Paparella: Volume IV: Plastic and Reconstructive Surgery and Interrelated Disciplines

Section 2: Disciplines Closely Associated With Otolaryngology

Chapter 32: Underwater Medicine: Otolaryngologic Considerations of the Skin and Scuba Diver

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History of Diving

Man, a terrestrial being, has long been entranced by the possibilities of exploring the atmosphere above and the sea below. Historical documentation through more than 1000 years points to the fact that kings and emperors employed breathhold divers to salvage sunken treasure, and offered them a percentage of the value as an incentive.

The open diving bell, an air-containing chamber, was invented in the 17th century, enabling humans to prolong their stay under water. In 1810 Augustus Siebe designed a copper helmet sealed to a watertight canvas suit that allowed fresh air to be pumped down from the surface via a hose; this is known as hard-hat diving.

Jacques-Yves Cousteau and Emile Gagnan designed the "Aqualung" in 1943, which today is known as *S*elf-*C*ontained *U*nderwater *B*reathing *A*pparatus; hence, the popular acronym SCUBA. This device consists of a tank that delivers pressurized air to a diver through a hose and mouthpiece at a pressure comparable with that of the aquatic depth attained. The pressure differential is controlled by a demand regulator attached either at the high pressure tank or at the mouthpiece.

After its initial use in World War II, the low cost and reliability of scuba gear made it the basic equipment of the recreational diver. The duration of a diver's stay under water with scuba equipment is limited only to the amount of air in the tank and the physics that govern his being. The marvels of the deep captured through underwater photography inspired many enthusiasts to see this different world of beauty for themselves.

There are approximately 6 million scuba divers in the world, of whom 4 million are in the United States. Two million of the latter are active, diving over five times each year. There 250,000 sport divers trained in the USA annually.

In addition to scuba, the helmet and diving suit design of more than a century ago was refined by industry and the military as a hookah unit (hard hat) with a surface air supply that limits the diver to approximately 100 foot water depth.

In deeper dives, helium is substituted for nitrogen - a mixture known as heliox - to prevent nitrogen narcosis; however, use of heliox does not prevent helium bends, which necessitates decompression.

In earlier times, military physicians handled the medical problems associated with diving. However, with the ever-increasing numbers of divers entering into the sport, all physicians should familiarize themselves with the medical problems and dangers inherent in skin and scuba diving. Most of the common problems fall within the specialty of otolaryngology.

Physics of Diving

Man's habitat on this earth is at the bottom of a veritable sea of air. The weight of this air at sea level is defined as one atmosphere, and is determined by a 1-sq inch vertical column of air as far as the atmosphere extends. Measured atmospheric pressure is approximately 14.7 pounds per square inch (psi). This is the pressure exerted on the body's surface at sea level by the weight of one atmosphere and is ideal for human life (Box).

Box. Pressure Equivalents at One Atmosphere

1 Atmosphere	=	33 feet of seawater (fsw)
	=	34 feet of freshwater
	=	14.7 pounds per square inch (psi).

Water is incompressible, but air is easily compressible, and the slightest difference in volume because of pressure change can be readily observed. Just as atmospheric pressure is caused by the weight of air, so water pressure is caused by the weight of water. Hence, a body submersed in water experience absolute pressure, or the sum of the atmospheric pressure added to the water pressure. Water pressure alone is recorded as gauge pressure.

Every cubic inch of water beneath the surface weighs 0.445 pounds of pressure (torr). Each 33 feet of water equals one atmosphere or 14.7 psi. The atmosphere above the surface also exerts 14.7 psi, which means that a diver 33 feet beneath the surface experiences 29.4 pounds of pressure per square inch absolute or two atmospheres of pressure. This is equivalent to 14.7 psi or one atmosphere of gauge pressure. In general, divers refer to gauge pressure rather than absolute pressure, since this is what is monitored by the pressure gauge carried by every scuba diver. Figure shows the equivalents in pounds per square inch according to the depths reached. It is interesting to note that 18,000 feet into the atmosphere is equivalent to one-half an atmosphere of pressure or approximately 15 feet under water.

This information and the laws of physics assume paramount importance for the physician who treats divers. Included in these laws are the classic contributions of Boyle, Dalton, and Henry.

Boyle's law states that at a constant temperature the volume of a body of gas varies inversely to the pressure to which it is subjected ($P=K/V$), where K is a constant. Most otolaryngologic problems encountered by the diver fall within the scope of Boyle's law, because of the air-occupying spaces of the middle ear and sinuses. While tissues are composed primarily of water, which is nearly incompressible and thus minimally affected by pressures of underwater diving, the body's gases are compressible, with the resultant possibility of barotrauma (injury due to changes of barometric pressure).

Gas volume changes are most noticeable to the diver near the surface. When a diver reaches the depth of 33 feet of sea water, the pressure is one atmosphere gauge. As the pressure is doubled, the volume is halved. Figure depicting Boyle's law shows how essential the equalization of pressure becomes to the body air spaces of the diver, both in descent and in ascent. The risks of diving, whether in shallow or in deep water, are due largely to the breathing of gases as the ambient (surrounding) pressure at the level of the dive increases during descent or decreases during ascent.

Skin diving, also known as breathhold diving, free diving, or snorkeling, employs only a mask, flippers, and snorkel and is generally limited to between one and two atmospheres (33 to 66 feet) in depth. For the skin diver who is holding his breath, reaching 33 feet means that the lung volume has decreased to half its size because the ambient pressure has doubled. At 66 feet, the lungs are one-third their size in volume while the pressure is tripled. For the sake of simplicity, the numbers of pressure under water in the illustrations are rounded off to whole numbers.

Boyle's law has a different application to the scuba diver who is supplied with air from the tank through the open glottis to the lung at a pressure that equals ambient pressure. Thus, at 33 feet the scuba diver's lung volume remains the same, but the external pressure is doubled. At 66 feet the ambient pressure is tripled and at 99 feet it is quadrupled, although the lung size does not change.

Inspiration becomes more difficult as depth increases because the molecules of air are compressed; therefore, more molecules of air must flow through the orifice of the regulator and the upper airway for each breath. This difficulty is the result of increasing ambient pressure on air in the tank and in the lungs.

Dalton's law is concerned with partial pressures of individual gases and states that "in a mixture of gases, the pressure exerted by each gas is the same as it would exert if it alone occupied the same volume. The total pressure is the sum of the partial pressures of the component gases". In other words, as the ambient pressure increases, the total pressure of a gas mixture increases even though the percentage remains a constant. Thus, the partial pressure becomes the important consideration in the biologic effects of gases, because both diffusion and the amount of gas dissolved in a solvent are proportional to the partial pressure of each individual gas in the mixture.

It is important to keep in mind that the air we breathe from the tank is made up of approximately 78 per cent nitrogen, 21 per cent oxygen, and trace units of rare gases. Supplying the proper amount of oxygen to the tissues of the body depends on the partial pressure of oxygen, not on the total gas pressure. During the metabolic process, carbon dioxide is formed. The percentages of carbon dioxide and nitrogen are also dependent on the partial pressure of each of these respective gases.

The calculations of Dalton's law illustrate the danger of oxygen toxicity to the diver using compressed air and descending to great depths. At 132 feet below sea level the partial pressure of oxygen in the scuba air tank is equivalent to breathing 100 per cent pure oxygen at sea level (760 torr). Since the normal partial pressure of oxygen is much lower (160 torr), such a concentration of oxygen at this depth is toxic to the body, especially the lungs, and

can be fatal.

Henry's law states that at a given temperature the mass of a gas dissolved in a given volume of solvent is proportional to the pressure of the gas with which it is in equilibrium. Scuba diving application of this law is important in considering the increasing amounts of nitrogen that dissolve in the body fluids and tissues during descent and come out of solution during ascent.

