Level 1 – Dive Medicine Neurologic Issues and Diving

- May 14, 2025
- Chair Discipline of Anesthesia
- Associate Professor of Anesthesiology
- Hyperbaric Medical Staff Eastern Health
- Contract Physician Atlantic Offshore Medical Services
- **E-mail: gzbitnew@mun.ca**

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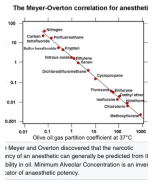
Objectives:

- Identify neurological conditions that would be contraindication or concern for diving
- Summarize pathophysiology and presentation of CNS oxygen toxicity
- Identify risk factors for CNS oxygen toxicity
- Common neurologic issues in diving

2

"Martini's Law" – Nitrogen Narcosis

- Relationship of depth to narcosis feeling
- For every ATM of pressure below 2 ATA, is the equivalent of having one martini
  - 3 ATA = 1 martini
  - 4 ATA = 2 martinis
  - 5 ATA = 3 martinis



Meyer and Overton discovered that the narcotic ncy of an anesthetic can generally be predicted from it bility in oil. Minimum Alveolar Concentration is an inver ator of anaesthetic potency.

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Narcotic Effect of Compressed Air Diving

Feet	Meters	Effect
0-100	0-30.5	Mild impairment of performance on unpracticed tasks. Mild euphoria.
100	30.5	Reasoning and immediate memory affected more than motor coordination and choice reactions. Delayed response to visual and auditory stimuli.
100-165	30.5-50.3	Laughter and loquacity may be overcome by self control. Idea fixation and overconfidence. Calculation errors.
165	50.3	Sleepiness, hallucinations, impaired judgment.
165-230	50.3-70.1	Convivial group atmosphere. May be terror reaction in some. Talkative. Dizziness reported occasionally. Uncontrolled laughter approaching hysteria in some.
230	70.1	Severe impairment of intellectual performance. Manual dexterity less affected.
230-300	70.1-91.5	Gross delay in response to stimuli. Diminished concentration. Mental confusion. Increased auditory sensitivity, i.e., sounds seem louder.
300	91.5	Stupefaction. Severe impairment of practical activity and judgment. Mental abnormalities and memory defects. Deterioration in handwriting, euphoria, hyperexcitability. Almost total loss of intellectual and perceptive faculties.
300	91.5	Hallucinations (similar to those caused by hallucinogenic drugs rather than alcohol).

NOAA Diving Manual 4th Edition

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Food for thought in assessing a diver?

- Could this condition lead to a loss of consciousness underwater?
- Does this condition affect the diver's ability to complete a strenuous task?
- Is the diver's **motor control or dexterity impaired**?
- Are there any **symptoms that could be confused with decompression illness**?

http://www.musee-rodin.fr/accueil.htm

- Is this condition associated with an increased risk of DCS?

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Medical Review of the Diver?

Initial Dive Medical

- No investment in training –YET
- What is career plan?
- Prior occupational exposures?
- If you find something what is the natural course – how could a condition affect the diver moving forward?
- A chance to divert from path

Subsequent Medical Review

- What type of diving?
- In-shore/Off-shore
- Saturation
- Has anything changed? Episodes of DCS? 50% recovery spinal cord DCS
- Will condition require disqualification or conditions imposed?

6

Review of Systems

- History of visual, hearing, speech, swallowing, motor, sensory, balance, coordination, bowel, bladder and sexual dysfunction
- Predisposition to episodes of impaired consciousness/awareness, convulsions, and disturbances of speech, vision, or motor control are incompatible with diving

- Cranial nerve function examined
- Motor and sensory systems
- Balance
- Co-ordination
- Gait
- Proprioception
- Vibration sense
- 2-point discrimination
- DTRs and Plantar response
- **Record baseline clinical findings to allow detection of subsequent variation**

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Contraindications

??

Absolute

- Any seizure activity, except febrile < 5 years (if seizure free for 10 years off medications)
- Recurrent loss of consciousness of unknown etiology or recurrent fainting
- Severe motion sickness
- Severe migraine (frequency and symptoms), especially with daytime somnolence

- Neurologic disease like multiple sclerosis, stroke, Parkinson's
- After stroke or TIA, 12 months symptom free
- Intracranial surgery –if no seizures, deficits, increased risk of seizure
- Moderate to severe head injury due to post-traumatic epilepsy (higher risk with depressed skull fracture, intracranial hematoma, unconsciousness, post-traumatic amnesia > 30 minutes, or focal neurological signs accompany injury)
- Mild head injury (<30 minutes unconsciousness or post-traumatic amnesia) –need 4-6 weeks off and ensure no post-concussion symptoms

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You will receive calls from Recreational Divers

