

A FUNDAMENTAL APPROACH TO  
THE PREVENTION OF DECOMPRESSION SICKNESS

SUMMARY

This article presents a hard look at the fundamental issues underlying the formulation of preventive decompression. The author's interest in this subject was captivated some fifteen years ago when taking instrument recordings of the remarkable decompressions routinely followed by pearl divers - particularly the Okinawans operating in the Torres Strait and elsewhere along the northern coast of Australia. This study revealed a very efficient decompression practice derived purely by trial and error at the expense of maybe several thousand lives and serious injuries. These remarkable practices were derived over half a century when that area supplied the world with the pearl shell which was in great demand before buttons were made of plastic. Working at Adelaide University the author and his aeromedical colleagues were just in time to put on record these practices before the pearling industry dwindled to a state at which that vast wealth of invaluable human experience would have been lost for ever.

The methods employed by those divers were both successful and much more economical on time than Naval practice. Moreover their emphasis upon spending much more time deeper at the start of decompression and surfacing directly from 25-35 feet was totally incompatible with the Haldane rationale and neo-Haldanian calculation methods for diving table formulation at the peak of popularity at that time. This discovery stimulated much scientific work at Adelaide, leading to concept of an equilibrium state rather than a supersaturated state as the most relevant in determining the imminence of bends. Publication of this approach in 1966 presented the first comprehensive challenge to the Haldane method of formulating decompression tables as elaborated by the US Navy in particular. The major point of divergence was to point out that only equations were used to formulate tables and that, whatever the accompanying words, conventional equations assumed that the bends-free dive was bubble-free, pointing out why the diver was so disadvantaged if this proved to be incorrect.

There is now much more scientific evidence to support the Thermodynamic Approach which has been updated recently in a book entitled "Decompression Sickness: The Biophysical Basis of Prevention and Treatment" (published by John Wiley's in New York and London). This paper is a distillate of that work. In order to avoid distraction from the main theme, some statements are made with minimal supporting data, if any, but the relevant references and detailed explanation can all be found in the book.

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The major medical problem in deep-sea diving is the prevention of decompression sickness, since any diver must decompress in returning to his normal environment and inadequate decompression can prove fatal or may lead to permanent disablement. By far the most effective way to avoid decompression sickness is to invoke gradual decompression; but this immediately raises the question of how gradual is gradual? Obviously one wishes to minimise the wearisome time spent by the diver in a chamber or suspended in the ocean and yet not jeopardise his safety. Consequently, a means is needed to optimise the whole environmental program needed to return the diver to the surface, ie. a simultaneous optimisation of:

- ( DEPTH versus
- ( TIME versus
- ( COMPOSITION OF BREATHING MIX

The methods of accomplishing this fall into four broad groups:

1. Devise a schedule by trial and error.
2. Compute a schedule from a calculation method or mathematical model.
3. Use a meter based upon one of those models or calculation methods in #2.
4. Monitor a body parameter, using the response to determine decompression.

The last would be the best if a good parameter were available and the relevant tissue to monitor could be identified anatomically. Although some encouraging advances have been made in monitoring tissues ultrasonically and conductometrically, such techniques must still be regarded as novelties until we can be sure of what to look for and where to look - issues discussed later.

The third approach (viz. the use of meters) is very good if the engineering is adequate but is really no more than a convenient form of #2 by providing a decompression unique to each particular dive history and so circumventing the fact that it is impracticable to compile a book of tables to cover all depth-time combinations. Most tables in operation today are hybrids of #1 and #2.

However, before discussing the formation of tables, a moment should be spent in considering whether it is worthwhile to calculate at all. Hence let us consider a major piece of purely experimental data - the bounce dive curves for air and heliox otherwise known as the no-stop decompression limits. Both depict a fundamental relationship between depth and time; so the fact that these curves can be so clearly defined for each individual can be taken as a manifestation of an underlying rationale which justifies efforts to them mathematically and even to invoke complex functions if needed.

Behnke emphasizes this clear demarcation by saying that maybe 5 feet in depth can separate serious injury from a state of wellbeing.

In formulating a preventive decompression by means other than pure trial-and-error there are basically two approaches involving either:

1. *calculation methods* in which a convenient equation is selected and constants determined empirically to offer the best fit to experimental data, adding more equations (and more constants) if needed, or,
2. *true models* based on the physiological and physical principles involved.

While the empirical approach (#1) is good for interpolating between dives already proven in the field, it has seldom been successful in extrapolating to greater depths or longer times. Thus a calculation method in which the constants have been adjusted to provide a safe table at 400 feet may fail completely when used to 500 feet. These discrepancies can always be accommodated by adding more hypothetical tissues (and more constants) until, with some 700-800 degrees of freedom which some designers use, one wonders whether it is worth invoking calculation at all. The calculation methods arising from the Haldane rationale have divagated into incredulous complexity to force a "fit" to experimental data.

The alternative approach - that of synthesizing a mathematical model from fundamental physics and physiology - would seem ideal until we realise how little we really know of the mechanism of decompression sickness. The symptoms are so varied that they tell us little; while pathological studies seem to show bubbles in most organs so, as Haymaker points out, nothing really pertinent to a specific model emerges from that vast mass of material.

However the symptoms do seem to fall into five broad categories:

1. Limb bends and other essentially *local* manifestations.
2. Cerebral symptoms - which are rare.
3. Spinal "hits".
4. Vestibular DCS
5. Dysbaric osteonecrosis?

#### *Dysbaric osteonecrosis*

This disease induced by diving is little understood and, at this time, cannot be used to program decompression. Its principal features are:

1. No correlation between the incidence of bends and bone lesions when the data are analysed very carefully.
2. No bone lesions in aviators - at least, no more than the incidence in the "normal" population.
3. Greatly increased decompression time (eg. the Blackpool tables) greatly reduced the bends rate, yet did not change the incidence of bone lesions.
4. The time course for dysbaric osteonecrosis is several orders longer than anticipated for an acute insult at the time of the last dive.

As many as nine hypotheses for the mechanism of dysbaric osteonecrosis can be found in the literature, six based on acute infarction or vessel occlusion and three on a more subtle form of insult occurring at a more microscopic level of bone physiology.

In fact, it is just possible that dysbaric osteonecrosis may not be caused by the decompression

#### *CNS Symptoms*

Cerebral symptoms are virtually identical to those caused by undisputed arterial air embolism, eg. when occurring after submarine escape, that their aetiology is seldom questioned. This is not true of spinal decompression sickness which occurs roughly three times more frequently. These CNS symptoms can always be produced by a decompression far in excess of one known to induce mild limb bends, yet it is probably fair to say that they are rarely the presenting symptoms for *marginally* unsafe decompressions. The factors predisposing the subject to the rare exceptions to this general rule are discussed later (p. 25).

#### *Limb Bends*

Most empirical *calculation methods* work to a "trigger point" for each hypothetical tissue, eg. violating an 'M' value in the conventional approaches. Calculation effectively stops at that point as though whatever is "triggered" must occur. However, let us consider the man performing a dive on which he has developed bends some time after return to the surface. He now repeats that exposure, presumably violating the same hypothetical "trigger points", but recompresses to 20 feet shortly before he knows he will develop bends (and *limb bends* are quite reproducible\*), stays there for 30 mins and then returns to the surface with no problem. The recompression to 20 feet for 30 mins obviously averted what would otherwise occurred, so the process leading to bends must have taken place in at least two steps:

\* generally occur in the same individual for the same exposure and decompression.

- ( 1. a primary event "triggered" by decompression
- (
- ( 2. a critical insult producing symptoms

Moreover, there would appear to be a continuous variation in the insult, bends occurring only if it reaches a threshold level for pain. See Figure 1.