Deep sea divers can experience nitrogen narcosis, commonly known as "rapture of the deep", which is an intoxication-like phenomenon. Its explanation was given by Captain Albert Behnke, who found that nitrogen under pressure acts as an anesthetic. The deeper the depth of the dive, the greater is the pressure pushing nitrogen gas into the body fluids and tissues, according to Henry's law. For every 50 feet of descent, a scuba diver breathing tank air experiences the equivalent of drinking one martini on an empty stomach. Thus, at 150 feet, the sport diver would be subjected to the intoxicating effects of three martinis. The phenomenon is commonly known as "Martini's law". For this reason, commercial deep divers substitute helium (heliox) for nitrogen, which does not have an anesthetic effect. For the sake of safety, it is most important that the sport scuba diver limit the depth of the dive.

Physiology of the Ear

The very configuration of the ear, sinus, and contiguous structures makes them susceptible to diving injuries. The ear and eustachian tube are the weak links in the anatomy of a diver.

Well over 50 per cent of the medical problems related to diving are within the specialty of the otolaryngologist, and 90 per cent of these involve the ears.

The normal ear maintains a pressure equal to that of the immediate environment. The eustachian tube, which ventilates the middle ear, provides the only means of equalizing pressures of ascent and descent in diving. The nasopharyngeal ostium of the eustachian tube is normally closed except when there is positive pressure in the nasopharynx, or when it is opened by the muscular contraction of the tensor veli palatini, levator palatini, or salpingopharyngeus. Armstrong and Heim (1937) demonstrated that upon descent the eustachian tube acts as a flutter valve and remains closed under pressure, resulting in middle ear barotrauma or "squeeze", unless the diver opens it reflexively or voluntarily. Upon ascent, as the volume of air increases in the middle ear, the eustachian tube usually opens with little difficulty, venting the air.

Because the middle ear does not equalize automatically, every diver must pressurize the ears to ambient pressure. The most popular method is the Valsalva maneuver, so called after the eighteen century anatomist Antonio Valsalva. By gently holding the nostrils, closing the mouth, and exhaling against the closed glottis, most divers can readily inflate air up the eustachian tube to the middle ear space. Common conditions predisposing the diver to middle ear squeeze include upper respiratory infections, uncontrolled nasal allergy, obstructing nasal polyps, and deviation of the nasal septum.

Major Problems of SCUBA Diving

The major problems of the scuba diver can be categorized as (1) problems occurring during descent, (2) problems occurring during ascent, and (3) other conditions that may occur at the depth of a dive.

Problems Occurring During Descent

External Ear Canal

When the external auditory canal becomes occluded by impacted cerumen, ear plugs, or a tightly fitting hood, a diver may develop pain in the ear during descent. Air pressure in the ear canal deep to the occlusion remains the same as surface pressure, while the surrounding ambient pressure and middle ear pressure increases. This vacuum or negative pressure in the ear canal causes a "squeeze" or congestion of the skin of the canal and tympanic membrane, resulting in petechiae. As the pressure increases, hemorrhagic bullae can form in the skin and tympanic membrane. These may rupture and cause bleeding within the canal as well as perforation of the tympanic membrane. The amount of pain experienced by a diver suffering external ear barotrauma can range from minimal to considerable, usually varying with the amount of pressure it takes to rupture the bullae. Prevention can be accomplished by never using ear plugs or occluding the external ear canal. Unless infection develops, it is unnecessary to treat this type of injury, except to provide symptomatic relief for the pain and swelling. However, it is important that the diver stay out of the water until the symptoms disappear and medical clearance is received.

A very common problem among divers who are in the water frequently is external otitis (swimmer's ear). Some divers aggravate the condition by overzealous use of cotton applicators to dry the ear canals after a dive.

A diver who is prone to frequent external otitis should fill the ear canal after each dive with an acidified alcohol solution such as Swim Ear. This author provides an instruction sheet advising an inexpensive mixture of white household vinegar (1 tablespoonful) in 4, 5, or 6 ounces of 70 per cent isopropyl alcohol. This may be followed by one of the antibiotic-corticosteroid ear drops, if indicated.

For a diver who has a propensity for otitis externa, silicon oil, such as Dow Corning 200-pound fluid or Silicone spray, can be used as a protective coating for the skin of the ear canal before diving or swimming.

Box. Instruction sheet given to divers who are subject to otitis externa (swimmer's ear).

Instructions for Care of the Ears After Swimming

Some people are susceptible to inflammation or infection of the ear canal skin after swimming or getting water in the ears.

A. When shampooing the hair, seal the outer ear canal as follows:

1. Take a flap piece of cotton or lambswool, approximately 1 inch square.

2. Rub plain Vaseline into both sides of the square.

3. Roll the square into a ball and plug into the ear canal.

B. After swimming, the water can be removed and the ear canal dried by the following procedure:

1. In a 4 or 6 ounce bottle place 1 tablespoon of white vinegar and fill with isopropyl (70%) Rubbing Alcohol.

2. Warm the solution by placing the bottle in a basin of hot tap water.

3. Lie on your side and fill the uppermost ear canal with the solution for 2 to 3 minutes.

4. Cover the ear with a folded towel and turn over so the towel absorbs the solution as it runs out of the ear.

5. Repeat the procedure for the opposite ear.

6. The alcohol solution may burn slightly. If it is painful or burns excessively, this means that the skin surface is broken and is a warning signal. Do not go swimming for 48 hours. After this period of time, try the solution again. If there is no burning, swimming is permitted.

Middle Ear

Middle ear barotrauma (aerotitis media), commonly known to divers as "middle ear squeeze", is one of the most common complaints of the scuba diver. This condition occurs on descent when the eustachian tube does not allow air to enter the middle ear space. Air in the middle ear remains at surface pressure while the diver's tissues and the air in the external ear take on the ambient pressure of the surrounding water at that depth. The first 33 feet beneath the surface are the most critical in the diver's compensation for pressure changes. A diver who is unable to pressurize the middle ear can develop symptoms just under the surface of the water, and consequently will be unable to continue the descent without severe otalgia and possible rupture of the tympanic membrane. This can occur with a pressure differential

of as little as five psi and in just a few feet of water. The diver experiences hearing loss and frequently dizziness. The eardrum appears hyperemic and retracted, with possible hemotympanum from rupture of the delicate capillary network of the middle ear mucosa.

Measures Recommended for Preventing Middle Ear Squeeze

Decongestant Ear Squeeze. Oxymetazoline 0.05 per cent (Afrin, Duration, Neo-Synephrine 12-hour, Dristan 12-hour) is well tolerated, with a 10- to 12-hour duration of effectiveness. An illustrated instruction sheet is given to divers who have difficulty with middle ear squeeze. They are advised to spray the nose 20 to 30 minutes before entering the water, wait 5 minutes, and then repeat the spraying procedure. The first application opens the anterior nasal passages; the second reaches the sinus and eustachian tube ostia. Emphasis is placed on positioning the head facing the floor with the spray bottle aimed toward the back of the head, in order to effectively deliver the vaporized spray to the ostia of the eustachian tubes, as illustrated.

Box. Instruction sheet given to divers who are subject to barotrauma (middle ear squeeze). The nose spray is used 20 to 30 minutes before diving.

Instruction for Use of Nasal Sprays

1. With the head ERECT point the spray bottle in the direction of the EYE and squeeze briskly so the spray will come out in a fine mist.

2. With the head FACING THE FLOOR point the spray bottle toward the TOP of the EAR and spray briskly.

3. Wait FIVE minutes - this will allow time for the FRONT nasal passages to open. The repeat procedures 1. and 2. to open the BACK nasal passages.

4. When blowing the nose always do so GENTLY and with the MOUTH OPEN.

Do NOT use nose drops or nose spray for more than one week.

Systemic Decongestant. Pseudoephedrine 120 mg (Sudafed 12-hour, Afrinol) can be taken every 12 hours, or phenylpropanolamine 25 mg (Propagest) every 4 hours.

An antihistamine is prescribed in combination with the decongestant if the patient has allergies; however, the latter is contraindicated if drowsiness occurs. There are antihistamines available for the allergic diver that rarely cause somnolence (Seldane, Hismanal). It is imperative that all medications that are taken for the first time be used on a trial basis well in advance of the dive, to avoid adverse effects that could result in an underwater tragedy. For example, oxymetazoline is an excellent, long-lasting decongestant nasal spray that is usually effective when used twice a day for 5 to 7 days without resulting in rebound hypercongestion and complete obstruction of the nasal passages. On rare occasions a susceptible diver may experience an idiosyncratic reaction to the first use of the spray, causing rebound. If this happens at depth, the diver is unable to equalize the pressure change that occurs during ascent, and there is resultant distention of the tympanic membrane from

expansion of air in the middle ear according to Boyle's law. This is known as "reverse squeeze".