- They have the option of choosing not to dive
- Their livelihood does not depend upon them going in the water*
- Unlike Commercial divers who must perform on job site (often remote location)
- Your job here is to educate the diver about the condition and risks associated with it
- *** Seizures known triggers it is still a NO***
- Some people clear if no recurrence and 5 years no medications, only fever related as child, related to alcohol withdrawal/hypogylcemia

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Dive Phases for Loss of Consciousness

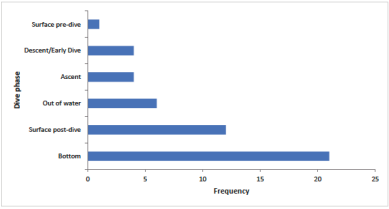

Figure 1-9. Phase of dive when diver lost consciousness (n=48)

DAN 2019 Annual Diving Report page 22

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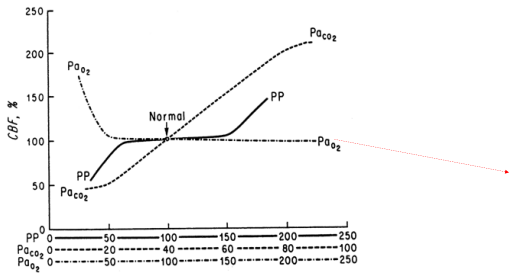
Association of Diving Contractors International, Inc.

- Disqualifying conditions:
  - History of seizure disorder other than early childhood febrile conditions
  - Significant central or peripheral nervous system disease or impairment
  - Chronic alcoholism, drug abuse or dependence or history of psychosis
  - Hearing impairment, **better ear should be 40 dB average in 500, 1000, 2000 Hz frequencies**

CSA Z275 Audiogram initially and every 2 years 250-8000 Hz then as needed 12 hours after loud noise or barotrauma

11

Michenfelder JD: Anesthesia and the Brain, pp 6, 94–113. New York, Churchill Livingstone, 1988



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Ontario Code for Medical Examination of Divers

regulation 629/94 (Diving Operations) OHSA

- Must consider: age, physical fitness, disability and functional loss, mental health (schizophrenia, bipolar affective disorder, recurrent depression, disorders asymptomatic due to treatment, agoraphobia and claustrophobia
- Contraindications: recurrent, unprovoked **loss of consciousness** of unknown etiology, recurrent fainting episodes, epilepsy (unless seizure-free for ten years without treatment or medication and with expert assessment), neurologic disease such as **stroke**, **multiple sclerosis** or **Parkinson's disease**, **severe motion sickness** and **severe migraine**, with complicated aura and excess daytime somnolence

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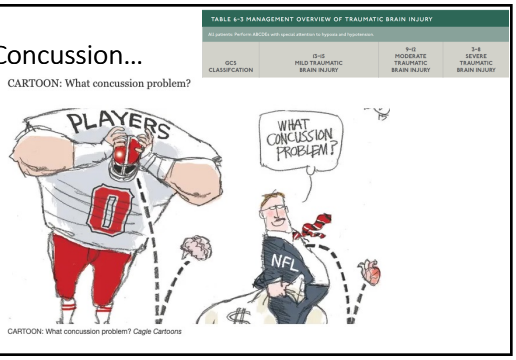
Prior Concussion…



CARTOON: What concussion problem?

CARTOON: What concussion problem? *Cagle Cartoons*

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A systematic review of potential long-term effects of sport-related concussion

Manley G, *et al. Br J Sports Med* 2017;**51**:969–977. doi:10.1136/bjsports-2017-097791

Concussion: Long term effects????

- Back to 1920s – boxers with dementia pugilistica
- Chronic Traumatic Encephalopathy (CTE) some evidence former NFL players have mild cognitive impairment, neuroimaging abnormalities, altered brain metabolism for their age
- Identified 7145 articles and 3819 were then screened and 47 articles included
- Some former athletes of concussive sports have cognitive deficits or decrements and psychological health problems
- Depression is more common in those with prior concussions

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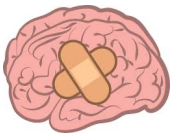
Traumatic Brain Injury

- Mild TBI GCS 13-15 at 30 min post injury
- Moderate GCS 9-12
- Severe ≦ 8
- Males 2-2.8:1 Females



- Signs/Symptoms:
- Stumbling, inability to walk tandem/straight line
- Vacant stare, delayed verbal expression, inability to focus attention, disorientation, slurred or incoherent speech, emotionality, memory deficits
- May see transient neurologic deficits like global amnesia or cortical blindness

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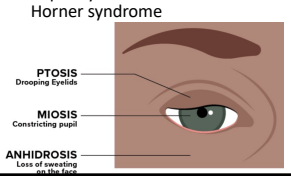
TBI – Red Flags

- Focal neurologic deficits: limb weakness or hemiparesis
- Visual field deficit
- Pupillary abnormalities or Horner syndrome



- Post-traumatic seizure in < 5% of mild or moderate TBI
- More common severe especially with intracerebral hematoma
- 25% first hour
- 50% within first 24 hours
- Earlier generalized seizure, after one hour > 50% are simple partial or focal with secondary generalization

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Symptomatology and Functional Outcome in Mild Traumatic Brain Injury: Results from the Prospective TRACK-TBI Study

J Neurotrauma. 2014 Jan 1; 31(1): 26–33.

