Hyperbaric Oxygen Therapy for Animals

Basic Course

Manual

VETERINARY HYPERBARIC ASSOCIATION

2023

VHA – Hyperbaric Oxygen Therapy for Animals Basic Course 2023

Hyperbaric Therapy for Animals - Basic Training Course

Schedule

September 2023

	Day 1 September 20th 2023	Day 2 September 21st 2023	Day 3 September 22nd 2023	
Time	Topic/Presenter	Time/ Topic/ Presenter	Time/ Topic/ Presenter	
9:00	**Welcome & Introduction** D Geiser	**9:00** Welcome Back S Hoberg	Welcome Back S Hoberg	
9:15	**VHA & CHT-V Certification Process** S. Hoberg	**9:10 Patient Selection & Evaluation - Large Animal** D. Geiser	**9:10- 10:00** Discussion/Q&A/Exam Prep*	
9:45	**Physics & Physiology of Gases** D. Geiser	**10:10** Break/Questions	**10-10:15** Break	
10:45	Break/Questions	**10:20 Chamber Types & Designation** S. Hoberg	**10:15-12:00 Examination**	
11:00	**Hyperbaric Facility Operations/Safety** S. Hoberg	**10:50 Selected Indications Small Animal** M. Johnson	**12:00-12:30** Exam review	
12:00	Break/Questions	**11:50** Break/Questions	**12:30** Discussion/Close	
12:10	**Decompression Theory & Arterial Gas Embolism** D. Geiser	**12:00 Selected Indications Large Animal** D. Geiser	*If no discussion or questions exam will start early.	
1:00	Lunch 45mins	**1:00** Lunch 1hr **A personal success story of HBOT in small animal wholistic practice care** S. Goodworth (45mins)		
1:45	**Physical Effects of Pressure** D. Geiser	**2:00 Safety- Standards/Codes/Facility Oversight** S. Hoberg		
2:30	Break/Questions	**2:45 The Value of CHT-V** L. Hewlett		
2:40	**Patient Selection & Evaluation- Small Animal** M. Johnson	**3:15** Break/Questions		
3:40	Break/Questions	**3:25 Contraindications – all species** D. Geiser, M. Johnson, S.Hoberg		
3:50	**Equine Practice- how HBOT assists in treatment of difficult cases.** A. Rullan	**4:25 Discussion/Q&A**		
4:50	**Q&A/Discussion**	**4:45** Dismiss		
5:00	**Dismiss**			

VETERINARY HYPERBARIC ASSOCIATION

Module 1

Overview and Learning Outcomes

Module 1 is designed to provide the student with the basic physical concepts surrounding the use of pressure and oxygen as a treatment modality in animals. This module discusses important common terminology, units of pressure and gas concentration, laws that govern the development and effects of pressure, and the theory of decompression and the prevention of decompression sickness. Mastering this module will provide a basic foundation in the theory behind the delivery hyperbaric oxygen to a patient.

Module 1 - Learning Outcomes

Following completion of this module each student they should be able to:↘

- *Section 1 Terminology and Definitions*
  - ↘verbally, from memory define or explain…
    - pressure, gas density, and the terms associated with a hyperbaric treatment (surface, dive, ascent, descent, depth etc.), hyperbaric vs. hypobaric.
  - ↘ distinguish between atmospheric pressure vs. absolute pressure vs. gauge pressure
- *Section 2 Units and unit conversions*
  - ↘discuss the commonly used units of pressure and be able to covert between units, especially atmospheres absolute and psi and mmHg and torr.
- *Section 3 Gas Laws and their application*
  - ↘verbally, from memory state the commonly used gas laws in hyperbaric medicine.
  - ↘apply the common gas laws to specific physical and physiologic phenomenon that occur or may occur during the application of oxygen under pressure.
- *Section 4 Decompression theory*
  - ↘discuss the pathophysiology of decompression sickness (DCS), apply appropriate gas laws to the initiation of DCS, the dynamics of nitrogen gas and how bubble form.
  - ↘discuss the causes of DCS, risk factors, signs/symptoms, and treatment of DCS.
- *Section 5 No - decompression tables – application*
  - ↘verbally define the terms associated with the use of no-decompression dive tables.
  - ↘use the no-decompression dive tables provided to determine dive profiles for given single or repetitive dive scenarios.

Physics of Hyperbaric Oxygen Therapy

Section 1

A. Hyperbaric terminology and definitions

Learning Outcomes

↘Verbally, from memory define or explain pressure, gas density, and the terms associated with a hyperbaric treatment (surface, dive, ascent, descent, depth etc.), hyperbaric vs. hypobaric

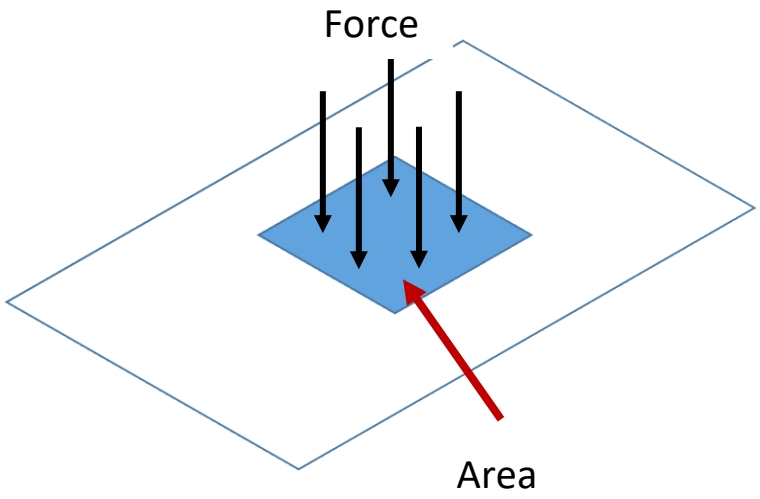
↘Distinguish between atmospheric pressure vs. absolute pressure vs. gauge pressure

1. Definitions
   a. Pressure – ratio of force to an area over which that force is distributed = force per unit area, e.g. **P**ounds (lbs.) per **S**quare **I**nch (in.) or **psi.** Force measured in pounds that is exerted on every inch of your body. The pressure of a gas is created as each gas molecule moves around and hits an object or body *(Diagram 1).*
   b. Gas density – the number of molecules of gas per unit of volume. In general the smaller the volume the denser the gas, molecules are compacted together and therefore there are more per unit of volume.
   c. Surface – sea level, ground level, the point or pressure from which a hyperbaric treatment begins.
   d. Dive – a hyperbaric treatment, each exposure to increased pressure.
   e. Descent – the period during which the pressure increases (compression).
   f. Ascent – the period during which the pressure decreases (decompression).
   g. Depth – the maximum exposure pressure
   h. Atmospheric pressure – the pressure exerted at ground level, caused by a column of air the reaches into the atmosphere. Usually described as a column of air 1 inch square. At sea level this column produces 14.7 psi.
   i. Absolute pressure – the total pressure exerted, that is the pressure produced by the atmosphere + the pressure developed by diving in water or being placed in a hyperbaric chamber. Its actually atmospheric pressure + gauge pressure.
   j. Gauge pressure - is the pressure measured in the hyperbaric chamber or measured underwater at a certain depth. Total pressure – atmospheric pressure.
   k. Hyperbaric - higher than normal surrounding pressure, underwater, in a cave below sea level.
   l. Hypobaric - lower than normal surrounding pressure, in an airplane, in space.

Diagram 1. Definition of Pressure

Pressure = Force per unit of area

$$\mathbf{P}\ (\text{lb.s/in}^2) = \frac{\mathbf{F}\ (\text{lbs.})}{\mathbf{A}\ (\text{in}^2)}$$



Section 2

A. Units of pressure and gas concentration

Learning Outcomes

↘discuss the commonly used units of pressure and be able to covert between units, especially atmospheres absolute and psi and mmHg and torr.

1. Pressure is force per unit of area
   a. Force (weight) uses units of weight like pounds (lbs.) Pounds per square inch or psi
   b. Area uses English units like square inches
      1) mmHg = millimeter(s) of mercury - a unit of pressure equal to the pressure that can support a column of mercury 1 millimeter high.
      2) Cm H_2O = It may be defined as the pressure exerted by a column of water of 1 cm in height at 4 °C (temperature of maximum density) at the standard acceleration of gravity.
   c. Barometric pressure – created by a column of air around us that reaches into the atmosphere. Also called atmospheric pressure.
2. *At sea level this pressure equals 14.7 **p**ounds per **s**quare **i**nch (psi) or 760 mmHg, or 29.9 inches of mercury, or 0.99 torr.*
   a. If we ascend to mile high Denver this pressure is less because the column of air around us decreases. Likewise, if we descend below sea level the column of air increases and consequently the pressure around us increases.
   b. Water is heavier and denser than air and therefore weighs more and, thus, it exerts more pressure if we descend into it. For each 33 ft. of sea water the pressure increases another atmosphere or 14.7 psi. This is water we do when we place a patient in the hyperbaric chamber, we just don't have the water.
3. Gauge pressure – is pressure difference between the system pressure and the surrounding atmospheric pressure *(Table 1).*
   a. A gauge reading zero does not mean that there is no pressure, it means that there is no pressure in excess of the atmospheric pressure
      Gauge Pressure = Total Pressure – 1 atmosphere at sea level
4. Absolute pressure – absolute pressure is the total pressure = sum of the gauge pressure and the atmospheric pressure *(Diagram2).*
   a. Always includes the atmospheric pressure plus the pressure generated in the system and in our case the system is the hyperbaric chamber.
      1) E.g. if we produce 14.7 psi in the chamber the gauge pressure is 14.7 psi but the total pressure is 14.7 psi + the atmospheric pressure. If at sea level the atmospheric pressure is 14.7 psi, thus the absolute pressure is 29.4 psi absolute. This is equivalent to 2 ATA (2 atmospheres absolute).
      2) Remember that every 33 ft. of sea water (fsw) generates another atmosphere of pressure. The absolute pressure on a body at 33 fsw is 2 ATA, one from the water pressure and one from the atmosphere above the water (atmospheric pressure).

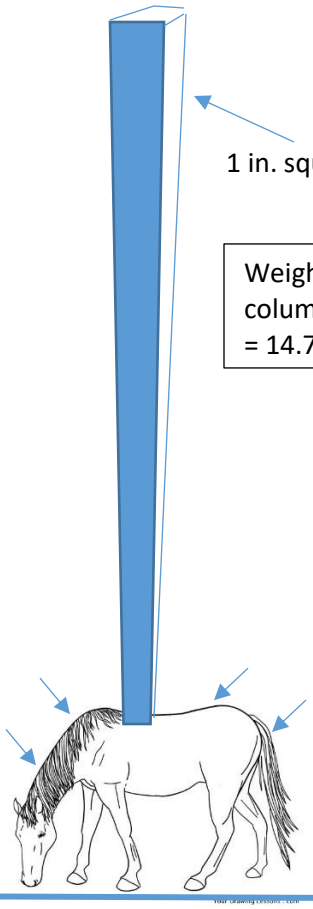
Table 1. Gauge Pressure

Gauge Pressure

The difference between the pressure being indicated and the atmospheric pressure.
If we are at sea level and the pressure you have developed in the chamber is 1 ATA and the atmospheric pressure at sea level is 1 ATA then the gauge pressure is zero.

Absolute pressure			**Gauge Pressure**	
ATA	*mmHg*	*psi*	*fsw*	*psi*
0	0			
1	760	14.7	0	0
2	1520	29.4	33	14.7
3	2280	44.1	66	29.4
4	3040	58.8	99	44.1
5	3800	73.5	132	58.8
6	4560	88.2	165	73.5

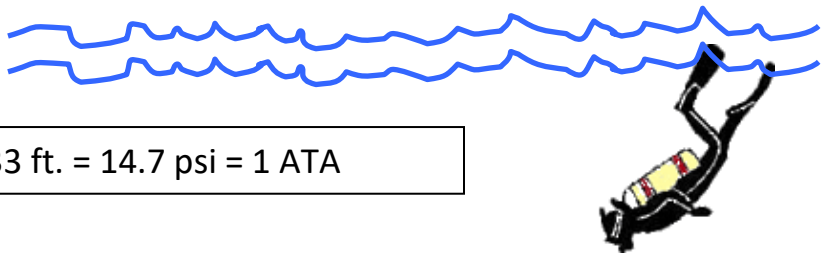
Diagram 2. Absolute Pressure



ATA = atmospheres absolute

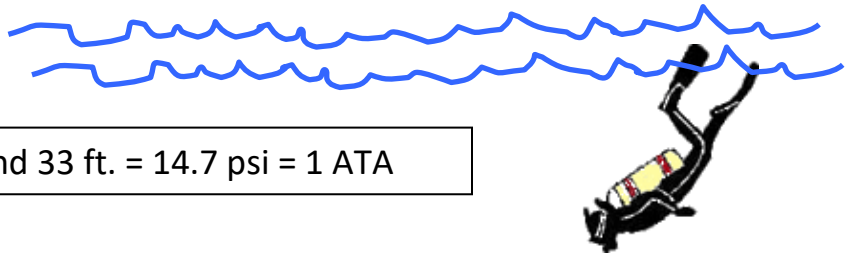
Surface……14.7…….. 1 ATA (760 mmHg)
First 33 ft……29.4…….. 1 ATA
Second 33 ft….44.1…….. 1 ATA
Third 33 ft……. 58.8…….. 1 ATA
Absolute Press 99 ft. = 4 ATA

Surface (sea level) ……..14.7 psi …… 1 ATA



First 33 ft. = 14.7 psi = 1 ATA

2 ATA Total



Second 33 ft. = 14.7 psi = 1 ATA

3 ATA Total

b. In the hyperbaric world we use atmospheres absolute as our common language for describing the pressure on a patient in the chamber.

5. Conversion of pressure units
   a. Conversion chart and formulas (Table 2).
      Psi to ATA
      ATA to psi
      ATA to mmHg
      mmHg ATA
      mmHg to psi
      psi to mmHg
      ATA to torr
      torr to ATA

Table 2. Nomenclature

Nomenclature and Conversion of pressure Units

Equivalents	**1 Atmosphere**	**1 mmHg**	**1 Torr**
Atmospheres	-	0.0013	.0013
mmHg	760	-	1.000
Pounds/sq.in. (psi)	14.7	0.0680	0.0019
Feet of sea water (fsw)	33.0	0.0303	
Meters of sea water (msw)	10.0	0.1000	
Conversions			
ATA to mmHg	ATA x 760 mmHg		
ATA to psi	ATA x 14.7 psi		
ATA to fsw	(ATA-1) x 33 fsw		
psi to ATA	(psi + 14.7) ÷14.7 psi		
fsw to ATA	(fsw +33) ÷ 33 fsw		

Section 3

A. Gas laws

Learning Outcomes

↘verbally, from memory state the commonly used gas laws in hyperbaric medicine.

↘apply the common gas laws to specific physical and physiologic phenomenon that occur or may occur during the application of oxygen under pressure.

1. General gas law
   a. Individual gas laws
      1) **Boyles law** – The **volume** of a gas **varies inversely** with the **pressure** at a constant temperature. As the pressure increases and the volume decreases the density of the gas increases, more molecules of the gas per unit volume or area.

         V = 1/P

         a) **Applications:** Creation of high blood oxygen concentration, barotrauma, DCS

      2) **Dalton's law** – in a mixture of gases the total pressure exerted is equal to the sum of the partial pressures of each individual gas.

         $P_T = P_1 + P_2 + P_3 + P_4 +$ ……

         a) **Applications:** Creation of high blood oxygen concentrations, DCS

      3) **Fick's law (diffusion)** – the movement of molecules is from the area of high concentration to the area of low concentration.

         a) **Application**: Creation of high blood oxygen concentrations, tissue oxygen concentrations, DCS

      4) **Henry's Law (solubility)** – the amount of gas that can be dissolved in a liquid is proportional to the partial pressure of the gas above the liquid.

         a) **Application**: Creation of high blood oxygen concentrations, DCS

      5) **Pascals Law** - A change in pressure at any point in an enclosed fluid at rest is transmitted undiminished to all points in the fluid.

         a) **Application:** barotrauma, patient safety, devices placed in the chamber

      6) **Charles's law** – at a constant pressure the volume of a gas is directly related to the temperature.

         a) **Application**: Gas cylinders and other closed pressurized containers

         $V \propto T$

      7) **Gay-Lussac's law** – pressure of a gas is directly proportional to the temperature at a constant volume.

         a) **Application**: Gas cylinders and other closed pressurized containers

         $P \propto T$

Section 4

A. Decompression theory

Learning Outcomes

↘discuss the pathophysiology of decompression sickness (DCS), apply appropriate gas laws to the initiation of DCS, the dynamics of nitrogen gas and how bubble form.

↘discuss the causes of DCS, risk factors, signs/symptoms, and treatment of DCS.

1. Decompression illness
   - a. Pathophysiology –
     - 1) The effect of elevated partial pressure of nitrogen
       - a) Composition of air
         21% O_2 and 79% N
         Nitrogen is an inert gas in the body – not consumed by metabolic processes
       - b) Dalton's Law of partial pressure
         At sea level 750 mmHg
         Air 21% x 760 = 159.6 mmHg O_2
         79% x 760 = 600 mmHg N
     - 2) Effects of pressure
       - a) As we increase the pressure around a patient/person *breathing air* we increase the amount of nitrogen and oxygen they inspire.
         At 2 ATA we double the pressure of O2 and N
         O2 = 319 mmHg and N = 1200 mmHg
         [760 x 2 (ATA) = 1520 mmHg x 21% = 319 mmHg & 1520 x 79% = 1200 mmHg]
   - b. Nitrogen uptake and elimination
     - 1) Uptake and elimination occur in the lungs by a diffusion gradient (high to low concentration of N, (Fick principle)
     - 2) Uptake and elimination also depend on respiratory rate and depth (tidal volume).
     - 3) Nitrogen is more soluble in fat than water
     - 4) Tissue uptake/elimination depend on blood flow and tissue lipid content.
   - c. Tissue compartment models
     - 1) Tissue compartments – each tissue compartment will eliminate N into the blood at a different rate.
     - 2) The rates are described as "half times" = the time it takes to reduce the N concentration in that tissue by 50%. e.g. a 5 minute tissue gives up 50% of its nitrogen in 5 min. and 50% of the remaining N in the next 5 min. and so on. After 30 min. this tissue would be almost rid of its nitrogen.
   - d. Critical ratios
     - 1) A critical ratio is the relationship of the tissue N pressure to the total pressure = P_{N2}/P_B

2) Each tissue has a different ratio
3) On ascent the ratio should not be exceeded – if nitrogen comes out of the tissue to fast that it cannot be eliminated by the lungs it will accumulate in the blood with the increased risk of bubble formation.

e. Bubble formation

1) On ***descent*** (pressure increasing)
N has a high concentration in the lungs and therefore the gradient causes more N to be transferred to the blood and subsequently to the tissues.
2) On ***ascent*** (pressure decreasing) N concentration in the lungs decreases and the gradient causes N to be transferred from the tissue to the blood and subsequently to the lungs and it is expired (eliminated).
3) If the ascent is too rapid (pressure decreasing rapidly) or breath holding occurs, the respiratory system cannot eliminate the N coming out of the tissue. The rapid decrease in pressure also allows the N to come out of solution as a bubble(s).
4) Bubbles can then cause several reactions in the body that are associated with Decompression Sickness (the bends).

f. Body response
1) Bubbles act as foreign bodies
Vasoconstriction
Intra and extravascular fluid leakage
Blood vessel blockage, pressure on tissues
ischemia/infarction
WBC chemotaxis
Oxygen radical production
Coagulation activated
Release endothelial derived mediators
Mechanical effects
Obstruction
Distortion
Disruption

g. Causes of decompression illness
1) Rapid ascent
2) Breath holding
3) Flying immediately after diving

h. Symptoms
1) Cutaneous
a) Formication - prickling, tingling sensation known as "pins and needles”
b) Pruritis
c) Pitting edema

d) Reddening, blotching of the skin, rash

2) Musculoskeletal
   a) Pain mostly in large joints

3) Neurologic
   a) Predominance with spinal symptoms as opposed to central, cerebral symptoms numbness, headache, nausea, dizziness, paresthesia, weakness, nerve pain

i. Risk factors
   1) Obesity
   2) Exercise during decompression
   3) Increased CO2 levels
   4) Patent foramen ovale
   5) High cholesterol
   6) Flying after diving
   7) Physical condition
   8) Cold temperature
   9) Dehydration

j. Treatment and prevention
   1) Immediate first aid
      a) O2 by mask
         Aides in de-nitrogenation of tissues
         Oxygenation of hypoxic tissues
      b) Supportive care – hydration, warmth, etc.
      c) Recompression
         Bubble compression
         Re-dissolving nitrogen
         Hyper-oxygenation
         Decrease edema
         Reducing reperfusion injury

Section 5

A. No-decompression tables

Learning Outcomes

↘verbally define the terms associated with the use of no-decompression dive tables.