Valsalva Maneuver. Autoinflation of the middle ear space is accomplished before the dive is begun, either on the deck of the boat or preferably on the surface of the water after leaving the boat. It is better to slightly overinflate the middle ear before the descent, since the greatest change of volume per unit of descent occurs close to the surface. The diver should be cautioned against too vigorous autoinflation because of the possibility of trauma to the inner ear with resultant permanent hearing loss. This type of sudden deafness is usually in the high frequency range, is frequently associated with tinnitus, and fortunately is most often unilateral. Although the mechanism is not clear, it has been proposed that exhaling too forcefully against the pinched nose and closed glottis can lead to an increase in thoracic and abdominal pressure, which is transmitted to the cerebrospinal fluid (CSF) by engorged veins of the spinal column. The increase of pressure in the CSF is transmitted to the fluids of the inner ear by way of the endolymphatic and cochlear ducts. The perilymphatic pressure elevation can result in the rupture of the round or oval window membranes. It is during this process that permanent damage can occur to the inner ear. An alternative, safer method of equalizing the middle ear pressure is the Frenzel maneuver, which involves increasing pressure within the pharynx by holding the nose, closing the glottis, and at the same time contracting muscles of the pharynx. Thus, air can be forced up the eustachian tube without a change in thoracic pressure. Since the oral cavity is blocked by the tongue being pressed against the soft palate during this procedure, the Frenzel maneuver can be accomplished with the mouth open or with a regulator in place.

Feet-First Descent. This author has long pioneered descending under water feet first because it is easier to inflate the middle ear in the head-up position, there being less gravitational hyperemia of the mucosa around the eustachian tube orifice. In addition, the diver pays more attention to effecting middle ear inflation of air than to the surrounding environment. Autoinflation should be accomplished at every 2 to 3 feet of descent. Since it is difficult to judge change of depth under water, it behooves the diver to descend down the anchor line if diving from a boat, to determine more precisely every few feet when autoinflation is important. If a diver experiences a squeeze, he should ascend several feet, which will decrease the volume of the air in the middle ear, thereby relieving the negative pressure so that he can work on autoinflation.

There are other ways to facilitate middle ear inflation:

1. A mask that form-fits around the nose allows for more effective nasal compression. Some divers can easily accomplish autoinflation by the mere act of swallowing.

2. Jaw and head movement. Sometimes it helps to move the mandible from side to side, or to stretch the neck, extending the head with the troublesome ear facing upward.

Although most barotrauma occurs on descent, a reverse squeeze may occur on ascent. If the eustachian tube remains blocked, the gradient of pressure in the middle ear will increase as the volume of air expands during ascent, resulting in progressively severe otalgia often associated with dizziness. Reverse squeeze could cause a diver to suffer middle ear hemorrhage, a ruptured ear drum, or even permanent hearing loss. Although the pain can be

relieved by descent, the diver will eventually exhaust the air supply and must return to the surface. For this reason it is imperative that the scuba diver who plans a safe dive should start the ascent with a minimum of 500 pounds of air reserve in the tank.

Treatment for Barotrauma of the Middle Ear

The scuba diver who has had problems equalizing middle ear pressure should refrain from entering the water until receiving medical clearance. Mild middle ear squeeze associated with minimal symptoms such as fullness or slight pressure in one or both ears usually resolves promptly with no treatment. Persistent blockage usually clears in 10 days or less with the aid of a systemic decongestant such as pseudoephedrine (120 mg every 12 hours). The addition of an antihistamine is indicated only if the diver has allergies for which the specific medication has a history of proven effectiveness; however, this class of medication may dry and thicken the secretions, slowing resolution. The combination of a mucus-thinning agent with the decongestant (Guanifed or Entex LA every 12 hours) may more effectively resolve the condition.

It should be kept in mind that a medication that works well for one diver is liable to have an adverse reaction on another. This is particularly true for antihistamines, which produce varying effects from extreme drowsiness to hyperexcitability. The potential impairment of a diver's judgement under water could become life threatening. It is not uncommon for divers to question their buddy about the medication they observe being taken, whereupon the other diver extols its effectiveness in preventing barotrauma and freely offers the medication. Obviously, this is extremely hazardous before diving. Any antihistamine, decongestant, nose spray, or other medication that has never been used in the past must be taken on a trial basis at least a day, or preferably several days, before a dive to make sure that no adverse side effects will occur under water. When in doubt, the diver should check with his physician.

Daily, periodic autoinflation is advised as long as blockage persists and there is no associated discomfort. Persistent otalgia usually indicates a middle ear infection requiring medical attention and antibiotic therapy in addition to the aforementioned treatment.

For continued severe pain with the findings of a bulging eardrum on physical examination, myringotomy is indicated. If fluid persists in the middle ear over an extended period, myringotomy with the insertion of a ventilation tube may be necessary. The judgement for surgical intervention is based on the same criteria as those for otitis media and serous otitis of any other cause.

When there is a discharge, the tympanic membrane is usually perforated, and the addition of otic drops combining topical antibiotics with a steroid may speed resolution. External otitis as the etiology of otalgia can be ruled out in most cases if there is no pain associated with tragal movement; however, both external otitis and otitis media may be present at the same time. Water should not be allowed to enter the ear until the diver receives medical clearance. On rare occasions a large perforation may not close on prolonged conservative medical management, necessitating tympanoplasty. Scuba divers are frequently advised to abandon future underwater activities after the surgical repair of a perforation. The experience of this author, having treated a significant number of divers who have had this

complication, is contrary to this opinion. With the use of tragal perichondrium, which provides a thin, strong graft, the repaired area has been found to be stronger than other areas of the tympanic membrane. Once the drum is repaired, the diver must pay meticulous attention to preventing middle ear squeeze by the methods previously described. If self-inflation is not possible or is very difficult, which is rarely the case, scuba diving should be abandoned.

Inner Ear

Middle ear barotrauma may also seriously damage the inner ear. If a diver does not successfully inflate air up the eustachian tube upon descent, the middle ear will remain at sea level pressure, while the tissues, nasopharynx, and ear canals take on full ambient pressure at the depth of the dive. The external pressure of water in the ear canal, as well as negative pressure in the middle ear space, result in depression of the tympanic membrane, which is transmitted through the ossicular chain to cause pressure changes in the inner ear by depression of the stapes footplate. If the diver is suddenly able to perform a Valsalva maneuver, the air rushing up the eustachian tube has an abrupt, outward force on the tympanic membrane and chain of ossicles. The sudden pressure wave in the inner ear created by the pull on the stapes footplate results in distortion of the inner ear membranes. Simmons postulated that the sudden increase in intracranial and intralabyrinthine pressure causes a rupture, break, or dislocation of structures such as Reissner's membrane, the basilar membrane, the saccule, the utricle, or the semicircular canals. Eichel and Landes theorized that the inner ear damage is either a result of the shearing force of the rapid fluid movement in the membranous labyrinth and cochlea or due to hemorrhage from torn blood vessels.

In addition to intracochlear structural damage, air rushing up the eustachian tube forces the tympanic membrane and consequently the stapes footplate outward. The negative pressure produced by the pull of the stapes is transmitted as a shock wave through the scala vestibuli, passing through the helicotrema to the scala tympani, and resulting in the bulging inward of the round window membrane with the possibility of "implosion" and leakage of perilymph into the middle ear. The outward pull on the stapes footplate can also result in a tear in the annular ligament of the oval window or the development of a crack in the footplate. Anson and colleagues demonstrated that the fissula ante fenestram is one of the weakest points in the otic capsule, making the anterior area of the oval window more susceptible to fracture.