- 1 year post 75% reported atleast one symptom
- Almost 1/3 failed to retune to full functional status at 3 and 6 months
- Dive conservative profiles, avoid decompression

- Mild TBI and return to normal in a week
- Mild TBI and recovered in 1 month – Return to diving in 6 months
- Mild TBI and recovery in <1 year – return to diving in 1 year
- Mild TBI and continuing symptoms – likely need review by neurology and symptom resolution

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History of Seizure?

- 1-2% emergency visits
- 8-10% population over lifetime
- Treatable systemic process or intrinsic dysfunction of CNS
- Acute Symptomatic – time of systemic insult or in close temporal association with a documented brain insult, metabolic derangements, drug or alcohol withdrawal, acute neurologic disorders (stroke, encephalitis, or acute head injury)

- 1 week stroke, traumatic brain injury, anoxic encephalopathy, intracranial surgery
- First identification of subdural hematoma
- Active phase of CNS infection
- 24 hours of severe metabolic derangement
- Acute symptomatic lower risk of future epilepsy but some go on to develop seizure disorder

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Acute symptomatic seizure causes?

- Acute ischemic or hemorrhagic stroke
- Subdural hematoma
- Subarachnoid hemorrhage
- Cerebral venous thrombosis
- Traumatic Brain Injury (TBI)
- **Eclampsia**
- **Posterior Reversible Encephalopathy Syndrome (PRES)**
- Hypoxic-Ischemia injury
- Brain abscess
- Meningitis/Encephalitis
- **Hypoglycemia**
- **Hyperglycemia**
- **Hyponatremia**
- **Hypocalcemia**
- **Hypomagnesemia**
- Uremia
- **Hyperthyroidism**
- Acute intermittent porphyria
- **Withdrawal states**

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Epilepsy

- **2 unprovoked seizures more than 24 hours apart** (unprovoked is seizure unknown etiology as well as one that occurs in relation to preexisting brain lesion or progressive nervous system disorder)
- One unprovoked seizure and probability of further seizures occurring over the next 10 years(as in structural lesions such as stroke, CNS infection and some TBI)
- Diagnosis of epilepsy syndrome

- **Positive Family history is a risk factor**
- EEG in adults with first seizure shows epileptiform activity in 25%, much higher likelihood second seizure over next 2 years
- If ≥ 4 EEGs 80-90% may find inter-ictal epileptiform discharges
- Better yield with – photic stimulation, sleep deprivation, hyperventilation

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And soooooooo?

- New diver medical presents with left arm motor seizure and retains awareness?
- Has Todd's paralysis for 15 minutes post event resulting in mild left arm weakness?
- What would you do????

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Medications Associated with Seizures

- Analgesics – Phenylpiperidine opioids (meperidine, tramadol)
- Anti-Neoplastic drugs –Busulfan, Chlorambucil, Cytarabine, Doxorubicin, Etoposide, Fluorouracil, Interferon alfa, Methotrexate, Mitoxantrone, Nelarabine, Platinum-based drugs (cisplatin, carboplatin, oxaloplatin), Vinblastine, Vincristine
- Anti-Microbials – Carbapenems (imipenem, Ertapenem, Meropenem, Doripenem), Cephalosporins (4th gen Cefepime, Cefpirome), Fluoroquinolones (Ciprofloxacin, Levofloxacin, Moxifloxacin, Delafloxacin), Isoniazid, Penicillins
- Immunosuppressants- Azathioprine, Cyclosporine, Mycophenolate, Tacrolismus

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Medications Associated with Seizures

- Pulmonary drugs-Aminophylline, Theophylline
- Stimulants-**Methylphenidate, Amphetamines**
- Sympathomimetics and decongestants- Anorexiants(diethylpropion, phentermine, phenylpropanolamine), Phenylephrine, Pseudoephedrine
- Psychiatric-Antipsychotics (**Clozapine highest risk**), Atomoxetine, Bupropion, Buspirone, Lithium, **Monoamine Oxidase Inhibitors** (furazolidone, Isocarboxazid, Linezolid, Moclobemide, Pargyline, Phenelzine, Procarbazine, Rasagiline, Selegiline, Tranylcypromine), **Selective Serotonin Reuptake Inhibitors** (Citalopram, Escitalopram, Fluoxetine, Paroxetine, Sertraline) **Norepinephrine Reuptake Inhibitors** (Venlafaxine, Duloxetine, Desvenlafaxine, Milnacipran, Levomilnacipran), **Serotonin Modulators** (Vortioxetine, Vilazodone), **Tricyclic Antidepressants** (Amitriptyline, Amoxapine, Desipramine, Doxepin, Imipramine, Nortriptyline, Protriptyline, Trimipramine)