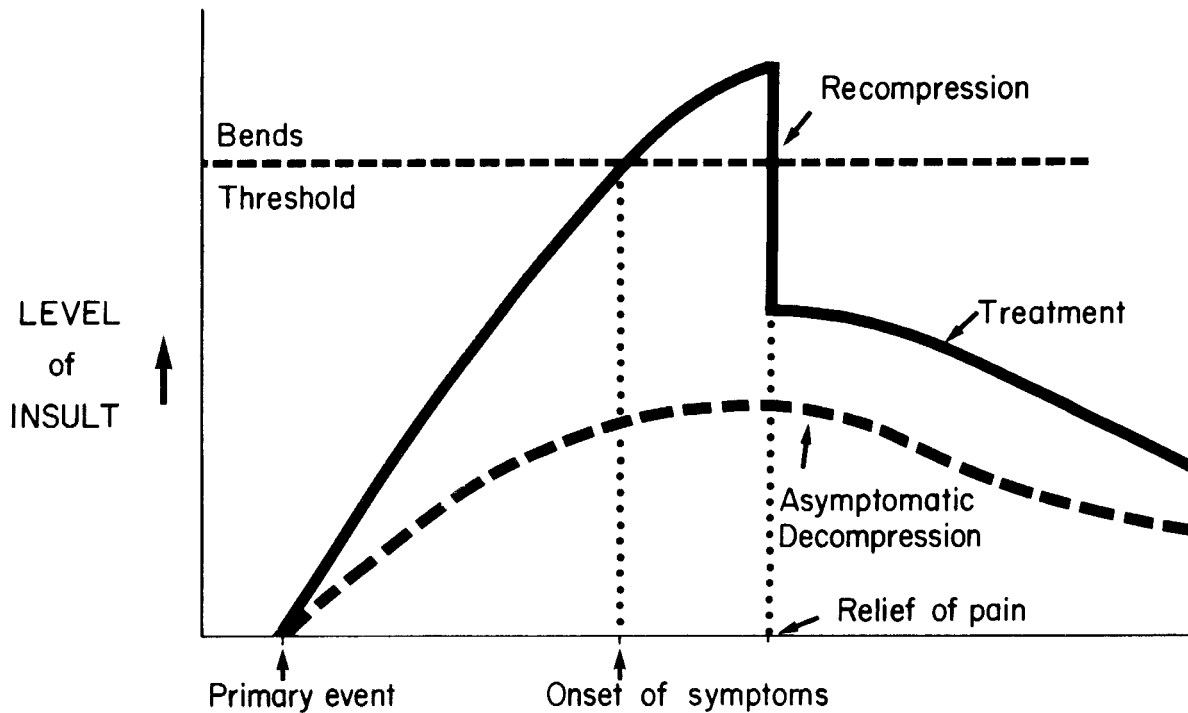


FIG. 1

This raises questions concerned not only with identifying these processes but with other queries needed to be answered in formulating a mathematical model from fundamental considerations. This list of questions includes:

1. What is the primary event?
2. What is the mode of insult and what is its critical level for pain?
3. What conditions initiate the primary event?
4. What is the cause of delay in reaching the critical

insult?

5. How is gas taken up by tissue?
6. How many tissues are involved?
7. Does the prevention of limb bends avoid other forms of decompression sickness and what factors tend to predispose the diver towards those symptoms?

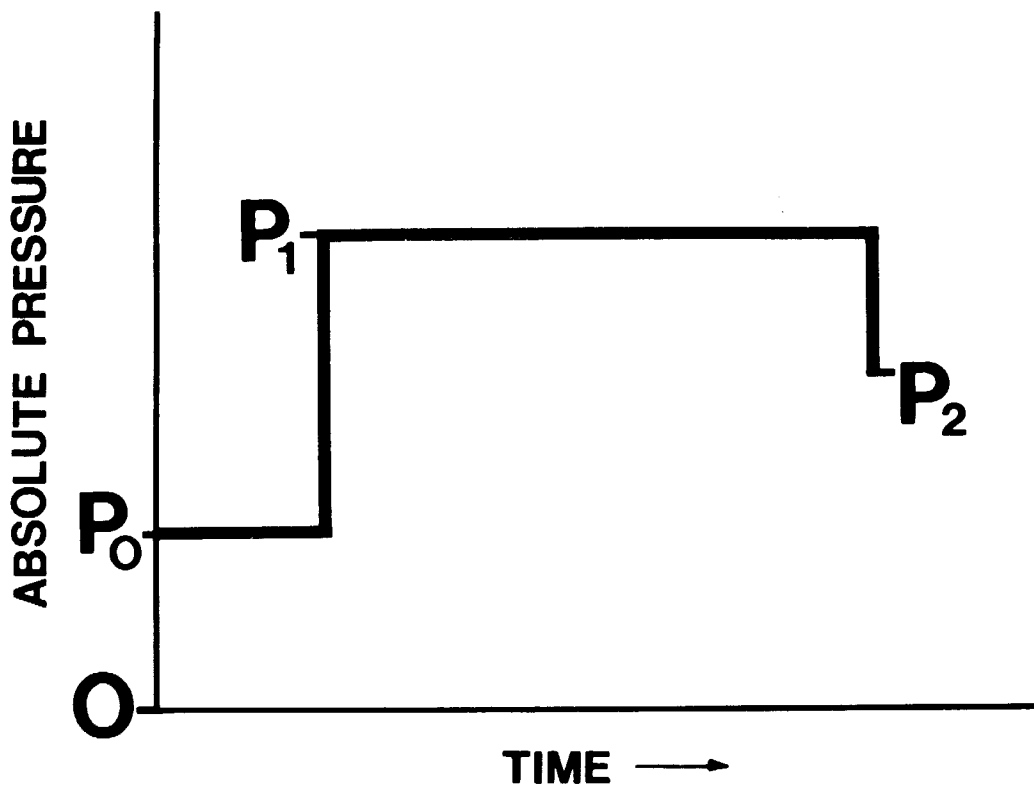


Figure 2

*Primary event*

Let us consider a simple exposure to a pressure  $P_1$  followed by a decompression to a pressure  $P_2$  - see Figure 2. Now the likelihood that limb bends will occur at  $P_2$  is determined by numerous factors which can be reduced to two primary ingredients:

1. The extent of the decompression ( $P_1 - P_2$ )

2. The inert gas content immediately prior to decompression as determined by:
  - (a) time "on the bottom",
  - (b) depth of exposure,
  - (c) solubility of inert gas breathed (eg. nitrogen or helium),
  - (d) exercising "on the bottom",
  - (e) substituting oxygen for inert gas in the breathing mix, etc.
  - (f) obesity (increased body fat)

The dominance of these two factors leaves few alternatives, if any, to the popular view that the primary event is the inception of a stable gas phase.\* It is very difficult to conceive other initiating processes which are so dependent upon the *combination* of these two dominant features listed above without invoking the principle of gas separating from solution. So far this introduces no controversy since, ostensibly, all designers of calculation methods and models claim that their indices for limiting decompression are thresholds for bubble inception; although whether they do so in practice is quite another matter. However this agreement ends abruptly when we proceed to the next question (#2 on p. 4) and consider the mode of insult leading to limb bends.

*Mode of insult*

Several mechanisms have been proposed or assumed by which the primary event can lead to the critical level of insult needed to induce limb bends. These differ according to the type of pain - whether induced by ischaemia or by mechanical means - and whether the insulting entity is a bubble or one of several degradation products known to be produced by a gas-blood interface. These approaches can be summarised as follows:

ischaemia		intravascular bubbles
	by	OR
OR		blood degradation products

PAIN INDUCED by

mechanical		extravascular gas phase
	by	OR
means		tribonucleation (joint gas)

Of these, tribonucleation is most unlikely since gas injected between the articular surfaces of the joint, or formed by various disease processes, does not induce pain (aeroarthrosis). In limb bends the pain is not within the joint but around it. Moreover it requires foreign particles much harder than bubbles to penetrate the synovium or articular surfaces to the depth of any nerve endings - such as sodium urate crystals in gout and the pain of gout is much different to that of limb bends.

