HYPERBARIC MEDICINE NEWSLETTER 1999

Introduction

This is the long-awaited newsletter from Davis Hyperbaric Laboratory, Brooks AFB, TX. With a new editor comes changes in presentation, and focus. New, is the fact that this comes to you in paperless format. It will be published at least yearly with possible interim updates as time or circumstances dictate. Some topics will not be written as articles but as powerpoint or excel presentations. The emphasis will be to provide the reader-participant concise information in the most timely and stimulating learning environment possible. It is published on our website where we decided to dispense all information on hyperbaric medicine. *Editor's commentary is italicized, 10 point.* So let the fun begin.

USAFSAM/FEH, 2602 West Gate Rd,
BROOKS AFB, TX 78235-5304
DSN: 240 - 3281
COMM: 210 - 536 - 3281
http://www.brooks.af.mil/web/hyper/

Fellows Forum

Much of the wound healing we all take for granted is governed by this little-discussed cell. There has been a great deal of research done to understand its biochemistry and while much is known, understanding in some areas is still nascent, e.g., how does oxygen and in particular HBO, modulate cell activity. Dr Ramirez presents the fundamentals of fibroblast function followed by a synopsis of some current literature.

BIOLOGY OF FIBROBLAST

HECTOR A. RAMIREZ, MD, FS
FELLOW, HYPERBARIC MEDICINE,
1998-'99

Overview:
Morphology
Histology
Biochemistry
Development
Differentiation
Function
Pathology of Fibrosis
Role of HBO

A fibroblast is a cell, usually elongated but can also be round. It has a nucleus and a nucleolus. It contains cytoplasm, which is usually eosinophilic. Has one or more expanded pseudopodia (ruffling - like) or non-pseudopodia (bipolar spindle - like), differing in the amount of collagen synthesis. Physiologically, is a unit of independent action entering into form, generating interactions with high motility properties and prevented by contact inhibition but moving in random fashion, except when constrained by cellular interactions.

Its morphology, histology and biochemistry enables the fibroblast to mature and function. For example, chondroblast tend to be round and immobile in contrast to the more mobile fibroblast of skin. Also, chondroblast synthesize little hyaluronic acid (mucopolysaccharide). Their synthesis of collagen is different. The collagen from skin and bone differs in amount. Another chemical component is chondroitin sulfate, another

mucopolysaccharide. Still, little is known of its biochemistry. Their reactivity, an essential characteristic of all living cells, is extremely high which results in intercellular matrix and fibers forming granulation tissue and providing stability.

A lot is known about connective tissue but less attention has been paid to its precursor cell; the fibroblast. These cells have been cultured in Petri dish and have been observed to proliferate and stabilized from monolayer and independently of one another lying parallel with its immediate neighbor. A transient stage, the confluent monolayer patchwork, leads to multilayer stacks of cells built over and over until cessation where it differentiates, completing its morphological process and inhibited by collagenase; a total stabilized tissue by self - maintaining and self - replacing (i.e., cuts).

A lot of these findings have been documented since 1958 in an attempt to integrate the connective tissue research from a biological and medical point of view. Back then the medical work was concentrated mainly on rheumatoid arthritis, genetic disorders and wound healing. Fibroblast was the model cell in which it was started. Chicks' embryos and human peripheral blood were the sources for the fibroblast cultures. In 1960, collagen was found in human WBC by ascertaining hydroxyproline, an aminoacid, expanding more knowledge on fibroblast.

Fibroblasts differentiate into chondroblast, collagenoblast and osteoblast. Chondroblast arises from the mesenchyma and forms cartilage. Collagenoblast produces collagen mainly at the site of inflammation. Osteoblast produces bone. You see, when the stimulus of the connective tissue (fibroblast) stops, an involution process takes place, at which time differentiation occurs, resulting in permanent connective tissue.

Fibroblasts form the fibrous tissue in the body, tendons, and aponeurosis, supporting and binding tissue of all sorts. Fibroblast is the connective tissue cell responsible for most collagen formation in the body; other tissues include smooth muscle cell, Schwann cells. The main action of the fibroblast are extracellular, their membrane contains the receptors of its function, and because of its biochemical environment which is acidic, it provides them the proliferative and synthetic phases for eventual stabilization and strength of tissues. Fibroblast plays a role in the formation of granulation tissue and in pathological processes, such as rheumatoid arthritis, hypertrophic scar (keloids, adhesions), sclerosis (otosclerosis, atherosclerosis, nephrosclerosis), some degenerative disorders (osteoarthrosis), in hereditary morphological defects (hernias) and fibrosis (liver cirrhosis). As a contractile cell it behaves like the smooth muscles cell. Still, little is known about what triggers fibroblast proliferation particularly, during the process of wound healing. Silver from the Department of Pathology in England in early 1969 studied the growth of fibroblast in rabbit's ears. He measured PC02, P02, pH and PNa via microelectrodes and the output was recorded via amplifier and

then polygraphs. His results of the normal fibroblast environment during the inactive phase, demonstrated parallels lie among the fibers in which the P02 was normally decreased. Of interest, their mucopolysaccarides were also decreased. On the other hand, fibroblast in their active phase, during the wound repair process, lived in 3 zones: the growing edge, the synthetic and the crosslinking zones. In the growing edge zone, the morphology was that of the rounded type, less mobile, where they lived in an environment in which the P02 was decreased and did not appeared to divide unless they were closed to a perfused capillary bed. Their pH was 6.7 - 6.8 and PC02 were 40 - 60 mm Hg. In the synthetic zone the fibroblast was elongated, more mobile, collagen was abundant, P02 was normally higher and PC02 was less with a pH 6.9 - 6.95. This zone was well perfused by arteriolar capillaries. In the crosslinking zone the cells were sparser and the capillary network was less profuse.

Fibroblast growth takes place within a narrow range of P02 while synthesis occurs with increased P02. At tissues < 20 mm Hg collagen synthesis stops. Growing cells can not survive in a hostile environment that may be slightly different from the one in which they are normally found; a delicate balance. Body homeostasis is crucial for the growing zone. Nutrition is important and must be maintained in order to achieve healing during the fibroblastic stages of collagen formation. Steroids diminished fibroblast proliferation, diminished collagen synthesis, and retard epithelialization. However, the exact mechanism of fibroblast damage is unknown but perfusion and oxygenation are key. Damage is also associated with an anaerobic environment as a consequence of decreased P02 with increased lactic acid and sodium pump failure. When this pump is affected the osmotic and the ionic balance of the cell cease to be maintained resulting in increased intracellular sodium and water and decreased potassium. Swelling of the mitochondria and finally disruption with irreversible damage follows this. This is where oxygen plays a big role. Hunt et al (67) and Silver (69) stated that the fibroblast in the growing zone would utilize extra oxygen if it were supply artificially.

Fibroblast can be beneficial but also its pathological processes are significant. Fibroblastic activity in inflammation can be divided into acute and chronic inflammation and cryptogenic fibrosis. In acute inflammation the fibroblasts proliferate and synthesize collagen and mucopolyssacharide but only when there is tissue destruction. If this is lacking, the inflammation exudate disappears and resolves a good example is the lobar pneumonia. On the other hand, with destruction of tissue, suppuration can become continues with eventual tissue loss secondary to its own biochemical effect or ischemia. In chronic inflammation, in an attempt to heal, granulomas are formed, like in tuberculosis, leprosy, sarcoidosis, syphilis and rheumatoid arthritis. Cryptogenic fibrosis is rare but the classic example is cirrhosis with extensive fibrosis, chronic glomerulonephritis, fibrosing alveolitis and diffuse systemic sclerosis, where there is dense collagen deposit.

Niinikoski and Kiviassri, Department of Surgery and The Department of Medical Chemistry, University of Turku, Finland evaluated the role of HBO and Wound Healing in 1969. Oxygen dynamics of wound healing gained practical importance after it was shown that the rate of tissue repair varied directly with the supply of oxygen. This was also observed by Stephen's and Hunt, et al.,(71). The main targets of oxygen in wound healing appeared to be the synthesis of collagen and differentiation of fibroblast. They also observed that the oxygen supply of the cells was diffusion limited, because oxygen gradients are steep between the capillary and the healing tissue and significant portions of any injured tissue exist in conditions of low oxygen tension (Silver 69 and Niinikoski 72). These findings clinically were not feasible to locate therefore oxygen measurements were initiated. In our facility here at Brooks we perform transcutaneous oxygen studies in our problems wounds, not only provides us with the oxygenation of the wound but helps the surgeon with mapping the anatomy if amputation is needed.

Joseph V. Boykin, Jr., MD from the Hyperbaric Medicine Problem Wound Center, Metropolitan Hospital and the Division of Plastic and Reconstructive Surgery, Medical College of Virginia, Richmond, presented a study titled Hyperbaric Oxygen Therapy (96). A Physiological Approach to Selected Problem Wound Healing, where he stated that hyperbaric oxygen (HBO) therapy has clinically demonstrated to significantly ($P<0.05$) accelerate the healing of chronic wounds. In a randomized, double blind study of human volunteers with non-diabetic chronic ulcers, adjunctive hyperbaric oxygen therapy has demonstrated to reduce the size of the ulcers and promote healing, compared to routine clinical measures not utilizing hyperbaric oxygenation. Oxygen is absolutely necessary for normal wound healing. However, more is not necessarily always better. Improperly utilized, oxygen is potentially damaging.