See UpToDate Table 6 Evaluation and Management of the first seizure in adults

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Traumatic Brain Injury

- 4% epilepsy cases are attributed to trauma, **13% of known cause are posttraumatic**, TBI largest cause of epilepsy persons aged 15-24 years
- Early post-traumatic seizures within 1 week are NOT felt to represent epilepsy
- Higher likelihood with depressed skull fracture and intracerebral hematoma requiring evacuation, more severe injury and penetrating head injury
- Alcohol withdrawal is a common cause of early and late trauma in patients with head trauma
- Patients with early seizures are at higher risk for post-traumatic epilepsy
- **Seizures after 1 week** reflect more permanent structural and physiologic changes
- 10-year-incidence posttraumatic epilepsy post TBI 2%

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And soooo?

- 48-year-old diver mostly surface supply in-shore work on piers and hydroelectric work
- Review of systems: waking up with tingling in his radial 3 fingers, aggravated when doing paperwork on computer
- What is going on? Is it a concern

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Case Reports > Ann Emerg Med. 1996 Jul;28(1):90-3. doi: 10.1016/s0196-0644(96)70143-9.

Acute carpal tunnel syndrome in a diver: evidence of peripheral nervous system involvement in decompression illness

A P Isakov [1], J R Broome, A J Dutka

33 year old USN diver in simulated 150 fsw dive

- Carpal Tunnel is most common mononeuropathy
- 2.4% patients have a peripheral nerve disorder, increases to 8% in elderly
- Half of diabetic patients have neuropathy
- This does present us with a diagnostic dilemma when assessing a patient for DCS
- **Temporarily unfit until issue resolved**

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Headache

- A common complaint of recreational and commercial divers
- Diving headache (ICHD-3) is due to hypercarbia

ICHD-3 Diving Headache

Description:

Headache caused by diving to a depth greater than 10 metres, occurring during the dive but often intensified upon resurfacing, in the absence of decompression illness. It is usually accompanied by symptoms of carbon dioxide (CO_2) intoxication. It remits quickly with oxygen or, if this is not given, spontaneously within 3 days after the dive has ended.

Diagnostic criteria:

A. Any headache fulfilling criterion C
B. Both of the following:
  the patient is diving at a depth >10 metres
  no evidence of decompression illness
C. Evidence of causation demonstrated by at least one of the following:
  headache has developed during the dive
  either or both of the following:
  a) headache has worsened as the dive is continued
  b) either of the following:
  headache has spontaneously resolved within 3 days of completion of the dive
  headache has remitted within 1 hour after treatment with 100% oxygen
  at least one of the following symptoms of CO_2 intoxication:
    a) mental confusion
    b) light-headedness
    c) motor incoordination
    d) dyspnoea
    e) facial flushing
D. Not better accounted for by another ICHD-3 diagnosis.

Bifrontal, Bitemporal, Bi-occipital
Throbbing quality
Mild to severe
Resolves within 3 days
Often resolves quickly with 100% oxygen
Slow, deep breaths, avoid vigorous exercise

Current Pain and Headache Reports (2019) 23: 46

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Headache

- May be due to squeeze in mask or facial sinuses
- More likely if cold or allergies
- Excessively tight mask compressing supraorbital nerves
- Excessively tight neck compressing
- Or excessively loose allowing cold stimulus headache
- Consider CO contamination of gas
- Also migraine and tension type headaches
- Diving ascent headache – similar to airplane travel headache

https://acidcow.com/pics/48773-the-danger-of-scuba-diving-7-pics.html

Hypercapnea – facial flushing, lightheadedness, confusion, ultimately loss of consciousness

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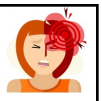
Migraine



- Caused by cortical spreading depression a self-propagating wave of neuronal and glial depolarization that spreads across the cerebral cortex
- Causes: aura, activates trigeminal afferents, alters blood-brain permeability by Matrix metalloproteinase activation
- **12-15% population** (17% females 6% males)
- Without aura in 75%
- Major cause of disability after low back pain
- 25% focal neurologic symptoms (Aura) no longer than 1 hour: **positive symptoms** (bright lines, shapes, objects, tinnitus, noises, music, burning, pain, paresthesia, jerking or repetitive movements) **negative symptoms** absence or loss of function (loss of vision, hearing, feeling, motor control

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Migraines

- With Aura – much higher likelihood of a PFO (up to 50%)
- May increase their chances of DCS up to 5X
- Counsel and offer option of TTE screening to look for PFO
- What is frequency? How would they cope with aura in water?
- What is frequency of occurrence