\* we are simply concerned with whether bubbles form or not and mechanism(s) of nucleation/activation of nuclei, etc. are of largely academic interest.

Returning to the other mechanisms, the pain of *limb bends* is unlikely ischaemic in origin for the following reasons:

1. Ischaemic pain must be induced by *arterial* emboli, but bubbles only form *de novo* in the *arterial* system with explosive decompression, while venous bubbles are trapped by the lungs.

2. Known arterial air embolism does not produce limb bends, eg. after an accident in submarine escape training.

3. Diseases known to produce infracting agents such as thrombi, fat emboli, platelet aggregates, etc. do not produce the pain of limb bends.

4. Compression affords immediate relief of pain in most cases of limb bends while ischaemic pain is greatest upon restoration of blood flow.

5. If pain were ischaemic in origin, one would expect that further decreasing tissue oxygen supply by lowering the inspired oxygen partial pressure would exacerbate the situation, and yet hypoxia has been found to have a mild protective action - if any.

6. Similarly one would expect elevated oxygen to help relieve ischaemic pain and yet hyperoxia *per se* (as opposed to oxygen substitution for inert gas) potentiates the bends.

This would lead us to deduce that the mechanism of limb bends is a bubble pressing on a nerve ending.

### *Mechanical approach*

This simple approach to the pain of limb bends implies that the gas would probably need to be located in an extravascular site in order to bend a nerve ending as far as its pain threshold. This is easily justified by the fact that, even after an extensive recompression for a few minutes, limb bends will re-occur in the same site upon a further decompression same pressure at which they occurred in the first place. Intra-arterial bubbles actually observed in vascular window preparations can be totally displaced by such recompressions and washed away in the circulation, gas causing the local pain of *limb bends* is fairly certain to be extravascular.

In selecting an extravascular site for the insult, it becomes more important to identify a tissue anatomically since it would then eliminate major problem of programming a decompression by following a direct tissue monitor - viz. the question of knowing where to look. It would also provide for the physiological parameters in the model. Thus the requirements the critical tissue type can be listed as follows:



1. a preponderance of nerve endings;
2. insult of those nerve endings by other mechanical means must provoke a pain similar in nature to bends;
3. there should be a positive correlation between gas content and bends in that tissue;
4. it needs to be a "tight" tissue since a compliant tissue would enable gas to expand freely when its pressure would be less likely to reach the pain threshold for bends (Figure 1);
5. a small change in blood flow with exercise of that limb.

All of these conditions have been satisfied by tendon, but could also apply to certain other connective tissues.

### *Pain threshold*

The simple mechanical concept of pain (in limb bends only) has been particularly well demonstrated by Inman and Saunders who found that the identical pain could be produced by injecting Ringer's solution into tendon and other connective tissues. This pain was not determined by the volume of the solution injected but by the pressure differential. Moreover it was reversible and appeared or disappeared at the same pressure threshold

If we return to the situation of a gas rather than Ringer's solution pressing on that nerve ending, the net deforming pressure is the net gas pressure (internal less interfacial effects -  $\delta_g$ ) plus pressure ( $\delta_f$ ) due to any fluid accumulation. Thus bends pain can occur if:

$$\delta_g + \delta_f > \delta_t \quad \dots (1)$$

This very simple criterion for pain can be related to the volume gas (v) separated from solution in unit volume of tissue by:

$$\delta_g = Kv \quad \dots (2)$$

where K is the modulus (reciprocal of compliance) resisting expansion of the tissue.

Thus the unknown  $\delta_g$  can be eliminated, so that

$$\text{BENDS can occur if:} \quad v > (\delta_t - \delta_f)/K \quad \dots (3)$$

This simple quantitative relationship is compatible with the fact that limb bends are more likely to occur for a greater exposure or decompression ( $v \uparrow$ ), in an older subject ( $K \uparrow$  with age) for nerve endings sensitized by release of serotonin or other humeral factors ( $\delta_t \downarrow$ ) and trauma ( $\delta_f \uparrow$ ) while ameliorated by acclimatization ( $K \downarrow$  with creep) or plasma expanders ( $\delta_f \downarrow$ ). Moreover, in absolute terms, the  $\delta_t$  value from Inman and Saunders combined the value of  $v$  estimated for a diver whose minimum bends depth is fsw gives a  $K$  value within 10% of that for excised tendon.

### Relation to dive parameters

While the simple mechanical model can interpret many of the widely differing features of limb bends, we need to know whether this extends to the parameters of a dive.

Let us again consider the simple case of an exposure to a pressure ( $P_1$ ) by immediate decompression to  $P_2$ .

If the nitrogen tension at  $P_1$  is  $P_{N_2}$  immediately prior to decompression and is then reduced to  $P'_{N_2}$  by 'dumping' nitrogen into the gas phase until a quasi phase equilibrium is established at  $P_2$ , then a simple nitrogen balance gives:

$$n P_{N_2} = S_{N_2} \cdot P_{N_2} - S_{N_2} \cdot P'_{N_2} \quad \dots (4)$$

( $N_2$  dumped from solution)
( $N_2$  initially in solution)
( $N_2$  left in solution)

where  $S_{N_2}$  is the solubility of nitrogen in the tissue.

This now relates  $v$  to the nitrogen tensions before and after decompression. Before decompression:

$$P_{N_2} = F_{IN_2}(P_O - P_W) + F_{IN_2}(P_O - P_W) \quad \dots (5)$$

(nitrogen before compression to  $P_1$ )
(nitrogen taken up in time  $t$  at  $P_1$ )

where  $P_O$  is normal atmospheric pressure,  $P_W$  is water vapour pressure at body temperature,  $F_{IN_2}$  is the volume fraction of nitrogen at body temperature and  $\phi^t$  is the function of time ( $t$ ) for uptake [for the particular case where a steady state has been reached at  $P_1$ ,  $\phi(t) = 1$ ]:

$$\text{Steady state: } P_{N_2} = F_{IN_2}(P_1 - P_W) \quad \dots (6)$$

The only remaining unknown in relating the pain threshold  $\delta_t$  to dive parameters is now  $P'_{N_2}$ . This can be resolved by a simple pressure balance - but only for the "worst possible case".

*Worst Possible Case*

This is the state where at least one out of many millions of micro-regions of the tissue has 'dumped' all gas in excess of saturation to come to phase equilibrium. This is the worst possible not only because it represents the maximum volume of gas which can separate from solution, but there is then the lowest driving force remaining to eliminate that gas from the tissue via blood - see p. 18.

In any bubble the absolute gas pressure is determined by the external pressure, the pressure needed to push tissue aside in forming the bubble and the effect of surface tension ( $\gamma$ ) as described by the Laplace equation ( $2\gamma/r_{SDO5(b)}$ ). According the Dalton's Law this total absolute pressure must equal the sum of the partial pressures as depicted in Figure 3.

$$P'_{N_2} = P + B - m \quad \dots(7)$$

where  $B$  is a small constant as defined in Figure 3 and  $m = P_{vO_2} + P_{vCO_2} + P_w$  and is approximately constant provided  $P_{vO_2}$  does not exceed about 100 mm Hg.