Goodhill proposed that the mechanism of membrane rupture is either the result of a forceful Valsalva maneuver against a blocked eustachian tube or of straining, which can occur in both diving- and non-diving-related injury. An elevation of CSF pressure is transmitted through a patent cochlear aqueduct or (less likely) through the internal auditory meatus, raising intracochlear fluid pressure. If there is a sufficient pressure differential between the perilymphatic space and the middle ear, an "explosive" outward rupture of the round window membrane or the oval window ligament will occur into the middle ear space with leakage of perilymph. If, on the other hand, the eustachian tube suddenly opens, an "implosion" can occur as previously described.

Pullen reported a round window fistula in a 40-year-old diver who developed a sudden sensorineural hearing loss 3 days after a 30-foot asymptomatic dive. The hearing returned to normal after middle ear exploration and surgical repair. He described the round window

membranes, in three cases of fistulae that he explored, to be quite visible and less angled than usual, there being no true niche of the window.

Schuknecht and Gacek reported a 21-year-old patient who experienced a plugged sensation and sharp pain in one ear, with a loss of hearing as well as vertigo, nausea, and vomiting, shortly after a 12-foot dive. The vertigo resolved in 2 days, whereas the sensorineural hearing loss persisted. One week after the injury, the diver sought medical attention, and immediate exploration revealed a marginal linear tear in the round window membrane. Hearing progressively improved after repair of the fistula except for a persistent high-frequency sensorineural loss.

Healy and colleagues reviewed 40 cases of perilymph fistula, finding the chief complaint to be episodic as well as positional vertigo. Hearing loss and tinnitus were less frequent symptoms. Surgical exploration revealed 31 oval window leaks, five round window leaks, and four leaks involving both windows. These authors advised that high-power magnification may be necessary to identify the draining fistula; in some cases, it took as long as 5 to 10 minutes before perilymph reappeared after blotting the window. In some patients, bilateral jugular compression was necessary to precipitate the flow. In this series, no patients with a hearing loss of more than 3 weeks' duration regained significant hearing, which argues for early exploration, especially when there is a history of deterioration in hearing.

Zannini and associates followed 160 professional divers with serial audiograms. Compared with a control population, the divers had significantly worse hearing, the greatest loss occurring in the group with the longest duration of diving activity. The frequencies most affected fell in the 4000- to 8000-Hz range; divers who had difficulty autoinflating suffered the most severe sensorineural hearing loss.

Edmonds and colleagues described bilateral round window fistulas in a diver whose hearing subsequently improved significantly in both ears after repair.

Paparella and associates reported that traumatic round window perforations in 36 chinchillas healed spontaneously and completely in 9 days; all but one had healed by the seventh day.

MacFie studied barotrauma in Royal Canadian Navy divers in whom he documented the occurrence of both hearing loss and vertigo, as well as hearing loss without vertigo, and vertigo without hearing loss. The loss of hearing usually occurs immediately but may develop over the subsequent few days. It is sensorineural, most often involving the high frequencies, but it can occur in all frequencies. A conductive component may also be present if there is a barotrauma involving the middle ear. Tinnitus, nausea, and vomiting are frequently present and otoscopic examination may be normal. Tinnitus is very often permanent when the high-frequency sensorineural hearing loss persists.

Divers with a loss of hearing and dizziness following a scuba dive that does not require decompression should be considered to have inner ear barotrauma necessitating immediate medical management. This includes bed rest with the head elevated and avoidance of any stressful act such as coughing, nose blowing, autoinflation, or straining that would cause an increase of pressure in the CSF, which is transmitted to the perilymphatic fluids.

Parell and Becker emphasized that since the history and physical findings for both middle and inner ear barotrauma may be very similar, air and bone conduction audiometry is imperative for differentiation. This should be accomplished as soon as possible because treatment of these forms of barotrauma may be entirely different. It is possible for both inner and middle ear barotrauma to coexist, in which case audiometry reveals a mixed conductive and sensorineural loss. The hearing loss documented in cochlear injury is usually nonprogressive and sensorineural, with a greater drop in the high frequencies. The sensorineural hearing loss in cases of perilymph fistula may be progressive or fluctuating, with either a flat audiogram or more high-frequency deficit. Surgical exploration of the middle ear was the only positive means of differentiation between perilymph fistula and cochlear damage before the introduction of moving platform posturography, because all other fistula tests based on the vestibulo-ocular reflex have not correlated well with tympanotomy findings. Black and colleagues reported a 97 per cent test sensitivity when measuring the isolated vestibular control of postural reflexes after the removal of both visual and support-surface orientation references. Sinusoidal changes in external auditory canal pressures produced an increased and, at times, phase-locked postural sway in patients with perilymph fistulas subsequently confirmed by surgical exploration. Since tympanotomy is contraindicated for patients in whom there are no clinical indications for surgery, Black and colleagues submitted that the determination of absolute specificity by posturography evaluation is not possible. If the loss of hearing is progressive or nonserviceable, tympanotomy is indicated.

Parell and Becker divided cochleovestibular (inner ear) barotrauma into the following categories:

1. Hemorrhage within the inner ear (the basal turn of the cochlear is the most vulnerable site).

2. Rupture of Reissner's membrane with the mixing of endolymph and perilymph.

3. Fistula of the round or oval window.

4. Mixed pathology, involving any combination of the above three.

The vestibular symptoms associated with inner ear hemorrhage are transitory. The sensorineural hearing loss may be diffuse and mild, moderate, or severe, usually sloping to the high frequencies. This tends to stabilize or return to normal during the subsequent 2 to 3 months. If there is no progression of the hearing loss or increased severity of vestibular symptoms, nonstrenuous activity can be resumed in 2 weeks and full activity in 6 to 8 weeks.

The forms of medical management that have been recommended for inner ear barotrauma include vasodilators, histamine, steroids, and inhalation of a mixture of 95 per cent oxygen and 5 per cent carbon dioxide (carbogen) to increase cerebral perfusion and oxygenation. Antibiotics are not necessary unless otitis media is present.

Other Otolaryngologic Problems Associated With Barotrauma

Aerosinusitis. If the ostium of a sinus becomes occluded during descent, a diver usually develops barotrauma of the sinus, commonly known as "sinus squeeze". Equalizing pressure changes in the sinuses is usually much less problematic than in the middle ear, unless the sinus ostium is obstructed by hypertrophied mucosa, as during upper respiratory infection (with or without sinusitis) or uncontrolled nasal allergy. Other conditions predisposing a diver to sinus squeeze include obstructing nasal polyps or deviation of the nasal septum. Only a slight pressure differential across the sinus wall may lead to a negative pressure or vacuum in the sinus, resulting in edema of the sinus mucosa and possible hemorrhage into the sinus cavity, accompanied by immediate pain. The pain of sinus squeeze is frequently excruciating during the first few feet of descent under water, forcing the diver to terminate the dive.

Should a diver feel the sensation of fullness or pain over the upper teeth or sinus cavity, or a numbness in the front of the face, he should immediately abort the dive and return to the surface. Treatment for aerosinusitis is the same as that for aerotitis media. Other methods of treatment include management of nasal allergy or surgical intervention by maxillary sinus lavage or frontal sinus trephine, nasoantral window, nasal polypectomy, functional endoscopic sinus surgery, and submucous resection.

Aerodontalgia (Tooth Squeeze). The most common cause of pain in the teeth while descending under water is aerosinusitis; however, a sharp sudden pain may occur in a tooth because of a minute air pocket beneath a filling or dental cap (Boyle's law). Diving may need to be discontinued until the filling or cap is replaced. Aerodontalgia is not a common problem.

Face Mask Squeeze. Face mask squeeze occurs through failure to equalize the pressure change in the air-containing space of the face mask during descent. The diver may incur tissue damage from the resultant pressure differential between the air pocket in the rigid mask and the flexible tissues of the face. The most vulnerable tissues are those around the eyeball and the lining of the eyelids. Such a problem can easily be prevented by exhaling through the nose into the face mask during descent; most divers do this subconsciously.

Epistaxis. Bleeding from the nose into the mask under water is a fairly common occurrence that most uninformed divers attribute to bleeding from the sinuses. In fact, the bleeding results from pressure changes on the small vessels of the anterior nasal septal mucosa. Because of the selective absorption of the color spectrum by the blue-green water, which acts like a photographic filter, the blood in the diver's mask appears bluish-green until the diver surfaces and realizes what has happened. In over 20 years of diving, this author has never seen or heard of epistaxis that continues at the surface of the water or is not controlled by a few minutes of external nasal compression. Epistaxis of this type is not a contraindication to scuba diving.