↘use the no-decompression dive tables provided to determine dive profiles for given single or repetitive dive scenarios.

1. Why I need to know about decompression sickness and the dive tables
   Although at the present time we do not worry about DCS in our animal patients, an understanding of the theory of decompression illness and its prevention indicates a good working knowledge of pressure, gases, and physiology.
2. Terminology
   a. *Descent* – compression from surface to depth

b. *Depth* – the greatest pressure reached in feet of seawater (FSW) during a dive

c. *Bottom Time* (total bottom time) – TBT = the total elapsed time from departing the surface to the time you begin ascent from the greatest depth (actual dive time = ADT)

d. *Ascent* – decompression from depth to surface

e. *Decompression stops* – a specific depth at which a diver must remain for a specific length of time (Stop Time).

f. *Total Decompression Time* (TDT) – The time computed from leaving the bottom to reaching the surface.

g. *Total Dive Time* (TTD) – The time computed from leaving the surface to returning to the surface.

h. *Repetitive Group Designator* (REPET) – A letter used to relate directly to the mount of residual nitrogen remaining in the diver's body. Used for any dive made in less than 24 hrs. after a previous dive.

i. *Surface Interval Time* (SIT) – The amount of time in hours & minutes elapsed from the and of one time until the start of another dive.

j. *Residual Nitrogen Time* (RNT) – The amount of excess nitrogen, in minutes, from the previous dive.

k. *Equivalent Single Dive Bottom Time* – the combination of RNT from the previous dive + the actual total bottom time (TBT) of the current dive.

l. *Adjusted Maximum Dive Time* (AMDT) – Maximum dive time for a depth minus the residual nitrogen time (RNT).

m. *Decompression Schedule* – A specific decompression procedure for a given combination of depth and bottom time as listed in the decompression table. Expressed as fsw/minutes. In the no deco tables always round "up" to the next depth and time.

3. No decompression tables – theory and use
   a. Nitrogen
      1) A function of depth and bottom time
      2) Represented by letters A-Z (A = least, Z = most)
   b. Residual Nitrogen
      1) Excess nitrogen left in the body from the previous dive(s)
      2) Diminishes over time (exhaled over time at the surface)
      3) Residual nitrogen time (RNT) = amount of excess nitrogen represented in minutes
   c. Decompression stops
      1) Allow more time for nitrogen to leave
      2) Necessary when large quantities of nitrogen are accumulated in the body
   d. No decompression table
      1) Example applications
         a) Sample decompression tables – There are many recreational no-decompression tables out there, but most are similar in how they are used. The Navy also

has no-decompression tables as well as decompression tables that are more sophisticated.

b) Also in use today for recreational diving are some very sophisticated underwater computers.

e. How to use the NAUI recreational dive tables (Diagram 3&4)

1) Composed of three tables:

a) End of dive letter group

-Find the depth of the first dive in the left column labeled “Start Depth”. Always round up e.g. if the depth is 65 ft. use 70 ft.

-Find the number of minutes of the dive across the table.

-The number of minutes in red type with a blue circle is maximum time you can dive at that depth without a decompression stop (no-decompression)

-Find the dive time and follow that column down to discover your nitrogen letter group for the first dive.

b) Surface interval Time (SIT Table)

-The surface interval is the time between the end of the first dive and the beginning of the second dive (SIT). The time that you sit on the surface and blow off nitrogen.

-Find that time in the column below your letter group and then go horizontally from the box to your new letter group crediting you’re the nitrogen you have eliminated.

c) Residual Nitrogen time (RNT)

-In this 3rd table find the depth of the next dive at the top of the table (blue text).

-Go horizontally across the table from the box of your new letter group until you intersect with the vertical column corresponding to the depth of the next dive.

-In this box will be two numbers, one in blue and one in red. The blue number is your residual nitrogen time (nitrogen left in your body after your surface interval. The red number is you adjusted maximum dive time. Adding those two numbers together gives the maximum time you can spend at the new depth during the second dive. If you now follow from Table 3 back to Table 1 you will be able to determine your new nitrogen letter group after the second dive.

The best way to learn this procedure is to practice using the table with a few sample dives.

2) Sample no-decompression calculations:

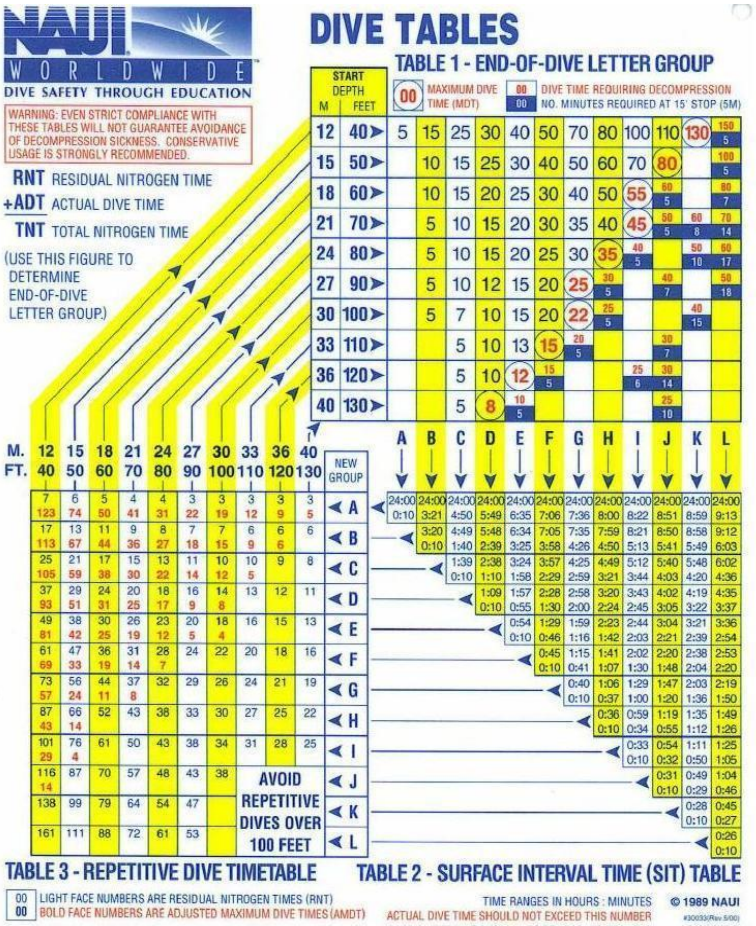
Sample #1

A diver plans his first dive to a maximum depth of 60 fsw. His actual dive time ends up being 50 min.
After completion of the first dive the diver stays on the surface for 2 hrs. and 15 min.
After the diver stays on the surface he decides to do a second dive to 50 fsw and he his actual bottom time is 42 min.

What is the no-decompression maximum dive time for this dive.
What is the diver's nitrogen letter group after completion of this dive?
What is the diver's residual nitrogen group after the surface interval?
What is the maximum depth the diver can go to on his 2nd dive without a required decompression stop? For how long?
What is his residual nitrogen time (RNT) before he goes on this second dive?
What is the diver's total nitrogen letter designation after the 2nd dive?

Please see Diagram 3&4 and follow the description below:

Diagram 3. NAUI No-decompression Dive Table



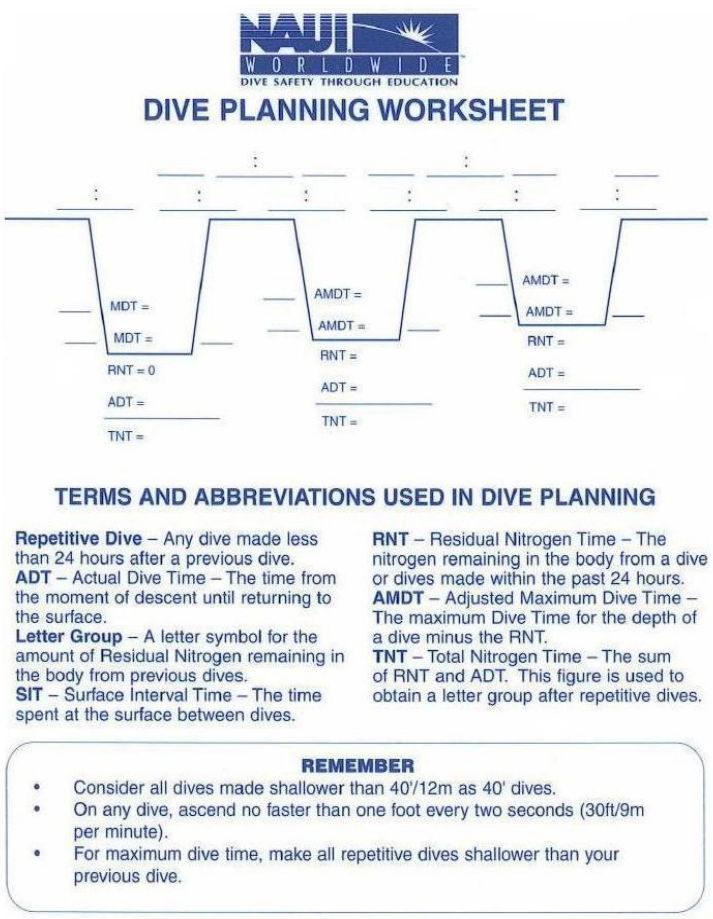
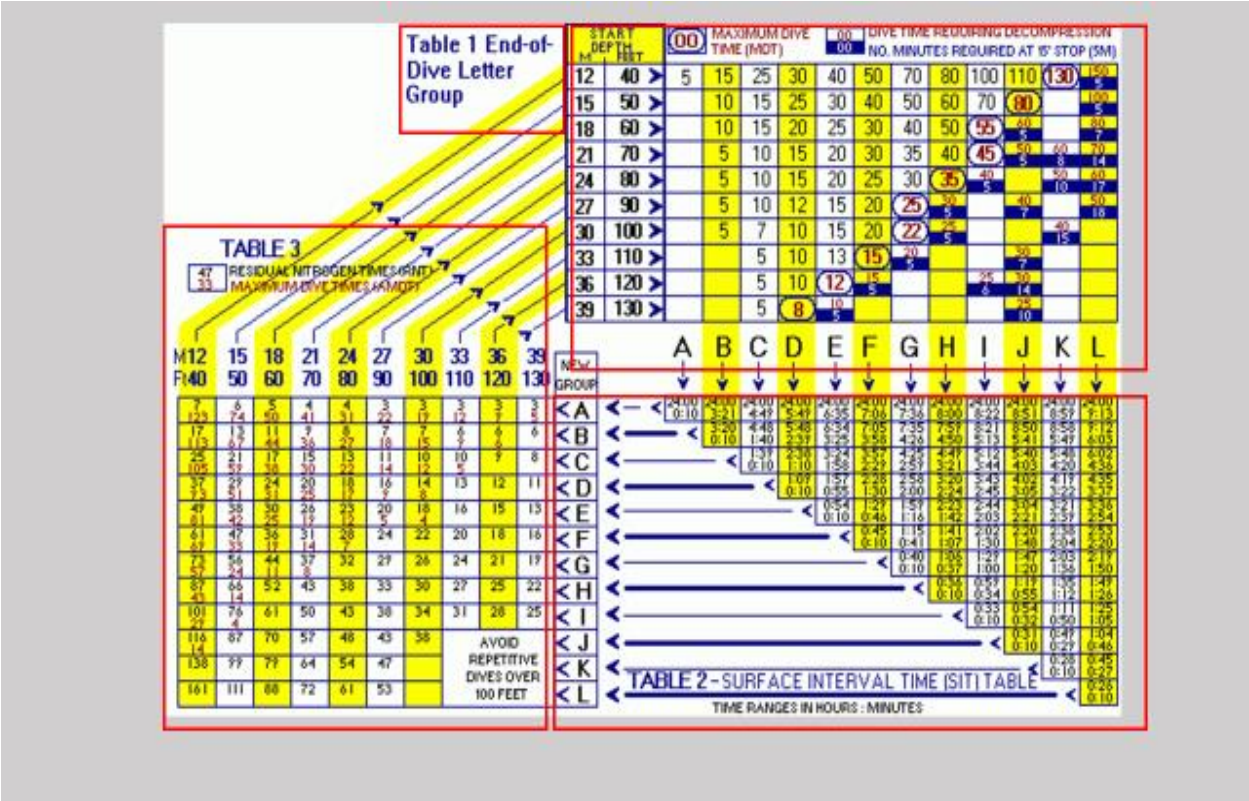


Diagram 4. NAUI No-decompression Dive Table – Three Table Configuration



1. Table 1 End of Dive Letter Group – Find depth of first dive in left hand column. (always round up) 60 fsw.
2. Follow the horizontal column across to the actual dive time of 50 min. (always round up if needed).
3. Follow the vertical column down from the 50 min. column to the line of letters And find than the nitrogen letter for the first dive is **"H".** This letter represents a particular level of nitrogen in the divers system.
4. Table 2 Surface Interval Time (SIT) – Continue down the vertical column from the letter H to the box that includes the surface time of 2 hrs. and 15 min (between 1:42 and 2:23). Now go horizontally across the this table from the box to see the new nitrogen letter group, which is **"E"**. This letter represents the nitrogen still left in the divers body after he has blown off some nitrogen while sitting on the surface.
5. We now go to Table 3 Residual Nitrogen Time and Adjusted Maximum Dive Time – Continue across horizontally to the left, in the letter E column to the vertical column that represents the depth of the second dive, 50 fsw. Find the intersection of the 50 fsw vertical column and the horizontal column from the E letter. IN the box you will see two numbers, one blue and one red. The blue number the amount of residual nitrogen still in the diver's body and is expressed in min (Residual nitrogen time or RNT) = 38 min.. The red number is

the adjusted maximum dive time for this new dive (AMDT) considering that the diver still has a certain amount of residual nitrogen on board = 42 min. S the diver can only stay at this new depth for 42 min. to do a no decompression dive (ordinarily he would be able to stay 80 min.). Add the red and the blue numbers together to obtain the Total Nitrogen Time (TNT) after the 2nd dive = 80 min.

6. Back to Table one to determine the diver's new nitrogen letter group after this 2nd dive. Follow the vertical column up to the 50 ft. depth and then diagonally to the 50 ft. box in Table 1. Then proceed horizontally across table 1 in the 50 ft. column to 80 min. The once again down this column to see the new letter designation which is **"J".**

   So you can see by the letter designation that the diver went from no residual nitrogen to being an "H" diver, then eliminated some of his nitrogen during the surface interval which made him an "E" diver. He then accumulated more nitrogen during the second dive which was added to the residual nitrogen from the first dive to make him a "J" diver. The further down the alphabet you go the more nitrogen you have accumulated.

Please see Diagram 5&6 - Useful schematic for organizing dive profiles

1. Follow the diver in the first example through his 2 dives.

Please see diagram # - Use this template to complete additional problems provided.

Other dive profiles to practice

1. Diver starts at 9 am and makes a single dive to 45 fsw for 50 min.
2. Diver starts at 8:30 am and makes a dive to 40 fsw for 60 min.
3. Diver stats at 8 am and makes a dive to 63 fsw for 45 min.
4. Diver starts at 8 am and makes a dive to 70 fsw for 45 min. and then starts a second dive at 11:15 am to a depth of 50 fsw for 30 min.
5. Diver starts a dive at 9 am to a depth of 35 ft. for 60 min. and then does a second dive at 12:20 pm to a depth of 55 fsw for 60 min.