Criswell and W. J. Mehm, Division of Altitude and Hyperbaric Physiology, Armed Forces Institute of Pathology, Washington, DC, reported on the effects of hyperbaric oxygen and growth factor on fibroblast infiltration in mice in the Journal of Hyperbaric Medicine (92). It's worth mentioning that growth factors are new in wound healing. They are proteins, naturally occurring in the body. Found in platelets and macrophages. They function as mitogen and chemoatractants. Falanga, et al., states that their main overall effects in wound healing consists of epithelialization, angiogenesis and collagenase activity. One difficulty of growth factors is determining how does it work. The fact is that different effects are observed between the in vitro and in vivo research; one can be a potent inhibitor of endothelial proliferation but in vivo can result in rapid angiogenesis. More research is on going. But for this study, a polyvinyl alcohol (PVA) sponge was implanted in mouse s. c. tissue to investigate two treatments [Intermittent Hyperbaric Oxygen (100% oxygen for 90 min twice a day at 250 kPa) and Epidermal Growth Factor (EGF) which may modulate fibroblast infiltration. Two conditions were established for treatment exposure of animals to chronic

hypoxia (12% oxygen for 23 h/day), simulating low oxygen tensions in problem wounds, and normoxia (21% oxygen). In experiments evaluating EGF, sponges were implanted whose core contained EGF covered with a slow-release polymer, the other group with placebo. Sponges were harvested 15, 25, or 32 days after implantation. The area of the disc infiltrated by fibroblasts was measured by planimetry. After 32 days exposure to hypoxic conditions (7 days before sponge implantation and 25 days after) EGF slightly increased (not significant) the area of fibroblast infiltration compared to placebo under both hypoxic and normoxic conditions. No significant differences were observed between the hypoxically conditioned groups and normoxic controls. Neither chronic hypoxia alone nor chronic hypoxia with intermittent hyperbaric oxygen administered 21 - 32 days after disc implantation affected the area of fibroblast infiltration. EGF significantly increased the area of the fibrous capsule around small PVA sponges after 15 days under normoxic conditions.

Tompach PC, Lew D, and Stoll JL, from the Department of Oral and Maxillofacial Surgery, University of Iowa Hospitals and Clinics, Iowa's City, USA, reported on the cell response to hyperbaric oxygen treatment (97). They investigated the effects of hyperbaric oxygen (HBO) on cells involved in wound healing. Cultured endothelial cells and fibroblasts were exposed to HBO. The effect of varied partial pressure, oxygen saturation, and duration and frequency of exposure to HBO on cell proliferation was determined by 3H-labeled thymidine incorporation. HBO caused an increase in the partial pressure of oxygen in the medium of cultured cells, leading to increased endothelial cell and fibroblast proliferation. Increased endothelial cell proliferation occurred after 15 min of HBO. Fibroblasts required 120 min of HBO to produce a response. A second exposure to HBO on the same day produced no additional increase in cell proliferation. A 120-min HBO exposure stimulated fibroblast proliferation for 72 h after the exposure. An increase in pressure from 2.4 to 4.0 atmospheres absolute did not enhance the proliferative response. These studies began to elucidate the effects of HBO on cells involved in wound healing and established a scientific foundation upon which to develop more efficacious and cost-effective HBO therapeutic protocols.

Roberts GP, Harding KG, from Wound Healing Research Unit, University of Wales College of Medicine, Heath Park, Cardiff, UK (94), examined the effect of hyperbaric oxygen treatment on the synthesis of glycosaminoglycans (mucopolysaccharide) by fibroblasts isolated from wounds and normal skin. Fibroblast cultures were exposed to seven treatments of intermittent hyperbaric oxygen, and then metabolically labeled with D- [6-(3) H] glucosamine (aminoacid). Hyaluronic acid and proteoglycan synthesis was determined by measuring the radioactivity precipitated with cetylpyridinium chloride before and after digestion with hyaluronidase. Hyperbaric oxygen treatment resulted in increased synthesis of hyaluronic acid and proteoglycans by fibroblasts from wounds and normal skin. Overall, the average increase in total

glycosaminoglycan synthesis after hyperbaric oxygen treatment was 28%, whereas fibroblast proliferation was decreased by 7%. These results suggest that one of the effects of this treatment on a wound may be to increase the ratio of extracellular matrix to cells. Such a changed could have important consequences for cellular activities essential for effective wound healing, such as migration of cells into the wound and control of cell function.

Kindwall EP, Department of Plastic and Reconstructive Surgery, Medical College of Wisconsin, Milwaukee (92), reviewed the uses of hyperbaric oxygen therapy in the 1990s. He states that hyperbaric oxygen increases the killing ability of leukocytes and lethal to certain anaerobic bacteria. It inhibits toxin formation by certain anaerobes, increases the flexibility of red cells, reduces tissue edema, preserves intracellular adenosine triphosphate, and maintains tissue oxygenation in the absence of hemoglobin. In addition, it stimulates fibroblast growth, increases collagen formation, promotes rapid growth of capillaries, and terminates lipid peroxidation. He also stated that the most important effects of hyperbaric oxygen (HBO) for the surgeon, are the stimulation of leukocyte microbial killing, the enhancement of fibroblast replication, and increased collagen formation and neovascularization of ischemic tissue.

Wu L, Pierce GF, Ladin DA, Zhao LL, Rogers D, Mustoe TA., Division of Plastic Surgery, Northwestern University Medical School, Chicago, IL., (95). They reported on Kaposi's fibroblast growth factor (K-FGF, FGF-4), a newer member of FGF family with uncharacterized wound healing properties. Basic fibroblast growth factor (bFGF, FGF - 2) has been well studied and accelerates repair in normal and impaired wound healing models. K - FGF and bFGF are known to have similar biological effects in tissue culture, and both stimulate fibroblast and endothelial cell proliferation. The rabbit dermal ulcer model was used to examine the effects of bFGF and K-FGF under ischemic and nonischemic conditions. They found bFGF was ineffective in stimulating healing under ischemic conditions even at high doses (30 micrograms/wound). However, when the ischemic wounds were treated with bFGF (5 micrograms/wound) plus hyperbaric oxygen therapy, it was highly effective, as previously found under nonischemic conditions ($P<0.05$). In contrast K-FGF stimulated repair in both nonischemic and ischemic wounds ($P<0.05$). These results suggest that wound oxygen content differentially regulate responsiveness to bFGF and that K-FGF is biologically active in hypoxic wounds.

Other studies includes, stimulation of angiogenesis to improve the viability of prefabricated flaps, by Bayati S, et al., (98). Hyperbaric oxygen and basic fibroblast growth factor promote growth of irradiated bone, by Wang X, et al., (98). Histology study of the effect of hyperbaric oxygen therapy on autogenous free bone grafts, by Sawai T, et al., (96). Shortening of the in vitro lifespan of human diploid fibroblasts exposed to hyperbaric oxygen by Honda S., et al, (83). The pericryptal fibroblast sheath in the human rectal mucosa, by

Wiernick G. Et al, (75), and numerous more.

I conclude this general information on fibroblast with more to come and to learn, as far as wound is concerned. I realized the basic is always difficult to grasp. Also, I hope that you feel like I do, that fibroblast is the precursor and model cell of connective tissue and above all, hyperbaric oxygen therapy together with fibroblast certainly has a role in wound management.

References

1. Kulonen and Pikkarainen, Biology of Fibroblast, 73.
2. D. W. Criswell and W. J. Mehm. Effects of hyperbaric oxygen and growth factor on fibroblast infiltration in mice. Journal of Hyperbaric Medicine, Vol. 7, No. 1. 92.
3. Joseph V. Boykin, Jr., MD. Hyperbaric oxygen therapy: A physiological approach to selected problem wound healing. Wounds 96.
4. Wu L, Pierce GF, Ladin DA, Zhao LL, Rogers D, Mustoe TA. Effects of oxygen on wound responses to growth factors: Kaposi's FGF, but not basic FGF stimulates repair in ischemic wounds. Growth Factors, 95.
5. Tompach PC, Lew D, Stoll JL. Cell response to hyperbaric oxygen treatment. Int. J. Oral Maxillfac Surg, 97.
6. Roberts GP, Harding KG. Stimulation of glycosaminoglycan synthesis in cultured fibroblasts by hyperbaric oxygen. Br. J. Dermatol. 94.
7. Kindwall EP. Uses of hyperbaric oxygen therapy in the 1990s. Cleve. Clin. J. Med. 92.
8. Kindwall EP, Gottlieb, LJ, Larson DL. Hyperbaric oxygen therapy in plastic surgery: A review article. Plast. Reconstr. Surg. 91.
9. Honda S, Matsuo M. Protective effect of glutathione against oxygen - induced growth inhibition of human diploid fibroblasts. Biochem. Int. 89.
10. Wiernick G, et al. The radiosensitivity of a mesenchymal tissue. The pericryptal fibroblast sheath in the human rectal mucosa. Br. J. Radiol. 75.
11. Bayati S, et al. Stimulation of angiogenesis to improve the viability of prefabricated flaps. Plast. Reconstr. Surg. 98.
12. Wang X, et al. Hyperbaric oxygen and basic fibroblast growth factor of irradiated bone. Int. J. Radiat. Oncol. Biol. Phys. 98.
13. Sawai T, et al. Histologic study of the effect of hyperbaric oxygen therapy on autogenous free bone graft. J. Oral Maxillofac. Surg. 96.
14. Honda S, et al. Shortening of the in vitro lifespan of human diploid fibroblasts exposed to hyperbaric oxygen. Exp. Gerontol. 83.

Dr Ramirez presents a second topic, this centered on Diving Medicine ranging from dangerous marine creatures to women in diving and medical issues.

