting. According to a report issued recently by the Centers for Disease Control and Prevention, almost one-fifth of people 18 and over exercise for less than 10 minutes a week. Only 46 percent of adults performed the recommended 30 minutes or more of brisk walking or other moderate exercise 5 days a week.

Whereas public health experts worry about the effects of a sedentary lifestyle on rates of heart disease, diabetes, and other health problems, Gómez-Pinilla is concerned that a lack of physical exercise could also foretell a wave of decreasing brain health for the United States. "Locomotion played a very important role in evolution. Animals had to move to find food and run away from predators. Exercise had a direct action on brain regions related to cognition," he says. "Normally, when two functions evolve in this way, you can't separate them."

But it's never too late to pick up an exercise program, according to psychologist David Albeck of the University of Colorado at Denver. Sedentary middle-aged and elderly rats placed on a walking program showed improvements in learning and memory, compared with sedentary rats of the same age, Albeck's team reported at the 2005 Society for Neuroscience meeting. Furthermore, memory tests given to 1,740 people over 65 during a 6-year project have linked moderate exercise to reduced risk of dementia. These results were published in the Jan. 17 Annals of Internal Medicine by a Seattle research team.

That's yet another piece of good news for the pre-swimsuit season rush of exercisers that will appear, like clockwork, at gyms across the country. For these new gym rats, starting to exercise is a smart move, says Zigmond, but the smartest move will be to stick with exercising for years to come. "If somebody were to stop me in the street and ask me what to do, I wouldn't have any problem telling them to exercise," he says. "There are lots of reasons to exercise, and virtually no reasons not to."

Source: Science News, 169:8

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EXERCISE/KEEPING BUSY

'Physical activity reduces the risk of depression, and has positive benefits for mental health including reducing anxiety, and enhancing self-esteem. It can help you relax, sleep well and cope with stress. It can help you

feel and look better' - NHS steps to health scheme

Many people find that physical exercise can be of value in helping to maintain good mental health. Those who suffer from depression, often resulting from some kind of social anxiety, sometimes find some relief by taking some brisk exercise, while those who are in the recovery process often find that exercise can help to speed it up. As well as having all round health benefits such as lowering blood pressure, maintaining good circulation and controlling weight, taking up a form of sport can be a very effective way of increasing social interaction, distracting ourselves from our anxieties and a good balance with other areas of our lives..

Anxiety in particular can be reduced significantly by a course of regular exercise as the stress reaction (i.e. the state we find ourselves in when tense and fearful at the prospect of blushing in front of others) encourages a state of high energy causing our bodies to stay in a tense state of arousal for hours at a time. Exercise can be a very effective way of dissipating this excess energy and encouraging a more otherwise relaxed and well-balanced attitude. Times when we experience insomnia are common times for worrying about future events or 'self-prophesising' about our social anxiety and blushing, and exercise can be a useful too in improving sleep and therefore reducing undue worries.

Other health benefits of exercise include the reduction of headaches, increased feeling of well-being, concentration and stamina. This is due to the chemicals called endorphins, which are released into the brain during exercise, morphine-like in their effect; they promote a sense of positivity and happiness, which can last for some time after exercising. This somewhat 'elated' mental state or 'improved mood' can help you to maintain a well-balanced attitude when faced with stressful situations where you feel you may blush by improving your overall mental health. So its well worth considering taking up a regular programme of exercise to help cope with and manage your social anxiety, blushing, and possible related depression.

The kind of physical exercise you choose to adopt is a matter for your own personal choice. However in order for exercise to be effective, it should be brisk rather than leisurely- brisk walking and cycling, even heavier types of gardening are ideal as out-door activities are particularly beneficial. At the very least, it is important to exercise three times per week for a minimum of 30 minutes each time. Other Aerobic activities like jogging, swimming, squash, football, aerobics classes and dancing are all suitable. Remember not to push yourself too hard in the beginning, to seek medical advice prior to taking up any new exercise programme and bear in mind that the body benefits more from short periods of regular exercise rather than infrequent bursts. Make sure to ease yourself into an exercise programme, as doing too much too soon could lead to physical exhaustion or injury.