Diagram 5. Example of Useful Dive Profile Work Sheet

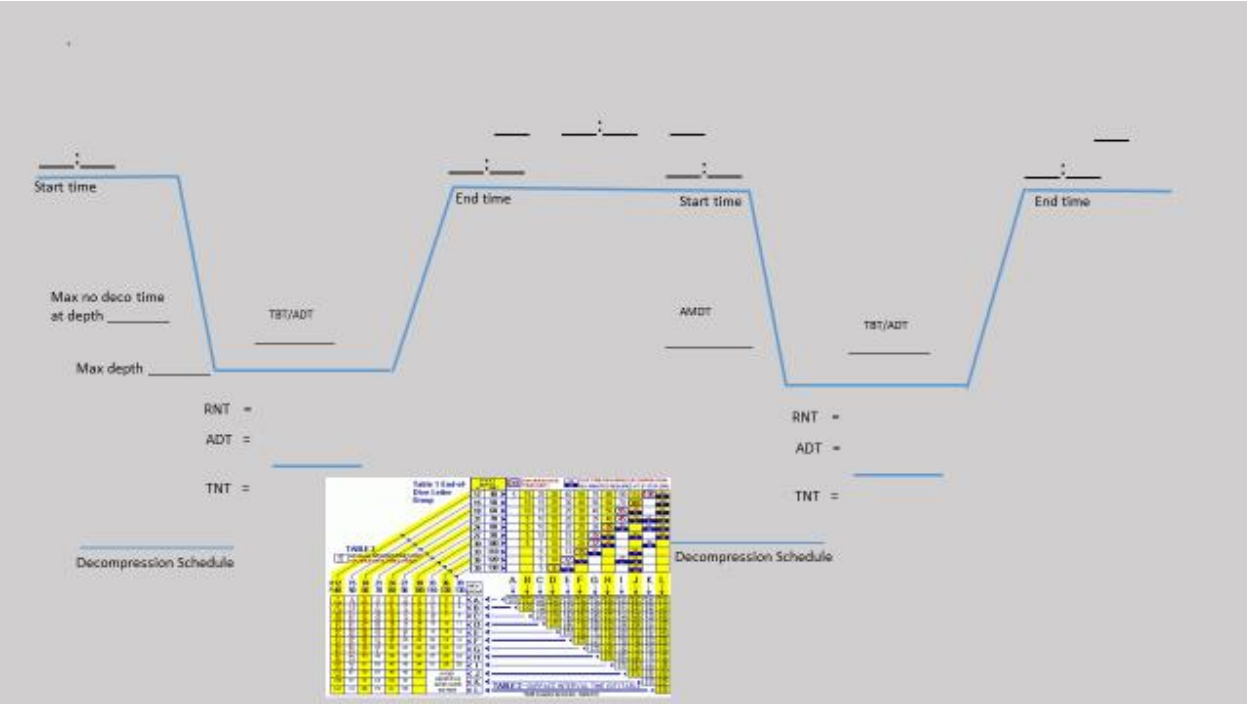


Diagram 6. Examples of Repetitive No-Decompression Dive Calculations

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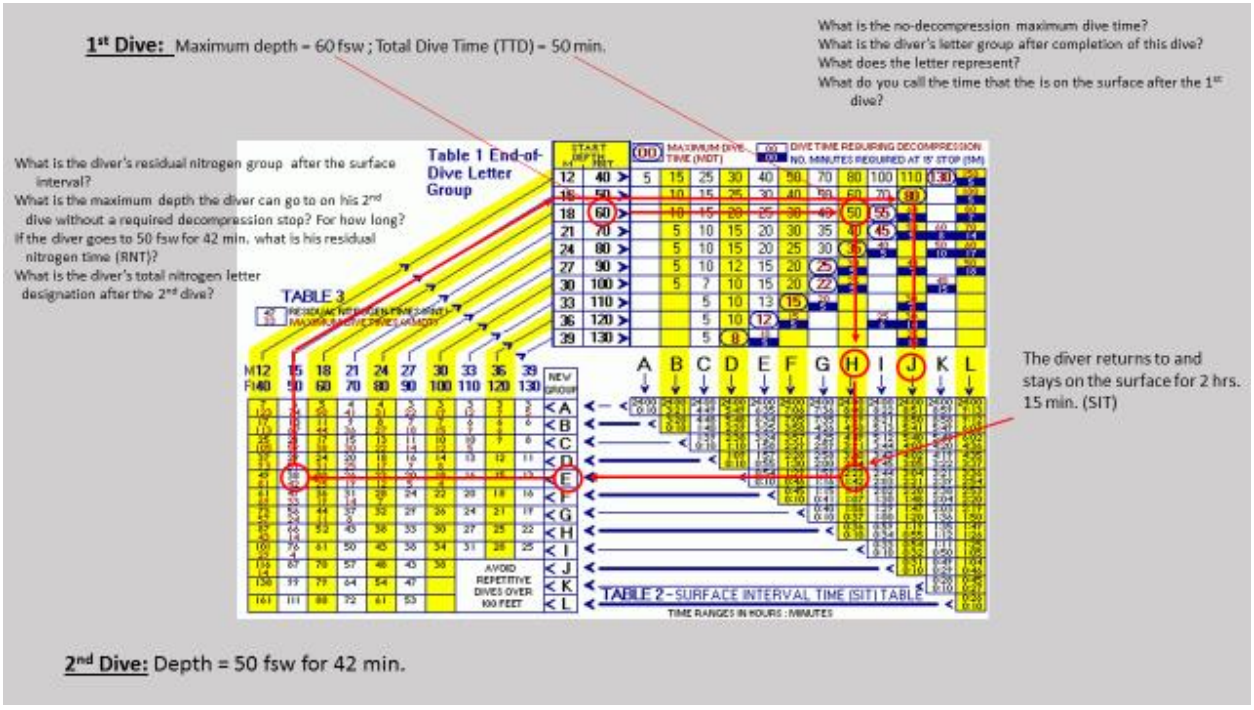
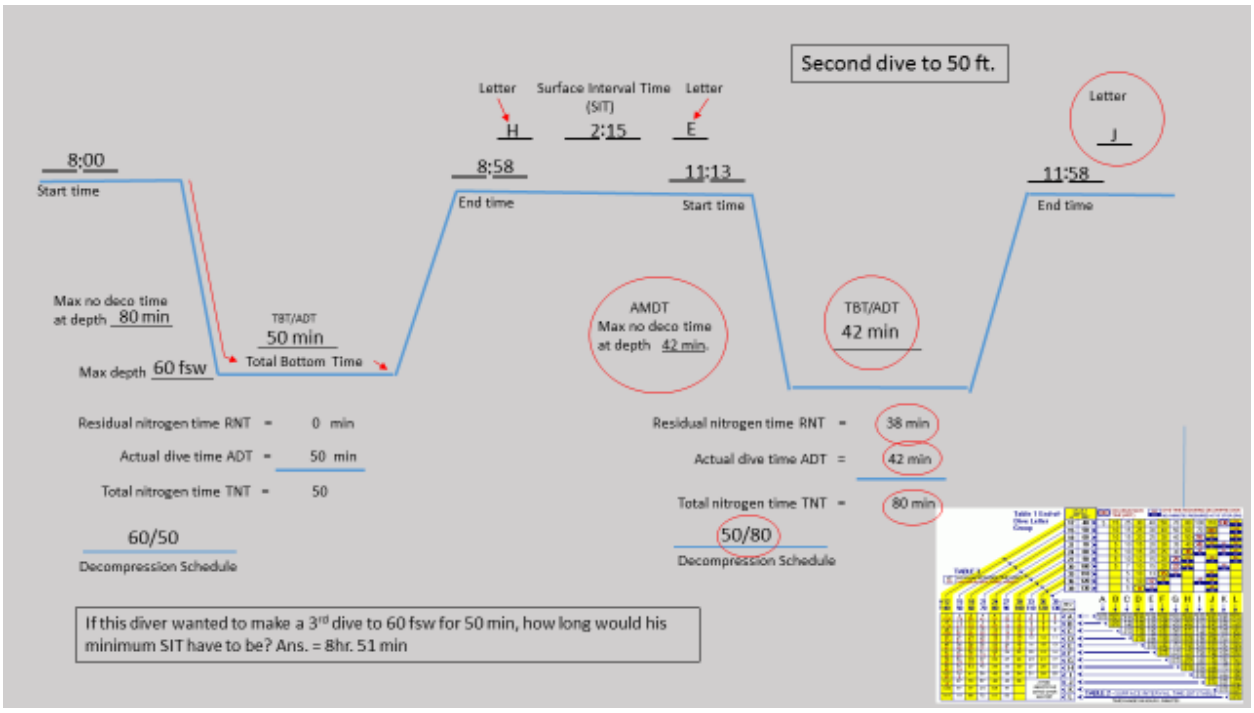


Diagram 6a. Sample Profile of Repetitive, No-Decompression Diving



VETERINARY HYPERBARIC ASSOCIATION

B. Arterial gas embolism (AGE)
   1. Description – a bolus of air within the blood vessels. Usually causes a vessel blockage.
      a. degree dependent on the ability of the cavity/organ to release extra gas into the environment (cavity compliance)

   2. Causes
      a. barotrauma, usually pulmonary put can associated with any gas containing body cavity or organ.
      b. Pulmonary barotrauma or overpressure syndrome.
         1) breath holding on ascent
         2) iatrogenic – bypass & vascular surgery, IV catheters, overventilation
   3. Signs/symptoms
      a. Symptoms occur very quickly and depend on region/location of the blockage
      b. Cardiovascular
         1) chest pain
         2) nausea, vomiting
         3) weakness
      c. CNS
         1) loss of ability to move, speak, think, touch
         2) ataxia
         3) seizure
   4. Treatment
      a) cardiovascular support
      b) 100% oxygen by mask
      c) HBOT – the protocol has been determined in people but not in animals at this time. The human protocol is very rigorous. Current chamber setups do not allow for animal treatment by using the human protocols.

Module 2

Overview and Learning Outcomes

Module 2 provides basic information physiology and pharmacology of high oxygen concentrations and the general beneficial effects on the body of most mammals. This module covers the major goal for developing high blood oxygen content and discusses how these concentrations are developed in the body. Also discussed are specific indications for the use of hyperbaric oxygen in clinical medicine. This module covers the potential side effects of administering oxygen under pressure and the contraindications for its use. Finally, patient selection, evaluation and preparation are discussed.

Section Learning Outcomes

Following completion of this module each student they should be able to:↘

- *Section 1 Physiology and pharmacology of oxygen*
  - ↘ explain and diagram how hyperbaric oxygen therapy increases tissue oxygen concentrations, including which gas laws are involved.
  - ↘ discuss the factors that affect the concentration of tissue oxygen.
  - ↘ discuss the general beneficial physiologic effects of oxygen as drug.
- *Section 2 Indications*
  - ↘ know and be able to list the common indications for the use of hyperbaric oxygen in animals.
- *Section 3 Side effects*
  - ↘ know and be able to list and discuss the direct indirect side effects of hyperbaric oxygen administration in animals.
  - ↘ discuss the signs of theses side effects in animals
  - ↘ discuss the prevention and management of hyperbaric therapy side effects in animals
- *Section 4 Contraindications*
  - ↘ discuss the absolute and relative contraindications for the use of hyperbaric oxygen therapy in animals.
- *Section 5 Patient selection and preparation*
  - ↘ discuss the thinking behind patient selection.
  - ↘ discuss the and/or design a protocol for the evaluation of a patient that is recommended for hyperbaric oxygen therapy.
  - ↘ discuss how a patient is prepared for hyperbaric therapy and the safety aspects that are involved.

Section 1

A. Physiology and pharmacology of oxygen

Learning Outcomes

Following completion of this module each student they should be able to:↘

↘explain and diagram how hyperbaric oxygen therapy increases tissue oxygen concentrations Including which gas laws are involved.

↘ discuss the factors that affect the concentration of tissue oxygen.

↘ discuss the general beneficial physiologic effects of oxygen as drug

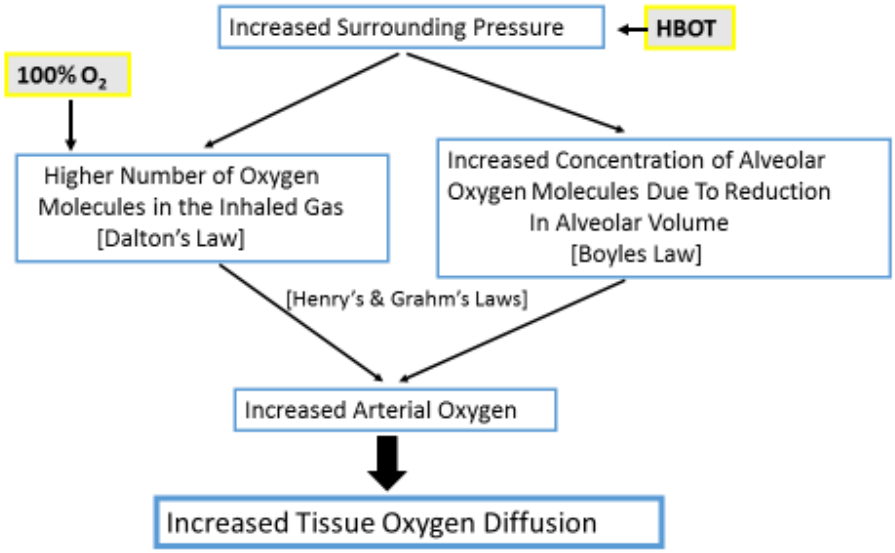
1. Physiology of oxygen
   a. Ultimate goal of hyperbaric therapy
      1) How hyperbaric therapy increases tissue oxygen concentration (*Diagram 7*)
         a) Increased inspired concentration of oxygen molecules
         b) Increased partial pressure of oxygen in the inspired air (↑ F_iO_2) 100% F_iO_2
         c) Increased surrounding pressure (use of the chamber) Dalton's Law of partial pressures
         d) Increased concentration (density) of oxygen molecules in the pulmonary alveoli and presented to the alveolar-capillary interface. Boyles Law

e) Increased diffusion of oxygen molecules into the capillary blood - Fick's principle of diffusion
f) Increased oxygen content of the blood and plasma
g) Henry's Law of solubility
1a. Increased transport and presentation of oxygen to the capillary-tissue interface
1b. Increased diffusion from the capillary blood to the tissue

b. A 20 x increase in PaO_2 → 4 fold increase in diffusion distance. Edema and inflammation increase the diffusion distance in diseased/compromised tissue. Normal capillary O_2 tension are not sufficient to drive O_2 into the diseased tissue. The use of HBOT helps to increase the diffusion of O_2 further into the tissue.

c. Tissue oxygen depends on:
FiO_2 (inspired oxygen concentration)
Pulmonary Function
Cardiac output
Blood flow
Cellular metabolism
Substrate availability

Diagram 7. Increasing Tissues Oxygen Concentrations



2. Pharmacology of oxygen – beneficial effects
a. General physiologic effects
1) Oxygen as a drug
a) FDA considers 100% medical grade oxygen as a drug

b) The use of 100% O_2 under hyperbaric conditions has a dose, frequency and duration. Dose: F_iO_2 = mmHg of oxygen inhaled = current barometric pressure x $\%O_2$ x ATA
Could also calculate F_iO_2/min
c) Duration: min of treatment at desired pressure
d) Frequency: daily, BID, EOD, etc.
e) Total duration: for 7 days, 10 days, 30 days etc.

b. ↑ tissue oxygen concentration and diffusion
1) bottom line goal for HBOT – increase tissue O2 in diseased tissue
2) most diseased tissue is hypoxic, degree depends on the tissue and the disease
c. Inflammation, edema, poor perfusion etc. impede tissue oxygenation.
d. Vasoconstriction in hypoxic tissues
1) aids in edema reduction
e. Increased white blood cell oxidative killing
1) killing of microbes requires a significant amount of tissue oxygen to complete the phagocytosis and disruption of microbes by WBCs (oxygen radical production). Number one effect of HBOT in treating infections.
f. Increased effectiveness of some antimicrobials
1) some antimicrobials are less effective in a hypoxic environment.
g. ↓ lipid peroxidation which contributes to an anti-inflammatory effect
h. Suppression of some autoimmune responses
i. Reduces platelet aggregation and increases RBC deformability
j. Decrease inert gas bubble size – especially important in treating DCS
k. ↓white blood cell endothelial adherence
1) important in preventing/treating reperfusion diseases
l. Tissue healing – especially wounds, bone, muscle etc.
1) Stimulates fibroblasts and collagen synthesis
2) ↑ growth factor production, synergism with O_2
3) Supports neovascularization and epithelialization
4) ↑ stem cell release from bone marrow

Section 2

A. Indications - Effects specific to diseases and/or body systems in animals - mechanisms

Learning Outcomes
Following completion of this module each student they should be able to:↘
↘ know and be able to list the common indications for the use of hyperbaric oxygen in animals

1. Neurologic system
a. physiologic effects
1) metabolism – in the injured brain ↑ O2 tends to decrease metabolism with an overall balancing of glucose metabolism. 1.5 ATA was beneficial but 2.0 ATA was deleterious.

2) blood flow – initially decreases due to vasoconstriction in the injured brain, but prolonged use may produce loss of vasoconstriction effects.
↓ in CBF → ↓ ICP
Dependent on brain region, insult, pressure and exposure time
3) BBB – not completely elucidated at this time, may protect disruption of BBB from ischemic and edema effects.
4) CSF – reflects increase in arterial O2 – in severe injury CV shunts occur that may decrease the effects of HBOT

b. Beneficial effects of HBOT
1) decrease cerebral edema ↓ CBF and ICP (acute injury?)
2) ↓ anaerobic metabolism, balance glucose metabolism
3) tissue/cell salvage
4) supports regeneration

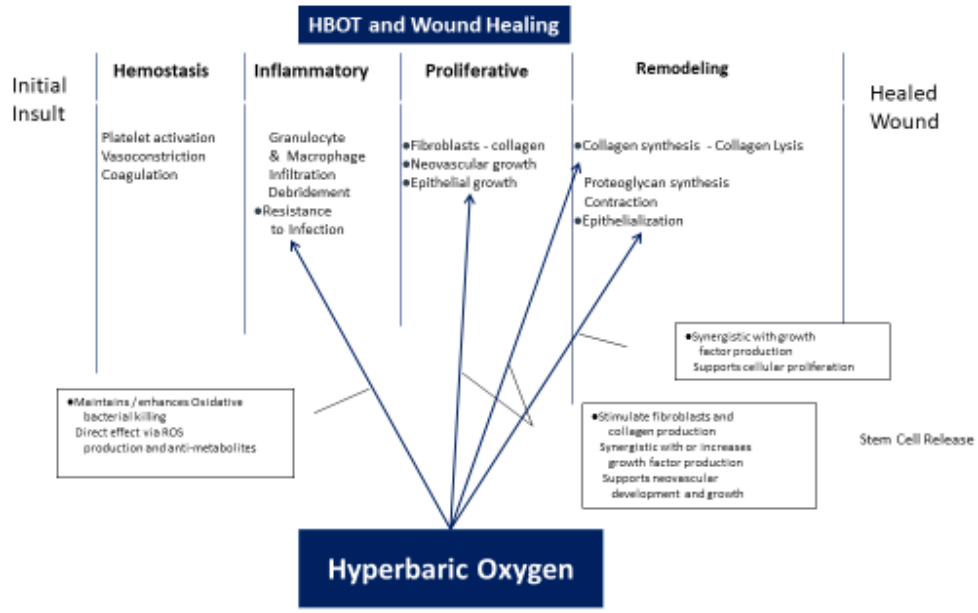
c. application
1) acute brain/cerebral injury (incl. acute cortical blindness)
2) hypoxic ischemic encephalopathy
3) brain/spinal chord infection/abscess
4) cerebral vascular accidents
5) compressive spinal chord disease and surgical manipulation
6) peripheral nerve injury

[Please see references on the final page of the this handout]

2. Cutaneous system

a. Wounds - basic beneficial effects of HBOT in wound healing enhancement of healing in selected problem wounds *(Diagram 8)*,
1) basic beneficial effects of HBOT in wound healing
2) types of wounds – etiology
mechanical
thermal
surgical
chemical
toxins
3) Phases of wound healing *(Diagram 8)*
Hemostasis & coagulation
Inflammatory
Proliferation & migration
Remodeling & maturation
4) Oxygen and wound healing
Initial wound hypoxia ↑ lactate levels stimulates macrophage production of angiogenic factors – capillary development. Not inhibited by O_2 administration
Essentially, O_2 is required for all other phases of wound healing
5) Compromised wounds occur for various reasons:
arterial vascular insufficiency, diabetes, neoplasia, infections, stress, steroid administration, wound size, etc.

Diagram 8. Phases of Wound Healing and Hyperbaric Oxygen



b. Compromised grafts and flaps *(Table 3)*

1) Early determination of potential graft/flap failure is ideal for best results
2) Early treatment is ideal – within first 24 hrs.
3) Elevated O_2 <u>and</u> pressure appears to be important. Elevated pressure in presence of 100% O_2, slowly increase pressure until graft/flap looks physically better.
4) Treatment times, 60-90 min.
5) Pre and post op treatments for free flaps/grafts seems to work best
6)
7) Application
   a) Small animals – 2.0 – 2.5 ATA, 30-45 min. initially may start at twice daily (tx. separated by 6 hr. surface interval). 7-10 day course of treatment
   b) Large animals – 2.5 ATA, 60– 90 min. initially may start at twice daily (tx. separated by 6 hr. surface interval). 7-10 day course of treatment
   c) Exotic animal – depends on the species, start low and increase time and pressure slowly.