Summation in Bubble

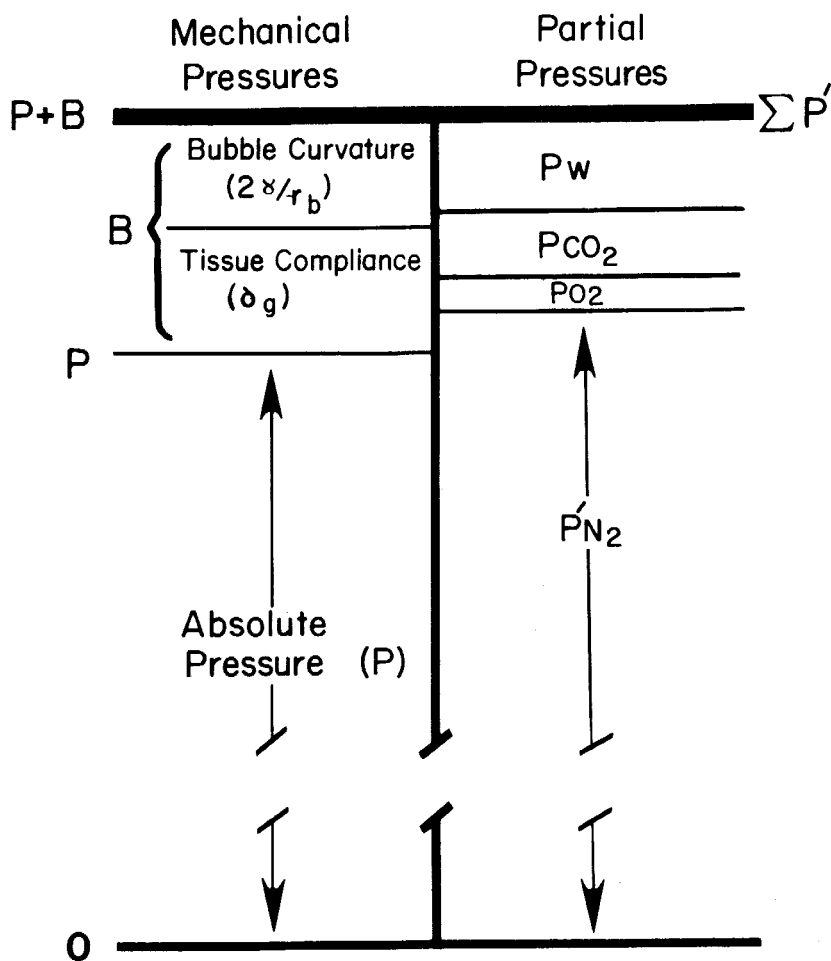


Figure 3

The sum of the mechanical contributions to bubble gas pressure must equal the sum of the partial pressures of all gases present - Dalton's Law

## Decompression ratio

In the past much attention has been paid to the use of decompression ratios of 'M' values, so it is important to see whether the simple mechanical approach to bends pain can explain the apparent adherence of decompression limits to a ratio concept.

Let us again consider the simple case (Figure 2) of a diver who has attained steady state at  $P_1$  being decompressed to a pressure  $P_2$  (ie.  $P = P_2$  in equation 7). Elimination of unknowns in equations 3, 4, 6 and 7 gives the simple relationship:

$$P_1 = WP_2 + Y \quad \dots(8)$$

where

$$W = (v + S_{N2}) / S_{N2} \cdot F_{N2}$$
$$Y = [(v + S_{N2})(B-m) + P_w \cdot F_{IN2} \cdot S_{N2}] / S_{N2} \cdot F_{IN2}$$

which is a small constant if  $v$  is constant.

Much more important is the fact that the gradient ( $W$ ) is constant if the volume ( $v$ ) is constant.

Equation 8 can be re-written in terms of a ratio ( $M = PSD05(1) / PSD05(2)$ ) as:

$$M = P_1 / P_2 = W + Y / P_2 \quad \dots(9)$$

Thus the simple mechanical approach gives a linear relationship between  $P_1$  and  $P_2$  - in fact, almost a ratio since  $Y$  is small. It predicts a decreasing ratio ( $M \downarrow$  as  $P_2 \uparrow$ ) as many proponents of popular calculation methods now prefer.

Actually a linear relationship offers a better separation of experimental bends and no-bends points as seen in Figure 4.

It also offers a much better correlation between diving and aerial bends where, by a simple ratio, a value of around 2 would apply for divers but 3 for aviators.

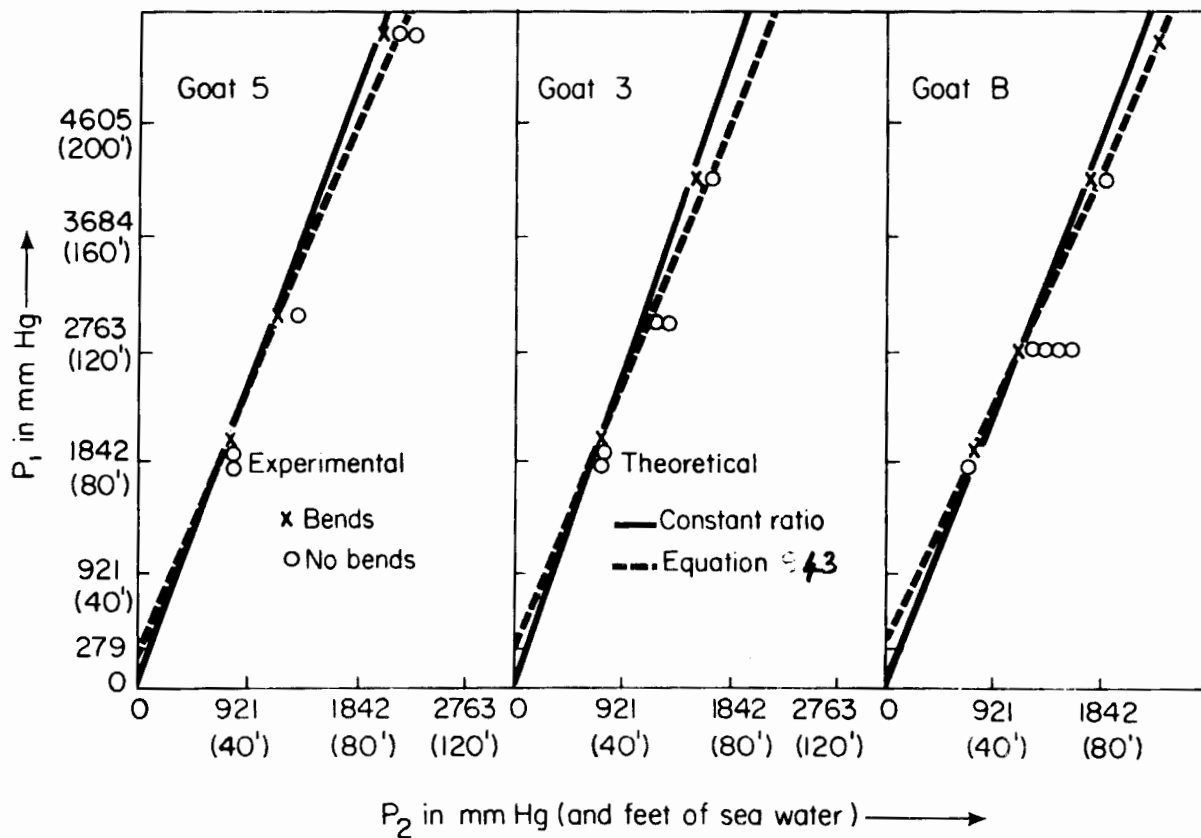
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### PULL THE OTHER LEG(S)?

Two patrons of a city hotel were startled by a large octopus on the floor of a ladies' toilet. The women told the management of the Hyatt-Regency Hotel they found the octopus outside a bar on the 22nd floor. The assistant manager, Mr Obie Collins, said the octopus was apparently placed there by two women dressed in army fatigues who were seen carrying a rubbish bag into the toilet. No one was hurt.