DIVING MEDICINE

Hector A Ramirez, MD, FS
Hyperbaric Medicine Fellow

Overview:

I. DANGEROUS MARINE LIFE

A. Venomous Marine Animals
1. Cone Shells
2. Octopuses
3. Fishes
4. Coelenterates (Jelly Fish)
5. Reptiles
6. Sea Urchins

B. Biting Marine Animals
1. Sharks
2. Crocodiles and Alligators
3. Barracuda
4. Grouper
5. Moray Eel
6. Marine Mammal
7. Turtles

C. Poisonous Marine Animals
1. Scombroid Poisoning
2. Ciquatera Poisoning
3. Paralytic Shellfish Poisoning
4. Tetrodotoxin Poisoning (Pufferfish)

II. WOMEN IN DIVING

A. Female/Male Differences in Diving
B. UHMS Recommendation
C. Navy Policy

III. FITNESS FOR DIVING

A. Medical Evaluation for Diving Operation
1. Ear
2. Ocular
3. Lung
4. Musculoskeletal
5. Mental Health
6. Neurologic
7. Skin
8. GI/GU
9. Dental
10. Heme and Endocrine
11. Miscellaneous
12. Studies

B. Medical Evaluation for Sport Diving
1. Sport Diving
2. Psychiatry
3. Neurologic
4. Ophthalmology
5. Endocrinology
6. Gastrointestinal Tract
7. Orthopedic Disorders
8. Oral and Maxillofacial/Dental Considerations
9. Medications and Diving

C. Medical Evaluation for Commercial Diving
1. Respiratory System
2. Cardiovascular System
3. Ear, Nose and Throat
4. Eyes
5. Endocrine System
6. Musculoskeletal System
7. Hematologic System
8. Genitourinary System
9. Neurologic, Mental Fitness and Skin

I. DANGEROUS MARINE LIFE

A. Venomous Marine Animals

1. Cone Shells: There are over 400 species and all have well developed

venom apparatus. The sting of some can be deadly. Because cone shells inject their venom with a harpoonlike structure located at the narrow end of their shells, persons handling these animals should grasp them at the wide end. There is 25% mortality rate if stung and also, die quickly. Symptoms varies but overall include mild to excruciating pain, numbness and tingling, may take less than 10 minutes to develop followed by diffuse skeletal muscle paralysis and respiratory paralysis followed by death. Clinical state may deteriorate for 1-6 hours, after which survival is likely. If the patient survives, mobility and activity usually returns within 24 hours. Full recovery takes a few weeks. Treatment is supportive. Must immobilize extremity and ace pressure bandage. No antivenoum available. Prevention is not to pick up living shells.

2. Octopuses: They usually are timid. Rarely non-toxic to humans except for the rare Blue Ring Octopus from Australia. A diver was bitten in the neck and died within 2 hours. It is the most dangerous octopus, also found in Western Pacific. Ringed markings on the tentacles and body become bright blue when feeding or angry. The bite is painless and may go unnoticed. The area around the bite becomes hemorragic and edematous. The toxin is Tetrodotoxin (identical to that found in puffer fish). Tetrodotoxin is a pure crystalline, highly lethal neurotoxic substance, C11 H17 N3 O3. Ingestion results within minutes, in malaise, dizziness, and tingling about the mouth, which may be followed by ataxia, convulsions, respiratory paralysis, and death. Treatment includes no tourniquet, instead use ace pressure bandage. No antivenom available, only supportive care until symptoms resolves.

3. Fishes: Most inflict venomous wound with their fin spines. They tend to injure divers who deliberately handle or provoke them. Include the scorpionfish, lionfish, stonefish and stingray. Many fish have spines with a venom apparatus for protection. Most of the envenomation result in localized tissue damage, but deaths do occur. First symptom is usually immediate local pain lasting minutes to hours. Puncture wound is usually anesthetized and cyanotic with area of painful surrounding edema and erythema. Can progress to generalized symptoms and signs such as, delirium, malaise, nausea, vomiting, elevated temperature, and leukocytosis. Infrequently leads to cardiovascular shock and death. Treatment should include elevation of the affected extremity. The toxin is heat labile and immersion of the affected limb in hot (110 – 115) water for 30 – 90 minutes should alleviate pain. No tourniquet. Remove visible portions of the spine or sheath. Scrub with soap and water and irrigate thoroughly. X-rays may be helpful is ensuring complete debridement. Definitely topical antibiotic and plus or minus oral antibiotic. Stonefish is probably the most dangerous. Grows about 12 inches in length. Lies in shallow water, and can survive many hours out of the water (on the reef). Its venom produces intense vasoconstriction and blocks conduction of both smooth and striated muscles. Antivenom available in Australia, zoos, aquariums and poison centers. Stingrays usually are bottom dwellers who often remain buried in sand. Can grow to 4 meters in diameter. They feed on shellfish, mollusk, crustaceans, and

worms. Non-aggressive, but capable of protecting itself. Their dorsal spine is serrated and sharp on both sides. Venom causes death of fat and muscle within one hour at the local site of injury. May take many months to resolve. Can cause death from penetration of body cavities (pleural, pericardial, peritoneal), from the venom itself, or both. The treatment is the same as for other venomous fish. Prevention includes, shuffling feet when walking in the water and swim well above bottom.

4. Coelenterates: There are over 10,000 species of which 100 are dangerous to man. Found in all oceans, but most common in tropical and temperate zones. Include the Scyphozoans (true jellyfish), Hydrozoans (Portuguese man-o-war) and the Anthozoans (Soft Corals, Hard Corals, and Sea Anemones). Common characteristics are their stinging apparatus named Nematocyst. A minute stinging structure used for anchorage, for defense, and for the capture of prey.

The Nematocyst is filled with venom. Usually, triggered by physical contact or osmotic swelling. An encounter with Portuguese Man-O-War could trigger several million nematocysts. Clinical presentation includes mild itch to burning or throbbing pain. The inflamed area can develop blisters or even necrosis. Treatment includes vinegar and alcohol to prevent further discharge of the nematocysts. Dislodge tentacle gently. Apply topical anesthetic (Lidocaine 5%) and topical steroids. Supportive care for cardiorespiratory complications. The Box Jellyfish is the most venomous marine creature in existence. Can induce death in less than 30 seconds. One Sea Wasp has enough venom to kill 3 adults. This jellyfish poses a much greater danger than any known shark (many fatalities each year). Death is attributed to hypotension, profound muscle spasm, muscular and respiratory paralysis and cardiac arrest.

5. Reptiles: Venomous snakes are a more widespread hazard in fresh water than in the sea. There are two types: surface swimmers and bottom dwellers. All are air-breathing reptiles. Found in many tropical and temperate waters. Most abundant in Indian Ocean, Western Pacific, and Persian Gulf. There are approximately 50 species. The venom is 2-10 times more potent than Cobras. Only 25% of those bitten develop symptoms and signs (this is due to the inefficient delivery system and small mouth). The venom is heat stable, which appears to block neuromuscular transmission at the postsynaptic membrane. Fang and teeth marks consist of small puncture wounds and may number from 1-20. Usually, there is a latent period of 10 minutes to several hours between bite and symptoms. Clinical presentation includes euphoria, anxiety, or restlessness, progressing to dry throat, nausea, vomiting, generalized weakness, and an ascending paralysis or a paralysis that radiates out from the bite site. Finally, respiratory distress. Can see myoglobinuria and renal failure. The treatment includes no tourniquet, use ace wrap pressure bandage to occlude the veins and lymphatics only (at bite, above and below). Immobilize extremity. Supportive care. Antivenoms are associated with anaphylaxis and are of unproven value. If no symptoms develop within 6 hours, unlikely poisoning occurred. Remove bandage after 24 hours, if unable to get clinical help and if patient remains

asymptomatic. Full recovery usually occurs. Prevention includes wet suit to offer some protection and avoid known areas of high concentrations.

B. Biting Marine Animals

1. Sharks: Include the Great White, the Maco, the Tiger, the Bull, which is the only one to travel upward stream, the Oceanic White Tip, the Hammerhead, the Blue, the Nurse and the White Tip/Black Tip Reef Sharks. There are approximately 350 species. Only 30 species have been reported as attacking man, although shark attacks are very rare. More people die from bee stings each year than from shark attacks. Drowning is 1000 times more likely than shark attacks. Some statistics include 25% of attacks for divers, 43% of those being free diving, not SCUBA. 20% of attacks are associated with spear fishing. 20% is associated with line fishing and males are 13.5 times more likely to be attacked than females. Most of the sharks are strictly marine inhabitants and most live in shallow waters. Most prefer temperate or tropical zones. Range in size from 2-60 feet and can weigh up to several tons. Can swim 20-40 mph. Their most well developed sense is smell. Almost 70% of brain is devoted to smell. Able to detect blood in concentrations of only 1ppm. Vision is poorly developed, however, ability to discriminate movements and minor variations in low illumination conditions is extremely efficient. Their sense of hearing is particularly sensitive to low frequency sounds (i.e. helicopters, explosives, sinking ships or thrashing in the water). Sharks have been known to congregate at site of plane crashes and sinking ships. The USS Indianapolis was torpedoed in the Pacific in WWII by Japanese submarine. After almost 5 days in the water, 900 survivors were reduced to 317 due to shark attacks. Attacks are more frequent when water temperature is > 20 C and when more potential victims at risk (holidays, summer, weekends). Two types of attack patterns have been described. The Feeding and the Territorial. In the Feeding type, the sharks are circling with gradual increase in speed and tightening of circle. They bump, bite and spit. In the territorial mode they exhibit awkward exaggerated lateral movements. Snout usually turned upward, with back arched and tail up. The initial bite during feeding is intended to critically injure. They will often wait for prey to bleed to death before continuing attack. Bite force can be up to one ton/cm2. Initial bite may cause feeding frenzy if other sharks in the area (schooling). Single bite is most common and teeth fragments are commonly found in wound. Treatment usually involves amputations and massive hemorrhage is the most significant problem.