You should consult your GP before embarking on a new course of exercise. .....Source: NHS, UK

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NEW SURVEY SHOWS CARDIOLOGISTS AWARE OF LIFE-SAVING DIET, YET FAILING TO RECOMMEND IT

Wider use of vegetarian diet would result in fewer surgeries and deaths from heart disease; Studies show patients transition easily to new diet

A pilot survey of cardiologists reveals that most know about the life-saving potential of a truly low-fat vegetarian diet for heart patients, but fail to recommend the diet in the mistaken belief that patients will not comply. Published studies actually show that patients transition fairly easily to a low-fat diet that contains no animal products, and most rate this diet as "good" or "extremely good." If cardiologists' knowledge of the acceptability of the vegetarian diet were equal to their familiarity with its efficacy, the result would be improved patient care and fewer deaths.

Instead, most cardiologists responding to the survey recommend the standard omnivorous low-fat (up to 30 percent of calories from fat) diet, which recently made headlines for its role in the Women's Health Initiative study. Omnivorous low-fat diets have not proven effective for treating or preventing heart disease. To experience dramatic improvement, heart patients must consume a diet that contains less than 15 percent of calories from fat and that excludes saturated fat from animal products.

"Patients hospitalized with life-threatening cardiac conditions should be advised by their doctor that they could head off another heart attack by switching to a low-fat vegetarian diet," says report co-author Amy Joy Lanou, Ph.D., a senior nutrition scientist with the Physicians Committee for Responsible Medicine and an assistant professor of health and wellness at the University of North Carolina." Dietary changes reinforced by a doctor's recommendation will make it even easier for patients to make simple changes that could add years to their lives." The lead author of the report is Keith Rafal, M.D., M.P.H., medical director of the Rehabilitation Hospital of Rhode Island.

Ninety-one percent of responding cardiologists were either "very familiar" or "somewhat familiar" with the research supporting very low-fat cardiac diets, the survey found. In 1990, cardiologist Dean Ornish, M.D., changed

cardiac care forever with the publication in the Lancet of a study showing arrest and even reversal of heart disease with a very low-fat vegetarian diet. Other researchers have published similar findings.

The simplicity of a vegetarian diet that excludes animal products appeals to people busy with work and family, and many familiar recipes are easy to adapt. At least four studies published in peer-reviewed journals show that patients give the low-fat vegetarian diet a high rating in terms of acceptability. ....Source: Physicians Committee for Responsible Medicine

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But then.....

LOW-FAT DIET DOES NOT CUT HEALTH RISKS, STUDY FINDS

The largest study ever to ask whether a low-fat diet reduces the risk of getting cancer or heart disease has found that the diet has no effect.

The $415 million federal study involved nearly 49,000 women ages 50 to 79 who were followed for eight years. In the end, those assigned to a low-fat diet had the same rates of breast cancer, colon cancer, heart attacks and strokes as those who ate whatever they pleased, researchers are reporting today. "These studies are revolutionary," said Dr. Jules Hirsch, physician in chief emeritus at Rockefeller University in New York City, who has spent a lifetime studying the effects of diets on weight and health. "They should put a stop to this era of thinking that we have all the information we need to change the whole national diet and make everybody healthy."

The study, published in today's issue of The Journal of the American Medical Association, was not just an ordinary study, said Dr. Michael Thun, who directs epidemiological research for the American Cancer Society. It was so large and so expensive, Dr. Thun said, that it was "the Rolls-Royce of studies." As such, he added, it is likely to be the final word. "We usually have only one shot at a very large-scale trial on a particular issue," he said.

The results, the study investigators agreed, do not justify recommending low-fat diets to the public to reduce their heart disease and cancer risk. Given the lack of benefit found in the study, many medical researchers said that the best dietary advice, for now, was to follow federal guidelines for healthy eating, with less saturated and trans fats, more grains, and more fruits and vegetables.

Not everyone was convinced. Some, like Dr. Dean Ornish, a longtime promoter of low-fat diets and president of the Preventive Medicine Research Institute in Sausalito, Calif., said that the women did not reduce their fat to low enough levels or eat enough fruits and vegetables, and that the study, even at eight years, did not give the diets enough time. Others said that diet could still make a difference, at least with heart disease, if people were to eat the so-called Mediterranean diet, low in saturated fats like butter and high in oils like olive oil. The women in the study reduced all kinds of fat. The diets studied "had an antique patina," said Dr. Peter Libby, a cardiologist and professor at Harvard Medical School. These days, Dr. Libby said, most people have moved on from the idea of controlling total fat to the idea that people should eat different kinds of fat.