*Australian, 24 April 1978*

DID THEY CHECK IT WAS A FEMALE OCTOPUS?



**Figure 4**  
Data from Hempleman (1957)

*Number of tissues*

Returning to the list of issues (p. 4) for which answers must be found or assumed in the development of any model based upon fundamentals, the next question concerns the number of tissues, or rather, tissue types involved in limb bends. This is important since it determines the number of independent constraints to be applied to the formulation of the decompression and, hence, the number of independent equations to be used.

One might expect that, if the Haldane rationale applied, then "triggering" the 10 min "tissue" would provoke a different response to "triggering" the 40 min "tissue" and yet no correlation between the symptom and any hypothetical "tissue" has ever been shown. Hence there would seem to be no good reason for assuming more than one tissue- at least, no more than one *anatomical* tissue is involved in *limb bends*.

If there were several tissues, then one would expect a transition point in the dose-time curve whenever one superseded another in the imminence of pain, ie. a 'kink' in the no-stop decompression limits and yet none is perceptible in the data for air or heliox.

Similarly, if we return to the case of simple decompression from  $P_1$  to  $P_2$  where  $P_2$  is 'titrated' to marginal bends, then we would expect a transition if one tissue were to 'take over' from another as bends-determining. However, once again, no transition point could be detected until the pressure was in excess of about 300 feet (Figure 5) when the  $P_1$  vs.  $P_2$  relationship follows a different linear relationship, but vestibular DCS are then the presenting symptoms. Hence another tissue must be included for depth of over 300 feet, but there is then ample justification from the symptomatology. However, for *limb bends* there would still appear to be no reason to assume that more than one tissue type is involved - and *tendon* would seem a likely candidate according to previous discussion (p. 8). The next question concerns how this tissue takes up inert gas.

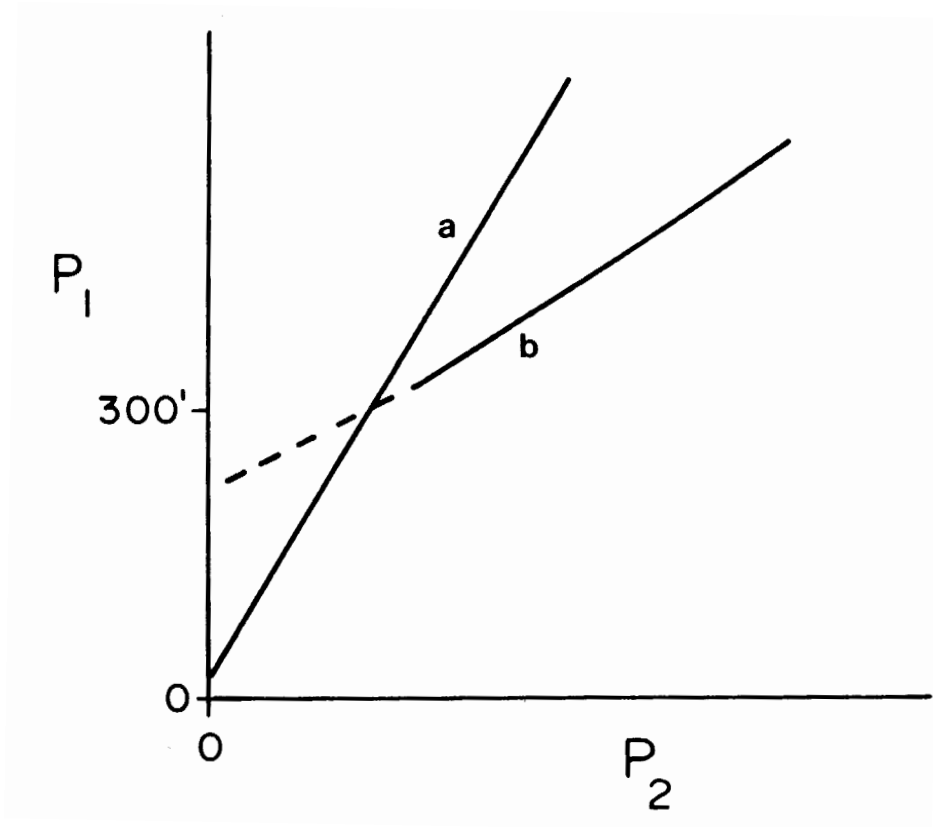


Figure 5

Data from Hempleman (1975).

How is gas taken up by tissue?

So far we have considered only those decompressions where the man has reached a steady-state before decompression, ie. where his pre-decompression nitrogen tension is independent of time as expressed by equation 6.

However, for shorter bottom times or further decompression to shallower depths, it is necessary to estimate tissue gas content in the light of the past history, ie.  $P_{N_2}$  is now a function of time. The question is then which function do we use. It is obviously an asymptotic function to allow for attaining steady state, but there are thousands from which to choose and the popular exponential (as used in the Haldane rationale) is just one possibility.

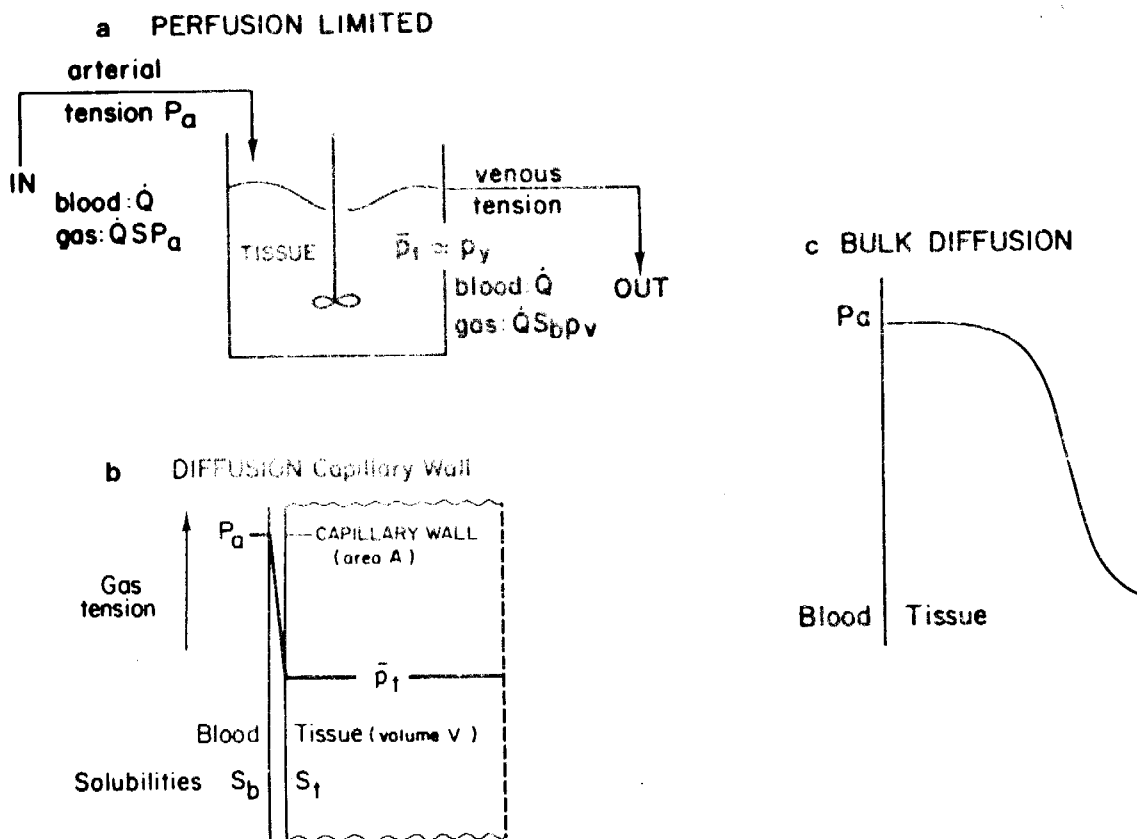


Figure 6

Various models which have been used to try to quantify inert gas uptake.