Therefore, control of bleeding is first priority; once out of the water direct pressure. If wet suit is being worn, assess wound and consider leaving it on (serves as compression). Prevention includes swimming with partner, avoid swimming with animals such as dogs and horses. Avoid swimming in turbid water, near drop-offs, deep channels and waste outlets. Remember risk increases if spearfishing. Avoid areas of explosives. Also, avoid urinating or swimming with wounds. Menstruating women do not seem to attract sharks, in fact menstrual blood seems to repel some species. If in plane crash or boat sinks, stay in life raft if possible. Avoid

known feeding areas. If confronted by a shark, swim away and stay close to bottom if possible. Kick or strike in a sensitive area (snout, eyes, gills). The Great White can weigh up to 2 tons and measure over 6 meters long. San Francisco is known as the Great White attack capital of the world (area between Tomales Point at Bordega Bay and Santa Cruz and Monterey Bay). The Tiger shark is the "Billy-Goat of the Sea" because trash is mostly found in their stomach. The Hammerhead can measure up to 6 meters in length and often travel in packs (seen in packs > 200 in the Gulf of Mexico). Cousteau described the White Tip Oceanic as the most dangerous of all sharks. They can be as large as Great White's. Finally, the Maco is a very fast swimmer and likes jumping into boats. Typically feed on deep sea fish.

2. Crocodiles and Alligators: They are very territorial and extremely dangerous to man. Most dangerous during nesting season. Can reach 10 meters in length and weigh more than a ton. Have been known to stalk prey, can cover short distances very quickly. Crocodiles are much more aggressive, faster moving, and more dangerous than alligators. However, both attack even when unprovoked. They may growl or exhale loudly as a warning. May be able to abort attack by throwing object at the animal or striking snout.

3. Barracuda: Usually, found in tropical and subtropical waters. They usually travel in groups and can grow 2 meters long. Occasionally, they will stalk a diver remaining only a few feet away. Can be easily chased away, but may return repeatedly. Danger of attack is greatest at night when diving with a bright light. Avoid colorful clothing, bright shinning objects and lights.

4. Grouper: Usually in the tropics. They are potential hazards. Their mouths can engulf a diver, and there are reports that they have done so.

5. Moray Eels: Can grow up to 3 meters in length. Rarely attack without provocation. They tend to be night feeders and bottom dwellers, and usually found in holes and crevices in the rock or coral during the day. No venom associated with bite. However, may be poisonous to eat. Avoid feeding and wear heavy gloves if petting.

6. Marine Mammals: The juvenile and female seals and sea lions frequently frolic in the water near divers. Underwater encounters with sea lions can be expected, but it is rarely dangerous. If bitten by a seal or sea lion, the diver should consult a physician because of the infectious diseases. Avoid large whales. Don't provoke whales. Muskrats are potential hazards in fresh water. They attack if feel threatened. A danger with rabies is a possibility. Therefore, if bitten by muskrats every effort should be made to capture or kill the animal for later examination.

7. Turtles: Not generally threatening. However, the Alligator Snapping Turtle and Common Snapping Turtle of America's fresh water can inflict major injuries.

C. Poisonous Marine Animals: Most seafood are edible and nourishing, however, several of the most toxic substances known are sometimes found in marine organisms. Mollusk shellfish,

such as clams, mussels and oysters, are sometimes poisonous because they feed on toxic dinoflagellates, which are microscopic plankton. Dinoflagellates are plantlike protozoa with flagella. Plankton is free floating organisms, vegetables and animals, which live in practically all natural waters. Violent intoxication and fatalities have also been reported from eating tropical reef crabs.

1. Scombroid Poisoning: Poisoning is caused by inadequate refrigeration of dark or red muscled fish. Typical fishes are Tuna, Mackerel, Skipjack, Bonito, Albacore, and Mahimahi. Bacterial decomposition causes histamine production. Taste may be sharp, bitter or peppery. Symptoms occur 15-90 minutes and include flushing, itching, burning, hives, difficulty breathing, headaches, heart palpitations, hypertension, nausea and vomiting, diarrhea, abdominal pain and a metallic taste. Treatment consists of steroids, epinephrine and antihistamines, but usually resolves in 8-12 hours without treatment. Death is unusual. Prevention should involve refrigeration, discard if pallor of the gills, or odorous and do not swallow fish that taste peppery.

2. Ciguatera Poisoning: Numerous species of tropical reef fish are known to be poisonous to eat because they cause a disease known as Ciguatera. An edible fish in one locality may be deadly in another. Common fishes are Grouper, Snapper, Sea Bass, Surgeonfish, Coral Trout, Parrotfish, Spanish Mackerel, Rock Cod, and Moray Eel. It is the most serious and common form of marine poisoning. The toxin is from algae and freshness and cooking have no bearing on its toxicity. Onset of symptoms may be 15-30 minutes but usually within 12 hours. Clinical presentation includes abdominal pain, nausea, diarrhea, numbness and tingling of arms, legs, and around the mouth, incoordination, paralysis, coma, and temperature variations. Treatment should include induction of vomiting if within 3 hours and low chance of aspiration. Also, gastric lavage with activated charcoal. For the mild case, it is ok to provide Benadryl but overall care is supportive treatment. Prevention includes feeding small amounts of fish to kitten before eating and also feed the old people first. Avoid re-exposure.

3. Paralytic Shellfish Poisoning: Clams, Oysters, Scallops, and Mussels are included. Again, caused by concentration of “red tide” plankton and protozoans (dinoflagellates). They are water soluble, heat and stomach acid stable. Neurotoxin (saxitoxin) is not destroyed by cooking. Onset may be very rapid. Symptoms are similar to Ciguatera but may have sensation of loose teeth. Peri-oral paresthesias is usually the first symptom. Can have weakness in the extremities, floating sensation, incoordination, difficulty in speech and vision. Symptoms are sometimes mistaken for drunkenness. Mortality from 1-10% have been reported. If victim survives 24 hours the prognosis is good. There is also some evidence that chronic exposure to small amounts of toxin may induce resistance. Treatment should include induction of vomiting if within 3 hours or gastric lavage with activate charcoal and of course, supportive care. Prevention is actually not that secure; there is no way to visually determine which shellfish are safe.

4. Tetrodotoxin Poisoning (Putterfish): Most Putterfish contain a deadly poison known as tetrodotoxin, and puffers and related species should be carefully avoided. Called "fugu" in Japan. Poisoning is caused by contamination of the muscle by liver contents while preparing. Tetrodotoxin from a medium sized putterfish could kill 30 people. Onset of symptoms is usually rapid but can be up to four hours. Clinical presentation includes sweating, nausea, numbness and paresthesias of the tongue, difficulty speaking and walking, paralysis, fixed dilated pupils in an apneic unresponsive patient and it is not a sign of "cerebral death". 60% of cases die within 6 hours. Treatment is supportive and includes induction of vomiting if within 3 hours, anticholinesterase and watch what is discussed in patient's presence.

II. WOMEN IN DIVING

A. Female/Male Differences in Diving

1. Women have played significant roles as divers for many years, beginning with their work as Hae-Nyu and AMA divers in Korea and Japan. The number of certified female sport divers, instructors, research, and commercial divers in America has increased significantly since the early 1970's and national certification agencies report that approximately 25% of newly certified divers are women. The participation of women in sport scuba diving increases every year. During the 1970's women were clearly underrepresented, comprising less than 20% of the diving community. Current estimates place the percentage between 33% and 35%. When performing a diving physical for a prospective woman scuba candidate, the examining physician must be aware of the anatomic, physiologic, mental, and medical factors that may predispose to diving accidents or mishaps. Regarding size and strength; smaller size is a disadvantage when brute strength is required. However, diving supervisors have always chosen the diver best suited for the job, (i.e. The brute diver gets the brute job). Women have less muscle mass than men because the male hormone, testosterone, which is needed for the development of large muscles, is present only in reduced quantities in women. A woman's heart and lungs are smaller than a man's.

2. Thermal stress represents one of the major energy burdens in diving. Staying thermally comfortable during a dive is important both for enjoyment and to accomplish the work planned for a dive. Despite the fact that women have a layer of subcutaneous fat that is a good insulator, many women quickly become chilled when they dive. Anatomically and physiologically women respond to cold in subtly different ways. The surface area or volume ratio is slightly higher in women, increasing the area of conductive heat loss. Also, women poses much smaller muscle mass, with less metabolically active tissue to generate heat during activity. Therefore suitable exposure suits, properly fitted, are recommended to ensure thermal protection. (i.e. The Neoprene wet suit, Dry suits, or Variable Volume Neoprene or Rubber dry suits).