- Emotional stress (80 percent)
- Hormones in females (65 percent)
- Not eating (57 percent)
- Weather (53 percent)
- Sleep disturbances (50 percent)
- Odors (44 percent)
- Neck pain (38 percent)
- Lights (38 percent)
- Alcohol (38 percent)
- Smoke (36 percent)
- Sleeping late (32 percent)
- Heat (30 percent)
- Food (27 percent)
- Exercise (22 percent)
- Sexual activity (5 percent)

Common Triggers

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Migraine therapies

- Triptans
- CGRP antagonists
- Lasmiditan
- **Dihydroergotamine**
- NSAIDS
- ASA
- Acetominophen
- Metoclopramide
- Valproate

- Many of the drugs are sedating
- Some prolong QT interval
- Many are potent coronary and other vasoconstrictors
- Delay prior to diving would be prudent after receiving many of these medications

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Pooled Analysis of PFO Occluder Device Trials in Patients With PFO and Migraine

JACC VOL. 77, NO. 6, 2021
FEBRUARY 16, 2021:667–76

Mohammad K. Mojadidi, MD,[a,*] Preetham Kumar, MD,[b,*] Ahmed N. Mahmoud, MD,[c] Islam Y. Elgendy, MD,[d] Hilary Shapiro, MD,[b] Brian West, MD,[e] Andrew C. Charles, MD,[f] Heinrich P. Mattle, MD,[g] Sherman Sorensen, MD,[h] Bernhard Meier, MD,[i] Stephen D. Silberstein, MD,[j] Jonathan M. Tobis, MD[b]

TABLE 3 Analyses of Migraine With Aura and Migraine With Frequent Aura, Comparing Patients Who Underwent Patent Foramen Ovale Closure With Control Subjects

	Migraine With Aura (n = 225)	Migraine Without Aura (n = 78)	Migraine With Frequent Aura (n = 125)	Migraine With Infrequent Aura (n = 100)
Mean reduction in migraine days	−3.2 ± 4.8 vs. −1.8 ± 4.4	−2.8 ± 3.4 vs. −2.2 ± 4.0	−4.3 ± 5.3 vs. −1.4 ± 4.8	−2.4 ± 3.8 vs. −2.3 ± 3.7
p value	0.03	0.53	0.002	0.99
% responder rate	38 vs. 29	37 vs. 31	48 vs. 25	33 vs. 31
p value	0.16	0.60	0.005	0.69
Mean reduction in migraine attacks	−2.0 ± 2.0 vs. −1.4 ± 1.9	−2.0 ± 1.8 vs. −1.0 ± 2.0	−2.7 ± 1.9 vs. −1.5 ± 1.9	−1.5 ± 2.0 vs. −1.4 ± 2.9
p value	0.09	0.03	<0.001	0.52
% complete headache cessation	11 vs. 1	5 vs. 0	13 vs. 1.5	6 vs. 0
p value	0.002	0.16	0.01	0.01

Values are mean ± SD, unless otherwise indicated.

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Diving and Hyperbaric Medicine Volume 50 No. 4 December 2020

A review of diving practices and outcomes following the diagnosis of a persistent (patent) foramen ovale in compressed air divers with a documented episode of decompression sickness

Christopher W Scarff[1], John Lippmann[2,3], Andrew W Fock[1,3]

Diving style before and after PFO diagnosis. Data are *n* (%). msw = metres' seawater

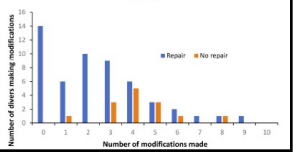
Phase	Not diving	Recreational 0–30 msw	Deeper > 30 msw	Technical	Commercial
Pre-intervention	–	8 (11.0)	31 (42.5)	31 (42.5)	3 (4.1)
Post-intervention	5 (6.8)	25 (34.2)	14 (19.2)	25 (34.2)	4 (5.5)

Of 73 subjects 55 had 1 or more procedures to close a PFO, 18 had no closure and 5 ceased diving

Comparison of PFO repair status and change in diving practice

PFO status	*n* (%)
Closure and no change	15 (20.1)
Closure and CDP	38 (52.1)
Closure and no diving	2 (2.7)
Not closed and CDP	15 (20.1)
Not closed and no diving	3 (4.1)
Total	73 (100)

Diving modifications post-presentation for divers with and without PFOC



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A review of diving practices and outcomes following the diagnosis of a persistent (patent) foramen ovale in compressed air divers with a documented episode of decompression sickness

Christopher W Scarff[1], John Lippmann[2,3], Andrew W Fock[1,3]