But the Mediterranean diet has not been subjected to a study of this scope, researchers said. And Barbara V. Howard, an epidemiologist at MedStar Research Institute, a nonprofit hospital group, and a principle investigator in the study, said people should realize that diet alone was not enough to stay healthy. "We are not going to reverse any of the chronic diseases in this country by changing the composition of the diet," Dr. Howard said. "People are always thinking it's what they ate. They are not looking at how much they ate or that they smoke or that they are sedentary."

Except for not smoking, the advice for a healthy lifestyle is based largely on indirect evidence, Dr. Howard said, but most medical researchers agree that it makes sense to eat well, control weight and get regular exercise. That is also what the cancer society recommends. Dr. Thun, who described the study's results as "completely null over the eight-year follow-up for both cancers and heart disease," said his group had no plans to suggest that low-fat diets were going to protect against cancer. Others cautioned against being too certain that a particular diet would markedly improve health, and said that whether someone developed a chronic disease might not be entirely under their control — genetics also plays a role.

David A. Freedman, a statistician at the University of California, Berkeley, who is not connected with the study but has written books on the design and analysis of clinical trials, said the results should be taken seriously. "The studies were well designed," Dr. Freedman said, "and the investigators tried to confirm popular hypotheses about the protective effect of diet against three major diseases in women. But," he added, "the diet studied here turned out not to be protective after all."

The study was part of the Women's Health Initiative of the National Institutes of Health, the same program that showed that hormone therapy after menopause might have more risks than benefits. In this case, the study addressed

a tricky problem. For decades, many scientists have said, and many members of the public have believed, that what people eat — the composition of the diet — determines how likely they are to get a chronic disease. But that has been hard to prove. Studies of dietary fiber and colon cancer failed to find that fiber was protective, and studies of vitamins thought to protect against cancer failed to show an effect. Many cancer researchers have questioned large parts of the diet-cancer hypothesis, but it has kept a hold on the public imagination. "Nothing fascinates the American public so much as the notion that what you eat rather than how much you eat affects your health," said Dr. Libby, the Harvard professor.

The study found that women who were randomly assigned to follow a low-fat diet ate significantly less fat over the next eight years. But they had just as much breast and colon cancer and just as much heart disease. The women were not trying to lose weight, and their weights remained fairly steady. But their experiences with the diets allowed researchers to question some popular notions about diet and obesity. There is a common belief that Americans get fat because they eat too many carbohydrates. The idea is that a high-carbohydrate, low-fat diet leads to weight gain, higher insulin and blood glucose levels, and more diabetes, even if the calories are the same as in a higher-fat diet. That did not happen here. Others have said the opposite: that low-fat diets enable people to lose weight naturally. But that belief was not supported by this study.

As for heart disease risk factors, the only one affected was LDL cholesterol, which increases heart disease risk. The levels were slightly higher in women eating the higher-fat diet, but not high enough to make a noticeable difference in their risk of heart disease. Although all the study participants were women, the colon cancer and heart disease results should also apply to men, said Dr. Jacques Rossouw, the project officer for the Women's Health Initiative. Dr. Rossouw said the observational studies that led to the hypothesis about colon cancer and dietary fat included men and women. With heart disease, he said, researchers have found that women and men respond in the same way to dietary fat.

The most recent study follows a smaller one, reported last year, on low-fat diets for women who had breast cancer. That study hinted that eating less fat might help prevent a recurrence. But the current study, asking if a low-fat diet could protect women from breast cancer in the first place, had findings that fell short of statistical significance, meaning they could have occurred by chance. Dr. Rossouw said he was still intrigued by the breast cancer data, even though it was not statistically significant. The women on low-fat diets had a 9 percent lower rate of breast cancer; the incidence was 42 per thousand per year in women in the low-fat diet group, compared with 45 per thousand per year in women consuming their regular diet. That could mean that fat in the diet may have a small effect, Dr. Rossouw said, perhaps in some subgroups of women or over a longer period of time. He added that the study investigators would continue to follow the women to see if the effect became more pronounced.

While cancer researchers said they were disappointed by the results, heart disease researchers said they were not surprised that simply reducing total fat had no effect, because they had moved on from that hypothesis. Of course, Dr. Libby acknowledged, the latest advice, to follow a Mediterranean diet and get regular exercise, has never been tested in a large randomized clinical trial. "If they did a study like that and it was negative," he said, "then I'd have to give up my cherished hypotheses for data." The low-fat diet was not easy to follow, said Dr. Rowan T. Chlebowski, a medical oncologist at Harbor-U.C.L.A. Medical Center and one of the study's principal investigators. Women were told to aim for a diet that had just 20 percent of its calories as fat, and most fell short. The diet they were told to follow "is different than the way most people eat," Dr. Chlebowski said. It meant, for example, no butter on bread, no cream cheese on bagels, no oil in salad dressings. "If a physician told a patient to eat less fat, that will do nothing," he said. "If you send someone to a dietitian one time, that will do next to nothing." The women in the study had 18 sessions in small groups with a trained nutritionist in the first year and four sessions a year after that.