Alternobaric Vertigo. Alternobaric vertigo is a transient vestibular dysfunction that was described by Lundgren in 1965 as occurring during ascent because of unequal equilibrium of pressure between the right and left middle ear spaces. In a series of 2053 Swedish divers, he found that 16.7 per cent of the group most probably experienced this type of dizziness. Divers who cannot easily autoinflate their ears are at risk. Edmonds and associates reported

this unequal vestibular end-organ stimulation occurring during descent. The duration of vertigo may be a few seconds to 10 minutes and is usually dominant in one ear. Reversing the direction of the movement of the body usually results in disappearance of the vertigo. Hearing loss and tinnitus do not occur with alternobaric vertigo. Diving should not be attempted if vertigo is experienced at the surface when the middle ear space is autoinflated or if clearing the ears is difficult.

Becker described a case of facial paralysis associated with alternobaric vertigo that resolved completely upon return to the surface.

Unequal Caloric Stimulation Vertigo. Vertigo can occur from cold water entering only one ear canal because of obstruction of the opposite ear, producing unequal caloric stimulation of the vestibular system. A diver is usually aware of unilateral blockage in hearing and seeks medical attention to resolve this annoying condition long before attempting to dive. A preexisting peripheral vestibular deficit in one ear can also produce vertigo; however, the diver experiences this symptom on the surface or in shallow water, and will most likely have had otolaryngologic evaluation including audiometric and electronystagmographic confirmation of the condition.

Anxiety. Anxiety is the most common cause of dizziness not related to the vestibular mechanism. A certain degree of apprehension, experienced by all novice divers, progressively resolves with experience. Strauss emphasized that panic is the most frequently listed serious problem in diving. Statistically, in 80 per cent of scuba diving deaths in Los Angeles County, panic was the underlying cause. Egstrom showed that the leading cause of scuba deaths is seen in divers who are in marginal medical or physical condition and become exhausted when they panic during a minor emergency. The University of Rhode Island, which is a clearing house for the reporting of scuba diving accidents, has documented that panic is associated with half of all diving accidents. Education of the diver is the most important way to prevent panic.

Temporomandibular Joint Syndrome. Otalgia from too forceful clenching on the mouth piece of the regulator or snorkel can result in a temporomandibular joint (TMJ) syndrome. This condition occurs more frequently in the anxious novice. In addition to pain, the diver can experience fullness or stuffiness of the ear as well as tinnitus and (less frequently) unsteadiness. If there are no other findings of external or middle ear pathology on physical examination, the diagnosis of TMJ syndrome is usually made by palpation of the joint while the mandible is opened and closed or moved from side to side. In most cases, little more than reassurance is necessary. Prolonged discomfort is relieved by application of heat to the joint, the use of analgesics, and a soft diet. A muscle relaxant is rarely indicated. For prolonged symptomatology, dental evaluation is necessary. A customized mouth piece for the regulator and snorkel can be fabricated for recurring problems.

Problems Occurring During Ascent

Pulmonary Overpressure Accidents

If a diver holds the breath during ascent, the air in the lung expands according to Boyle's law, resulting in distention of lung tissue with damage and possible rupture of the pulmonary alveoli. Pulmonary overpressure accidents can also occur in divers with obstructive lung disease such as asthma or pulmonary blebs in which the diver is susceptible to localized pulmonary air trapping. The escaping air can cause one or more of the medical problems described below:

Mediastinal Emphysema. The escaping alveolar air may dissect along the bronchi into the mediastinum, producing progressive discomfort in the chest and neck with shortness of breath as the predominant sign. This condition is not usually critical, and recompression in a chamber is not necessary unless an air embolism is present. The diagnosis can be confirmed by chest x-ray. The only recommended treatment is 100 per cent oxygen if breathing is impaired.

Subcutaneous Emphysema. The alveolar air may continue to dissect along the bronchi and trachea into the neck, producing the sensation of fullness with retrosternal discomfort, changes in the voice, and labored breathing. Air bubbles under the skin cause the crackling sensation of crepitus when the skin is touched. The onset of symptoms for both mediastinal and subcutaneous emphysema is frequently delayed. Like mediastinal emphysema, the condition is not critical unless associated with an air embolism. It can also be treated with 100 per cent oxygen.

Pneumothorax. If the air from the alveoli ruptures into the pleural cavity, the diver develops sudden, rapid, labored breathing with shortness of breath and mild to severe pleuritic pain. A nonproductive cough may be associated with these symptoms. Since the condition of the diver may be critical, prompt medical attention is indicated. The diagnosis is made by percussion and auscultation of the chest and confirmed by chest x-ray. Depending on the size of the pneumothorax, placement of a chest tube may be necessary. As an emergency measure for a life-threatening situation away from a medical facility, a large-bore needle (15 gauge) with a condom attached to the hub by a rubber band is inserted into the pleural cavity. An opening is made in the condom, which than acts as a one-way valve to vent the buildup of air pressure. Chamber recompression and the administration of 100 per cent oxygen is advisable.

Air Embolism. This is the most serious complication that can occur during scuba diving and frequently results in catastrophic neurologic complications. The diver often loses consciousness shortly after surfacing, if not before. Bloody, frothy sputum is usually present as well as shortness of breath, or, more commonly, cessation of breathing. The onset is sudden, with possible symptoms of chest pain, confusion, dizziness, fatigue, weakness, and blurring of vision. The diver may stagger and complain of feeling faint; this may progress to the development of shock. Air embolism occurs when alveoli and pulmonary blood vessels rupture, permitting air bubbles to be carried by the bloodstream to other parts of the body. When these bubbles enter the cerebral circulation, they may coalesce into larger bubbles blocking the cerebral blood flow, which results in an acute emergency (cerebral air

embolism). Immediate transportation to a chamber is critical in order that recompression be instituted to reduce the size of the bubbles and facilitate circulation before cellular brain damage occurs. During transportation to the chamber, if necessary 100 per cent oxygen should be administered and cardiopulmonary resuscitation given. Since air rises, the patient should be positioned with the head and chest low to discourage bubble flow to the cerebral circulation. Furthermore, the body should be positioned with the left side down so that outflow from the left ventricle is not blocked by frothy blood. Evacuation by air from the dive site to the recompression facility may result in enlargement of air bubbles because of the unavoidable elevation in altitude with a decrease in pressure (Boyle's law); however, the speed of the journey to the recompression chambers makes the risk worthwhile. The aircraft should fly at a low altitude to minimize air bubble growth. Residual problems resulting from air embolism may include any of the signs and symptoms of a stroke and may require repetitive hyperbaric oxygen treatment for several days up to 2 weeks.

To prevent pulmonary overpressure accidents, it is imperative that the diver never hold the breath under water, especially during ascent. Diving should not be permitted for a person with a history of obstructive lung disease (forced expiratory volume (FEV) < 80 per cent) or radiographic evidence of air trapping. If there is doubt, a pulmonary specialist should be consulted.

Decompression Sickness

Decompression sickness was first described in the mid-1800s in caisson workers who built bridge foundations in the compressed air environment of tunnels or caissons. Upon returning to the surface, some workers noticed steady, boring pain in the hip and knee joints. These men limped in a manner described by their fellow workers as "doing the Grecian bend", a stooped posture assumed by tightly-corseted fashionable ladies of the 1800s. Thus, decompression sickness was called "the bends" or "caisson disease".

The mechanism of this condition is related to the partial pressure of the inert gas nitrogen, making up 78 per cent of the air that is breathed from the scuba tank. Under water the partial pressure of this gas is increased, leading to absorption of nitrogen by the blood, which delivers the gas under increased pressure to the tissues. The amount of nitrogen gas absorbed by the tissues of the body is in direct relationship to both the depth and the duration of the dive. In other words, the deeper the dive, the greater the pressure gradient there is from lungs to blood to tissues, and the longer a diver stays at depth, the more time the gradient of pressure has to push the nitrogen gas in the direction from the lungs into the tissues.