To try and answer this question from fundamentals we need to determine the appropriate model (Figure 6) for gas exchange in the critical tissue and this immediately raises the very fundamental question concerning whether the uptake of an inert gas is limited by the circulation (blood perfusion rate) or by diffusion when the relevant diffusion barrier can be either a membrane or the whole bulk of cellular material - see Figure 6 for alternative models which have been proposed.

To put these in perspective, let us consider an individual who has just switched from air to heliox breathing at normal pressure. The macro distribution of helium will be effected by the circulation while its subsequent assimilation by the extravascular tissue must occur by diffusion. Thus blood perfusion and extravascular diffusion are two transport processes in series, but which is rate-controlling?

There is a vast literature relevant to this issue, but very little can be considered decisive. However, to put many of these studies in perspective, we might return to the above case of the individual who has just switched from air to heliox. The highest tension of helium will occur in arterial blood ( $p_a$ ) while mean tissue ( $-p_t$ ) will be the lowest. One can then argue that if the tension is at the interface between the perfusion and diffusion transport processes is closer to arterial, then uptake is largely diffusion-limited; otherwise, if this tension is closer to mean tissue tension, it is largely perfusion-limited. Unfortunately this gas tension at the capillary wall cannot be measured, but venous ( $p_v$ ) may be taken as a good reflection of it. Hence the fact that Kety and Schmidt found that venous tension ( $p_a$ ) lay so close to their *estimated* mean tissue for  $N_2O$  in monkey brains was taken as strong evidence that uptake was controlled predominantly by the circulation. This has provided the basis for the conventional concept that blood perfusion is the rate-limiting process and that venous blood leaves in equilibrium with tissue ( $p_v = -p_t$ ). This gives a simple exponential function as the time response for a single tissue as needed by the Haldane rationale for decompression formulation. The membrane model (*b* in Figure 6) would also predict an exponential.

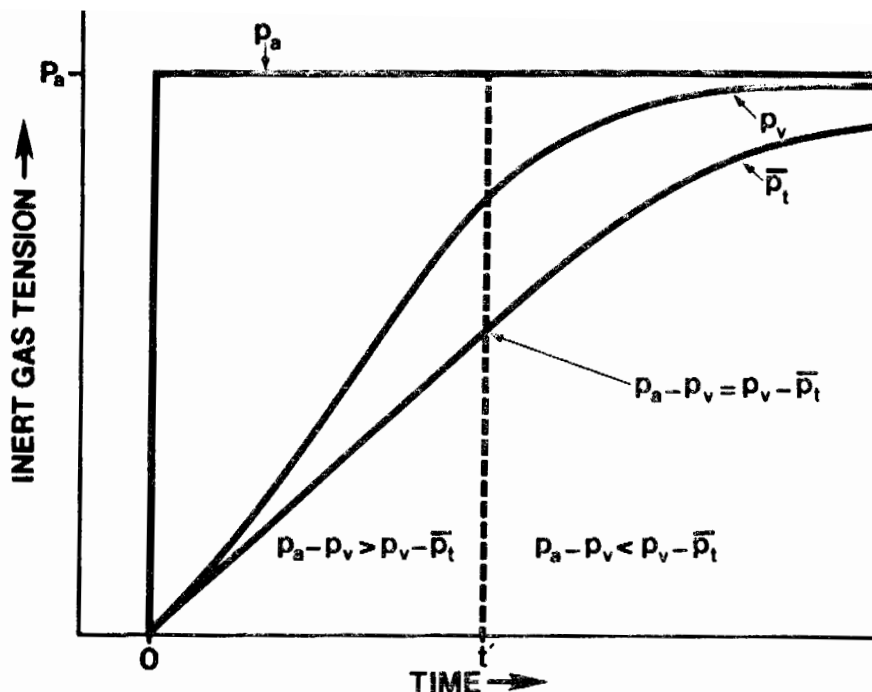


Figure 7

Arterial ( $p_a$ ), venous ( $p_v$ ) and mean tissue ( $-p_t$ ) tensions of inert gases in brain monitored after a sudden switch to that gas in the breathing mix.



However, more accurate analyses monitoring isotopes indicate that venous tension starts by following  $ACO1VS1(-,p)SD09(t)$  but then moves close to  $pSD05(a)$  with time (Figure 7), ie. uptake starts perfusion limited as the first of the new gas enters the system and then changes to predominantly diffusion limited as the increased assimilation reduces the gradients. However the same data can be equally well interpreted by saying that venous is the mixed venous outflow from many zones of the same tissue, so the only conclusion is that the final model is more than either (a) or (b) in Figure 6, and is either:

1. heterogeneous perfusion of the critical tissue in which each micro-region would have its own half-time (but this would still not explain a different M value needed for each), or
2. the rate-limited mode of uptake is bulk diffusion (model C in Figure 6), or
3. uptake is controlled by both processes.

### a DIFFUSION and PERFUSION

#### Two-phase model

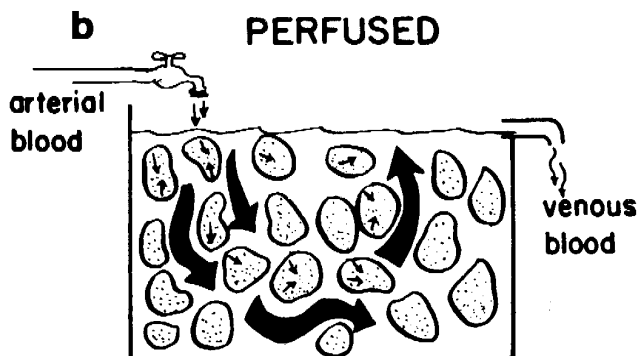
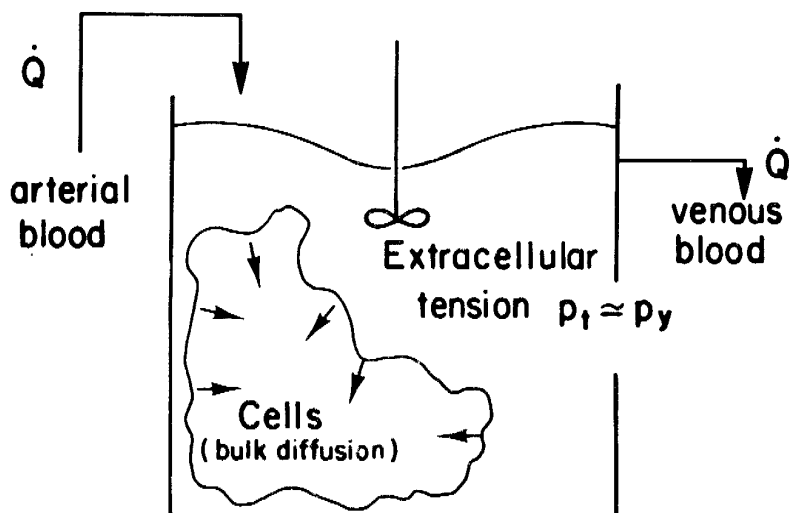


Figure 8

If we consider the most uniformly perfused organ large enough to viz. isolated skeletal muscle, the classical studies of Renkin have that the uptake of inert tracers is circulation-limited at low blood perfusion rates and diffusion-limited for high flow rates. Unfortunately, the normal physiological range lies intermediate between these two limiting cases so that neither diffusion nor perfusion can be ignored. This probably applies to the critical tissue also, so a compromise between the two is envisaged - Figure 8. This is the Kety concept of the stirred tank but taken only as far as the walls of the cell - immersed as though it were an irregular shape of effectively uniform permeability in which gas is assimilated by relatively slow bulk diffusion.