In the first year, the women on the low-fat diets reduced the percentage of fat in their diet to 24 percent of daily calories, and by the end of the study their diets had 29 percent of their calories as fat. In the first year, the women in the control group were eating 35 percent of their calories as fat, and by the end of the study their dietary fat content was 37 percent. The two groups consumed about the same number of calories.

Some medical specialists emphasized that the study did not mean people should abandon low-fat diets. "What we are saying is that a modest reduction of fat and a substitution with fruits and vegetables did not do anything for heart disease and stroke or breast cancer or colorectal cancer," said Dr. Nanette K. Wenger, a cardiologist and professor of medicine at Emory University School of Medicine in Atlanta. "It doesn't say that this diet is not beneficial." But Dr. Freedman, the Berkeley statistician,

said the overall lesson was clear. "We, in the scientific community, often give strong advice based on flimsy evidence," he said. "That's why we have to do experiments." .....Source: NYTimes.com

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WHY AM I USING OXYGEN?

Why am I using oxygen? This is a question that everyone using oxygen has, but very few probably ever get a satisfactory answer. In order to answer that question let's break it down into simpler terms.

What is oxygen? Oxygen is a part of the air that we breathe into our lungs every time we take a breath. Our body uses the oxygen to create the energy it needs to carry on all of the processes essential to living such as walking, thinking, breathing, and laughing. Without oxygen the body would shut down in less than 10 minutes.

Why do I need extra oxygen? Due to some heart and lung conditions, not enough oxygen is able to get into the blood to supply the body with the oxygen it needs. Once your condition has been stabilized, the only way to correct a low oxygen level is to use supplemental oxygen.

Won't I become addicted to it? This is a common myth that many people using oxygen have heard, sometimes even from a medical professional. The air we breathe is 21% oxygen. Most people using oxygen are only receiving a little higher percentage (about 28% at 2 l/m), just enough to bring your blood oxygen back to an adequate level. There is no clinical evidence that using oxygen is physically addicting or that using oxygen on a part-time basis will lead to full-time use. If anything, using the oxygen will help to stabilize your condition which may prevent increased use later.

How do you know I need the oxygen? Your blood has been tested and shown to be low on oxygen. This has been done in one of two ways. You may have had a sample of blood taken from your artery known as an arterial blood gas (ABG), or you may have been tested with a small probe on your finger or ear, called oximetry. Your oxygen may be measured at rest, during activity, or during sleep. If your level is low during any of these times, you should be using your oxygen at those times.

Can't I tell if my oxygen is low? Not necessarily, some of the symptoms of low oxygen can be shortness of breath, fast heart rate, changes in the color of lips and fingernails, but some people will have low oxygen level without experiencing any of these symptoms. Others may experience these symptoms without being low on oxygen. The only way to tell for sure is the testing mentioned above.

What are the benefits of using the oxygen? Multiple studies have shown that using oxygen as prescribed leads to a longer life and improved quality of life. Some people feel dramatically different after they start using the oxygen. Less shortness of breath, increased stamina, better tolerance of activity, more restful sleep, improved memory, and clearer thinking are some of the immediate improvements experienced. Others may see little or no change in the way they feel. But whether you feel differently or not, using the oxygen is necessary and will result in the long-term benefits.

When should I use it? Oxygen is not stored well by the body. In fact, once you quit using it, your blood levels will return to previous levels within minutes. For this reason, it is essential for you to use the oxygen at the times your oxygen level has been shown to be low. If you are low during activity you should wear it during activity. If you are low during sleep, wear it while you sleep. If you are low at rest, you should wear your oxygen continuously.