- Initially 80 DCS cases in 73 divers over 50107 dives
  - 16/10000 dives
- After PFO management 3 cases DCS in 19118 dives
  - 1.6/10000 dives
- 38 large defects, 26 small, 9 not classified
- 34 suffered migraines, 18 with aura
- 23 migraine sufferers closed, 17 had partial or complete cessation
- 38% decreased maximum depth
- 13.7% increased depth
- All who returned to diving without closure adopted a more conservative style, and 69% of those who underwent closure adopted modifications (nitrox, initiated or increased safety stops, decrease no deco limits, decreased repetitive dives per day, single dive per day, more hydration, less exertion, slower ascents

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Cerebrovascular Accident

Common medical complications of stroke

Complication	Percent
Falls	25
Urinary tract infection	24
Chest infection	22
Pressure sores	21
Depression	16
Shoulder pain	9
Deep venous thrombosis	2
Pulmonary embolism	1

Medical complications of stroke were frequent in a prospective multicenter study of 311 patients followed weekly through hospital discharge and again at 6, 18, and 30 months after stroke.

Data from: Langhorne, P, Stott, DJ, Robertson, L, et al. Medical complications after stroke: a multicenter study. Stroke 2000; 31:1223.

Serious medical complications of stroke

Complication	Percent
All pneumonias	5
Aspiration pneumonia alone	3
Heart failure	3
Gastrointestinal bleeding	3
Cardiac arrest	2
Angina/MI/cardiac ischemia	1
Deep venous thrombosis	1
Pulmonary embolism	1
Hypoxia	1
Urinary tract infection	1
Sepsis	1
Cellulitis	1
Peripheral vascular disorder	1
Dyspnea	1
Pulmonary edema	1
Dehydration	1

In a prospective study that analyzed the placebo group of the RANTTAS database (n = 279), at least one serious medical complication (defined as prolonged, immediately life threatening, or resulting in hospitalization or death) occurred in 24 percent of patients.

Data from: Johnston, KC, Li, JY, Lyden, PD, et al. Medical and neurological complications of ischemic stroke: experience from the RANTTAS trial. RANTTAS Investigators. Stroke 1998; 29:447.

UpToDate

Limited Physical Abilities, Elevated Medical Risk

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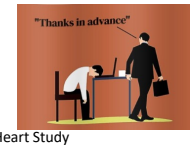
Transient Ischemic Attacks



- Numerous etiologies: embolic, dissection, venous thrombosis, aneurysmal dilation, atherosclerosis, decreased perfusion pressure, increased viscosity
- Sudden onset of focal neurologic symptom/sign lasting < 24 hours
- Transient decrease in blood flow rendering area producing symptom ischemic (risk of permanent injury if symptoms last an hour)
- Tissue-based definition transient episode of neurologic dysfunction caused by brain, spinal cord, or retinal ischemia

- Framingham Heart Study
- Age 45-54 0.22/1000 person-years
- Age 85-94 4.88/1000 person-years
- Overall 1.19/1000 person-years
- Prevalence in USA approximately 2%
- Often warning shot of coming stroke 1.5-3.5% in first 48 hours after
- Need brain, vascular and cardiac evaluation

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TIAs

- Patients are at high risk for acute stroke?
- Hard to meet targets for endovascular thrombectomy/thrombolysis underwater?
- If occurs on ascent or immediately after surfacing; how are we to differentiate it form DCI??? Do we recompress or thrombolyze??

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Neurodegenerative Diseases

- Most common are:
  - Alzheimer's disease
  - Parkinson's disease
- Others include:
  - Amyotrophic lateral sclerosis
  - Friedrich's ataxia
  - Lewy body disease
  - Spinal muscular atrophy
  - Huntington's disease

- These are all absolute contraindications to professional diving – limited capacity

? Recreational diver with suspected early Parkinson's

Do symptoms interfere with diving/swimming?

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CNS Oxygen Toxicity

Respiration Physiology (1978) **34**, 1–28

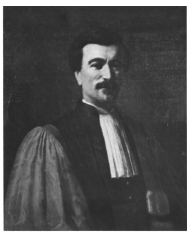
LA PRESSION BAROMÉTRIQUE: PAUL BERT'S HYPOXIA THEORY AND ITS CRITICS[1,2]

RALPH H. KELLOGG

Department of Physiology, University of California, San Francisco, CA 94143, U.S.A.

Was working on Altitude sickness and determined that partial pressure of oxygen was essential for life - not the concentration
"Father of Aviation Medicine"
Discovered the toxic effects of oxygen on the CNS – the **Paul Bert Effect**

"We conclude from all these experiments that oxygen does not kill by acting on the heart, the motor nerves, or the muscles, but the reflex acts of the spinal cord…The poisoning is characterized by convulsions which, according to the intensity of the symptoms, represent the different types of tetanus, strychnine, phenol, epilepsy, etc.; These symptoms, which are quieted by chloroform, are due to an exaggeration of the excito-motor power of the spinal cord" Translated 1943 F and M Hitchcock



Larks exposed to 15-20 ATA Air

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Oxygen Toxicity = VENTID

- V – Vision (tunnel)
- E – Ears (tinntis)
- N - Nausea
- T – Twitching (facial)
- I - Irritability
- D – Dizziness
- **CONVULSIONS**

Myopic Shift – hyperbaric exposure

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Reduction of a Molecule of Oxygen to Water

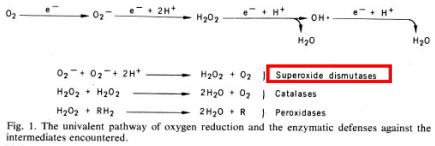
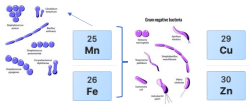


Fig. 1. The univalent pathway of oxygen reduction and the enzymatic defenses against the intermediates encountered.