As the scuba diver ascends to the surface from a routine dive, the partial pressure of the nitrogen gas is decreased, resulting in a progressive elimination of the dissolved nitrogen in the direction from the tissues to the circulating blood to the lungs where it is blown off: a process known as outgassing.

If, however, the diver remains at depth for an extended period, the blood and tissues become saturated by the inert nitrogen gas. If the diver then returns directly to the surface after the tissues are saturated, the nitrogen gas comes out of solution in the tissues as small bubbles because of the rapid reduction of partial pressure. In fact, the tissues of the body at the surface suddenly become supersaturated because the surrounding pressure is less than the

total gas pressure within the tissues. The scuba diver who is supersaturated should ascend to 10 or 20 feet or to a depth where there is enough pressure to hold the nitrogen gas in solution, but allow for outgassing at the diminished partial pressure of nitrogen at that depth. Underwater decompression tables have been worked out to determine the time that a diver must remain at specified shallow depths, after which return to the surface is permitted without the risk of developing decompression sickness. Figure indicates the relationship of the limits of depth and time for scuba diving to avoid the bends. These "no decompression limits" are the maximal times that the diver can remain at various depths, after which return directly to the surface is allowed without the necessity for decompressing. The formation of bubbles in the tissues of "bent" scuba divers on ascent is analogous to the formation of bubbles in a bottle of carbonated beverage after it has been uncapped. Small bubbles may coalesce into larger bubbles, which may lead to mild or serious problems, depending on the location and size of the bubbles. This is the basis for the development of decompression sickness.

The pathogenesis of the bends is directly related to bubbles within blood vessels impeding circulation and, in the extravascular areas, distorting tissues as the bubbles expand. Clinical manifestations may be noted immediately, but there is usually a delay of minutes to hours after surfacing. Symptoms rarely occur after 12 hours. Statistically, over 50 per cent of persons with decompression sickness develop symptoms within 1 hour and 90 per cent within 6 hours of the dive. The delay in the onset of symptoms can be explained by the time it takes for the growth of bubbles that cause tissue damage and edema, resulting in the gradual progression of pathologic processes.

The symptoms that occur depend on the location of the bubbles, whether in the skin, joints, muscles, bones, or nerves.

Skin Bends. This mild condition is seen more often after simulated dives in decompression chambers where the compressed gas is in direct contact with the skin. The skin of the trunk, especially the back, is more susceptible, with the diver experiencing itching, burning, and numbness, as well as mottling. The diver may complain of strange sensations in the skin. Since the rash disappears spontaneously over a few hours or days, skin bends are not treated unless other symptoms are present.

Bends of the Extremities. Localized pain is the most common symptom of decompression sickness. Musculoskeletal involvement has variously been described as "joint bends", "type 1 bends", "pain only bends", and "decompression arthralgia". At the onset the diver experiences a poorly defined discomfort, numbness, or weakness, which may develop over the next hour into a steady, deep, dull ache or throbbing pain, with occasional exacerbation of sharpness. In mild cases the symptoms are of short duration and are referred to as "niggles". Although any of the limbs may be affected, the shoulder is the most common joint involved in diving, being affected in approximately one-third of cases. Other affected joints include the elbows, wrists, hands, hips, knees, and ankles.

The symptoms are believed to be caused by bubbles in the tendons and ligaments, and usually resolve in several hours even if not treated. More severe cases may last 3 to 7 days. The areas involved usually look completely normal, with minimal tenderness, fairly good mobility, and an absence of symptoms. The pain may be relieved and subsequent tissue damage avoided by treatment in a recompression chamber.

Traumatic injuries to the extremities can easily occur under water without the awareness of the diver because of the excitement of the dive and possible numbing effects of the cold water. Pain from injury can easily be differentiated from bends of the extremities by the obvious edema, discoloration, tenderness to palpation, and discomfort with motion.

Pulmonary Bends. Bubbles formed in the vessels of the systemic tissues may be carried to the lungs by venous blood and may occlude small pulmonary vessels. Pulmonary manifestations of decompression sickness are rare, but if they occur can be extremely serious. The pulmonary syndrome or "the chokes" is characterized by substernal chest pain; rapid, labored breathing with shortness of breath; and a dry, nonproductive cough. Pulmonary edema is possible, and unless recompression procedures are instituted immediately, circulatory collapse and death may occur. Chamber treatment results in complete symptomatic relief in most instances, usually within minutes, by reduction in bubble size, which allows the establishment of circulation.

Central Nervous System Bends. Decompression sickness involving the central nervous system (CNS) is especially serious because it can result in permanent nerve damage and paralysis. Immediate treatment of this medical emergency in a recompression chamber can result in complete or almost complete resolution of symptoms. The onset of low back and abdominal pain, referred to as "girdle" pain, is the first sign of spinal bends and indicates a very serious problem. The diver may attribute early symptoms to a sprain or pulled muscle from lifting air tanks. It is usually not until paresthesias or hypesthesias of the legs develop, described as "pins and needles", that the diver realizes that something significant has occurred. This becomes further verified when the legs become weak and walking is unsteady. Another manifestation is the inability to urinate with bladder distention. Paralysis below the waist or neck may follow, with symptoms similar to those of a spinal cord injury from a fractured vertebra.

Brain damage from occlusion of any of the cerebral or cerebellar vessels by gas bubbles occurs less frequently than spinal cord involvement, and produces a host of clinical manifestations analogous to those of a cerebrovascular accident. These symptoms include visual disturbances, weakness on one side of the body progressing to hemiplegia, extreme fatigue, confusion, dizziness, syncope, staggering, shock, and loss of consciousness. With decompression sickness these symptoms rarely occur immediately after surfacing, in contrast to a cerebral air embolism, in which the symptoms are rapid in onset. Cerebellar damage can result in ataxia, incoordination, and tremor, as well as scanning speech and nystagmus. Although "the staggers" has been attributed to decompression sickness involving areas such as the spinal column, it is most likely due to cerebellar lesions, occurring without nystagmus. Gas bubbles can form in the inner ear, producing inner ear bends that result in hearing loss, tinnitus, and vertigo. MacFie reported cases in which any one or all of these symptoms occurred.

The scuba diver who has been "bent" rarely surfaces unconscious, unlike the diver with an air embolism, but the infirmity is acutely critical and requires recompression as quickly as possible within 6 to 12 hours, even in mild cases, to prevent progressive neurologic involvement. In no circumstances should the victim who has surfaced and developed decompression sickness be allowed to go back under water in an attempt to recompress; this may further complicate the problem and is rarely successful. The reason that decompression

stops on the way to the surface are allowable, if not mandatory, is that bubbles have not formed in the body. Stopping during ascent at the depths and for the times specified by the US Navy Dive Tables allows for outgassing without bubble formation. Once bubble formation has occurred, returning under water in an attempt to decrease the size of the bubbles and then recompress over a period of time is usually unsuccessful and, in fact, extremely dangerous. Administration of 100 per cent oxygen should be instituted immediately, and the "bent" diver transported to a chamber where he can be treated by recompression to reduce the size of the bubbles, thereby improving circulation and reducing pressure on nerves and tissues. The victim is then gradually decompressed to facilitate outgassing from the tissues through the bloodstream to the lungs. Transportation to the chamber should be accomplished with the diver's head and chest low and with the left side down as described in the section on air embolism. Pure oxygen is administered en route. Cardiopulmonary resuscitation is instituted if necessary. For the location of the nearest chamber, the Coast Guard or the state police should be contacted.

Information can be obtained from the following:

1. Divers Alert Network (DAN)

Duke University Medical Center, Durham, NC

(919) 684-8111

2. US Air Force School of Medicine

San Antonio, TX

(512) 536-3278 (mnemonic - LEO-FAST)

3. US Navy Experimental Diving Unit

Panama City, FL

(904) 234-4355.

These facilities have recompression chambers and qualified personnel available 24 hours a day. Transportation to the chamber should be accomplished as quickly as possible. If an aircraft is necessary, the cabin pressure should be as close to one atmosphere as possible. Although air transportation necessitates a decrease in ambient pressure, favoring the formation and growth of bubbles, the time saved in arriving at a recompression facility more than compensates for the problems of diminished atmospheric pressure.