What if I don't use it? Not using the oxygen as your doctor has prescribed will result in your blood oxygen level remaining low. This causes your entire body to be stressed, especially your heart. A low oxygen level increases the pressure inside your heart and with time can cause your heart to enlarge and work less efficiently, a condition known as cor pulmonale. As stated above, studies have shown that patients not using their oxygen do not live as long and have a decreased quality of life.
Will I ever be able to quit using it? Some people with a reversible condition such as pneumonia will get better and no longer need to use oxygen. Conditions such as congestive heart failure, emphysema, and chronic bronchitis are primarily irreversible and long-term use is often necessary. Oxygen should never be discontinued without testing showing that your blood oxygen level is adequate at rest, during sleep, and during activity. An important part of adapting to the use of oxygen is keeping a positive attitude. Don't dwell on the negative aspects but look at all the benefits mentioned above. It can help to increase your activity and improve your quality of life. In many ways it's like getting your first pair of glasses. Sure they feel funny and make you look different, but now you can see to read and drive.

STUDIES SHOW THAT USING OXYGEN AS PRESCRIBED WILL RESULT IN LONGER LIFE AND INCREASED QUALITY OF LIFE.

.....Source: Rotech Medical Corp.

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PLAN YOUR GARDEN

Spring is approaching rapidly. And for many of us, that means one thing: gardening. And it's never too early for planning.

Once you get your garden cleaned up, you may want to revamp it a bit. Or you may decide to design a completely new garden.

The BBC's gardening site will get you started with fresh ideas. The Virtual Garden tool walks you through the process of designing your garden. You can do your work online or download the program to work offline.

If you need more inspiration, visit the virtual garden show. It displays some of the best garden designs created by other visitors.

Note: You'll need the free Shockwave player to design your garden. If you don't have it, you'll be prompted to download it.
http://www.bbc.co.uk/gardening/design/virtualgarden_index.shtml

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ASPARAGUS FOR YOUR BRAIN

Help keep your mind sharp with a few tender spears of asparagus.

Low blood levels of folate and high blood levels of homocysteine were associated with greater memory problems for people 65 and older in a recent study. Protect against memory deficits by getting plenty of folate-rich foods such as asparagus. Increasing your blood levels of folate can help keep your homocysteine levels in check. Source: RealAge.com

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HORMONE LINKED TO GOOD HEARING AS WE AGE

Researchers have linked a hormone known to adjust levels of key brain chemicals to the quality of our hearing as we age. The more of the hormone that older people have in their bloodstream, the better their hearing is, and the less of the hormone, the worse their hearing is. The hormone, aldosterone, is known to regulate kidney function and also plays a role in controlling levels of two crucial signaling chemicals in the nervous system, potassium and sodium. For nerves to send signals crisply and work properly, potassium and sodium must be in precise proportion, without any disruption in the molecular channels or gates through which they move. Levels of potassium are particularly crucial in the sensitive inner ear, where fluid rich in potassium plays a central role in converting sounds into signals that the nervous system recognizes.

The team of scientists in Rochester, N.Y., put 47 healthy men and women between the ages of 58 and 84 through a battery of sophisticated hearing tests. Scientists also measured their blood levels of aldosterone, which is known to drop as people age. They found that people with severe hearing loss had on average about half as much aldosterone in their bloodstream as their counterparts with normal hearing. The researchers noted, however, that the levels of aldosterone found in all the participants is considered normal, and that no patients or physicians should consider altering aldosterone levels without more research.

The findings come from researchers at the International Center for Hearing and Speech Research (ICHSR), a group funded by the National Institute on Aging that is recognized as a leader in research on age-related hearing loss. The center includes scientists from the National Technical Institute for the Deaf at Rochester Institute of Technology and neuroscientists from the University of Rochester. "The inner ear is especially sensitive to any disruption in potassium levels," said Robert D. Frisina, Ph.D., professor of Otolaryngology at the University of Rochester Medical Center and an adjunct professor at Rochester Institute of Technology. "We know that potassium levels in the inner ear seem to decrease as we age and that these falling levels play a role in age-related hearing loss, and we also know that blood levels of aldosterone generally decrease with age.

"We found a direct link between blood levels of aldosterone and the ability of people to hear normally as they age. Depressed hormone levels may hurt hearing both in the inner ear and the part of the brain used for hearing. More research is needed, however, to understand the precise role that aldosterone plays – for instance, whether it's a cause of failed hearing, or whether it's symptomatic. Before we understand the issue more fully, people should not worry about their aldosterone levels or look to boost the amount in their bloodstream." The team led by Frisina published its results in the November issue of the journal Hearing Research. This week at the annual international meeting of the Association for Research in Otolaryngology in Baltimore, the team presented its latest results showing just how important potassium regulation is to age-related hearing loss.