3. Women are able to accomplish most diving jobs, especially with the advent of lighter dive dress and

power equipment. As far as pregnancy is concerned, there is no apparent problem for the mother. There are many theoretical dangers to the fetus during diving and recompression treatments. Teratogenic effects in animals, retrolental fibroplasia, premature closure of the ductus arteriosus and potential fetal DCS. Most workers, investigating DCS and fetal risk agree that the fetus is at no increased risk of bubble formation during decompression. In fact, three researchers demonstrated that the fetus is more resistant to bubble formation than the mother. Only Fife and colleagues found an increased risk in fetal lambs, but later Stock and colleagues repeated the experiment and asserted that the risk was an artifact of instrumentation. Pregnant women should not dive unless CO poisoning is diagnosed. There is concern that basic physiologic changes in pregnancy may compound diving risks. Many divers experience some anxiety at the outset of a dive. Combining the increased exercise demand, cold stress, pregnancy load and sympathomimetic reflex of anxiety, the potential for potent vasoconstriction is present; therefore, the possibility of decreased blood flow may be significantly increased for the pregnant diver. Every woman should be counseled on potential hazards to the fetus at the initial physical dive. If pregnancy is established she should be medically suspended. Birth control pills are not a contradiction (see further cautions that follow in this article). Although obstetricians encourage patients to continue their favorite sports during pregnancy, as long as they are comfortable and use common sense. Hyperbaric physicians take the most conservative position and recommend that their patient's discontinue diving while they are pregnant, since so much is still unknown about effects of diving on the fetus.

4. Early reports in the field of hypo and hyperbaric medicine promulgated the notion that women are at increased risk of DCS. In 1973, Basset sited 7 cases of DCS in 3,190 exposures in female nurse flight trainees, versus 2 cases in 9,056 exposures in male pilots. A second study in 1980 reaffirmed an apparent 3.6 fold, increased risk of DCS in a woman. However, most of the field studies on divers under observation and with protocols for testing and reporting do not confirm an increased incidence of DCS in women. An aerospace study of DCS during stimulated space missions, authored by Waligora and colleagues, found that the rates of reported pain and detectable Doppler bubbles in male and female subjects were similar. Of the 14 women, 9% reported pain and 18% were Doppler positive for venous bubbles. Of the 15 men, 6% had pain and 23% had detectable bubbles. Dunford's analysis revealed that in relation to the timing of menstruation, the relative risk for chamber dive related DCS appeared to have increased 7.6 fold during the menstrual phase of the cycle. Some data support the idea that increased body fat predisposes women to high rates of DCS. Many other factors are believed to increase an individual's susceptibility to DCS, including age and general vascular condition (i.e. BCP can affect circulation, clotting factors). Older divers should have an annual diving physical examination, and should swim several times a month with mask, fins, and snorkel to stay in good diving condition. They should also watch their weight, avoid fatigue, ascend and

descend at a reasonable rate, and consider the potential interactions between pressure and any prescribed medication before diving.

B. UHMS Recommendations: The 35th UHMS workshop in 1986 developed the following recommendations. There are no contradictions to diving for the normal, healthy, non-pregnant female. The fetus may be at greater risk than the mother; there is insufficient data to establish safe depth/ time exposures for the fetus and there are still a large number of unanswered questions. Robertson, in a letter to the Undersea and Hyperbaric Medicine Society, reported on 111 cases of DCS from the Australian Navy. Though no correlation existed in cases related to age dive experiences, dive profile and other measures, the rate of type II DCS for women was 4.3 times higher than the rate for men (confidence interval 1.2 to 15.8).

C. Navy Policy: Governed by NAVMED COMINST 6200.2 and states that females will be counseled on potential hazards to the fetus at the following times: At the initial dive physical, on the beginning initial diving, training, and at each periodic dive physical. Women will also sign an informed consent release at the above times, which will be kept in their medical record. The female diver will notify the medical department representative at the first presumptive sign of pregnancy (missed menstrual period). She will then be temporarily disqualified for diving until diagnosis is established. If pregnancy is established, she will be medically suspended from diving until the pregnancy ends.

III. FITNESS FOR DIVING

A. Medical Evaluation for Diving Operation: The majority of military, commercial, and recreational dives proceed, smoothly with medical problems being infrequent and generally minor. This excellent safety record may be attributed to high standards for diver health and fitness, rigorous diver training, sophisticated engineering of diver equipment, meticulous pre-dive planning and strict adherence to safe diving procedures. The most important function of medical personnel involved in a diving operation is to prevent diving accidents. In the Navy, article 15-66 of MANMED states who has the authority to endorse waivers. Waivers are endorsed by BUMED-21 and approved by BUPERS. DMO; USA/USAF FSO, or certified PA/NP from NDSTC conducts examinations. The general requirement is for a vigorous, emotionally stable individual who is free of cardiovascular, pulmonary, neurologic, and otolaryngologic disease.

1. Ear**:** Eustachian tube dysfunction or middle ear surgery, except tympanoplasty (120 days post-op) is disqualifying. Any inner ear surgery, pathology or hearing deficit is also disqualifying as well as any laryngeal or tracheal framework surgery.

2. Ocular: Ocular conditions that are disqualifying are: Visual acuity must correct to 20/20. DMO SCUBA can be clear with +/- 8 diopters. Seal and AF Special Forces must be 20/40 and 20/70. They must pass color and night vision. A passing FALANT test is a must for Special Forces and EOD.

Waivers can be requested for the above or individual basis.

3. Lung: A history of spontaneous traumatic pneumothorax (after 6 months, then may re-qualify). History of sarcoidoses, pulmonary barotrauma, RAD, or asthma after age 12.

4. Musculoskeletal**:** Osteonecrosis, particularly dysbaric, any fracture within 3 months of injury or bone or joint surgery (except scope) within 6 months.

5. Mental Health: Any diagnosis with potential to hinder performance, judgment and reliability. ETOH dependency without one year aftercare (level III).

6. Neurologic: Headaches requiring treatment (i.e. vascular type), head injury (penetrating), LOC, Syncope or recurrent vertigo and DCS or AGE with residual impairment. Heat stroke with residual neurologic impairment. Back pain secondary to disk disease. Spine surgery within 6 months.

7. Skin: Any disorder that is exacerbated by sun exposure.

8. GI/GU: Any disorder that is aggravated by diving.

9. Dental: Must be class 1 or 2.

10. Heme and Endocrine: As per initial.

11. Miscellaneous: History of DCS for initial candidates (must R/O PFO). History of severe or incapacitating motion sickness. Age over 35 for initial and over 28 for SEAL. Must pass pressure test.

12. Studies: All of the following need to be normal. CXR, ECG, FALANT, CBC, PPD, and cardiac W/U closest to age 45 (Lipid profile, EST as indicated).

B. Medical Evaluation for Sport Diving: Sport diving may allow a greater leniency than more rigorous or isolated diving modes associated with operational diving (military or commercial diving). The examining physician must know the type of diving planned as well as the specific conditions that may represent hazards in the diving environment. Whereas commercial, Naval, scientific, caisson and hyperbaric chamber divers are generally examined and certified according to a set of standards established by the employer or agency involved, there are no regulations or standard for sport divers, except those set forth by training agencies for acceptances of students into scuba classes. Once trained, a sport diver usually decides personally whether it is safe to dive and many never undergo a diving physical examination. Sport divers either may have no examination or may be examined by a physician with little or no training in diving medicine. You must ask yourself, what is at stake?

1. Psychiatry: The most common cause of death in sport scuba divers is panic, with ineffective behavior if an emergency develops underwater. Panic-prone individuals often have other phobias such as claustrophobia, a fear of new situations, or a lack of self-confidence. Such individuals should be disqualified from diving because an

event underwater would surely result in drowning and risk to buddy divers in rescue attempts. Alcohol or drug addicts or heavy users should be disqualified.

2. Neurologic: Seizures should be disqualified, regardless of control by anti-convulsant medications, if there is no neurologic or physical impairments in herniated intervertebral disc, diving can be allowed but with caution about further injury from climbing, lifting, and so forth. Diving can be resumed after 3 months following successful disc surgery with no complications. A history of stroke is also disqualifying when significant residual neurologic deficits are evident. Diving should not be advised in individuals with neuropathy that has resulted in skin or joint damage. A concern is the inability to differentiate neuropathy from DCS. The importance of documenting a detailed neurologic examination on an initial visit is a must.

3. Ophthalmology: It is important that the candidate have visual acuity and visual fields adequate for safe conduct on a boat or shore diving site for underwater orientation. Near vision adequate to read the pressure gauge, watch, compass, decompression tables, and depth gauge is necessary.

4. Endocrinology: The decisions on whether sport diving can be recommended ranges between individuals with the absolutely disqualifying evidence of end-organ disease. Ketoacidosis or frequent signs or symptoms of hyperglycemia or hypoglycemia, to those with mild abnormalities of glucose metabolism, fully controlled by weight loss and diet, who can be cleared for diving.

5. Gastrointestinal Tract: Peptic Ulcer disease and inflammatory bowel disease present relative contraindication. The fluid and electrolyte loses that can occur with acute exacerbations of the disease processes can render the individual more susceptible to DCS. An absolute contraindication, is any condition that could cause air trapping from gas expansion during ascent as it may increase the risk of vomiting underwater with the potential of drowning and panic ascent, with pulmonary barotrauma and air embolism. Other conditions to consider are GERD, achalasia because of pooling of secretions and food in the proximal esophagus. Paraesophageal and incarcerated sliding hiatal hernia are absolute contraindications because of the risk of massive overdistention of the gastric remnant in the hernia with rupture on ascent. Other conditions include partial chronic gastric–outlet obstruction of a high grade. Small bowel obstruction may dive after at least 6 months free of symptoms post surgery.

6. Orthopedic Disorders: Incompletely healed fracture or acute inflammation due to immobility and loss of dexterity with a cast, must be disqualified until fully healed. In scoliosis, must document normal PFT.

7. Oral and Maxillofacial/Dental Considerations: The ability to fit and hold a scuba mouthpiece is a prerequisite. However, major oral surgery with osseous or soft tissue deformity that would prevent proper fit of a scuba mouthpiece is a contraindication.