For this reason, a scuba diver who has completed an underwater session the duration of which is close to, but not reaching, that necessitating recompression as indicated by the US Navy Dive Tables should not fly for 12 hours or more, to allow for sea level outgassing. Air travel too soon after diving exposes the body to a decrease in ambient pressure, which may precipitate the bends. A diver who is at risk of developing the bends after getting out of the water should not be allowed to take a hot shower or bath, since this may precipitate bubble

formation.

Further supportive measures may be helpful as outlined below:

1. Steroids: dexamethasone, 10 mg intravenous push, followed by 4 mg intramuscularly every 6 hours for 72 hours. This help to decrease cerebral and spinal cord edema.

2. Heparin: 3500 units intravenously every 6 to 8 hours. This may halt deterioration when there is a progressive neurologic deficit such as paralysis or the loss of sensation or coordination. This low dose of heparin is not an anticoagulant dosage but prevents platelet clumping.

3. Aspirin: 10 grains every 4 hours to prevent or reduce blood sludging.

4. Diazepam (Valium): 5 to 10 mg every 4 hours to decrease anxiety. This is also an excellent anticonvulsant.

5. Low-molecular-weight dextran. Plasma expanders increase circulating blood volume.

6. Liquids. The intake of 1 liter of liquid by mouth every hour for 3 hours will expand the intravascular volume.

7. Catheterization of the bladder: this may be necessary.

Factors predisposing a scuba diver to the development of decompression sickness include poor physical condition, obesity, age, fatigue, exertion, exposure to cold, dehydration, and injury.

Prevention of the bends is accomplished by proper training leading to certification, as well as planning the dive ahead of time, then diving according to the plan. Diving without a buddy is foolhardy and should never be attempted.

The US Navy Dive Tables have been reorganized by this author into the No-Calculation Dive Tables, which is a simplified linear system for repetitive scuba dives. These Tables are more logically organized and arranged to facilitate their use in dive planning, which has been proved to reduce significantly the incidence of decompression sickness.

Other Conditions

There are other important medical conditions that are not directly related to the act of descending or ascending under water.

Oxygen Toxicity

It is difficult to count how many times this author has been asked about the "oxygen" tank used for scuba diving. The nondiver frequently mistakes the scuba tank as one containing pure oxygen instead of compressed air. Breathing 100 per cent oxygen at increased ambient

pressure under water can result in grand mal seizures and subsequent drowning. The initial symptoms include localized muscular twitching, especially noticeable in the face and lips. Tunnel vision is a predominant manifestation as well as anxiety, apprehension, clumsiness, incoordination, and difficulty in concentration. Fatigue and shortness of breath, with the possible cessation of breathing, may occur in addition to dizziness. The mechanism of CNS oxygen toxicity is not clear.

Scuba divers afflicted with either decompression sickness or air embolism are routinely treated in a recompression chamber, breathing 100 per cent oxygen by mask at a pressure of as much as 2.8 atmospheres, which is equivalent to 60 feet of sea water. To decrease the incidence of oxygen toxicity with the possibility of convulsions, the mask is removed and air breathing is periodically allowed for 5-minute intervals during oxygen treatment. Oxygen, rather than air, is used in the recompression chamber to increase oxygenation of the tissues as well as increase the gradient for diffusion of the inert nitrogen gas from bubbles in the tissues to the bloodstream and finally the lungs, where it is vented out of the body.

Nitrogen Narcosis

This condition, frequently called "rapture of the deep", was alluded to earlier in the consideration of Henry's gas law. Confusion, impaired judgment, and a false sense of well-being may gradually occur during descent from the anesthetic effect of nitrogen gas in the body under increased pressure. Concentration becomes difficult and abnormalities of vision and hearing may develop, as well as anxiety, apprehension, and dizziness. In a chamber, divers have been observed to have periods of profuse laughter at subjects that were not humorous. Continued descent may result in loss of consciousness. There is no specific treatment other than bringing the diver to a shallower depth. This further emphasizes the importance of one of the cardinal rules in diving: "Never dive alone".

Drowning

Each year more than 8000 individuals in the USA die by drowning. Between the ages of 5 and 24 years, drowning is exceeded only by motor vehicle accidents as the most common cause of death. A major contributing cause of drowning in adults is an overestimation of swimming ability, which results in poor swimmers wandering too far from the shore and good swimmers venturing where they unfamiliar with the surf, current, or tides. A high blood alcohol level that interferes with judgment contributes to an estimated 30 to 50 per cent of adult drownings. Many of the cases of "shallow water blackout" occur in young adults in good physical condition who are attempting to compete in underwater swimming or to set a time record for breath-holding under water. The victim hyperventilates vigorously just before submerging in an attempt to "increase the oxygen in the body", so that the length of time under water can be prolonged. In fact, the oxygen level in the body is *not* increased, but the partial pressure of arterial carbon dioxide ($PaCO_2$) is reduced. Under normal circumstances the major stimulus to breathing is the $PaCO_2$ tension. As this level increases, the *breathholding break point* is reached and the individual is forced to breathe. Thus, the mechanism for drowning is based on the fact that hyperventilation reduces the $PaCO_2$ level in the body, allowing for a delay in reaching the break point during which time the partial pressure of the arterial oxygen (PaO_2) can be significantly lowered, to a concentration that will not sustain consciousness. In essence, the victim consciously suppresses the urge to

breathe. The work of Craig provides the explanation for this phenomenon.

For the free diver who is attempting to reach greater depths or who is spearfishing and prolongs the duration of the underwater hunt, this situation is further complicated because the increased PaO_2 in the brain is sufficient to maintain consciousness as long as the diver is at depth (Henry's law). Eventually, enough CO_2 is produced by cellular metabolism to reach the break point, resulting in the irrepressible urge to breathe, at which point the diver begins the ascent. Two things then occur: (1) an increase in the consumption of O_2 demanded by the metabolic energy of exertion with the passage of time during ascent and (2) a reduction of cerebral oxygen tension (PaO_2) as the ambient pressure decreases during ascent (Henry's law). When the oxygen tension reaches a level below which brain cells will no longer function, cerebral hypoxia occurs and consciousness is lost. If this occurs before the diver reaches the surface, drowning can occur. Unfortunately, the unconscious victim does not appear to be in difficulty, often making seemingly coordinated movements that further delay attempts at rescue and allow time for the development of irreversible brain damage.

Treatment for shallow water blackout necessitates getting the unconscious diver to the surface as rapidly as possible where resuscitative measures can be initiated, preferably with administration of pure oxygen. Air embolism in this situation is not a hazard.

Education is the only solution to this problem, especially among the young, who frequently use hyperventilation in an attempt to remain under water for longer periods.

Near-Drowning

Drowning is defined as death by acute asphyxia during submersion, whether or not liquid has entered the lungs. Near-drowning, or shallow water blackout, occurs when the arterial oxygen level falls below the level needed to sustain consciousness during which, by definition, the victim survives at least 24 hours.

The most important physiologic result of a near-drowning accident is hypoxia and its accompanying metabolic acidosis. Initially, fluid is aspirated into the alveoli, which results in a greatly diminished gas exchange because venous blood is shunted through the lungs without being oxygenated. This process leads to the rapid onset of a severe hypoxia. The condition can worsen when atelectasis develops from a decrease in surfactant, a phospholipid that normally stabilizes the alveoli and prevents the development of atelectasis. Fresh water in the alveoli injures the surfactant, which is not the case with sea water; however, the latter may wash out the surfactant. The atelectatic areas may be so minute that they may not show up on x-ray. Further adding to the problem is injury to the alveolocapillary membrane by the aspirated fluid, allowing seepage of plasma from the capillary circulation into the alveoli and causing adult respiratory distress syndrome (ARDS) or noncardiogenic pulmonary edema. If vomiting and aspiration occur, a chemical pneumonitis may develop because of the low pH of the vomitus. In addition, the aspirate may contain infected material from the oropharynx or contaminated water, causing bacterial pneumonitis. All the complications that accompany near-drowning produce a dramatic and rapidly developing hypoxia leading to metabolic acidosis, myocardial depression and irritability, cerebral hypoxia, and death.