- 4 electrons required
- Dangers of superoxide: inactivate viruses, damage cell membranes, inhibit nucleic acid and protein synthesis

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Additional Mechanisms of Toxicity

⚠ CAUTION

- Alterations in cellular metabolism or hyperoxia induced enzyme inhibition
- Glutamic acid decarboxylase inhibited in CNS
- Reduced levels of Gamma Amino Butyric Acid are seen concomitantly with seizures
- GABA ↓↓ while Glutamate ↑ giving excess Glutamate activity
- Maximal working Limit for NOAA Diver is 1.4 ATM oxygen, 1.6 ATM oxygen for decompression
- Usual onset is 2-3 ATM
- Lower pressure onset with immersion, exercise, respiratory acidosis (hypercarbia), combined with Carbon Monoxide or inert gases
- Glycine may be involved linked to myoclonic activity in mammals

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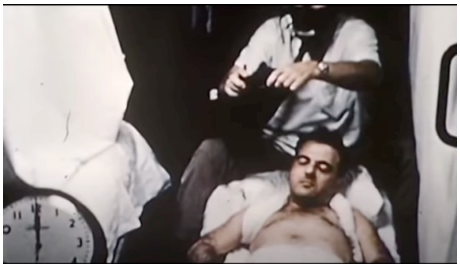
Oxygen Toxicity



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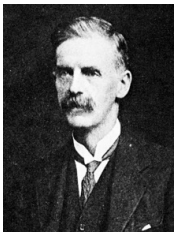
J. Lorrain Smith Effect



- 1899 was trying to induce "Paul Bert effect" on rats, left them on 73% oxygen for 4 days
- They died of pneumonia
- Pulmonary Toxicity of oxygen can arise after prolonged exposure to oxygen > 0.5 ATA
- Normal humans show toxicity after 10 hours at 1 ATA
- 3 phases: a) Tracheobronchitis b) ARDS c) interstitial fibrosis
- Loss of type 1 pneumatocytes
- Watch Vital capacity no more than 10% reduction

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Acta Neurochir (2016) 158:1269–1272
DOI 10.1007/s00701-016-2800-x

REVIEW ARTICLE - NEUROSURGERY TRAINING

Can patients with a CSF shunt SCUBA dive?

Dmitri Shastin[1] · Malik Zaben[1] · Paul Leach[1]

- In hyperbaric environment Huang et al. testing Level One Delta Valve flow characteristics with normal saline, Maximum pressure 4 ATA
- Cambridge shunt evaluation laboratory tested 26 valve models to 30°C – hypothermia had no effect on function
- Seizure risk of VP shunt – Copeland et al 24% of patients developed seizures after shunting
- 58% in first 4 weeks
- In the rest over 1-3 years post
- Others showed peak at day 20 and return to baseline at 1 year
- Neurosurgeons recommended avoiding diving first 2-3 months, may extend based on circumstances

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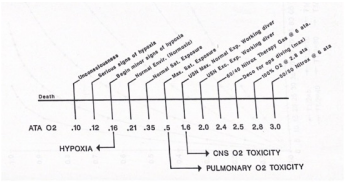
Effects of Different Levels of Oxygen Partial Pressures



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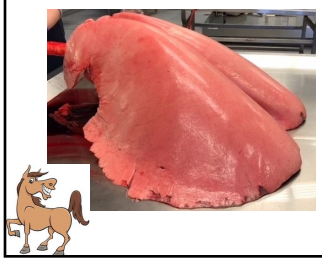
Unit Pulmonary Toxicity Dose



- Used to predict pulmonary damage with larger exposures
- 1 UPTD = 1 minute 100% oxygen at 1 ATA
- 1425 UPTD will lead to a 10% reduction in Vital Capacity

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UNDERSEA & HYPERBARIC MEDICINE

Severe carbon monoxide poisonings in scuba divers: Asia-Pacific cases and causation

John Lippmann, PhD [1,2,3]; Ian Millar MBBS [4,2]