Table 3. Compromised Wounds and Grafts

Pathophysiology of Compromised Wounds, Grafts/Flaps

Pathologic Effects Wounds	Rational for HBOT	Pathologic Effects Failing Grafts/Flaps
Hypoxia	Reverses tissue hypoxia	Hypoxia
Ischemia	↓ tissue hypoxia in face of poor perfusion, ↑ antioxidant production	Ischemic injury, ↑ free radical production
↓ energy metabolism	↑O_2 to meet metabolic needs	↓ O_2 for energy, ↑ necrosis
↓ collagen synthesis & quality	↑ fibroblast proliferation and collagen synthesis	
	O2 supports Na/K pump mechanism	Breakdown of Na/K pump mechanism
	Anti-inflammatory effects	Perivascular inflammation
↑ susceptibility to infection	Supports antimicrobial defenses	↑ susceptibility to infection
	↓ edema	Edema, vessel lumen constriction
	↓ platelet aggregation, supports microcirculation	Platelet aggregation, thrombus formation
	Blocks reperfusion - ↓ WBC adhesion, reverses hypoxia	Reperfusion disease
↓ neo-angiogenesis	Promotes neovascularization	Loss/decrease of vascularization
↓ growth factor production	↑ growth factor production and synergy	
↓ rate of epithelialization	↑ rate of epithelialization	
	↑ degree of cellular proliferation	

c. Envenomation
   1) Arachnids, venomous reptiles, chemicals
   2) Most information available is on brown recluse spider bites
   3) General effects of HBOT
      tissue salvage – prevention of necrosis
      edema reduction
      improved oxygenation of surrounding tissue even in the presence of poor perfusion
      improved healing and epithelialization
      may directly inhibit some toxins
   4) Application
      Small animals – 1.5-2.0 ATA 30 min, daily for 3-5 days, then re-evaluate
      Large animals – 2.0-2.5 ATA 45-60 min. daily for 3-5 days, then re-evaluate

d. Application:
   1) basic wound management
      a) debridement, bandaging, dressings, suction, etc.
   2) use of HBOT
      a) case by case dependent

b) probably not required for normal, healthy wounds, best for compromised wounds, ischemic wounds.
c) large wounds, vascular compromise, septic wounds, burns, radionecrosis,

3) period of hypoxia – often required for the initiation of the cascade of events that attracts appropriate cells and growth factors for initiation of healing. Ongoing hypoxia, however is detrimental for the healing process to proceed and HBOT then supplies adequate oxygenation.
4) Domestic companion animals (feline, canine and similar species)
1.25 – 2.5 ATA, 15-45 min. daily, total treatments dependent on nature of the wound
Equine and related animals
2.0 -2.5 ATA, 45 min. daily, total treatments dependent on nature of the wound
Exotic species – begin at low pressure and time and increase slowly monitoring the effects.

3. Gastrointestinal system
a. ***Ileus*** - defined as the functional inhibition of propulsive intestine activity, irrespective of its pathophysiology. Using the duration of clinical signs for classification, ileus can be either adynamic, resulting from short-term alterations of gastrointestinal motility, or paralytic, when motility is lost for a longer time.
1) An obstructive disorder of the GI tract resulting in a complete or incomplete functional obstruction of the intestinal tract
2) Causes
a) Paralytic - neural, hormonal metabolic → surgery, peritonitis, trauma, ischemia
b) Mechanical → secondary to GI obstruction, adhesions, neoplasia, inflammatory disease, luminal obstruction, intussusception etc.
3) Ileus can develop from diseases directly involving the digestive system, or be a consequence of diseases in other body systems, such as trauma to retroperitoneal structures or irritation of the peritoneum. Shock; electrolyte imbalances; hypoalbuminemia; peritonitis; endotoxemia; and distension, ischemia, or inflammation of the intestinal tract.
4) Pathophysiology
a) factors that may contribute to the development of ileus include:
autonomic (sympathetic) nervous system hyperactivity
neurotransmitter/hormone reductions NO, SP, vasoactive intestinal peptide
electrolyte imbalances (potassium reduction, calcium reduction)
infection (intestinal, peritoneal, other organs)

ischemia and ischemia reperfusion
inflammatory mediators, bowel manipulation
opiate administration
excessive gas accumulation and pressure
origin of air in bowel obstruction is swallowed air, bacterial and chemical production, diffusion from alveoli.

5) Effects of HBOT on ileus (reduced intestinal motility)
   a) reduction of excessive intestinal gas and bowel diameter – alteration of partial pressure gradients between bowl lumen and the blood.
   b) reduction of inflammation
   normalizes enzyme activity after bowel obstruction when removal of the obstruction did not normalize activity.
   c) prevents translocation of endogenous enteric bacteria and supports endogenous microbial defense mechanisms against pathogenic microbes.
   d) Things that govern diffusion rates of intestinal gases are:
   partial pressure gradients across intestinal mucosa to the blood;
   absorption coefficient in the blood,
   molecular weight of the gas,
   area of surface contact between gas and the semipermeable membrane,
   diffusion velocity
   molecular weight and absorption coefficient.
   one factor that can be influenced to increase absorption of gases from the intestine to the blood is the partial pressure gradient.
   e) Mechanical pressure effect to reduce size of gas filled bowel (Boyle's Law)
   f) improves intestinal viability and salvages compromised cells
6) Application
   a) Best results occurred in research/clinical trials if administered as soon as possible after reduction of any obstruction or post surgical manipulation (adynamic ileus).
   b) Small animals – 1.5-2.0 ATA, 45 min. daily, for 3-5 treatments
   c) Large animals – 2.0-2.5 ATA 60min., daily, for 5-7 treatments.
   d) Treatment frequency and number of Txs. Depend on cause and severity.

b. **GI ischemia-reperfusion disease** – re-perfusion injury, sometimes called ischemia-reperfusion injury or re-oxygenation injury, is the tissue damage caused when blood supply returns to tissue after a period of ischemia or lack of oxygen. The absence of oxygen and nutrients from blood during the ischemic period creates a condition in which the restoration of circulation results in inflammation and oxidative damage through the induction of oxidative stress rather than restoration of normal function

1) pathophysiology – please see details under cardiovascular system
2) causes in the GI system
   a) Primary cause would be those diseases that produce bowel ischemia
   b) bowel obstruction (most common)
      mechanical
      vascular obstruction
      Occurs when the ischemia is relieved (medically/surgically) and reperfusion occurs.
3) Application
   a) HBO treatment should be initiated as soon as the patient is stable enough to enter the chamber without the potential of untoward side effects. Research indicates that treatment within 12 hrs. is ideal.
   b) Frequency of treatment should be daily and patient re-evaluated at the end of three treatments.

c. **Pancreatitis**

1) Classification: acute (reversible), chronic (atrophy, fibrosis), Severe (systemic complications, mild (subclinical).
2) Causes: trauma to the abdomen, surgical manipulation, obesity, some medications, metabolic disorders, infections, high fat diets, genetic predisposition.
3) pathophysiology: the pathophysiology and associated beneficial effects of HBOT are summarized in *(Table 4).*
4) Application: Treatment initiation – As with most diseases, early initiation of treatment is usually met with the best results.
   a) Small animal protocol - 30min @10-14psi day 1 depending on chest, etc. sometimes treat twice the first day if severe/necrotizing. Use 30@14.7 once a day for about 2- 3 days after that.

Table 4. Pancreatitis Characteristics and Hyperbaric Effects

Pancreatitis and HBOT

Characteristics of Pancreatitis	Benefits of Hyperbaric Oxygen Therapy
Microcirculatory alterations	Supports microvascular health and vessel integrity
Tissue hypoxia	Increased tissue oxygenation, ↓ hypoxia
Tissue hypoxia, acidosis	Reduces tissue CO2 and lactate
Tissue edema	Tissue edema reduction
Ischemia-reperfusion disease	↓ hypoxia, ↓ neutrophil adhesion to venules
↓ red cell density and velocity in capillaries	Increased deformability of RBCs
Mitochondrial damage & oxidant release (ROSs) reduced peripheral anti-oxidants	Decrease tissue reactive oxygen species, increase endogenous anti-oxidants
Local and systemic inflammation	Anti-inflammatory
Recruitment of WBCs and ↑ cytokine production	Reduces neutrophil chemotaxis and cytokine production
Activation of coagulation cascade	Reduces platelet aggregation
Potential for bacterial translocation	Bacteriostatic and/or bacteriocidal
Hypoxia impairs neutrophilic bacterial killing	Restores optimal oxygen concentrations for neutrophil phagocytosis
Tissue cell death/necrosis	Reduces/blocks apoptosis
Abdominal pain	Analgesic

d. **Gastric ulcers**

1) causes

gastric pH, acid, pepsin secretion
stress
drugs – NSAIDs
infectious

2) pathophysiology

a) Not completely elucidated in most species
b) Gastric mucosa is mainly reliant on aerobic metabolism
c) Hypoxia can be caused by reduced blood flow, venous stasis, microcirculatory alterations.
d) Possibly the occurrence of hypoxia for whatever reason may predispose to infection and ulceration

3) Application of HBOT will have the following effects:

a) Reduction of hypoxia
b) Support of aerobic energy metabolism
c) Support of tissue healing
d) Antimicrobial effects

4. Ophthalmic system

a. Oxygenation of the ocular system

1) cornea & lens are avascular O2 from atmosphere, tear film, and aqeous of the anterior chamber

2) lens O2 from the aqueous humor and vitreous humor
3) retina and orbital O2 from dual blood supply = central retinal artery and choroidal vessels

b. Eye and pressure
1) aqueous, vitreous contain non-compressible fluid → not affected by pressure unless air is present.

c. Effects of hyperoxia on the eye
1) Retina (dual blood supply) – hyperoxia always one of the two retinal supplies to cover all of the retina's O2 needs. Therefore, if one supply is blocked oxygenation of the retina remain adequate.
2) Hyper-oxygenation constricts retinal vessels but does not decrease retinal O2 concentrations.
3) Hyper-oxygenation decreases intraocular pressure (IOP).
4) Normobaric O2 applied to the corneal surface increases vitreal O2 & hyperbaric O2 applied increases it even further.

d. Recommended indications for HBOT in ocular disease
1) ocular problems caused by DCS
2) Arterial Gas Embolism (AGE)
3) Central retinal artery occlusion
4) Necrotizing soft tissue and fungal infections of the orbit and eye
5) CO poisoning
6) Radiation optic neuropathy

e. Contra-indications and adverse effects
1) Retinal O_2 toxicity:
a) decreased peripheral vision, slow reversal on normobaric air
b) toxic to photoreceptor cells after long exposures in animals (canine, rabbits)
c) retrolental fibroplasia toxic to immature retinas – vasoconstriction, failure of normal vascularization, fibroplasia
2) lenticular O_2 toxicity – hyperoxic myopia, 3-6 reversal post therapy
3) cataract – prolonged exposures, may not be reversible
4) contraindications
a) hallow orbital prosthesis
b) intra-ocular gas (except from DCS and gangrene causes)

5. Respiratory system
a. HBOT may have limited application in pulmonary disease
1) individual variation in pulmonary response to pressure and high oxygen

b. Generally no adverse pulmonary physiologic effects at 1.5-2.0 ATA for 1 hour. Mechanics of ventilation may be altered at higher pressures and longer durations as well as after a number of treatments and in the presence of disease.

c. Potential clinical indications in pulmonary disease

1) Use in respiratory insufficiency and asthma produced variable results. Emphysema is a relative contraindication for use of HBOT
2) Hyperbaric air may be beneficial in COPD
3) Pneumonia, pulmonary inflammation, some infectious pulmonary diseases (e.g. Rhodococcus equi, Blastomycosis, Aspergillosis, etc.).
4) Pulmonary edema from neurogenic and cardiogenic as well as from smoke inhalation.
5) Anaerobic infections of the pleural cavity (screen for pulmonary bulla formation and pneumothorax)
6) Pulmonary air embolism (usually in association with embolectomy)

d. Contraindications for HBOT in pulmonary diseases
1) untreated pneumothorax
2) upper airway obstruction; e.g. collapsing trachea, laryngeal hemiplegia, soft palate elongation, combined syndrome brachiocephalic dogs, etc.
3) pulmonary bulla, emphysema,

e. Application
1) Use of HBOT for respiratory conditions should be considered with the utmost scrutiny and caution, since the respiratory system is very susceptible to direct and indirect effects of pressure the altered partial pressure of gases.
2) appropriate attention should be paid to pre-treatment evaluation and history (use of special diagnostics such as thoracic ultrasound and radiology.
3) a conservative approach to pressure, duration and frequency should be adopted
   a) Canine/feline – 1.25 -1.5 ATA, 20-30 min., daily (if tolerated)
      Maximum of 2.0 ATA
   b) Large animal – 1.5 – 2.0 ATA, 30-40 min., daily (if tolerated)
      Maximum 2.5 ATA
   c) Long treatment times and extended duration of therapy, may increase risk of pulmonary complications.

f. Air Embolism
1) Causes
   a) Pulmonary barotrauma – rapid ascent with exhalation of expanding pulmonary air.
   b) Trauma
   c) Heart and lungs – resuscitation – injury
   d) Iatrogenic
   e) Diagnostic and treatment procedures (e.g. IV catheter, arterial lines, angiography, + many others)

f) Intraoperative complications (cardiac, thoracic, vascular surgery, c-sections, etc.)

2) Pathophysiology/mechanisms
   a) Air introduced into veins or arteries (iatrogenic, barotrauma, trauma)
   b) Lung can filter bubbles up to a certain diameter (animals ??)
   c) Obstruction of blood flow, especially in the brain → ischemia, hypoxia, edema → platelet activation, leukocyte activation, reperfusion disease, blood vessel injury.

3) Clinical signs
   a) Signs vary with where the bubbles lodge – usually cardiac or CNS
   b) Posture may dictate their deposition
   c) Disorientation
   d) Coma
   e) Focal neuro effects
   f) Respiratory arrest
   g) Seizures
   h) Pneumothorax, dyspnea, myocardial ischemia

4) Diagnosis
   a) History of potential inciting cause – trauma, iatrogenic introduction of air into the vascular system, pulmonary barotrauma, etc.
   b) Neuro exam
   c) Doppler ultrasound
   d) Changes in mental function
   e) EEG

5) Treatment/Application of HBOT
   a) Steroids
   b) 100% O2 by mask
   c) Effects: HBOT for bubble compression and prevention of tissue hypoxia
      Treat immediately or as early as possible
      Table 6
   d) Unknown in animals what exactly the protocol should be. Table 6 is a very high pressure exposure and requires air breaks to prevent O2 toxicity.

6. Cardiovascular
   a. General effects of hyperoxia on the cardiovascular system
      1) ↓ HR, ↓ tissue blood flow, ↓ CO, vasoconcstriction, ↑ peripheral resistance, ↓ cardiac index, parasympathetic effects,↑ mean arterial pressure.
      2) Maintains or re-establishes adequate cardiac tissue oxygen for proper cardiac metabolism and function.
   b. Potential indications

1) Myocardial infarction and reperfusion disease – potential beneficial effects, reduction in infarct size, reduced mortality, angiogenesis, restoration of normal energy metabolism, decreased arrhythmias.
2) Heart failure, cardiac resuscitation – positive effects in heart failure through induction of heat shock proteins and in cardiac resuscitation - ↑ cardiac output, carotid blood flow, and arterial PO_2 (dogs) during cardiac resuscitation.

c. Shock
1) HBO may be necessary to achieve normal oxygen saturation and reduce mortality.
2) May have a roll in cardiogenic shock, especially if accompanied by cardiac infarction.

d. Peripheral vascular disease – ischemia and hypoxia of the limbs
1) Probably most applicable causes in animals include traumatic arterial obstruction (e.g. wire, rope around the limb), allergic vasculitis (purpura hemorhagica), lymphangitis, thromboembolism.
2) Raises tissue O_2 (improves metabolism)
    a) No vasoconstrictive effect on ischemic tissue
    b) Does increase tissue O2 in the presence of decreased flow
    c) Reduces pain
    d) Reduces edema
    e) Increases affinity of endorphins for receptors

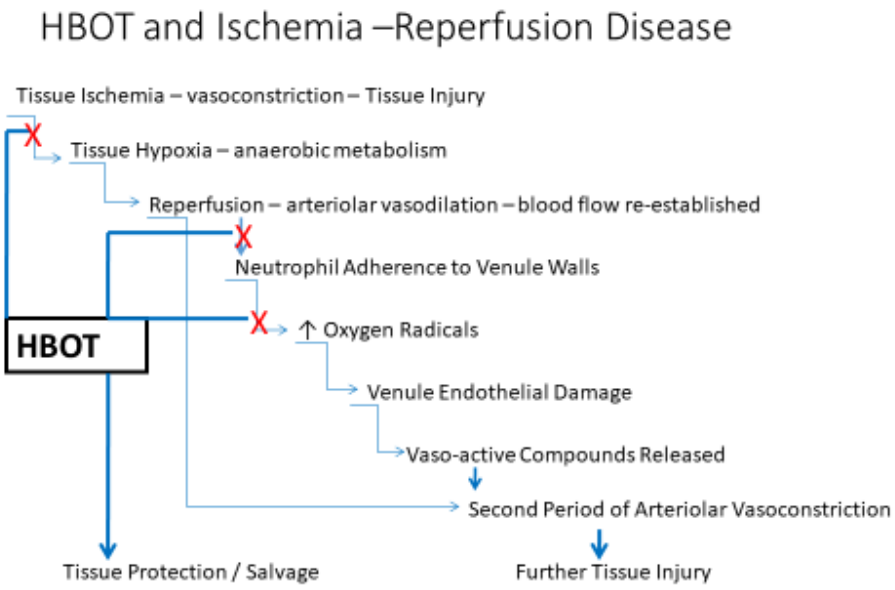
e. Ischemia-reperfusion disease (I-R)
1) Tissue damage caused when blood supply returns to tissue after a period of **ischemia** or lack of oxygen. The absence of oxygen and nutrients during ischemia creates a condition in which the restoration of circulation results in inflammation and oxidative damage through the induction of oxidative stress rather than restoration of normal function.
2) Can occur in any tissue that experiences an episode of ischemia, followed by re-establishment of blood flow:
    a) Common areas in veterinary medicine include the intestine, muscle, central nervous system, skin graft/flap.
3) Highlights of the pathophysiology:
    a) Injury causing an episode of tissue hypoxia, usually ischemia
    b) Lack of oxygen causes buildup of toxic by-products; oxygen radicals, inflammatory mediators, vasoactive substances, lactic acid, etc.
    c) Perfusion is then re-established either from endogenous healing or from treatment (e.g. surgically reliving an intestinal strangulation).
    d) Release of toxic substances and adhesion of WBCs to venules causes breakdown of capillary endothelium

releasing vasoactive substances that travel to the arterioles and produce a second round of vasoconstriction and ischemia further exacerbating the tissue damage.

4) Effects of hyperbaric therapy *(Diagram 9)*
   a) Supports reduction of initial ischemic/hypoxic event
   b) Decreases WBC adhesion to capillary endothelium
   c) Reduces oxidative stress
   d) Supports reduction/elimination of second ischemic/hypoxic event in the tissues upon reperfusion
   e) anti-inflammatory effects
   f) reduction of reperfusion effects persists after HBOT is discontinued

5) Application
   a) timing is important and early treatment is most effective
      Treat within 6-12 hrs. post reperfusion
      Anticipating potential reperfusion will help
   b) small animals: 1.5 – 2.0 ATA, 100% O_2, 20-30 min. daily for minimum of 3 treatments
   c) large animals: 2.0-2.5 ATA, 100% O_2, 45-60 min., daily for minimum of 3 treatments
   d) other species; dosing HBOT is species dependent

Diagram 9. Hyperbaric Oxygen and Reperfusion Disease



f. Carbon Monoxide Toxicity

1) General effects
   a) CO to Hb affinity is 200 times greater than O2
   b) Shifts oxyhemoglobin dissociation curve to the left – impairs O2 unloading at the tissue level
   c) Toxic to the BBB, produces cerebral edema
   d) Leukocyte activation and lipid peroxidation
   e) Hypoxic/ischemic injury
   f) Reperfusion disease
   g) Skeletal muscle binds a high % of the CO (15-20% with myoglobin) – becomes a storage sink and can lead to a 2nd rise of CO in the blood and body.
   h) In cardiac muscle – hypotension, arrhythmias, cardiogenic shock
   i) Metabolic acidosis – level of acidosis may be a good indicator of severity along with lactate
   j) Cerebral- altered mental status, delayed alteration, persistent alteration, coma, death.