Ten per cent of near-drowning victims known as "dry drowning". The apnea that develops is a result of laryngospasm or reflex breathholding, leading to progressive hypoxia and hypercapnia. Chest x-rays and arterial blood gases in "dry drowning" are normal.

Immediate first aid for a near-drowning victim is crucial. Cardiopulmonary resuscitation (CPR) is administered until an adequate pulse and spontaneous respirations are obtained. The airway is maintained by mild neck extension while lifting the jaw. Since 50 per cent of victims vomit massive amounts of gastric contents as well as aspirated fluid, anticipation is necessary to make sure that the head can be quickly positioned to prevent aspiration of the vomitus. Before moving the head, it is most important to determine that the obtunded victim has not sustained a fracture of the cervical spine from diving into a poor or lake. If possible, oxygen should be administered by mask during transportation to the hospital. CPR should be continued for extended periods if the victim has been submerged in cold water (below 10 to 15°C). Hypothermia will confer some degree of protection by shunting blood away from the skin, muscles, and viscera in addition to reducing the cerebral metabolic rate. This shunting is known as the "diving reflex" prominent in diving mammals such as seals and whales. When the face is immersed in cold water, a reflex bradycardia and marked constriction of the splanchnic visceral bed occurs with an increase of blood flow to the heart and brain. Victims may be apneic, cold, and cyanotic with no pulse and may appear clinically dead, but Theilade reported that they may be salvageable.

All near-drowning victims should be hospitalized, since there may be delayed complications of aspiration resulting in progressive hypoxia and possible death. The conscious patient should be evaluated immediately with a complete physical examination, a chest x-ray, and measurement of arterial blood gases. If these test results are all normal, the patient should be observed in the hospital for at least 6 hours or preferably overnight. The obtunded patient requires oxygen continuously with positive end-expiratory pressure (PEEP). The acid-base status should be normalized by correcting the metabolic acidosis with intravenous sodium bicarbonate. In addition to the previously outlined workup, a complete blood count and urinalysis, and electrolyte and blood alcohol levels should be obtained. Correction of the hypoxia and acidosis is of paramount importance and should be attended to before the electrolyte imbalance is corrected.

Although massive doses of steroids have been advised in the past, their current use is highly controversial. Steroids improve neither oxygenation nor the progress of the patient. In fact, they may increase the risk of infection. Prophylactic antibiotics are administered if the victim is rescued from heavily polluted water or has pneumonia upon clinical evaluation. The administration of antibiotics is indicated if the patient develops fever or if an increase of infiltrates is seen on chest x-ray. Blood and sputum cultures dictate the specific antibiotics to be used.

Neurologic sequelae can occur in victims of near-drowning who enter the hospital unconscious. Modell reported a 2 per cent incidence of neurologic complications in his series of 81 survivors.

Other medical conditions attributed to diving are outlined in Tables 5 and 6. These charts, along with a brief coverage of poisonous and venomous marine animals and the No-Calculation Dive Tables, are included in a handy, water-resistant publication entitled Scuba

Diving First Aid and Emergency Quick Reference Charts.

Medical Examination

The otolaryngologist is frequently called on to examine and qualify students for scuba instruction. There are absolute contraindications to scuba diving, which include the following:

1. Epilepsy: a history of seizure disorder. This does not include a history of febrile convulsions of infancy. When there is a history of post-traumatic epilepsy, evaluation by a neurologist is necessary.

2. Active asthma. Exertion- or cold-induced bronchospasm may lead to an increased risk of local air trapping and pulmonary overpressure accidents during ascent.

3. Air-containing pulmonary cysts or blebs.

4. A history of pulmonary overpressure accidents.

5. Significant obstructive pulmonary disease.

6. Coronary artery disease with a history of recent myocardial infarction. Underwater activities may involve extreme exertion, especially when there is a great deal of current or wave action on the surface.

7. Episodic vestibular disorders. Ménière's disease and other vestibular disorders are potentially fatal when occurring under water. Disorientation may occur as well as vomiting, with the possibility of aspiration and drowning.

8. Inability to equalize middle ear pressure. In addition to idiopathic conditions, there are several physical abnormalities, such as a deviated nasal septum, obstructing nasal polyps, or acute allergic rhinitis, which may contribute to difficulty in equilibration of pressure changes in the middle ear. Correction of these conditions may possibly resolve the problem.

9. Syncopal attacks. These may be on a cardiovascular or neurogenic basis.

10. Insulin-dependent diabetes mellitus. The diver runs the risk of an insulin reaction under water, especially during emergencies when there is a need for sudden bursts of energy. Diving is permitted provided it is confined to shallow depths in clear, warm water where there is little current, and provided the diver avoids stressful situations. Diabetes that is controlled by diet is not a contraindication to sport diving.

11. Middle ear surgery with stapes replacement prosthesis. Displacement of the prosthesis during sudden pressure changes while diving could result in permanent hearing loss.

12. Tympanic membrane perforations.

13. Psychiatric instability or mental retardation.

14. Chronic alcoholism.

15. Habitual drug abuse.

In a number of conditions, listed below, there is disagreement with regard to qualifying students for scuba diving instruction.

1. A history of pneumothorax. There is always the risk of air trapping by scarring and adhesive bands, whether the pneumothorax is on a spontaneous, traumatic, or surgical basis. An experienced diver whose chest pulmonary function tests are normal can be given a decompression chamber pressure test and be requalified by a pulmonary specialist.

2. Migraine. The headache associated with migraine may be confused with that of decompression sickness or air embolism. Whereas chamber decompression is mandatory and life saving for the latter, it is of no value for migraine headaches.

3. A history of neurologic decompression sickness with residual deficit.

4. Cardiovascular problems, such as a previous myocardial infarction. Diving can be resumed if the infarction has occurred over 1 year with no angina or arrhythmias and the exercise tolerance test is normal. Stress test electrocardiography and reevaluation should be performed every 6 months by a cardiologist.

5. Hypertension. Diving is allowed if the blood pressure is under medical control.

6. Repaired tympanic membrane perforation. If the tympanic membrane appears solid upon examination, diving is allowed as long as the diver has no difficulty with middle ear pressure equalization.

7. Pregnancy. Pregnant diver fall into a special category according to Fife. The incidence of fetal abnormality, spontaneous abortion, or decompression sickness is probably not increased; however, the long-term effects on the fetus are not known. It is generally felt that pregnant women should limit their diving to a depth of less than one atmosphere (33 feet).

The following are conditions that temporarily disqualify diving:

1. Upper or lower respiratory infections.

2. Active allergic rhinitis or hay fever.

3. Recent otitis media with fluid in the middle ear.

4. Acute alcoholic intoxication.

Examination of the diver for qualification should include a complete history as well as review of systems and a complete medical examination. There is a greater incidence of decompression sickness with increasing age, obesity, diving in cold water, physical injury,

exercise, and overuse of alcohol. The status of the otolaryngologic, pulmonary, and cardiovascular systems is most important. A baseline audiogram is advised and certainly necessary if the diver has had previous ear problems or difficulty with pressure equalization of the middle ear. Also recommended are chest x-rays on inspiration in both the posteroanterior and lateral positions, as well as a baseline electrocardiogram (mandatory for candidates over 40 years of age). If indicated, a stress electrocardiogram should be taken. Psychiatric evaluation is advised if there is any question of instability. Other tests that may be indicated include a complete blood count and urinalysis.

Davis and colleagues thoroughly reviewed the examination and physical standards for divers.

It is strongly recommended that the otolaryngologist take an active interest in the medical aspects of scuba diving and form a liaison with local diving instructors and dive clubs. When a student diver has a problem within the area of expertise of the otolaryngologist, the diver usually consults his or her family practitioners, who frequently advises abandonment of the sport. Many such divers, after consulting their family doctors, have been referred to this author by their instructor who did not want to lose a student. The great majority of these student divers, after a complete examination and discussion of methods of avoiding barotrauma as described in this chapter, have completed their instruction course and gone on to enjoy a most fascinating and challenging leisure pastime.