### *Summary*

So far, this discussion has been concerned with the mechanism for the occurrence decompression sickness rather than its prevention. This has indicated that the pain of limb bends has a simple mechanical basis which is easily quantified and most likely refers to just one anatomical tissue type (probably tendon or another tight well innervated connective tissue) in which gas uptake is controlled by both the blood perfusion rate and diffusion into the bulk of extravascular tissue. The next step is to see how this simple model can be used to optimise a decompression, but it was first necessary to test its compatibility for non-optimal situations. After all, you may disagree with the way the other fellow formulates his decompression, but you must still predict the outcome of the trials of his method by your model.

### OPTIMISATION

Let us consider a diver who has just completed his task on the bottom wishes to return to the surface safely yet without wasting time unnecessarily. The immediate question is how far does he decompress on the first 'pull'? Does he decompress all the way to a level just deeper than his bends point or is there some other criterion which determines his optimal depth?

If that particular phase of the decompression would enable the man to surface then the answer is obviously 'yes'. However, if bends would occur before surfacing, then do we decompress almost to his bends point or stop much sooner?

Conventional 'supersaturation' approaches to decompression, as presented in the multitude of neo-Haldanian calculation methods, assume that no gas phase is formed if you do not exceed the "trigger points" as expressed by ratios or 'M' values. The all-important question is does a sub-symptomatic decompression initiate the gas phase? By the model developed in this discussion, we may have a good correlation between the incidence of bends and other factors for single decompressions, but when does the primary event actually occur?

### *Point of inception of gas phase*

There is three basically different approaches to describing the point of inception of a stable gas phase in tissue:

1. The bends point
2. Point of phase equilibrium, and
3. Some intermediate "trigger point".

If the second of these is true, then it is most disturbing since it implies that tables formulated by the other approaches are provoking bubbles - including most conventional diving tables and US Navy tables in particular. However, before attempting to pursue this matter, it is desirable to know why this is such an important issue. After all, why should a few "silent" bubbles be so serious if they are not causing pain or other symptoms?

#### *Importance of supersaturation vs. equilibration*

The intention of all approaches to decompression formulation is to select conditions which will give the maximum rate of elimination of inert gas from tissue at each moment. Comparatively little can be done to change the resistance to the transfer of the gas, but a great deal can be done to select the optimal driving force for transfer of the tissue inert gas to blood for its elimination via the circulation, eg.  $\Delta P_{SDO5}(N_2)$  for air diving.

#### *Driving force for $N_2$ elimination*

The gradient for nitrogen elimination is simply the tissue-blood gradient where the blood tension for an arbitrary absolute pressure  $P$  is given by  $F_{IN2}(p-p_w)$  as per equation 6. The real problem is the value to use for tissue  $N_2$  tension, ie.

$P_{N2}$  for gas remaining dissolved, or  
whether

$P'_{N2}$  where gas in excess of equilibrium has been 'dumped'

This leads to two very different equations for the all-important driving force for nitrogen elimination from tissue, whether it is:

$$\text{IN SOLUTION } \Delta P_{N2} = P_{N2} - F_{IN2}(p-p_w) \quad \dots(10)$$

OR

$$\text{WITH SEPARATION: } \Delta P_{N2} = P(1 - F_{IN2} + B - m') \quad \dots(11)$$

$$\text{where } m' = m - p_w F_{IN2}$$

This comparison is extremely important since further decompression ( $P \downarrow$ ) would increase  $\Delta P_{N2}$  if all gas remains dissolved but decrease it if there is phase separation, since phase  $F_{IN2}$  must be less than 1 (0.8 for air). Thus the popular practice of decompressing as far as possible on the first 'pull' towards the surface, so characteristic of US Navy schedules, could have the diametrically opposite effect to that intended. In other words "by getting

the hell out of it" on that first long pull towards the surface, the driving force for nitrogen elimination is actually being *decreased* rather than *increased* - IF phase equilibration best describes the primary event. It therefore becomes imperative to establish whether a sub-symptomatic decompression can cause gas to separate from solution in the critical tissue(s).

### *"Silent" bubbles*

There is now overwhelming evidence that bubbles can be present during asymptomatic decompression. This includes examinations of sacrificed vascular window, X-ray studies, measurement of cerebrospinal fluid volume, conductometric monitoring of tissue and ultrasonic surveys. Even a device as crude as the ultrasonic bubble detector based on the Doppler principle indicates a host of venous bubbles in asymptomatic divers - often after the first long 'pull' towards the surface if using USN schedules.

However it can always be argued that these intravenous bubbles are irrelevant - particularly if we adopt the mechanical approach to bends pain discussed earlier. After all, nitrogen is five fold more soluble in fat and, from a structural standpoint, adipose tissue can be considered weak. Hence it is easy to envisage the large volume of extravascular gas bursting the capillary wall depositing bubbles, fat emboli and portions of endothelial cells into the circulation where they appear about the same time. Electron micrographs have been taken showing extravascular gas entering capillary blood in cutaneous tissue. The large volume of nitrogen would not give pain in adipose tissue due to the lack of nerve endings. Thus Doppler sounds probably refer to fatty tissues which would reflect the state of the critical tissue to some extent - hence the poor yet positive correlation between bends and 'venous' Doppler sounds.

It would therefore seem more relevant to turn to studies where a "tight" well innervated connective tissue has been monitored.

### *Conductometric studies*

The electrical conductivity of rat tail has been monitored during decompression, this being predominantly tendon. Electrical resistance was found to increase, the magnitude of the increase being greater for those animals which had respired a more soluble inert gas. Whereas this and the reversal upon recompression left no doubt that the electrical changes were caused by the separation of gas from solution, the interesting feature was the absence of any change until a minimal decompression of 95-145 mm Hg had been reached - whatever the inert gas present (Figure 9).

This threshold decompression for the appearance of the gas phase happens to coincide with the position of phase equilibrium in extravascular tissue (Figure 10) and agrees well with the altitude for the onset of bubbles as seen by X-rays.