2) Symptoms
   a) Nausea, vomiting
   b) Fatigue, exercise intolerance, gait abnormalities
   c) Dyspnea, tachypnea → respiratory failure
   d) Weakness, incoordination, collapse
   e) Altered mental status – normal mental status → drowsiness, mild confusion → loss of consciousness, coma
   f) Syncope
   g) Seizures
   h) Blindness
   i) Abortion, fetal death
   j) Red lips, gums, ears
   k) CO toxicity and COHb concentrations
      10-20% COHb = mild poisoning
      30-40% COHb = moderate poisoning
      40-60% COHb = severe poisoning
      >60% COHb = death likely

3) Treatment - Smoke inhalation injury can occur without clinical disease caused by CO
   a) Initial triage 100% O2 by mask.
   b) HBOT - Initially the use of HBOT was controversial but now should probably be the standard of care based on research data.
   c) HBOT mechanisms
      ↓ ½ life of COHb
      Inhibits reperfusion injury and lipid peroxidation
      Eliminates hypoxic-ischemic injury

Preserves ATP production, supports aerobic metabolism
Decreases cerebral edema
Aids in reversal of lactic acidosis

d) Specific treatment protocols, based on research, have not been developed in animals. Protocols for humans usually include air breaks.

e) The ½ life of COHb may be used to develop protocols

<u>½ life of COHb in humans</u>

On room air = 5 hr. and 20 min.
100% O2 – 1 ATA = 1 hr. 20 min.
100% O2 - 3 ATA = 20 min.
100% O2 – 2.5 ATA = 30 min.
Fetal CO ½ life room air = 7-9 hrs.
With HBO = 3-4 hrs.

<u>½ life CO in dogs (one study)</u>

Room air – 1 ATA = 188 min. (3.13 hrs.)
100% O2 – 1 ATA = 32 min.
100% O2 – 1.9 ATA = 25 min.
100% O2 – 2.8 ATA = 12 min.

g. Severe Anemia

1) Types:
   a) Acute blood loss anemia
   b) Blood transfusion may not be available, donors may not be compatible
   c) Fluid may maintain blood pressure but dilute remaining RBCs and may affect osmotic pressure without adequate protein concentrations (plasma).
2) Chronic blood loss anemia
   a) Patient decompensates
   b) ↓ viscosity, ↓ resistance to blood flow in periphery
   c) Hypoxia causes vasodilation
   d) ↑ cardiac work load and cardiac output
   e) Exercise intolerance
   f) Same issues as above with treatment
3) Hemolytic anemia
   a) Multiple potential causes
4) Application of HBOT:
   a) Primary effect of HBOT would be to provide adequate tissue oxygen until the primary inciting cause is under control and red cell production has adequately replaced lost cells/hemoglobin.
   b) HBOT may temporarily replace the use of blood transfusions.

c) HBOT may lessen the effects of the blood loss anemia and speed recovery of hemoglobin production.

7. Infectious diseases
   a. General Effects of HBO
      1) Direct effect on microorganisms
         a) Depends on the organism's ability to produce antioxidants
         b) Usually bacteriostatic – inhibits microbial metabolism
         c) Bacteriocidal for anaerobes and microaerophilic species
         < 1.5 ATA can promote aerobic bacterial growth
         d) Killing by phagocytosis and oxidation by oxygen generated radicals
         e) Dependent on local oxygen tensions
            Major ↓ in killing power begins at tissue O2 tensions < 30 mmHg
         f) HBO is more effective in increasing local tissue O2 than 100% O2 delivered by insufflation.
      2) Indirect effects
         a) Supports neutrophilic microbial killing (phagocytosis)
         b) Killing by phagocytosis and oxidation by oxygen generated radicals
         c) Dependent on local oxygen tensions
            Major ↓ in killing power begins at tissue O2 tensions < 30 mmHg
         d) Continual exposure to high O2 concentrations is detrimental to phagocytosis
      3) Effects on antimicrobial agents *(Table 5)*
         a) ↑ antimicrobial effectiveness by reversing hypoxia in the tissues, optimum effects of some antimicrobials are oxygen dependent.
         b) Acts in synergy with some antimicrobial agents
         Antimicrobial and microorganism dependent
      4) General tissue support effects
         a) Reversal of tissue hypoxia and lactic acidosis
         b) Reduces necrosis due to infection
         c) Growth factor production and synergism
         d) Supports collagen deposition and angiogenesis

   b. Specific Indications
      1) Necrotizing faciitis, gas gangrene, refractory osteomyelitis, mucormycosis, intracranial and intra- abdominal abscesses (UHMS approved).
   c. Other indications
      1) Septic arthritis, septicemia, endotoxemia, blastomycosis, Lyme's disease, infectious pleuritic, Rhodococcus, most anaerobic infections.

2) Possible efficacy against fungal infections (actinomyces, mucor, candida, nocardia, phycomycotic infections).

d. Osteomyelitis

1) Refractory osteomyelitis usually has low bone O2 tensions, < 30 mmHg.
   a) Low O2 can be caused by O2 consumption or inadequate delivery due to perfusion abnormalities.
2) HBO mechanisms of action
   a) ↑ tissue oxygen tensions even in the face of poor perfusion
   b) Enhances endogenous host defense mechanisms, oxidative killing by WBCs.
   c) Enhances osteogenesis and neovascularization
   d) Enhances osteoclastic activity – reabsorption of necrotic bone
   e) Improves effectiveness of some antimicrobial agents
   f) Used in conjunction with antimicrobials, debridement, drainage, removal of foreign bodies.

Table 5. Effect of Oxygen Concentrations on Antimicrobial Activity

Oxygen Tensions Influence Antimicrobial Activity

Antimicrobial	Anoxia	Hyperoxia/HBO	
Aminoglycoside	↓ bacteriocidal effects	↑O2 required for optimal activity	
Fluoroquinolones		↑O2 required for optimal activity	
Sulfas, Trimethoprim	↓bacteriostatic effects	↑O2 required for optimal activity, ↑static effect	
Vancomycin		↑O2 required for optimal activity	

[Kindwall EP, Whelan HT. Effects of Hyperbaric Oxygen in Infectious Diseases: Basic Mechanisms. In: Hyperbaric Medicine Practice 2 ed. 2004]

8. Musculoskeletal and Rehabilitation
   Injuries - Bone healing, Ligament and tendon treatment, muscle injuries
   Training -
   Crush injury, reperfusion disease, compartment syndrome, fractures, acute traumatic ischemias.

   a. General effects of HBO
      Roles in treating primary disease and as a complementary therapy in post injury recovery
      1) CNS disease rehabilitation
         a) Supports neuron function and repair/regeneration
         b) Decreases edema
         c) Facilitates movement and exercise
         d) Improves mental capacity
      2) Musculoskeletal injury rehabilitation
         a) Decreases edema and pain
         b) Supports soft tissue healing (similar to wound healing)
         c) Improves facture repair
         d) Osteomyelitis, delayed or non-union fractures, interruption of blood supply

      3) Training
         a) Improves post exercise recovery and helps eliminate oxygen debt
         b) Decreases recovery time and increases fitness to compete again

9. Bone healing
   a. Pathophysiology
      1) Lack of O_2 is major detriment to bone healing
      2) Low O_2 tensions in healing fractures until medullary canal is formed
   b. General effects
      1) Improves oxygenation for:
         a) fibroblastic bone formation instead of cartilage formation
         b) support of the fracture repair process
         c) remodeling (via osteoclastic activity)
         d) faster, stronger bone formation
         e) ↑ bone mineral density, BMD (Ca, Phos, Mg, Na, K, Zinc & collagen)
         f) ↑ callus formation
         g) ↑increased vessel ingrowth
         h) ↑torsional strength of fracture repair
   c. Application of HBOT
      1) General considerations:

a) Start treatment early after fracture/fracture reduction-repair
b) Taper treatment after 5-6 treatments
c) Consider intermittent application of HBOT
d) Bone healing appears to be pressure and time sensitive
Too much for too long may reduce repair of the fracture because of increased osteoclastic activity.

2) Protocols are species dependent
a) Large animals 2-2.5 ATA 60-90 min daily for 5-7 treatments
Reevaluate, then every other day for another 5-7 txs.
b) Small animals (canine, feline)
1.68, 30 min. test Tx.
1.68-2.4 ATA, 30-40 min. daily 3 txs. and re-evaluate
Feline-low end, canine – higher side.

10. Soft Tissue – ligaments and tendons
a. Pathophysiology
1) The low metabolic rate and well-developed anaerobic energy-generation capacity are essential to carry loads and maintain tension for long periods, reducing the risk of ischemia and subsequent necrosis.
2) A low metabolic rate results in slow healing after injury. Collagen type I accounts for 65% to 80% and elastin accounts for approximately 2% of the dry mass of tendons.
3) Histological examination of tendinopathy shows disordered, haphazard healing with an absence of inflammatory cells, a poor healing response, non-inflammatory intra-tendinous collagen degeneration, fiber disorientation and thinning, hypercellularity, scattered vascular ingrowth, and increased inter-fibrillar glycosaminoglycans.
4) Frank inflammatory lesions and granulation tissue are infrequent and are mostly associated with tendon ruptures.

b. Tendon healing
1) Phases similar to wound healing. Tendon healing occurs in three overlapping phases:
inflammatory → 1-2 days
proliferative → 30-70 days
remodeling → 70-365 days

c. Application of HBOT
1) Although normal tendons may function under increased anaerobic conditions, repairing tendons benefits from higher oxygen concentrations.
2) HBOT may be of benefit in the inflammatory and proliferative phases of healing which ultimately influences and benefits the results of the remodeling phase.
3) ↑ production of Type 1 procollagen RNA expression

4) ↓ Type III to Type I collagen ratio (less Type III collagen produced)
5) Synergism between growth factors and HBOT to decrease Type III collagen and increase Type I collagen.
6) Type I collagen associated with a stronger, more functional repair
7) One might extrapolate from this information that the use of HBOT to treat tendon and ligament injury might be most beneficial when applied immediately post injury and up to 45 days post injury. Early application would be more beneficial than later in the healing process.

11. Crush Injury, Compartment Syndrome, Other Acute Traumatic Ischemias
    a. Definition/descriptions
        1) A crush injury occurs when more than one tissue is involved in the trauma and tissue survival/viability is at question.
            a) Often a zone between tissue that will survive and tissue that will not survive – questionable gray zone.
            b) Functional deficits can occur despite successful recovery of the injured tissue.

        2) Compartment syndrome occurs when the pressure in a muscle compartment that is encased in a facial sheath increases and produces:
            ↓ perfusion
            ischemia
            cell/tissue death
            loss of function
        3) Characteristics
            a) Edema is a primary result and continuing insult
            b) ↓ capillary perfusion
            c) ↓ capillary integrity, leakage, WBC/RBC accumulation, edema
            d) ↑ muscle tissue compartment pressure
            e) Hypoxia
            f) A vicious circle develops as all of these changes contribute to
            Poor perfusion → hypoxia → more edema and pressure
    b. Effects of HBOT
        1) By elevating tissue oxygenation, ↓ hypoxia, → breaks the vicious cycle that occurs
        2) ↓ edema
        3) Restores aerobic energy metabolism
        4) Prevent reperfusion disease (see IR under cardiovascular section).
        5) Many diseases can result in ischemia, perfusion issues and loss of function due to this vicious cycle of events:

a) Direct trauma
b) Vasculitis (purpura hemorrhagica)
c) Frostbite
d) Envenomation (snake, spider and others)
e) Vascular diseases
f) Ergot poisoning

12. Delayed Radiation Injury (Soft Tissue and Boney Necrosis)
    1) Treatment of tumors with radiation is becoming more refined and the area of normal tissue that is affected is decreasing with the advent of precise application of the radiation.
    2) Radiation doses are also being refined to limit normal tissue destruction.
    3) HBOT has been used to also limit normal tissue loss and loss of function.
    4) Effects of radiation
        a) Energy transfers occur in the tissue
        b) Protein damage
        c) Lipid peroxidation
        d) DNA damage
        e) Currently reproducing cells are most sensitive
        f) Different tissue types have different sensitivity to radiation
        g) Blood vessels, bone, skin are very sensitive
        h) Some effects of radiation may not occur for years post therapy

    5) Application of HBOT
        a) Beneficial Effects
            ↑Tissue PO2
            Supports collagen formation
            Supports neovascularization
            Stimulates epithialization
            Similar to those effects in failing graphs and flaps
        b) HBOT benefits have been realized in post radiation therapy in a variety of tissues and systems:
            Osteoradionecrosis (mandible, skull, vertebrae)
            Neuropathies
                Soft tissue (extremities, head and neck, abdomen, bladder, rectum)
            Radiation hemorrhagic cystitis

13. Oncology
    a. Three scenarios may occur when a patient with a diagnosis of neoplasia is presented for hyperbaric oxygen therapy;
        1) patient is presented for the treatment of a problem not necessarily associated with the neoplastic diagnosis
        2) patient is being presented for treatment of the neoplastic condition using HBOT
        3) patient is currently receiving chemotherapeutic agents for the neoplastic condition and is presented for HBOT for another condition or to complement the chemotherapeutic modality. This often presents the clinician with a dilemma.

Currently there is not a wealth of evidenced based information on the treatment of neoplasia using HBOT. Here are some points to consider:

a) Based on previous reviews of the literature there is good evidence that HBOT does not increase tumor growth or increase metastasis of most tumors.
b) There is some evidence that HBOT may actually aid in the reduction of tumor growth.
c) There have been some research animal studies, but not many, on the clinical use of HBOT for neoplasia in animals.
d) There is some evidence that the concurrent use of HBOT and some chemotherapeutic agents may be synergistic in treating neoplasia. Consideration must be given to the mechanism of action and the side effects of both modalities, as there could also be synergism present in the side effects.

b. Conclusion: weight or evidence does not support tumor acceleration or metastasis with using HBOT
c. Conclusion: A history of malignancy should not be considered a contraindication for HBO2 therapy.
d. Evidenced based clinical guidance on HBOT and chemotherapeutic agents is currently lacking.
   1) Review mechanisms of both modalities to make risk assessment analysis.
   2) Some chemotherapeutic agents have a relative contraindication for the use of HBOT

Section 3

A. Side effects

Learning Outcomes

Following completion of this module each student they should be able to:↘

↘ know and be able to list and discuss the direct indirect side effects of hyperbaric oxygen administration in animals.
↘ discuss the signs of theses side effects in animals
↘ discuss the prevention and management of the side effects of hyperbaric therapy in animals

1. Physical Effects of pressure - Effects of elevated partial pressure of gases
   a. Review basic gas laws
      1) Boyles law
      2) Daltons Law
   b. Barotrauma (Baro = pressure, trauma = injury, injury due to pressure)
      1) Direct effects of pressure
         a) Definition - those effects that result from the changes that occur between the atmospheric pressure and the pressure in internal, air filled cavities of the body.

a) Air filled areas of the body include: ears, sinuses, teeth, intestine, alveoli

c. Pressure equalization: Pressure difference – as the external pressure increases to a level above the pressure in some air filled spaces, the pressure in the spaces must eventually equalize to be the same as the external pressure level.
   1) Failure to equalize may produce pain, anxiety, restlessness and even tissue damage (common example is the ears).
   2) Pressure can be equalized by several methods – animals cannot perform some of these methods but the first three are common maneuvers.
      Automatically occurs
      Swallowing
      Yawning
      Chewing
      Valsalva maneuver
      Toynby maneuver
      Others

d. Systems often affected include: respiratory, GI and auditory

e. Types of direct barotrauma include;
   1) **Squeezes/Blocks** - usually occurs on descent, external pressure is not equalized as it increases and squeezes the gas filled space.
   2) **Reverse Squeezes/Blocks** - usually occur on ascent, gas filled space expands as the external pressure decreases and excess pressure from the expansion is blocked from being released.

f. Causes
   1) Normal soft tissue that is collapsible
   2) Existing pathology affects function (swelling, inflammation)
   3) Rapid ascent or descent

g. Signs
   1) In animals must rely on behavioral changes
   2) Changes are usually due to pain
   3) Anxiety, restlessness
   4) Head shaking
   5) Swallowing
   6) Neck stretching
   7) Yawning
   8) Horse – phleming response

h. Pulmonary barotrauma – another direct effect of pressure changes
   1) Injury to pulmonary tissue due to excessive pressure
      a) Characteristics
         1a. Involves the alveoli
         2a. Usually occurs on ***ascent*** as the external pressure decreases and air filled spaces expand
         3a. Usually associated with rapid ascent and/ or breath holding on ascent.
         4a. What gas law governs this phenomenon?

5a. Usually the result of over expansion of the lung, rupture of alveoli allowing air to escape into the tissue or surrounding spaces

b) Types (ascending severity, one could lead to another, more than one can occur concurrently)

1a. Subcutaneous emphysema
Air under the skin, release from the thorax via the thoracic inlet to the skin of the neck& chest.
Air under the skin crackles to the touch, handling cellophane.

2a. Mediastinal emphysema
Air in the area between the lung lobes and at the hilus of the lung
Labored breathing, chest pain, reluctant to move or lie down.

3a. Pneumothorax
Air in the chest cavity and around the lung.
Labored breathing (dyspnea), anxiety, restless, diminished breath sounds on auscultation, hyper-resonant, cyanosis if severe.

4a. <u>Arterial gas embolism (AGE)</u>
Air that has escaped from the alveolus and enters the arterial capillaries
Sudden unconsciousness, seizure, hemiplegia, focal weakness, blindness, cranial nerve defects, confusion sudden onset.

2. Effects of the elevated partial pressure of gases - Indirect effects of pressure - those effects that occur because the change in pressure causes an excessive change in gas concentrations which may produce an adverse effect in the body.

a. Oxygen Toxicity

1) Types:

a) Central nervous system toxicity

b) Pulmonary toxicity

(What gas law governs the inspired oxygen concentration during hyperbaric therapy)

2) Description: oxygen toxicity occurs when elevated oxygen levels in the body increase the levels of oxygen radicals beyond the body's antioxidant system's ability to neutralize them. Imbalance between oxygen radicals and antioxidants.

3) At pressures <3 ATA hyperbaric therapy does not significantly increase oxygen radicals in the body.

4) Signs and Contributing factors

a) <u>Signs</u>: prodromal seizure signs, facial muscle twitching, anxiety, confusion, vision disturbances, fatigue, convulsions.

b) <u>Contributing factors</u>: ↑ CO_2, exercise,↑ body temperature, individual differences.

5) Treatment and prevention

a) Treatment: Lower the pressure, decrease oxygen concentration (only in chambers equipped). Signs generally subside when pressure is decreased.
b) Prevention: limit oxygen exposure, intermittent O_2/air breathing, no 100% O2 at pressures > 3ATA.

6) Pulmonary Oxygen Toxicity
a) Time/dose relationship, usually occurs with extended single oxygen exposures or exposures over a long period of time (cumulative damage).
b) Signs: labored breathing, cough, short breaths
c) Cause: direct oxygen effect, ↑ CO2, inert gases, surfactant changes.
d) Treatment: remove from oxygen source
e) Prevention: intermittent O2/air breathing, limit exposure

b. Nitrogen narcosis
1) All inert gases may have some narcosis or side effects associated with them, depending on their solubility in lipids.
2) Occurs when the inhaled nitrogen concentration is high, e.g. breath air, and the pressure increases.