8. Medications and Diving: No medication is considered "absolutely" safe. There is no official approved medication list. Must answer the question of why is this medication being given? People should feel their best diving. Relatively safe are pure decongestants, simple analgesics, NSAIDS, topicals, antibiotics, BCPs. Keep in mind that antacids can produce gas, recommend the non-effervescent.

In summary, scuba diving introduces unique and specific risks that lead to restrictions set forth above. Sudden alterations of consciousness underwater can lead to drowning or serious pulmonary overpressure accidents during rescue to the surface. The physician must protect against legal action by documenting conservative, yet sensible, recommendations. Every diver a physician approves is someone's diving buddy.

C. Medical Evaluation for Commercial Diving: Commercial divers must be medically, mentally, and physically fit to perform there varied tasks in a safety environment. A periodic examination seems necessary and acceptable. Protecting divers from the adverse effects of diving on their future health, is difficult to fulfill. Dysbaric osteonecrosis, is an occupational health hazard of diving that becomes manifest either by x-ray examination while the diver is still asymptomatic or later by joint pain because of physical damage to an articular surface. Also, medical examination of divers relates to the protection of the employer against future court action. For criteria of fitness that do not relate to safety, it is reasonable to have, in saturation diving, for example, a different standard from the other categories of diving. In saturation diving the patient can be isolated from treatment at normal atmospheric pressure by the need for a decompression that extends over several days. Commercial divers follow one or more of a wide range of activities from offshore, deep diving to professional instruction of sport divers. Neither rigid naval standards nor the relaxed approach for the amateur diver is appropriate for a person for whom diving is a regular and possibly lifetime job. The Health and Safety Executive (HSE) in the United Kingdom was the first government agency to issue detailed guidance on the medical examination of commercial divers.

1. Respiratory System: Conditions that can preclude diving are any acute or chronic respiratory infection; a history of spontaneous pneumothorax; the presence of lung cyst, blebs, or bullae; chronic bronchitis; emphysema; pleural effusion; fistula; bronchiectasis; fibrosis and neoplasm. Stable asthmatics who is not triggered by provocation testing (histamine or methacholine) could be considered fit provided they demonstrate less than 20% reduction of peak flow or forced expired volume in 1 sec (FEV) after as long as 20 minutes of hard exercise.

2. Cardiovascular System: Any organic heart disease should be a cause for rejection unless hemodynamically unimportant. Consider referral to cardiologist for fitness.

3. Ear, Nose and Throat: Similar to the Navy standards. The presence of attic or posterior marginal perforations

of the drum indicates middle ear disease and contraindicates diving.

4. Eyes: The diver should be able to see well enough to accomplish the necessary functions. A person who can read a car license plate at 25mph probably has adequate vision for diving. Modern gas-permeable contact lenses are acceptable. Color vision is not essential except for boat navigation, especially at night.

5. Endocrine System: Hypothyroidism on replacement therapy can be compatible with professional diving. Caution is diabetic on oral hypoglycemics.

6. Musculoskeletal System: The candidate must have unimpeded mobility and dexterity and must be sufficiently physically robust to meet the demands of the proposed work.

7. Hematological System: A hematocrit of 40% and a hemoglobin of 12g/dl in men and 10.5 g/dl in women are the minimum acceptable levels. Sickle cell trait is not necessarily a cause for rejection.

8. Genitourinary System: The presence of renal stones is usually a cause for rejection.

9. Neurologic, Mental Fitness and Skin: Are similar to the navy standards; Urticaria is a transient condition and if it does not affect the mucous membranes, it is not a contraindication.

In conclusion, fitness examinations are essential for diving safety. They can reveal medical conditions that not only are incompatible with an individual's safety in the water but also could put the safety of companions at risk. Assessment of knowledge and audit of performance of the examining physician are important contributions to the safety of divers in the water.

Dr Wright began his hyperbaric medicine fellowship in July '99 and presents a primer on plastic surgery fundamentals from his plastic surgery experience.

An Introduction to Flaps and Grafts

James K Wright, MD, FACS
Hyperbaric Fellow 1999-'00

The use of flaps and grafts in plastic surgery dates back thousands of years to ancient India where forehead flaps were utilized to reconstruct amputated noses. In modern times flaps and grafts did not come into widespread use until World War I when thousands of soldiers received head and neck wounds in the trenches of Europe. The mechanized nature of warfare produced large numbers of burns requiring reconstruction with skin grafts. Since then the techniques have been expanded and refined, but the principles remain unchanged.

A graft is a piece of tissue which is entirely separated from its blood supply and transposed to another area of the body without an attempt to re-anastamose its blood vessels. It entirely depends on the blood supply of the recipient site for survival, hence the better the blood supply of the recipient site or graft bed, the better the chance of the graft surviving. The survival of a graft is a race between ingrowth of new blood vessels from the recipient bed and necrosis of the graft. The proper

execution of every type of graft has been defined through surgical experience, and if the limitations are recognized, graft survival is usually possible, though by no means assured. Usually autografts are performed in which the patient serves as his own donor. In special circumstances, a heterograft may be performed, i.e. from one person to another, such as in the use of cadaver skin as a temporary wound dressing in burn patients. Xenografts are grafts from one species to another, and with the exception of frozen or lyophilized pig skin as a temporary wound dressing, are rarely performed. Any graft other than an autograft will be rejected unless the host is immunosuppressed.

Most grafts are skin grafts, which are used to close skin defects ranging in size from a few millimeters in the case of the eyelid, to large areas of the body surface in burn patients. The thickness of the grafts may vary from full thickness in which the entire thickness of the skin is transplanted including the hair and skin appendages such as sweat and oil glands to think split thickness skin grafts a few thousandths of an inch in thickness. Full thickness grafts leave a donor site, which must be closed by suturing the edges together, with a flap, or with another skin graft – usually split thickness. Full thickness skin grafts retain the qualities of the donor site skin, are usually supple and elastic, and if obtained from an area near the reconstructed defect, can offer a good color match. Hence, for facial reconstruction they are an ideal choice.

Split thickness skin grafts range in thickness from about 8 to 20 thousandths of an inch and may include the subepidermal keratinocytes only to large amounts of dermal tissue. As a general rule they do not contain oil or sweat glands and are incapable of supporting hair growth. The donor site heals by secondary intention, and depending on the thickness of the graft, may leave a significant scar. Split thickness grafts are used to close large defects such as burns, large skin resections, or the donor site of a large transposed flap. The skin graft contracts as it heals; the thinner the graft, the more contraction can be expected. Thin grafts are also not very durable and a slight injury may remove significant portions of a healed thin split thickness graft. The thinnest split thickness grafts are used when early wound closure is the primary goal, such as in large burns. The donor site of a thin split thickness graft can be harvested several times every few weeks, if necessary to supply needed skin. Thicker split thickness grafts are nearly as durable as full thickness grafts and can be used to resurface the palm of the hand or sole of the foot.

Not all grafts are skin grafts. Practically every tissue, with the exception of muscle has utility as a graft. Fat is used, often in conjunction with the dermis to add bulk to subcutaneous defects, such as those encountered in the face. The dermis is useful in facial reconstruction and scar revision. Tendon grafts are used to bridge the gap between segments of tendon resected because of scar entrapment or lost due to trauma. Nerve grafts have long been used to reconstruct segments of missing nerve and ultimately to restore function. Bone grafts can restore bony integrity across an are of fracture nonunion or small bony defects. Cartilage has been used for joint and nasal reconstruction. Since all grafts depend on the blood supply of the recipient site bed for survival, they

must be small in at least one dimension. That is, larger grafts of dermis and fat, or bone must be thin so that the ingrowth of new blood vessels can reach all areas of the graft in time to prevent necrosis. Larger bone grafts mat loose their osteocytes and serve as a scaffolding for the ingrowth of new bone, but this new bone may be of poor quality and lack the tensile strength necessary to prevent fracture. Because nerve and tendon are thin, their revascularization is usually adequate, and long grafts are possible. Cartilage grafts are a special case. Cartilage is relatively avascular; larger grafts usually survive, though resorbtion and calcification are possible.

Flaps are based of a different biological principal than grafts. A flap always has its own blood supply. This is present in the pedicle of flap. The blood supply may contain large named arteries an veins such as the thoracodorsal artery and veins which supply the latissimus dorsi muscular flap, or may contain the near capillary sized vessels of the dermal plexus as in a skin flap used to reconstruct the eyelid. If the blood supply is interrupted, the flap is nearly always incapable of surviving as a graft, and it will die. The blood supply in the pedicle of the flap is capable of supplying any type of tissue, and if properly designed, large amounts of tissue may be transferred. In addition, the presence of a blood supply makes it possible to transfer a functional flap to another area of the body – something which is impossible with grafts. Intestinal flaps and nerve-muscle flaps can restore lost function, as well as vascularized bone which is used to restore bony union across large defects. Sensate nerve-skin flaps are possible which can restore feeling to anesthetic areas of the body. With the advent of tailor made flaps which are prepared with tissues such as bone, cartilage, nerve, and skin in combination prior to transfer, the possibilities are almost endless.

There is a problem however. Since flaps must be transferred with their blood supply intact, it stands to reason that they can only be transferred only as far as this blood supply can reach. In the past this problem was overcome by shaping the pedicle into a tube and transposing the pedicle insteps or "walking" the flap to the desired recipient area. This only worked for skin flaps, but the idea was rather ingenious. After the flap had healed for three weeks the pedicle could be divided and the flap "inset." In this way tissue could be transferred from the donor site to a distant recipient site. Today the problem of distant transfer of a flap has been solved through the use of free flaps in which the vasculature is re-established through microvascular anastmoses at the recipient site.