CO POISONINGS IN ASIA-PACIFIC DIVERS – UHM 2022 VOL 49 NO 3

- Of 4 deaths in 645 dive fatalities
- 2 fatal involved internal combustion engine exhaust gases from compressor system entering the air intake
- 2 deaths resulted from combustion within compressor system
- 1 victim COHb 56%
- Uncommon
- Under-reported
- Required to have gas samples verified by CSA approved lab every 6 months
- Poor compressor function may lead to contamination with CO, CO2, and volatile hydrocarbons

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UNDERSEA & HYPERBARIC MEDICINE

Severe carbon monoxide poisonings in scuba divers: Asia-Pacific cases and causation

John Lippmann, PhD [1,2,3]; Ian Millar MBBS [4,2]

CO POISONINGS IN ASIA-PACIFIC DIVERS – UHM 2022 VOL 49 NO 3

- Usually 4.6/million population lethal CO poisonings
- Health Canada recommendations for indoor air: Long term 10 ppm over 24 hours, Short term 25 ppm over 1 hour
- US FAA aircraft fire survivability: immediate incapacitation 6850 ppm, 5 min exposure 4200 ppm
- Diving gas set at 5 ppm
- Remember 5 ppm at 1 ATA becomes…
  - 50 msw (165 fsw) = 6 ATA
  - 6 x 5 ppm = 30 ppm
- Immediate danger to life is 1200 ppm (NIOSH limits)
- US National Research Council's Acute Exposure Guidelines is 10 min for 1700 ppm for lethal limit

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Pneumocephalus, a rare complication of diving

OLE H. BUDAL M.D.[1], JAN RISBERG M.D., Ph.D.[2], KARI TROLAND[3], GUNNAR MOEN M.D.[4],
STEIN HELGE GLAD NORDAHL M.D., Ph.D.[5], GURO VAAGBOE M.D.[6], MARIT GRØNNING,M.D., Ph.D.[7]

UHM 2011, VOL. 38, NO. 1 – PNEUMOCEPHALUS AFTER DIVING

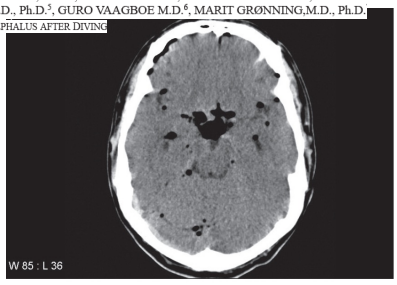

FIGURE 1 – Head CT axial projection aquired approximately two hours after injury. Air located sportlike in the subarachnoidal space, particularly around dorsum sellae and in the supracellar cisterna.

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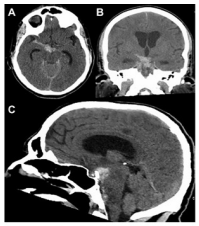
Nonaneurysmal Subarachnoid Hemorrhage in Scuba Diving

Keaton Piper, Ryan Screven, Sivero Agazzi, Waldo R. Guerrero, Keith Dombrowski



igure 1. Computed tomography scan of the head showing prepontine subarachnoid hemorrhage with extension to the right sylvian fissure: A) axial (B), coronal, and (C) sagittal.

Headache
Confusion
Nausea
Emesis

No relief with oxygen at home

- 60 yo male, presented 2 days post rapid ascent scuba diving
- Unremitting in spite of home oxygen
- CT head showed SAH, with no underlying vascular abnormality
- Discharge home intact

- **Remember: Differentials**

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Contraindications

- Unprovoked LOC
- Recurrent fainting
- Epilepsy*
- CVA/TIA
- MS
- Parkinson's
- Severe Migraine

- Past intracranial surgery*
- CNS tumour
- Prior spinal cord injury
- Prior DCS with residual neurologic deficits
- Degenerative neuromuscular diseases

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What we really want to know?

- Are there any cognitive or behavioral effects making individual unsafe?
- Has a seizure disorder developed that places the individual at risk of loss of consciousness or drowning? Lowered threshold?
- Is there level of consciousness normal?
- Does the condition impair physiologic reserve? Can the diver perform assigned tasks?
- Will condition increase the risk of decompression illness?
- Will pressure aggravate the condition?

https://onmilwaukee.com/articles/irgens-pensive

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Questions ???

- Best Wishes in your
- Dive Medicine Careers
- gzbitnew@mun.ca

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Components of neurologic exam

Exam Component	Pathways Tested	Possible Finding in Diving Injury
Mental Status	Cortex	Confusion, aphasia (AGE, cerebral DCS)
Cranial Nerves	Brainstem	Diplopia, facial droop, dysarthria
Motor Strength	Corticospinal tract	Weakness, hemiparesis, paraplegia
Sensory Function	Spinothalamic, Dorsal Columns	Patchy or asymmetric loss
Reflexes	Reflex arcs, UMN/LMN distinction	Hyper/hyporeflexia, Babinski
Coordination	Cerebellum	Ataxia, intention tremor
Romberg	Proprioception/vestibular	Instability with eyes closed

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