3) Signs:
a) euphoria, mood change
b) loss of concentration and judgment
c) slow reflexes/reactions
d) difficulty completing tasks
4) On average begins to create an effect at around 100 fsw and severity of narcosis increases as the depth increases.
5) Prevention
a) remain or return to lower pressure/shallower depth
b) reduce nitrogen in breathing gas (use oxygen = nitrox, e.g. helium)

c. Contaminant gases
1) carbon monoxide, carbon dioxide, hydrocarbons,

Section 4

A. Contraindications

Learning Outcomes
↘Following completion of this module each student they should be able to:↘
discuss the absolute and relative contraindications for the use of hyperbaric oxygen therapy In animals

1. Often divided into absolute and relative contraindications. Hyperbaric oxygen therapy is a very safe treatment modality and contraindications for its use are few. Each patient must be evaluated and examined to determine its suitability for the hyper-oxygenated, pressurized environment. The contraindications discussed here are known conditions that may place the use of HBOT at a different risk level, but other scenarios may preclude its use. As more research

and information is accumulated about some of these situations their status may change.

2. Relative contraindications – may or may not prevent the use of hyperbaric oxygen as a treatment. Decision is made case by case and following a risk assessment.
3. Absolute contraindication – uniformly prevents the use of hyperbaric oxygen therapy as a treatment regardless of the case situation.
4. Once an animal patient is in the chamber we lose some immediate control over the patient. When a complication occurs immediate intervention is not possible since we do not have attendants in the chamber with the patient.

b. Absolute contraindications (historical)
   1) Medications
      a) Doxorubicin – potentiation of cardiac toxicity (3 day withdrawal time)
      b) Bleomycin – Pulmonary toxicity, modest ↑ in inspired O2 triggers interstitial pneumonitis even years later
      c) Disulfiram – Increase seizure risk, blocks superoxide dismutase (SOD), multiple HBOT Txs.
      d) Cis-platinum – HBOT ↑ cytotoxicity, impedes wound healing
      e) Mafenide acetate - ↑ CO2 retention → peripheral vasodilation, HBOT → central vasoconstriction, HBOT + Mefenide → synergistic for both effects.
   2) Pre-existing conditions
      a) Untreated pneumothorax - may create a tension pneumothorax, dyspnea and severe distress.

c. Relative contraindications
   1) Abnormal temperature
      a) hyperthermia and hypothermia
      b) hyperthermia may predispose a patient to CNS O2 toxicity
      c) hypothermia may decrease the effects of trying to produce high tissue oxygen concentrations. This may be particularly a problem in our very small animal patients as temperatures fluctuate in the chamber..
   2) Opioids
      a) depends on the type of opiate that is used
      b) some opioids produce respiratory depression and produce CNS stimulation which can increase the risk for seizure activity.
      c) the use of transcutaneous delivery of medications does have the potential for increased heat between the patch and the skin as well as the potential for alteration of the amount of drug that is delivered in the hyperbaric environment.
   3) Sedatives
      a) Each patient should be evaluated for its temperament at the time of therapy. Tranquilization might be

necessary for the safety of the patient and the equipment. However…….

b) In our experience most small animals do not routinely require sedation or tranquilization. A drug such as diazepam (oral, rectal) would be a good choice.

c) Large animals, may require sedation or tranquilization more frequently, especially equines. Detomidine, IM and IV works well and most horses seem to tolerate it well. Using a small amount IV and concurrently using an IM dose will give an immediate sedative effect plus a long lasting effect.

4) Active Seizures
   a) review the history and current therapies for any history of seizure or drugs that are associated with seizure control. A history of seizure does not completely rule out the use of HBOT but does increase the risk for possible seizures.

5) Uncontrolled hemorrhage
   a) a patient that is bleeding and placed in the chamber may run the risk of potentiation of the bleeding due to the increased pressure, this coupled with the fact that we do not have ready access to triage a patient creates a potential serious complication for the patient.

6) Obstructed Airway
   a) limits inspired air flow and pulmonary oxygen delivery
   b) may exacerbate airway collapse
   c) airway obstruction may prevent exhalation of pulmonary gases as those gases expand on decompression producing pulmonary barotrauma

7) Equine Guttural Pouch Diseases
   a) Originally thought to be a contraindication but many horses having this condition have been successfully treated in the hyperbaric chamber.
   b) could effect equalization of pressure in the ears

8) Severe sinus pathology
   a) has the potential to produce a sinus squeeze or block

9) Splenic hemangiosarcoma
   a) A very fragile tumor that could result in hemorrhage in the pressurized environment

10) Other conditions
   a) pregnancy – anecdotally not contraindicated in the horse, other species we do not know
   b) malignancy – there is not overwhelming evidence that HBOT accelerates tumor growth. In some cases it has had a beneficial effect in suppressing tumor growth

and has been used synergistically with chemotherapeutic agents to suppress growth.

c) Pacemakers – there are pacemakers that have been approved for the hyperbaric oxygen environment, but these are not routinely used in animals.

11) Completing a risk to benefit analysis of patients with relative contraindications is a sound practice when one of these and other situations are encountered

Section 5

A. Patient selection and preparation

Learning Outcomes

Following completion of this module each student they should be able to:↘

↘discuss the thinking behind patient selection.

↘discuss the and/or design a protocol for the evaluation of a patient that is recommended for hyperbaric oxygen therapy.

↘discuss how a patient is prepared for hyperbaric therapy and the safety aspects that are involved.

1. Patient selection
   a. The selection of patients for hyperbaric therapy relies on a ***sound knowledge of the beneficial effects of and the indications for HBOT***, as well as knowing ***what situations might cause untoward effects in the patient.***
      1) The main purpose of thoroughly evaluating the selected patient is to assure that the patient can ***tolerate the pressurized environment***.
   b. The selection of animal patients for HBOT is based on:
      1) a knowledge of the physiologic and therapeutic effects of hyperbaric oxygen.
      2) a knowledge of the potential side effects
      3) contraindications
      4) correlation of the first three with the patient at hand based on the condition to be treated, history, diagnosis and current physical status.
2. Patient evaluation
   a. The components associated with patient evaluation, suitability for HBOT;
      1) diagnosis
      2) physical status
      3) concurrent therapies
   b. Physical status is determined by examining the history and performing a pretreatment examination.
      1) Is there anything in the history that would indicate the development of a problem during therapy or that would be a No Go situation.

2) A physical exam should be performed immediately pre-treatment regardless of how many other exams have been performed that day – TPR, mucous membrane status, heart rhythm, respiratory sounds/character, nasal discharge etc. (those system that might be affected by the pressurized environment)
3) First hand observation of the attitude of the patient, his/her temperament. The hyperbaric environment is one that many patient have not experienced and there are all sorts of changes, noises etc. Compatibility may require sedation for safety and good patient outcome.

c. Concurrent therapies
1) compatibility with the hyperbaric environment
2) Establishing a GO-NO Go list is helpful and a quick reference in the clinical setting. Naturally not everything imaginable can be placed in such a list, but common things and general categories of things can be listed as a reminder and reference.
3) For uncommon things a risk assessment can be performed.

d. A risk assessment is performed if there are any physical problems that might be adversely affected by HBOT and if there are concurrent therapies that require the introduction of materials, devices, medications that are not on the Go No Go List.

3. Patient preparation
a. Patient and personnel safety
1) Elimination of potential ignition sources
a) No conscious patient non-compliance
b) Personnel as a potential source (inadvertent, poor training)
c) equipment, concurrent therapeutic modalities
2) Elimination or reduction of unneeded fuel sources
a) Hair is a major fuel source in most animals
b) removal of grooming products and other topicals (medications et.)
c) removal of bedding, dirt, feces
d) assess materials/equipment, medications, and other compounds for use in hyperbaric oxygen environment. Risk assessment may be required.
3) Provide best possible outcomes

Module 3

Overview and Learning Outcomes

Module 3 provides a basic information in three areas; 1) equipment and facilities, 2) safety, and 3) standards and codes. We will discuss types of chambers utilized in animal hyperbaric medicine and the common components of a hyperbaric chamber system. Key points will be mentioned concerning the associated with the chamber and facilities, chamber operations, and the patient. Lastly, we will discuss codes and standards that are associated with hyperbaric chambers and their operation.

Section Learning Outcomes

Following completion of this module each student they should be able to: ↘

- *Section 1 Animal Hyperbaric Systems Characteristics*
  - ↘ describe the NFPA classification of hyperbaric chambers and explain how animal chambers for clinical use fit into this classification.
  - ↘ be able to list the common components of any hyperbaric chamber and explain their purpose.
  - ↘ discuss some of the major factors in determining a safe location for a hyperbaric chamber
- *Section 2 Operational chamber safety*
  - ↘ be able to describe the fire triangle and how fires can be prevented in a hyperbaric chamber using the knowledge of fire chemistry.
  - ↘ be able to describe the concepts or risk assessments and risk management
  - ↘ describe and/or demonstrate the proper handling of compressed gas tanks and lines associated with the delivery of hyperbaric oxygen therapy.
  - ↘ discuss how chamber construction, testing, and maintenance are related to safety.
  - ↘ describe the philosophy associated with ignition sources and the 100% oxygen environment, including the concern over static electricity in our animal patients.
  - ↘ describe the major components of safely dealing with potential emergency events associated with hyperbaric oxygen therapy for each of the following: fire inside the chamber, fire outside of the chamber, loss of power, loss of O2 source, major patient incident such as seizure, dyspnea, etc.
  - ↘ Describe the responsibilities of the safety director, medical director, chamber operation team
- *Section* 3 Guidelines*, recommendations, standards, codes, and laws*
  - ↘ Provide the actual name of the following organization acronyms and describe their general area of guideline for hyperbaric oxygen therapy

    NFPA
    CGA
    ASME

ASTM
FDA

Section 1

A. Animal Hyperbaric Systems Characteristics

Learning Outcomes
↘ describe the NFPA classification of hyperbaric chambers and explain how animal chambers for clinical use fit into this classification.
↘ be able to list the common components of any hyperbaric chamber and explain their purpose.
↘ discuss some of the major factors in determining a safe location for a hyperbaric chamber

1. Chamber classification
   a. National Fire Protection Association (NFPA) classifies chambers by occupancy in their codes and guidelines:
      Class A – Human – multiple occupancy
         Most of these chambers are pressurized with air and the patient breathes oxygen via a mask.
      Class B – Human – single occupancy
         Most of these chambers are pressurized with 100% oxygen
      Class C – Animal, no human occupancy
         Most of these chambers used in a clinical environment are pressurized with 100% oxygen and some can accommodate more than one patient.
   b. Large animal chambers may be as large as a Class A human chamber, but are pressurized with 100% oxygen. One large animal design is very similar to the Human Class A chamber as it is pressurized with air and the patients wear a mask that delivers 100% oxygen.
   c. Most animal chambers for clinical use are:
      Small animal - cylindrical and horizontal
         steel or acrylic
      Large animal – cylindrical or rectangular
         Steel
2. Components - Characteristics common to most animal chambers
   a. Chamber – design, materials, integrity, regulations
   b. Gas delivery – oxygen, air
      1) oxygen systems – bottled gas, liquid oxygen (LOX)
      2) air - compressor systems, bulk tank
   c. Control panel -
      1) monitoring and control – gas, pressure, temperature, humidity.
      2) often operated via gas pressure, electricity or both.
   d. Chamber construction –

1) Codes and standards – ASME and PVHO animal chambers built to human standards
2) Testing – hydrostatic to 1.5 times the working pressure, if chamber boundary is compromised after initial testing additional hydrostatic testing is necessary after penetration or damage.
3) Acrylic portals – periodic examination, replacement if significant decrease in structural integrity or damage occurs.
   - exposure to UV light
   - chemicals
   - use approved cleaning agents

e. Pressure relief – valve that releases oxygen/air when the chamber pressure exceeds its designed limits. Requires periodic testing and replacement.

f. Exhaust – piping that expels chamber gas to the outside, location and configuration.

g. Other components
1) fire suppression systems
2) penetrators
3) video monitoring
4) burst discs
5) humidification

3. Review small and large animal hyperbaric systems
Systems available at this time:

B. Facilities and installation

1. Facilities
a. space and location
b. dimensions
c. access, security
d. oxygen source, exhaust
e. environmental control

2. Installation

Section 2

Learning Outcomes

- be able to describe the fire triangle and how fires can be prevented in a hyperbaric chamber using the knowledge of fire chemistry.
- be able to describe the concepts or risk assessments and risk management
- describe and/or demonstrate the proper handling of compressed gas tanks and lines associated with the delivery of hyperbaric oxygen therapy.
- discuss how chamber construction, testing, and maintenance are related to safety.
- describe the philosophy associated with ignition sources and the 100% oxygen environment, including the concern over static electricity in our animal patients.
- describe the major components of safely dealing with potential emergency events associated with hyperbaric oxygen therapy for each of the following: fire inside the chamber, fire outside of the chamber, loss of power, loss of O2 source, major patient incident such as seizure, dyspnea, etc.
- Describe the responsibilities of the safety director, medical director, chamber operation team

A. Operational chamber safety

Incident – an event that occurs which does not result in loss but may have involved an unsafe condition or unsafe act.

Accident – a sudden, unplanned event that causes loss.

Risk control – any conscious act or decision not to act intended to reduce the frequency, severity or loss from an accident.

1. Chamber fire safety
   a. Fire chemistry
      1) components of fire – fire triangle
      2) Ignition temperature and flash point
         a) Flash point = temperature required for a fuel source to reach a vapor phase.
         b) Ignition temperature = that temperature which allows the combination of fuel and oxygen to combust.
         c) Burn rate – in chemistry, the burn rate is a measure of the linear combustion rate of a compound or substance. It is measured in length over time, such as mm/second or inches/second.
   b. Fire prevention
      1) key factors:

- Always be more conservative in chamber filled with 100% oxygen.
- Never place anything in the chamber that could be an ignition source or produce an exothermic reaction.
- Keep amount of potential fuel sources in and immediately outside the chamber to a bare minimum.
- Ideally, materials that need to be in the chamber should have high flash points and high ignition temperatures.
- Always use common sense, make decisions on the conservative side, evaluate risks, consult.

Eliminate ● Control ● Limit

2. Risk assessment and management - A risk assessment is a systematic examination of a task, job or process that you carry out at work for the purpose of identifying the significant hazards, the risk of someone being harmed and deciding what further control measures you must take to reduce the risk to an acceptable level.
   Risk control – any conscious act or decision not to act intended to reduce the frequency, severity or loss from an accident.
   Used routinely in patient preparation and evaluation.
3. Operational safety
   a. Standard operating procedures
      1) each facility should develop standard procedures based on best practices, facilities, and type of chamber.
      2) SOP's should be written and copies available to all personnel involved.
      3) Detailed chamber operation manual should be available at the chamber.

4. Hyperbaric Chambers
   a. manufactured according to ASME-PVHO standards and codes
   b. initial pressure test and additional pressure test if any alteration to the vessel, purposeful or accidental.
   c. tested to 1.5 times the working pressure
   d. chamber maintenance is a critical part of operational safety
      visual inspection – acrylic, doors, penetrators
      pressure relief valve testing and or replacement
      leak testing – door seals and other penetrations with seals
   e. maintenance logs must be kept "if it's not written, it didn't happen".
5. Pressurized gases
   a. label all cylinders/color code,
   b. purity, reputable supplier
   c. appropriate valve and fixed storage
   d. label pipes – gas type and flow direction; valve open & close direction
6. Electricity and Ignition Sources
   a. 100% O2 environment - keep all electrical items **outside of the chamber.**
   b. chamber must be adequately grounded
      difficult to ground animal patients but may be able to ground restraint containers housing animal patients
   c. static electricity prevention
      maintain adequate humidity levels in the chamber, ideal = 50% measure
      humidifiers, wet towels
      grounding
      grounding mats
      moistening the patients hair/fur
   d. evaluate substances/equipment etc. for heat production potential

7. Other operational safety items
   a. establishment of emergency protocols for potential scenarios
      e.g. loss of electrical power, loss of control panel operation, patient complications, loss of oxygen supply, loss of compressed gas supply, fire inside and outside of the chamber.
      written protocols available for operators
   b. rapid decompression procedures
   c. exhaust system – location of external exhaust pipe opening and inspection
   d. chamber deluge systems
   e. air compressors for control panel operation

B. Training
   1. Basic and advanced training
      a. Complete basic animal hyperbaric training course
      b. Experience in managing and handling animals
      c. Training and experience in chamber operations
      d. Completion of a safety director course
      e. Board certification

   2. Continuous training
      a. safety drills and procedures
      b. chamber operations
      c. chamber maintenance

C. Emergency procedures
   1. Policies and Procedures – Emergency
      a. Emergency procedures should be posted with easy access
      b. Plan for potential emergency scenarios
         1) Fire in the chamber
         2) Fire outside the chamber
         3) Loss of power supply
         4) Loss of O2 supply
         5) Loss of compressed gas supply
         6) Patient seizure or other incident in the chamber
         7) Others…..
      c. Staff training, emergency drills, training log
   2. General emergency *Shut Down* procedures
      Make decision on patient and operators status and movement
      "Human Safety is the Primary Concern"
      a. Discontinue oxygen flow
      b. Decompress
      c. Rapid decompression schedule
   3. Possible rapid decompression rates
      a. Base rate is 0.5 ft./sec
         1) From 2.0 ATA 33 ft. = 1 min.
         2) From 2.5 ATA 49.5 ft. = 1.5 min.
         3) From 3.0 ATA 66 ft. = 2.2 min
   4. Situational analysis
      a. Type of emergency

b. Loss of electrical power/compressed gas
c. Chamber mal-function
d. In-chamber patient incident or emergency – life threatening or non-life threatening
e. In-chamber fire
f. External fire

5. Safety plan
   a. Dependent on situational analysis
   b. Personnel safety is #1 – Patient safety – Chamber & Equipment
      1) Personnel and patients in the immediate area
      2) Evacuation strategy
      3) Emergency chamber shut down and decompression procedures
         a) Shut down procedures are chamber and facility dependent

6. Policies and Procedures - ***Fire inside the chamber****
   a. Eliminate incoming oxygen
   b. Activate chamber fire suppression (if available)
   c. Initiate rapid decompression and pressure relief
   d. Evaluate need to evacuate the area
   e. Activate building fire alarm system?
   f. Contact Hospital Operations

* Most in chamber fires in the monoplace environment are fatal to the patient.

7. Policies and Procedures - ***Fire outside of the chamber***

1) Situation analysis – determine location of the fire
   a) Immediate area or remote
   b) Relationship to fire doors (e.g. other side of 2 hr. fire door/wall)
2) Fire in immediate area
   a) Don face mask
   b) Eliminate incoming oxygen
   c) Initiate decompression and pressure relief
   d) Evacuate the area
3) Fire in remote area
   a) All procedures for fire in immediate area
   b) Remove patient
   c) Evacuate the area

8. Policies and Procedures – Emergency – ***Lost of Electrical Power***
   a. Loss of control panel function
   b. Less of a problem for pneumatically controlled chambers
      1) Continue with treatment or discontinue – standard decompression
      2) Pneumatic control supplied by compressor – reservoir tank
   c. Chambers with an electrical component to the chamber control

1) Use of emergency backup power
2) Use of back up battery power
3) Determine if decompression can occur without power – chamber dependent

9. Policies and Procedures - ***Loss of lighting in and around the chamber***
   a. Contingency lighting sources
10. Policies and Procedures – Emergency – ***Loss of O_2 Supply***
   a. Monoplace chamber
      1) Decompress and remove the patient - Standard decompression procedures
   b. Multiplace chamber
      1) Mask delivery of 100%O2 - Continued treatment might cause decompression sickness
      2) Initiate standard decompression
11. Policies and Procedures – Emergency – ***Major Patient Incident***
   a. Patient complication
      1) Determine the complication
      2) Determine the degree of patient risk
         a) life threatening
         b) Serious, not life threatening but might become so
         c) Not serious, not life threatening
      3) Decide on a course of action
         a) Immediate and rapid decompression
         b) Decompression to a level of less pressure
         c) Moderately rapid decompression schedule
         d) Standard decompression schedule

D. Program Development and Oversight *(Table 6)*
1. Program oversight (medical and safety)
   a. Team composition
      1) Hyperbaric medical director
      2) Hyperbaric safety director
      3) Hyperbaric chamber operations team
      4) Primary care team
   b. Responsibilities of medical director, safety director and chamber operation team should include completion of a quality basic hyperbaric medicine course.
   c. Role of safety director connotes additional training in a quality safety director certificate course.
   d. Table 2 lists some of the responsibilities of each team responsible for the patient before, during and after hyperbaric oxygen therapy.
   e. Multiple roles can be assumed by one individual.
   f. Adequate redundancy in training, so coverage of the hyperbaric treatment program is consistent.