In Baltimore, Otolaryngology medical resident Jared Spencer, M.D., presented results from "knockout" mice whose genes controlling the potassium channels in the inner ear don't function properly, and confirmed that malfunctioning potassium channels are central to age-related hearing loss, or presbycusis. The channels are highly concentrated in a part of the brain that plays an important role providing feedback from the brain to the

ears. Frisina's team previously discovered that the feedback system is one of the first things to go wrong in age-related hearing loss, often declining in people who are in their 40s and 50s, usually before they even realize their hearing is declining. "We are now working out some of the underlying biology about how the decline occurs," said Frisina. "We have evidence that these potassium channels may play an important role in the failure of the feedback system, which is a big part of age-related hearing loss."

Nearly everyone wrestles with failing hearing at some point. While some people suffer from hearing damage as a result of exposure to loud noise, or from other causes such as the side effects of some medications, for many people hearing problems occur with no known cause. Some people notice problems in their 40s and 50s, but the process becomes very noticeable for most people in their 60s and older. Frisina said that until the biology of the problem is better understood, the best advice for people concerned about hearing loss is to limit exposure to loud, damaging noise and to medications that are toxic to the ears. He also counsels people to eat healthy and to exercise – "all those things you know you should be doing to stay healthy with age," he said.

Meanwhile, his team is looking at the possibility of using gene therapy to try to correct the problem. It may be possible some day to modify a person's inner ear to correct the potassium imbalance that is central to hearing loss. Such an approach might also address the biggest cause of congenital deafness, which involves a genetic mutation that mucks up the potassium balance in the inner ear. Source: Univ. of Rochester Med Ctr

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OBESITY STUDY CONTRADICTS MYTH THAT NIGHT EATING CAUSES WEIGHT GAIN

A study in the current issue of Obesity Research dispels the common belief that late night eating promotes weight gain. Judy Cameron, senior scientist at Oregon Health and Science University and a psychiatry professor at the University of Pittsburgh, says the myth is based on the fact that our metabolic rate slows at night. "And, if you were to eat while your metabolic rate was slowing down," she says, "you might be more likely to store energy in the form of fat than to use it immediately."

To test that idea, Cameron and her researchers studied 16 female rhesus monkeys, who were placed on high fat diets, similar to the diet consumed by many Americans. "Some animals ate most of their food during the day at the meals they were served," she says. "Others were nibblers and nibbled a lot at night. There was a spread of eating only five percent of your calories in the evening hours and eating 65 percent of your calories in the evening hours. It turned out that the ones that ate a large percentage of their calories at night were no more likely to gain weight than the ones that ate during the day."

Cameron was surprised by the results, she says, because it was a popular notion and the recommendation of many diets to limit food intake in the evening. "It also surprised us that some animals that really increased their food intake were not gaining weight, while others that increased their food intake just a little bit gained weight," she says. "The total amount of food intake didn't correlate very well with weight gain."

Cameron says the best predictor of weight gain is activity. The monkeys in the study that gained the most weight were the ones that just sat around.

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SHRIMP SCAMPI

2 tsp olive oil
4 medium garlic clove(s), minced
1 tsp dried oregano
1 pound shrimp, medium, peeled and deveined
1/4 cup wine, dry white or vermouth
3/4 cup reduced-sodium chicken broth, fat-free
1/8 tsp salt, or to taste
8 oz uncooked linguini, cooked and kept hot
2 tsp cornstarch
1/8 tsp black pepper, or to taste
1/4 cup parsley, fresh, chopped

Heat oil in a large skillet over medium-high heat. Add garlic and sauté 1 minute. Add oregano and stir to coat. Add shrimp and sauté until bright pink, stirring frequently, about 3 minutes. Add wine and simmer about 1 minute.

Dissolve cornstarch in chicken broth and whisk until blended. Add mixture to skillet and simmer 2 minutes, until sauce thickens. Season to taste with salt and pepper.

Transfer cooked linguini to four individual shallow bowls and spoon shrimp and sauce over top; sprinkle with parsley. Yields about 1 1/3 cups of pasta, 1/4 cup of sauce and 3 ounces of shrimp per serving.

We renovated Shrimp Scampi by:
Substituting healthier olive oil for butter — and using much less of it.
Adding extra garlic to kick up the flavour.
Substituting fat-free broth for traditional chicken stock.

"What fits your busy schedule better, exercising one hour a day or being dead 24 hours a day?"

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