Flaps can be classified in a variety of ways, but usually are defined according to their blood supply since that is all-important in insuring flap survival. Random pattern flaps are skin flaps which rely on the dermal and subdermal vascular plexuses for survival. These are the most commonly used skin flaps and are ideal for reconstruction of small facial defects such as those arising from excision of skin cancers. Large random pattern flaps are sometimes used and an example is a large rotation flap used to close a sacral pressure sore. As a general rule the length of the random pattern flap must not exceed one and one-half times the width of the base or

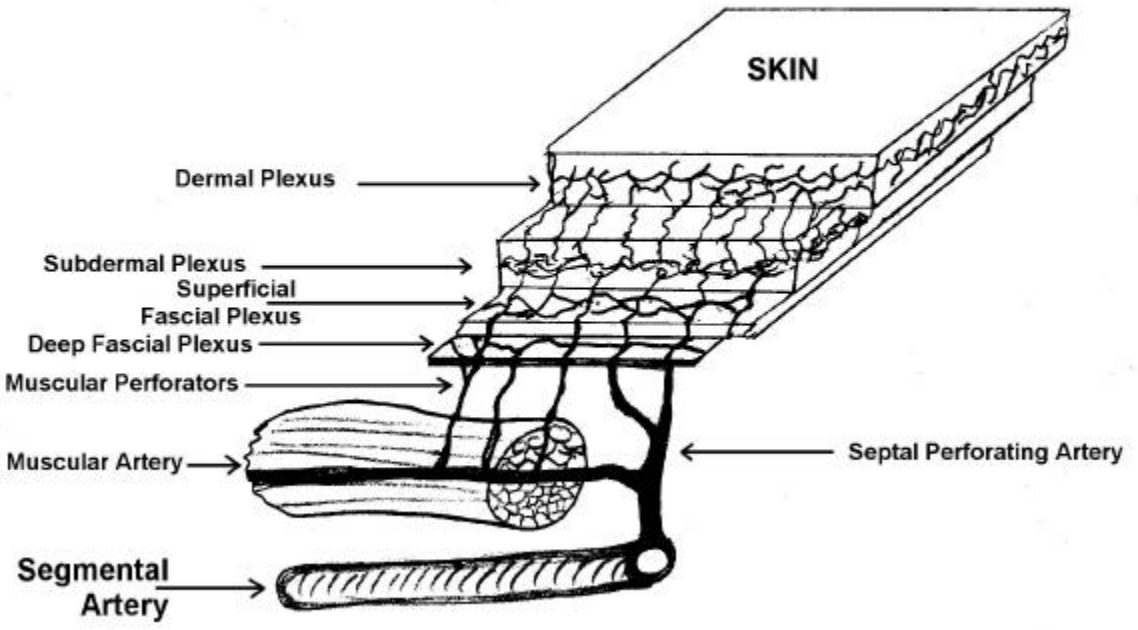
necrosis is likely to occur. One way to overcome the problem of inadequate flap length in a random flap is to perform a "delay" procedure in which the flap is elevated in stages allowing time for the dilatation of existing flap blood vessels to support the entire length of the flap.

Axial pattern flaps are based on the blood supply provide a named vascular pedicle which courses most of the length of the flap. Because axial pattern flaps are entirely dependent on one vascular pedicle, the base can be quite narrow and may comprise only the vessels themselves. Examples of axial pattern flaps are forehead flaps based on the superficial temporal artery, or the supratrochlear artery, either of which can be used for nasal reconstruction. Another example of an axial flap is the groin flap based on the superficial circumflex iliac artery. The deltopectoral flap based on tributaries of the internal mammary artery has long been used in head and neck reconstruction.

Fascial flaps utilize the perifascial plexuses – superficial or deep to the fascia and may include the fascia only, fascia and skin, or fascia, skin and bone. The superficial temporal flap includes the superficial temporal artery and fascia and can be utilized to reconstruct the ear. Because it has no skin it must be covered with a skin graft. A compound fasciocutaneous flap which incorporates bone is the radial forearm flap which may incorporate, skin, subcutaneous fat, and a portion of the radius and can be utilized for thumb reconstruction. The skin and bone are supplied by perforating vessels from the radial artery.

Musculocutaneous flaps are comprised of one or more vascular pedicles, muscle, the dermal perforators from the muscle, and skin. They are transferred as a unit and the muscle is used because the vascular perforating vessels travel through the muscle and subcutaneous fat to the skin. The muscle usually has no

Blood Supply of Muscle, Fascia, & Skin



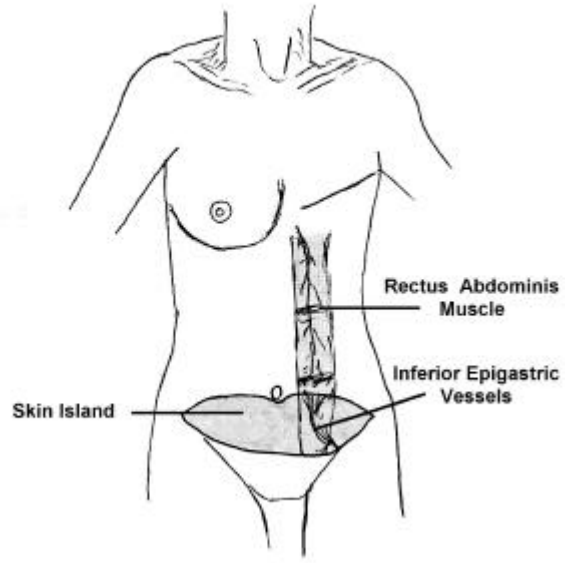
function except to support the perforating vessels; without it the flap would be nonviable. Because it is not innervated it atrophies over time and may eventually be completely replaced with scar tissue. The transverse rectus abdominis musculocutaneous flap is an example of this type of flap which has broad utility in breast reconstruction. An exception to this use is the transfer of innervated muscle such as the latissimus dorsi to reconstruct a lost muscle unit such as the biceps brachii in the arm. In this case the innervated muscle or musculocutaneous flap continues to function takes over some of the lost function of the reconstructed muscle.

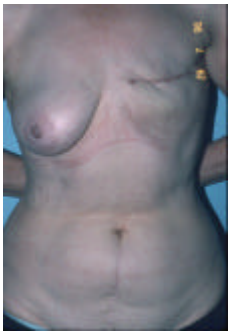
Free flaps are flaps that are completely detached from their blood supply and then re-attached to a blood supple at the recipient site. Because all that is needed for a free flap to work are suitable donor and recipient vessels, flap transfer problems are greatly reduced. Free flaps can be transferred to recipient sites as long as the blood supply can be re-established. In addition composite flaps consisting of bone, muscle, fascia, fat and skin can be fashioned and transferred. Bone flaps are especially valuable in reconstituting areas of bony loss from trauma or cancer resection.

Prior to the advent of free flaps successful re-establishment of large areas of bony discontinuity was frequently unsuccessful. Another useful innovation is the use of innervated free flaps. In addition to the re-establishment of vascular continuity, a sensory or motor nerve is also anastamosed to a donor nerve. Function can thus be restored to a paralyzed area, such as in the use of the serratus muscle free flap for facial nerve loss. The serratus muscle and its vascular supply are placed in the place of the lost facial muscles, the blood supply re-established through a micro-anastamosis and the long thoracic nerve to the serratus muscle is attached to the stump of the damaged facial nerve. One disadvantage of free flaps is the long operating time for completion of this technically demanding operation. With clinical experience and the use of operating teams the free flap has become a reliable mainstay of tissue reconstruction.

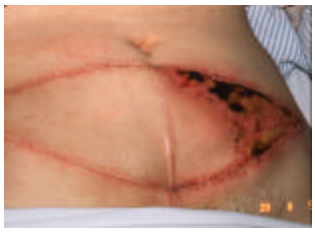
The use of an appropriate graft or flap depends on the experience and judgement of the surgeon. While numerous solutions to a problem wound or defect are often possible, the real art of surgery matches the one most appropriate solution to the patient's needs and risk factors. This can only be done with a complete armamentarium of flaps and grafts on the part of the surgeon, tempered by experience and sound judgement, and an intimate knowledge of the patient's requirements. Only then can an appropriate flap or graft be selected.

THE ANATOMY OF THE RECTUS ABDOMINIS

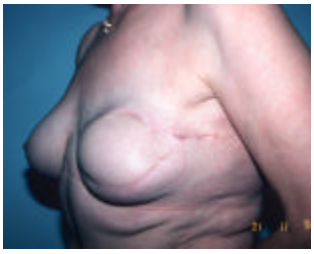




Patient after mastectomy with vertical lower abdominal scar



Rectus abdominus flap delayed showing necrotic area to be discarded



Right breast after reconstruction with rectus abdominus musculocutaneous flap

Clinical Hyperbaric Medicine-Report of Interesting Cases

This section represents a series of real cases which in most instances were evaluated and treated here at Brooks or cases derived from telephonic consultations from flight surgeons or other clinicians in the field. These are chosen for their teaching value and applicability to operational and clinical medicine.

Wound Care:

Brown Recluse Spider Bite

By: Editor

The patient, a navy medical officer, noted a slight pain on the edge of his right ear while reading in bed four days prior to hyperbaric medicine consultation. He had fallen asleep and when he felt the pain he awoke and looked around, never seeing the offending agent. Over the next two days he experienced increasing erythema and mild swelling of the helix and reported to the FSO. He was started on erythromycin and by this time, three dark brown, dry, necrotic appearing spots had appeared in the area of erythema. The patient was referred to Davis Hyperbaric Laboratory for evaluation. His past medical history included borderline glaucoma for which he took Timoptic and Xylantin. In addition to the previous history, he did not smoke, or drink alcohol; imbibed three cups coffee/day.