Table 6. Responsibilities for Hyperbaric Patient Care and Safety

Position	Responsibilities	
Hospital Director	Manages all hospital operations – financial, facilities, personnel, communications, etc. Might be the owner of a practice-	
Hyperbaric Med Director	Veterinarian	
	Working knowledge of HBOT	
	Knowledge of the side effects, contraindications and their prevention/management	
	Makes recommendations concerning the efficacy of HBOT in patients in consultation with the primary care team	
	Recommends HBOT protocols for patients	
	Works in collaboration with the safety director to assure patient and personnel safety	
Hyperbaric Safety Director	Recognition of hazards	
	Policies and procedures development	
	Oversight of safety training and education	
	Analysis/problem solving – decision making and risk assessment	
	Equipment & facilities maintenance needs	
	Documentation	
Chamber Operations Team	Day to day chamber operation	
	Patient management	
	Chamber maintenance and hygiene	
Primary Patient Care Team	Works with HBOT medical director in making decisions for HBOT for each patient	
	All other care associated with the patient including treatments, diagnostics, etc. outside of HBOT	
	Directs and coordinates other members of the primary care team	

Section 3

Learning Outcomes

↘ Provide the actual name of the following organization acronyms and describe their general area of guideline for hyperbaric oxygen therapy
NFPA
CGA
ASME
ASTM
FDA

A. Guidelines, recommendations, standards, codes, and laws
1. least mandatory to most mandatory
2. Important to the safety program
3. Also used in day to day operations

B. Important Governing and Guiding Organizations *(Table 7)*
1. ASME = American Society of Mechanical Engineers - Guidance for the production, inspection and certification of animal hyperbaric chambers.
   a. The Boiler and Pressure Vessel Code (BPVC, 2017), Pressure Vessels for Human Occupancy PVH-O 1&2]
2. NFPA = National Fire Protection Association
   a. NFPA 99 - Health Care Facilities code – contains information applicable to animal hyperbaric facilities
   b. NFPA 50 - Standards for Bulk Oxygen Systems
   c. NFPA 101 - Life Safety Codes
   d. NFPA 150 – Fire and Life Safety in Animal Housing
3. CGA = Compressed Gas Association
   a. Use of and handling compressed gases, gas containers, gas purity, gas identification.
4. ASTM = American Society for Testing and Materials
   a. Compatibility of materials for a high oxygen environment
   b. Materials for oxygen systems design
5. FDA = Food and Drug Administration
   a. Approval of medical devices and applications of medical devices and drugs.
6. OSHA = Occupational Health and Safety Administration
   a. Oversees facilities, equipment, and operations with regard to personnel safety.

Table 7. Important Governing and Guiding Organizations

Organization	**Acronym**
American Society of Mechanical Engineers	ASME
National Fire Protection Association	NFPA
Food and Drug Administration	FDA
American Society for Testing and Materials	ASTM
Compressed Gas Association	CGA
Resources	
Undersea and Hyperbaric Medical Society	UHMS
National Board of Diving and Hyperbaric Medical Technology	NBDHMT
Veterinary Hyperbaric Medicine Society	VHMS
International Hyperbaric Medicine Association	IHMA

Resources

Textbook of Hyperbaric Medicine. KK Jain 6th Ed. 2017.
Physiology and Medicine of Hyperbaric Oxygen Therapy. TS Newman, SR Thom. 2008.
Hyperbaric Medicine Practice. HT Whelan, EP Kindwall, 4th Ed. 2017.
Hyperbaric Facility safety: A Practical Guide. WT Workman, JS Wood, 2nd Ed. 2019.
Hyperbaric Oxygen for Neurologic Disorders. J Zhang, 1 st Ed. 2008
NFPA 99 Health Care Facilities Code Handbook. 2018 Ed.

Table 2. Nomenclature

Nomenclature and Conversion of pressure Units

Equivalents	**1 Atmosphere**	**1 mmHg**	**1 Torr**
Atmospheres	-	0.0013	.0013
mmHg	760	-	1.000
Pounds/sq.in. (psi)	14.7	0.0680	0.0019
Feet of sea water (fsw)	33.0	0.0303	
Meters of sea water (msw)	10.0	0.1000	
Conversions			
ATA to mmHg	ATA x 760 mmHg		
ATA to psi	ATA x 14.7 psi		
ATA to fsw	(ATA-1) x 33 fsw		
psi to ATA	(psi + 14.7) ÷14.7 psi		
fsw to ATA	(fsw +33) ÷ 33 fsw		

Table 1. Gauge Pressure

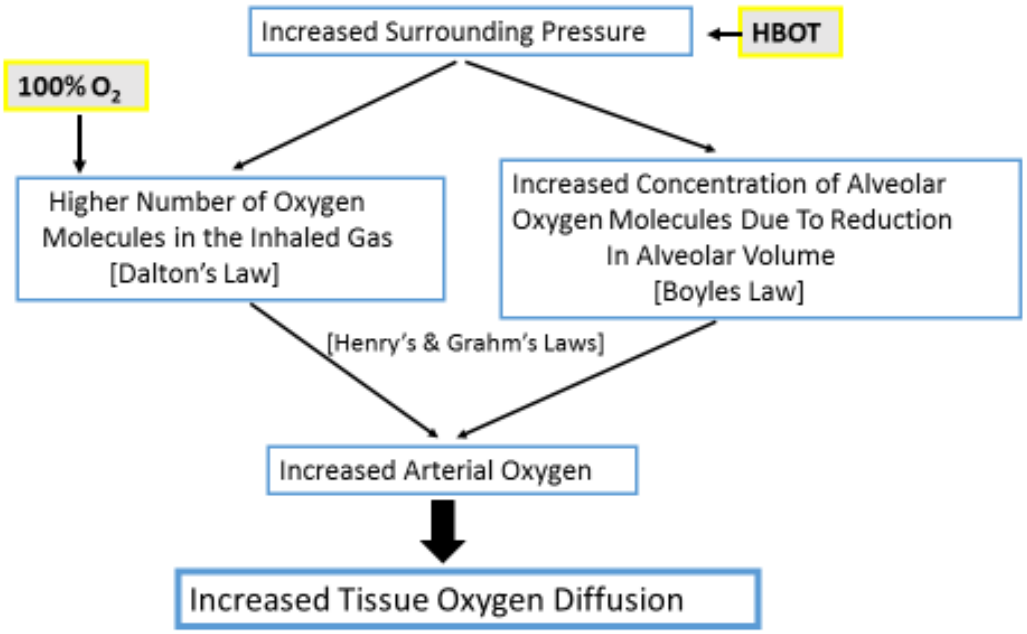
Gauge Pressure

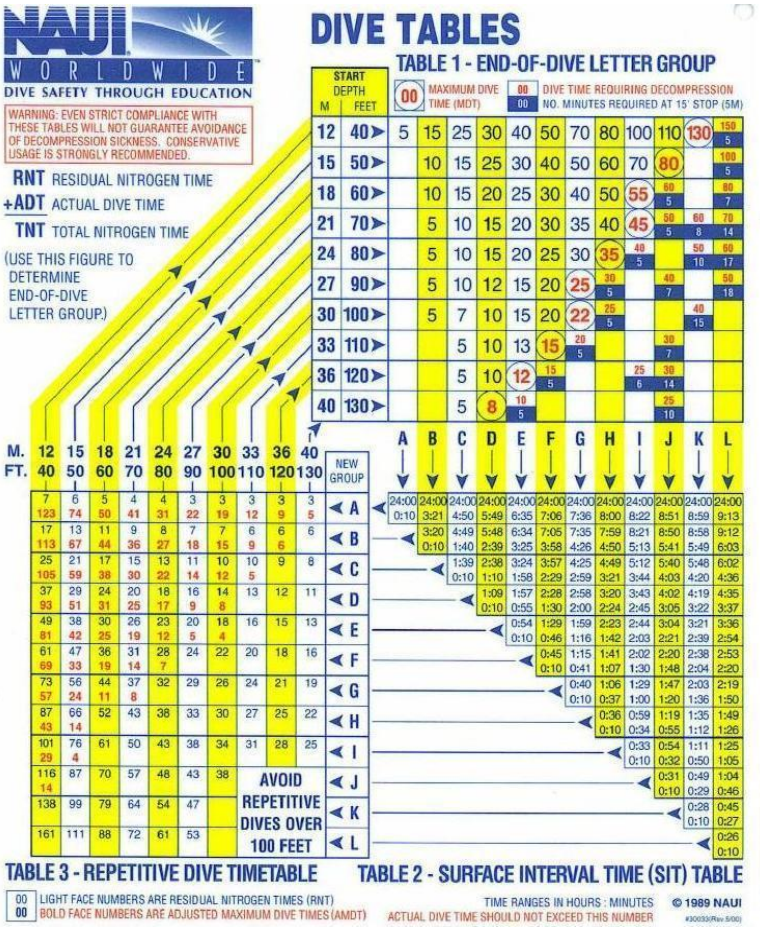
The difference between the pressure being indicated and the atmospheric pressure.

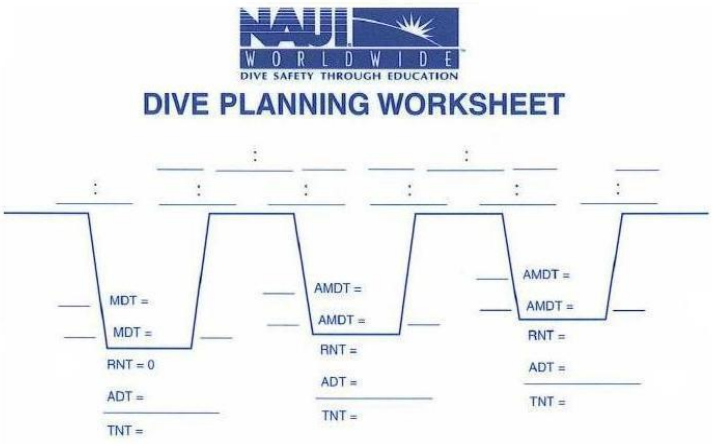
If we are at sea level and the pressure you have developed in the chamber is 1 ATA and the atmospheric pressure at sea level is 1 ATA then the gauge pressure is zero.

Absolute pressure			**Gauge Pressure**	
ATA	*mmHg*	*psi*	*fsw*	*psi*
0	0			
1	760	14.7	0	0
2	1520	29.4	33	14.7
3	2280	44.1	66	29.4
4	3040	58.8	99	44.1
5	3800	73.5	132	58.8
6	4560	88.2	165	73.5

Diagram 7. Increasing Tissues Oxygen Concentrations







TERMS AND ABBREVIATIONS USED IN DIVE PLANNING

Repetitive Dive – Any dive made less than 24 hours after a previous dive.
ADT – Actual Dive Time – The time from the moment of descent until returning to the surface.
Letter Group – A letter symbol for the amount of Residual Nitrogen remaining in the body from previous dives.
SIT – Surface Interval Time – The time spent at the surface between dives.
RNT – Residual Nitrogen Time – The nitrogen remaining in the body from a dive or dives made within the past 24 hours.
AMDT – Adjusted Maximum Dive Time – The maximum Dive Time for the depth of a dive minus the RNT.
TNT – Total Nitrogen Time – The sum of RNT and ADT. This figure is used to obtain a letter group after repetitive dives.

REMEMBER

- Consider all dives made shallower than 40'/12m as 40' dives.
- On any dive, ascend no faster than one foot every two seconds (30ft/9m per minute).
- For maximum dive time, make all repetitive dives shallower than your previous dive.

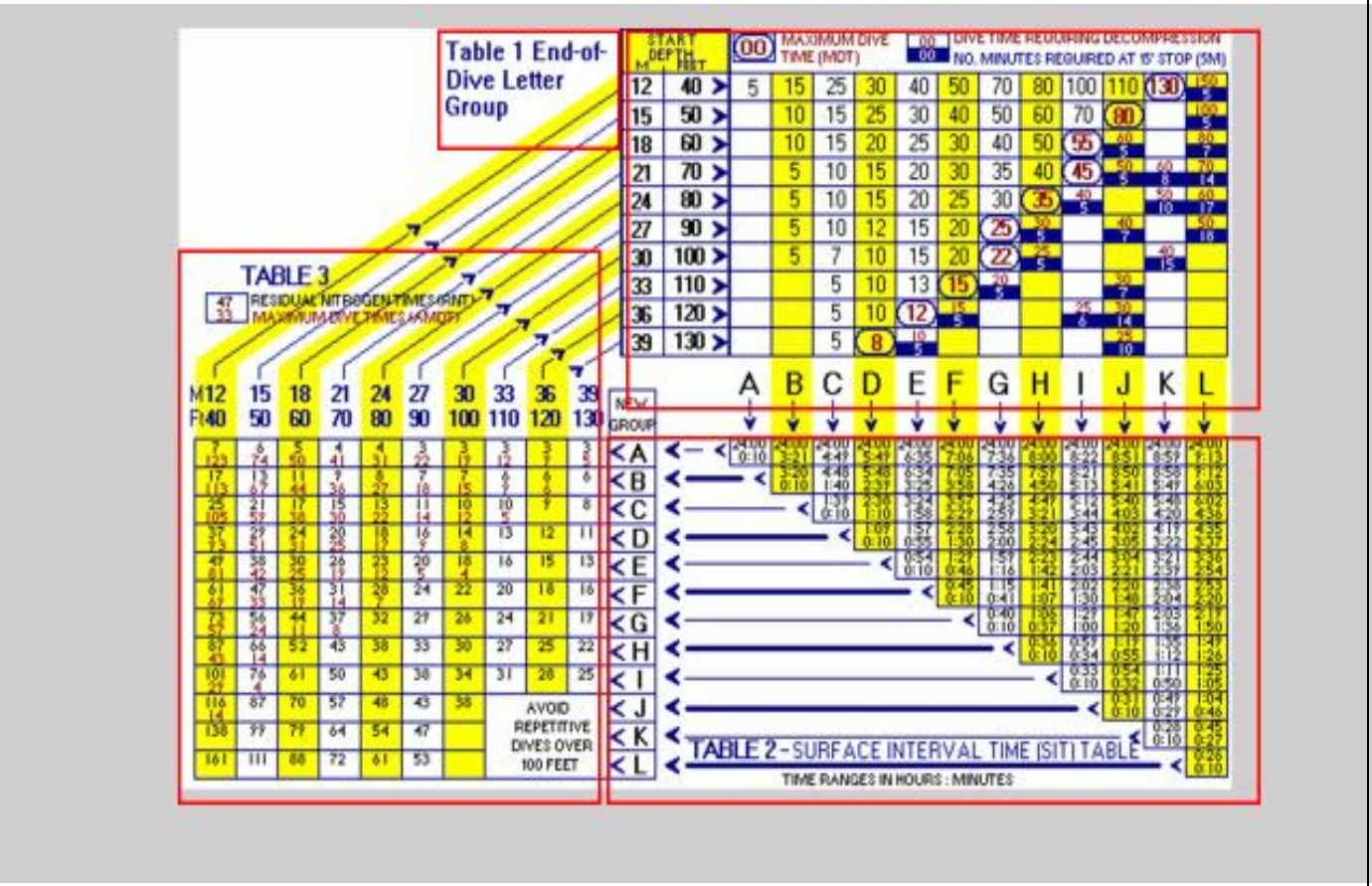


Diagram 3. NAUI No-Decompression Dive Tables

Diagram 4. Example Use of No-Decompression Dive Tables

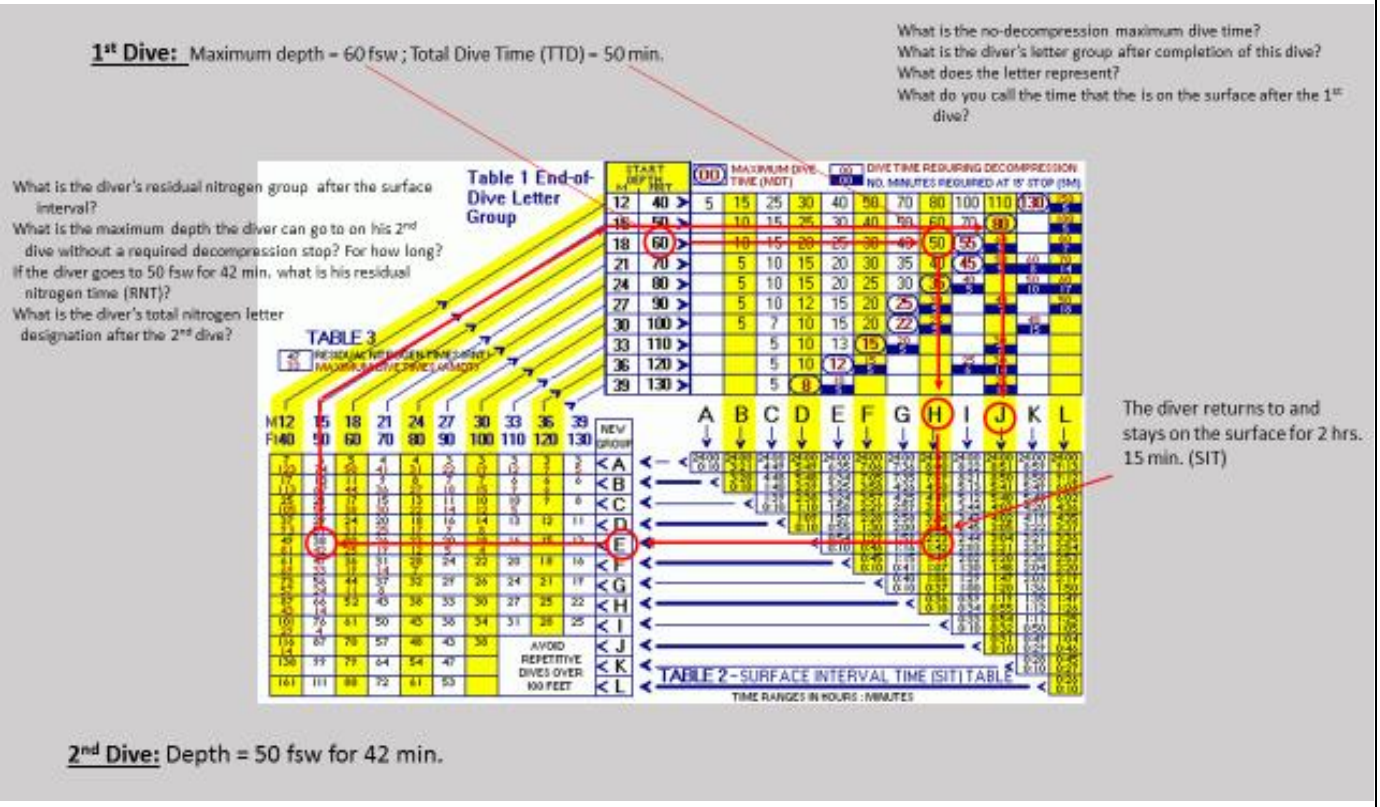

1st Dive: Maximum depth = 60 fsw ; Total Dive Time (TTD) = 50 min.
What is the no-decompression maximum dive time?
What is the diver's letter group after completion of this dive?
What does the letter represent?
What do you call the time that the is on the surface after the 1st dive?
What is the diver's residual nitrogen group after the surface interval?
What is the maximum depth the diver can go to on his 2nd dive without a required decompression stop? For how long?
If the diver goes to 50 fsw for 42 min, what is his residual nitrogen time (RNT)?
What is the diver's total nitrogen letter designation after the 2nd dive?
Table 1 End-of-Dive Letter Group
TABLE 3
TABLE 2 - SURFACE INTERVAL TIME (SIT) TABLE
TIME RANGES IN HOURS : MINUTES
AVOID REPETITIVE DIVES OVER 100 FEET
The diver returns to and stays on the surface for 2 hrs. 15 min. (SIT)
2nd Dive: Depth = 50 fsw for 42 min.

Diagram 6. Examples of Repetitive No-Decompression Dive Calculations

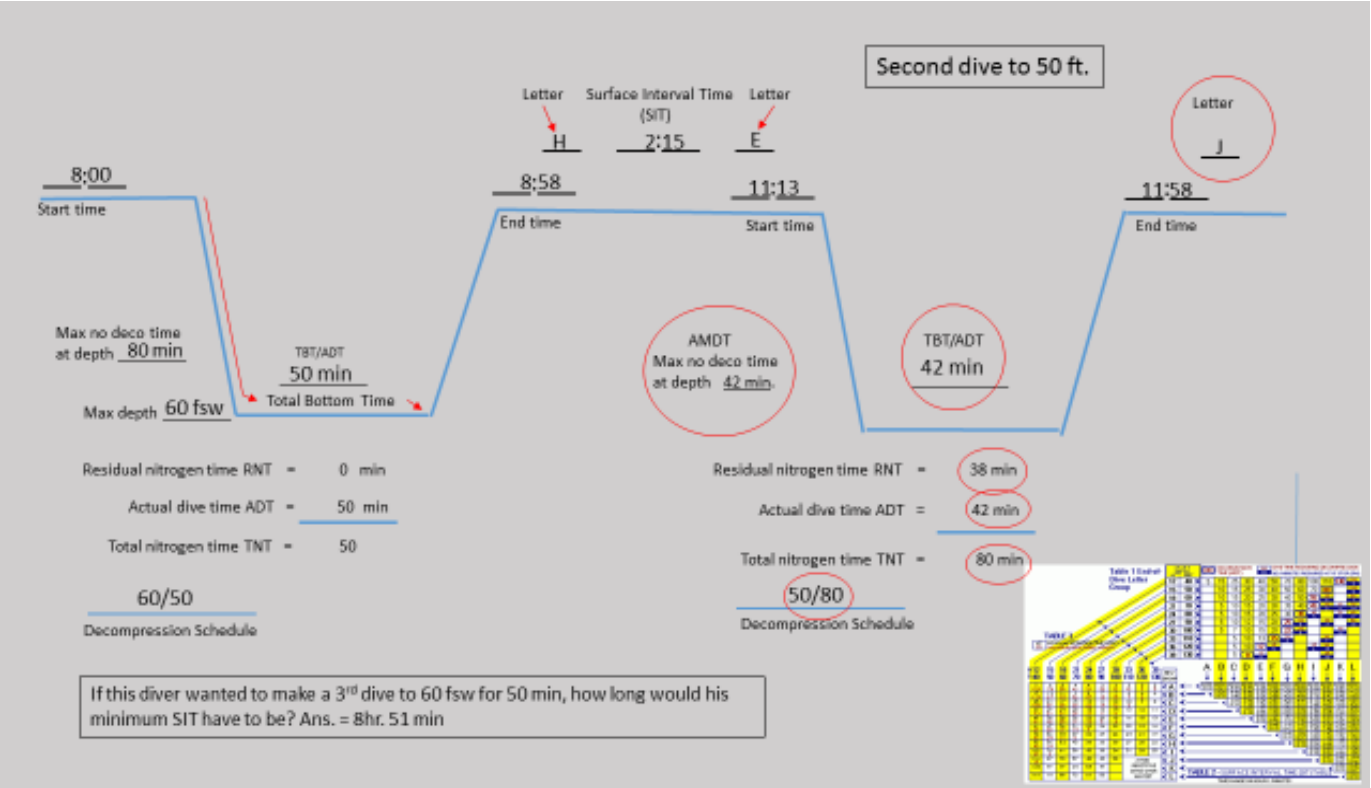


Diagram 5. Example of Useful Dive Profile Work Sheet

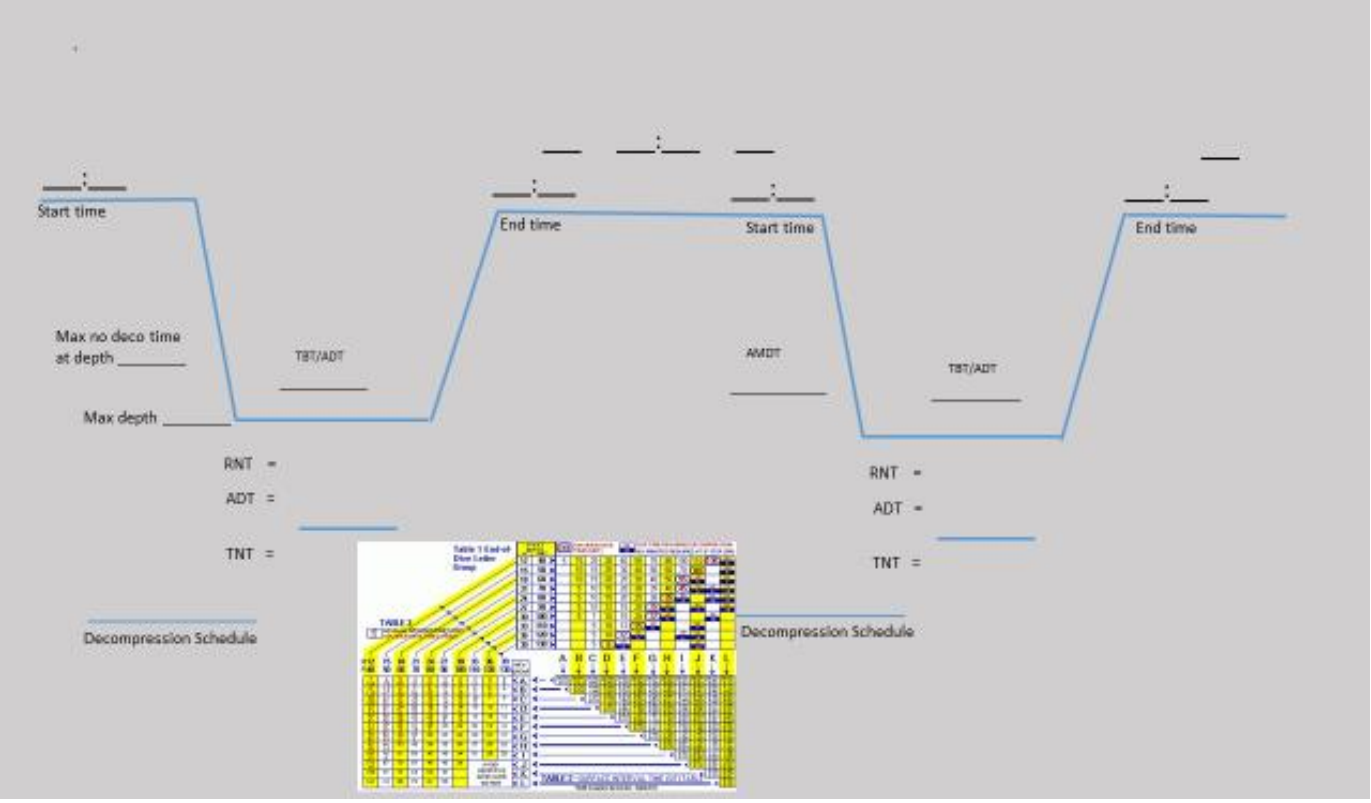


Table 5. Effect of Oxygen Concentrations on Antimicrobial Activity

Oxygen Tensions Influence Antimicrobial Activity

Antimicrobial	Anoxia	Hyperoxia/HBO	
Aminoglycoside	↓ bacteriocidal effects	↑O2 required for optimal activity	
Fluoroquinolones		↑O2 required for optimal activity	
Sulfas, Trimethoprim	↓bacteriostatic effects	↑O2 required for optimal activity, ↑static effect	
Vancomycin		↑O2 required for optimal activity	

[Kindwall EP, Whelan HT. Effects of Hyperbaric Oxygen in Infectious Diseases: Basic Mechanisms. In: Hyperbaric Medicine Practice 2 ed. 2004]

Diagram 8. Phases of Wound Healing and Hyperbaric Oxygen

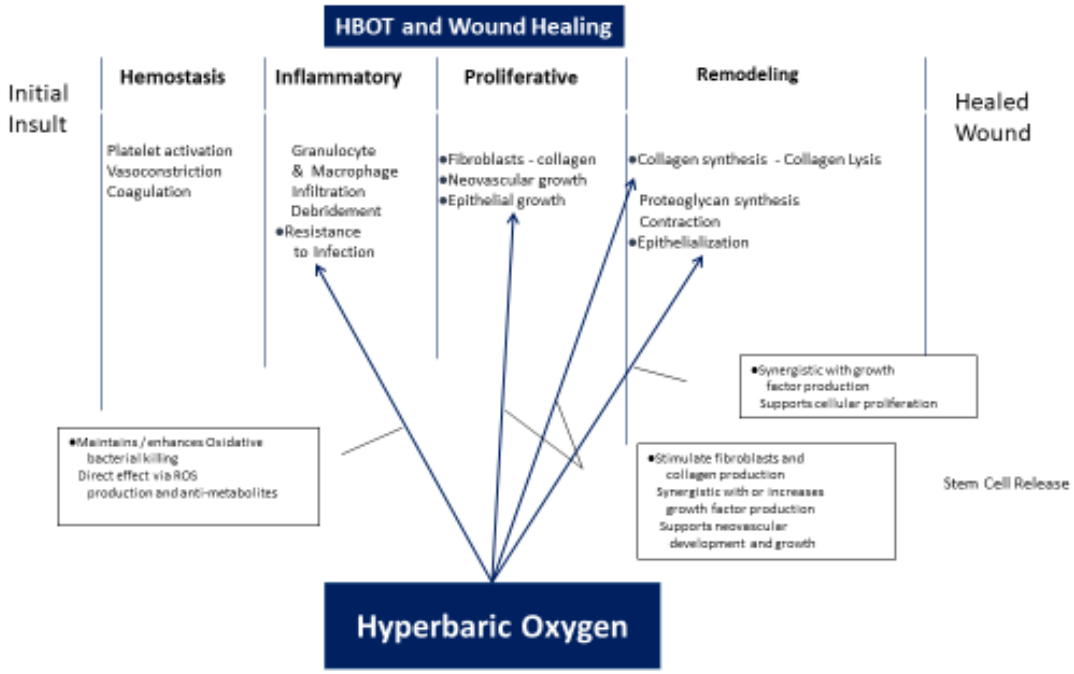


Table 3. Compromised Wounds and Grafts

Pathophysiology of Compromised Wounds, Grafts/Flaps

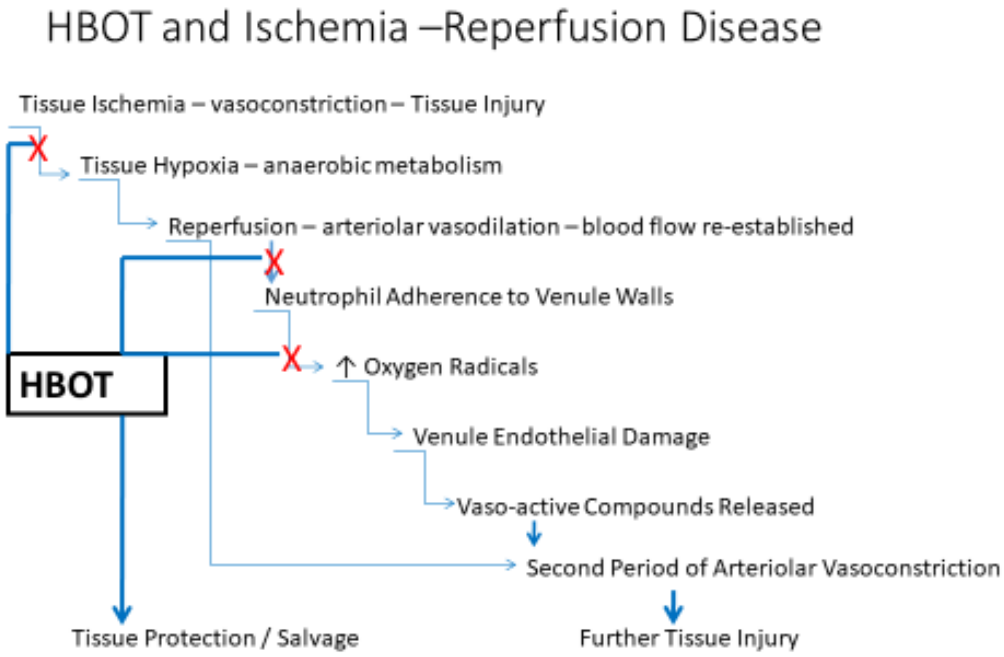
Pathologic Effects Wounds	Rational for HBOT	Pathologic Effects Failing Grafts/Flaps
Hypoxia	Reverses tissue hypoxia	Hypoxia
Ischemia	↓ tissue hypoxia in face of poor perfusion, ↑ antioxidant production	Ischemic injury, ↑ free radical production
↓ energy metabolism	↑O_2 to meet metabolic needs	↓ O_2 for energy, ↑ necrosis
↓ collagen synthesis & quality	↑ fibroblast proliferation and collagen synthesis	
	O2 supports Na/K pump mechanism	Breakdown of Na/K pump mechanism
	Anti-inflammatory effects	Perivascular inflammation
↑ susceptibility to infection	Supports antimicrobial defenses	↑ susceptibility to infection
	↓ edema	Edema, vessel lumen constriction
	↓ platelet aggregation, supports microcirculation	Platelet aggregation, thrombus formation
	Blocks reperfusion - ↓ WBC adhesion, reverses hypoxia	Reperfusion disease
↓ neo-angiogenesis	Promotes neovascularization	Loss/decrease of vascularization
↓ growth factor production	↑ growth factor production and synergy	
↓ rate of epithelialization	↑ rate of epithelialization	
	↑ degree of cellular proliferation	

Table 4. Pancreatitis Characteristics and Hyperbaric Effects

Pancreatitis and HBOT

Characteristics of Pancreatitis	Benefits of Hyperbaric Oxygen Therapy
Microcirculatory alterations	Supports microvascular health and vessel integrity
Tissue hypoxia	Increased tissue oxygenation, ↓ hypoxia
Tissue hypoxia, acidosis	Reduces tissue CO2 and lactate
Tissue edema	Tissue edema reduction
Ischemia-reperfusion disease	↓ hypoxia, ↓ neutrophil adhesion to venules
↓ red cell density and velocity in capillaries	Increased deformability of RBCs
Mitochondrial damage & oxidant release (ROSs) reduced peripheral anti-oxidants	Decrease tissue reactive oxygen species, increase endogenous anti-oxidants
Local and systemic inflammation	Anti-inflammatory
Recruitment of WBCs and ↑ cytokine production	Reduces neutrophil chemotaxis and cytokine production
Activation of coagulation cascade	Reduces platelet aggregation
Potential for bacterial translocation	Bacteriostatic and/or bacteriocidal
Hypoxia impairs neutrophilic bacterial killing	Restores optimal oxygen concentrations for neutrophil phagocytosis
Tissue cell death/necrosis	Reduces/blocks apoptosis
Abdominal pain	Analgesic

Diagram 9. Hyperbaric Oxygen and Reperfusion Disease



Hyperbaric Oxygen Therapy
Patient Management Checklist

Item	√	Comments
Patient evaluation		
Review medical history and signalment		
Review diagnosis		
Review current treatments		
Red Flags	Yes	
	No	
Patient Examination		
TPR completed		
Auscult cardiopulmonary system		
Upper respiratory exam/signs		
Temperament evaluation (sedation necessary?)		
Red Flags	Yes	
	No	
Risk Level		
Patient Preparation		
Evaluate for potential ignition sources		
Evaluate for reduction of fuel sources		
Groom		
Bathe		
Cover devices		
Remove substances		
Wet the patient		
Evaluate for the need for sedation		
Determine need for physical restraint		
Documentation		
Complete General Hyperbaric Record – Update daily		
Determine current barometric pressure and record		
Complete Hyperbaric Patient Monitoring Form for each treatment		
Determine need for pre and post pictures, video etc.		

Risk Level Criteria

Level	Evaluation Criteria	
1	Nothing in the medical history that would contraindicate treatment in high pressure high oxygen environment No current treatments that preclude the use of HBOT No absolute or relative contraindications present in this patient Pre-treatment examination is within normal limits, no respiratory complications Nothing present that is listed on the "No-Go" list No devices, substances, medications etc. that require a risk assessment No potential ignition or heat sources present on the patient Excellent to good temperament	
2	History may contain information that could result in complications Primary reason for HBOT is a respiratory problem Disease present that has produced systemic compromise, requires risk assessment Nothing present that is on the "No-Go" list No absolute contraindication present in this patient Relative contraindication present that may be manageable in the hyperbaric oxygen environment Presence of treatment/substances (topicals etc.) that could be a fuel source, minor risk assessment, requires removal and bathing Presence of devices that cannot be removed and may require covering	
3	One or more of the following is present: Item present which is on the "No-Go" list Significant pulmonary system disease or alteration Presence of an absolute contraindication High fever, multiple relative contraindications Treatments that would predispose the patient to the development complications Devices, substances, medications etc. that have an unknown response to high oxygen/high pressure and will require a significant risk assessment Things present that would could in ignition or heat generation	

Clinical Hyperbaric Oxygen Program
Patient Check List
Chamber Operational Check List
2017

Patient Check List

	Review diagnosis	
	Review current problem list	
	Review current treatments	
	Review patient history	
	Reason for requesting hyperbaric oxygen therapy	
	Topical treatments or care – question patients medical team about use of topicals	
	Pre-treatment physical exam (TPR)	Include cardiopulmonary auscultation, ear exam
	Assess anything attached to the patient	
		Remove if not required
		Risk assessment if required or requested
	Assess patient personality and attitude	
	Is sedation necessary and appropriate	Yes [ ] No [ ]
	Assess presence of any potential ignition sources	
	Assess grooming status (clean, free of debri)	
	Prepare patient individual treatment form	Determine & record barometric pressure (mmHg)
	Prepare hyperbaric summary sheet	
	Complete hyperbaric daily treatment log	
	Wet the patient or portions of the patient (case dependent)	

General Chamber Operational Check List

√	Item	
	Inspect chamber – cleanliness, contents	
	Inspect supply and exhaust lines	
	Provide extra chamber humidity	Wet towels, wetting the floor. Water pans, etc.
	Review step by step chamber operation procedures	
	Open main oxygen valve	
	Engage compressed gas supply for control panel if appropriate	Check compressed gas supply pressure
	Introduce patient to the chamber	
	Follow step by step manual for chamber operations	
	May want to perform leak tests during when at desired pressure.	
	Complete patient treatment monitoring form as Tx progresses	
	Following treatment completion	
	Close main oxygen inlet valve	
	Turn off control panel electronics	
	Shut off compressed gas supply and check tank pressure if used	
	Clean chamber if needed, follow manufacturers recommendations on disinfection procedures	