Physical exam revealed an irregular ~5 cm erythematous patch with multiple black confluent necrotic areas of the external helix extending from the posterior helix anteriorly to the concha.

A presumptive diagnosis was made of Brown Recluse Spider bite and the patient was immediately treated with a wound care dive to 45 fsw on a bid schedule for two days. With each dive, the cyanotic appearance of the tissue pinked up and the area of involvement lessened. There was some intermittent regression of the appearance of the lesion between dives, initially, but after the fifth dive, each successive dive showed consistent progress. There was no change over the subsequent weekend and treatment dives followed for the next week with a plateau of improvement by the ninth treatment, then nearly 2 weeks from the initial injury. HBO was terminated; all the necrotic areas were replaced with newly granulated tissue and wound was contracting. There was no slough of chondral tissue. There was normal sensation and just an eraser-sized spot of erythema present. The patient was discharged from HBO with bacitracin oint q day and will follow up in 1 month. He returned to FSO, ear intact, for follow-up checks.

Decompression Sickness and Other Hyperbaric Indications/Diving:

CO Poisoning in a Pregnant Patient

By: *Editor*

Pt is a 32 y/o G5P3A1 with a 38wk IUP who moved into base housing 3 days prior to presentation. During this time, she had turned up the thermostat due to cold spell in the early fall weather. She complained of flu-like symptoms, eg, nausea, fatigue, irritability, and headache. The illness ran rapidly through the house as all became sick within a day. Same symptoms were present in the husband and all three children were not eating, and fussy.

Mother had no weakness or LOC but had noted less fetal movement. The whole family checked into the BAMC ED with complaint of flu symptoms. They were seen by a sharp intern who quickly picked up on the suddenness of the illness and the fact that none of the family members had any elevations in temperature to go along with their flu symptoms. (Question is, can an elevated temp be a sign of CO poisoning?) A diagnosis of carbon monoxide poisoning was made, labs drawn and O2 by facemask rebreather was started. IV fluids (NS) were given for mother's orthostasis. Mother's COHgb level was 18.7% (after 90 minutes of leaving house, breathing 100 % O2 via non-rebreather mask—how many half-lives could that be? What could have been her immediate post-exposure carboxy-Hgb level). In the ED, fetal heart tones were heard and fetal movement was present. A fetal rhythm strip was run showing decreased beat-to-beat variability. Pt c/o uterine tightness but no cervical changes were noted from last exam and no contractions were palpated nor documented on the strip nor was there any fetal tachy/bradycardia. Pt was current with regular prenatal care, taking prenatal vitamins and iron supplements. OB resident did not perform uterine ultrasound.

The telephone rang across town at DHL with OB requesting an urgent consult. ED had elected to treat the father and kids there with 100% O2 by mask. Pt was brought by ambulance on an estimated 95% O2 via non-rebreather facemask. A concise history was reviewed; her immediate post-exposure COHgb was estimated to have been ~40%, fetus possibly 45%+. PE focused on a 6-part neurological survey and documenting FHT while the team was preparing the chamber. She complained of an occipital headache and nausea. An IVwas started and she was given 2 liters over 2 hours of NS. A snack was obtained from the hyperbaric kitchen to take into the chamber for later when she felt better. Pt's BP was 126/84, FHT were 145. She appeared tired but was normal on neuro testing except for some slowing in verbal responses and complaint of "thinking was funny, like an out of body experience". Within minutes, the patient had been consented, changed into dive clothing, and brought to equivalent of 66 fsw pressure on 100 % O2 per the CO HBO protocol. Her headache and nausea resolved at depth and she was able to tolerate p.o. fluids and crackers so IV was slowed to 100cc/hr. Her facial presentation brightened and verbal responses quickened and were appropriate. Neuro

Carboxy-Calculator

Current CO-Hgb Level	FiO2 95% Hours of O2	Hours of Air	Estimated Incident CO-Hgb Level
18.7	1.5	0	39.27

Instructions: Enter the current carb oxyhemaglobin level, the number of hours the patient received O2, the FiO2, and the # of hours the patient air following the acute CO exposure. The result is the estimated maximum carboxyhemaglobin level at the time of the acute exposure

Estimated T-1/2 (min) 84

exam was normal. Fetal activity was present throughout the hyperbaric treatment. She was admitted overnight for observation and fetal monitoring. She was seen in HBO the next am and had normal vital signs and neuro exam. She reported normal fetal activity. Pt was induced four days after this incident and delivered a healthy infant. Infant has done well in the 1st year of life, meeting all her developmental milestones.

Chokes in a Hypobaric Environment

By: James K Wright, MD, FACS
Hyperbaric Fellow, '99-'00

The morning quiet of the Jefferson Davis Hyperbaric Laboratory was shattered by the intercom announcing "Doc to the chamber! Doc to the Chamber!" "What could this be?" I wondered, as I hurried to the hyperbaric chamber thinking which of our wound care patients had gotten into trouble. No it wasn't here I was wanted but at the altitude chamber where the research subject, a healthy 30 year old male was complaining of substernal chest pain which worsened with taking a deep breath. He had been on 100% oxygen since an hour before ascending to 35,000 feet until now and his chest pain had begun about 45 minutes after reaching altitude. I quickly ran through the differential diagnoses – cardiac pain was unlikely in this healthy subject and clearly indicated that the pain was pleuritic and substernal. It could be oxygen toxicity, but that too was unlikely after only an hour and 45 minutes of O^2, part of which was at altitude. That left the chokes as the likely diagnosis.

During the descent to ambient pressure the symptoms vanished, confirming the diagnosis of the chokes – a fairly rare type of decompression sickness caused by nitrogen bubbles irritating the J receptors in the lungs. As the pressure increased during the descent to 1 atmosphere the bubbles recompressed and went into solution relieving the symptoms. But then there was the danger of decompression sickness returning as more nitrogen bubbles left solution during the re-equilibration process in which the tissues exchanged nitrogen as they readjusted to one atmosphere of pressure. To further ameliorate this situation of inert gas exchange, 100 % O2 is used at depth to drive nitrogen from the tissues, replacing it with a respirable gas. We therefore decided to treat the subject, who by this time was feeling quite well, with a Navy Treatment Table 6 – a five and one half hour hyperbaric treatment schedule specifically used for decompression sickness. He completed the table 6 without any further ill effects of decompression sickness. He was re-examined the next morning and released after having no more symptoms.

The chokes were first described in 1840 by the French engineer Charles-Jean Triger, the first to describe decompression sickness. After descending in a caisson to about 2.5 ATA which he designed for the coal mines of Lyon, Mssr. Triger suffered a certain "breathlessness" or "moins essoufles" about one half hour after ascending. Triger hired two physicians, Drs. B. Pol and T. J. J. Watelle who gave the first medical description of decompression sickness and the chokes and correctly surmised that decompression sickness is caused by too

rapid an ascent and that the symptoms could be alleviated by re-compression.[i] The "chokes" is pulmonary decompression sickness caused by an overwhelming number of bubbles in the pulmonary arterioles. Altitude chamber subjects frequently have small bubbles pass through the pulmonary arterioles to the pulmonary capillaries where they are cleared before the blood enters the pulmonary venous system. Small amounts of bubbles cause no symptoms and are rapidly cleared from the pulmonary circulation. When the bubbles accumulate in sufficient quantity they cause sub-sternal discomfort which worsens by deep breathing and may provoke paroxysms of coughing (Behnke's sign). This is thought to be from stimulation of the J receptors in the lungs.[ii] In cases where there is sudden ascent from depth, such as in divers who become too buoyant, the right heart may be overwhelmed and fail leading to cyanosis and circulatory collapse.

As in other types of decompression sickness, the chokes must be treated by hyperbaric oxygen. Failure to immediately begin treatment may result in progression of the symptoms to the point where the patient is in great danger and may even die. As the bubbles accumulate in the pulmonary vasculature, the leukocytes adhere to the vascular endothelium, release free radicals, and damage the endothelium. The result is pulmonary edema, which may progress to adult respiratory distress syndrome.[2] The chokes, a potentially serious form of decompression sickness, is treated with a U. S. Navy treatment table 6 which can be extended, if appropriate. Fortunately in our case resolution was rapid and no further treatment was needed.

References

[1] Phillips, J. L., *The Bends*, Yale University Press, New Haven, 1998.

[2] Dutka, A. J., and Francis, T. J., *Pathophysiology of Decompression Sickness,* in Bove. A. A. (ed.), *Diving Medicine, 3rd Edition,* W. B. Saunders Company, Philadelphia, 1997.

Editor's Comments

Robert N Bertoldo, DO, MPH

I caution all those reading literature involving an evaluation of the clinical effectiveness of HBO that there is a lot of bad science out there; many poorly designed studies, and unjustifiable "conclusions" made about HBO which do not hold water or are not up to the all-searching eye of scientific investigation. I have decided that there are some ignorant people who have no appreciation for the bias they openly infuse into their "studies" or others who do so purposely to promote their own agendas. It becomes incumbent upon those of us who hear of or read such articles to write to the journal editors and expose this heresy. We cannot fail to do this and with the same breath, complain that someone in our hospital is a non-believer because of some unsubstantiated article they read. I find there is a direct correlation to level of education or experience in HBO and the number of good working relationships with referring physicians/surgeons: termed, Positive Engagement. Bottom line: Positive Engagement translates into more consults and helping more patients heal. There's a lot of good science that yet needs to be done as well as a great deal of education and fostering of relationships, which we all need to promulgate. Best wishes to all readers for Y2K.