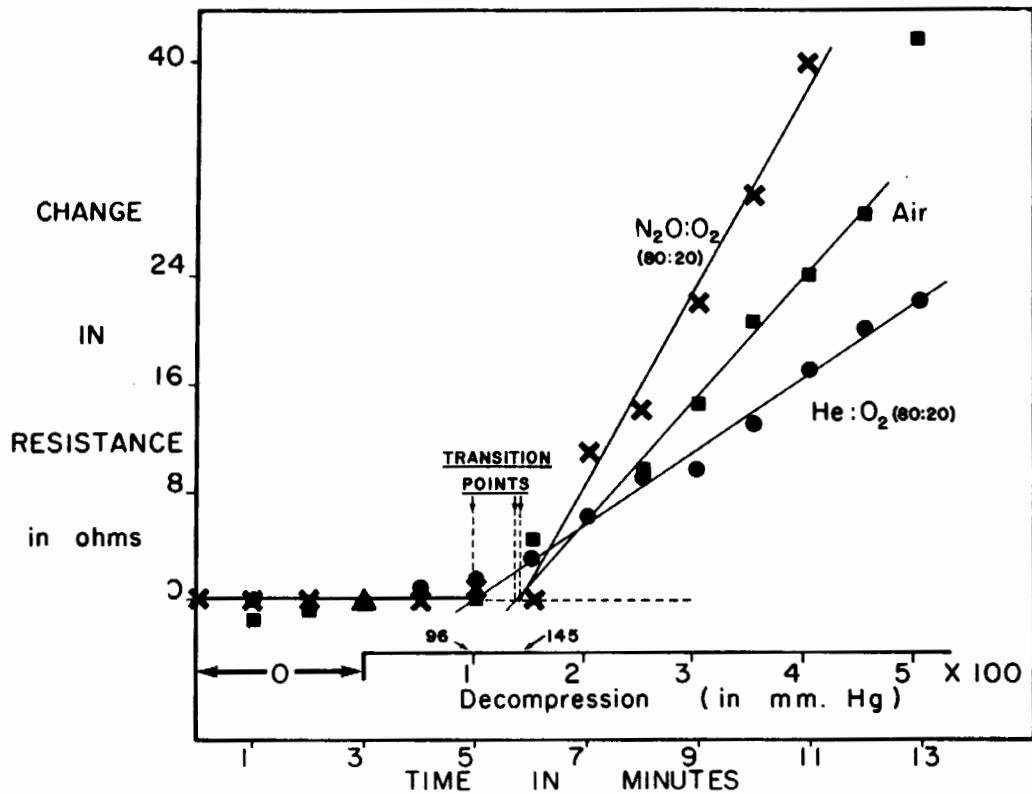
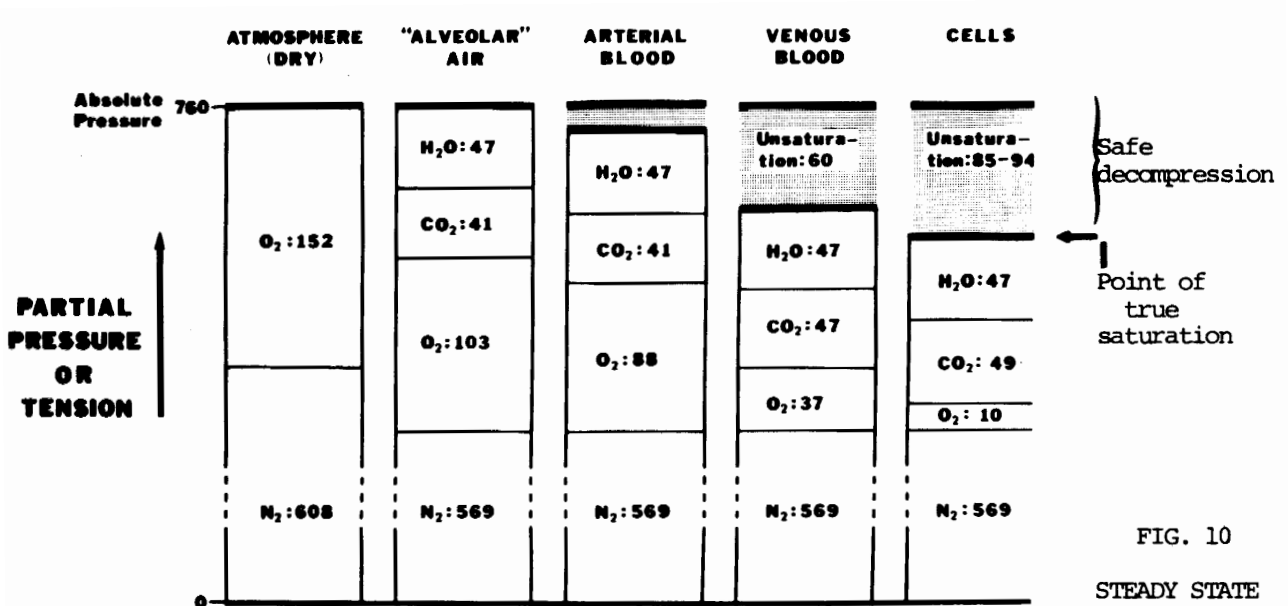


Figure 9

Electrical conductivity of a rat tail monitored during decompression to altitude at a uniform rate of pressure change. Data from Hills (1971).



### *Inherent unsaturation*

This immediately raises the question of why the position of phase equilibrium differs from normal atmospheric pressure in a subject who has always breathed normobaric air and might be considered "saturated". The reason is that, while Dalton's Law of partial pressures must apply to the gaseous phase, it need not hold in liquids; so that the conversion of a relatively insoluble gas ( $O_2$ ) into a much more soluble one ( $CO_2$ ) by metabolism causes a permanent deficit in the total gas tension of tissue relative to absolute pressure. This inherent unsaturation is depicted by the shaded area in Figure 10. Such reasoning implies that the term "saturation" diving is a misnomer and "steady state" might be more appropriate, since only a dead diver could reach true saturation before the start of decompression. The inherent unsaturation is very important not only because it determines the position of phase equilibrium upon decompression, and hence the point at which the first bubbles can start to form, but it provides a permanent driving force for dissolving gas in the body. This includes not only bubbles but intrapleural gas, gas in an occluded bronchioles or a blocked sinus, etc.

It has been demonstrated very simply by a sealed subcutaneous tube made from a non-collapsible plastic permeable to all gases and water vapour. Over a period of a few hours, the tube develops a partial vacuum of 80-100 mm Hg and stays at that value *ad infinitum*. Moreover, if the breathing mix or pressure is changed it moves to another value determined largely by the magnitude of the inspired  $PO_2$ . Thus the subject who has breathed pure  $O_2$  for a few hours at normal pressure has an inherent unsaturation of 600-700 mm Hg, so that he can decompress by this amount without any fear of inducing bubble formation.

Moreover the unsaturation also provides the driving force for dissolving bubbles. Let us consider the transcutaneous tube which has reached a steady state by virtue of all gases and water vapour equilibrating with the adjacent tissue. If the rigid tube were suddenly removed, the gas would be compressed by the inherent unsaturation and this compression would disturb the equilibrium and, in so doing, provide a driving force for dissolving the gas equal in magnitude to the original inherent unsaturation. Thus the inherent unsaturation is particularly desirable and fundamental to the whole problem of formulating decompression.

### *Decisive tests*

The foregoing evidence indicating that gas could separate from solution in tissue for much lesser degrees of supersaturation, if any, than implied in conventional calculation methods led this writer to claim that such diving (including USN schedules) were not preventing bubbles but were really *treatment tables* for containing subsymptomatic gas below the pain-provoking threshold. However, such a serious implication needed a definitive test, since previous experiments to try to settle this issue had involved searching for gas in one way or another and all such *direct* methods may have been monitoring an irrelevant tissue.

A crucial test which avoids identifying the critical tissue anatomically

has exploited the difference in  $\Delta PSD_{05}(N_2)$  depending upon whether gas remains in solution (equation 10) or is 'dumped' into the gaseous phase (equation 11), the significance being emphasized on page 18. Let us therefore consider a man who has spent one hour at 160 feet on air and has followed the appropriate US Navy air table to the end of the time normally allotted to the 20-foot stop - Figure 11.

**Figure 11**



Titration of a last stop at 10, 20 or 30 fsw on the same goats. Data from Hills (1968).

If no bubbles have been formed to that point, as assured in the formulation of the table based upon  $P_{N_2}$  rather than  $P'_{N_2}$ , ie. by neo-Haldanian reasoning, then decompression to the 10-foot stop at that time should provide more driving force for nitrogen elimination (equation 10) and hence a safer decompression. On the other hand, if much gas separated from solution on that first long 'pull' to 60 feet, and the subset decompression has simply been "treating" them, then we should use  $P'_{N_2}$  and equation 11 rather than equation 10 to determine the true outcome. It would then be better to remain at 20 feet when the driving force is greater than at 10 feet ( $\Delta P'_{N_2} \uparrow$  as  $P \uparrow$  in equation 11) and surfaced directly from 20 feet. When total decompression times were 'titrated' to bends

points on the same animals, it was found to be more efficient to surface directly from 20 feet than to include a 10-foot stop (see Figure 11) indicating that the gas phase *is present in the critical tissue* during this particular USN decompression and probably during many others based on the same calculation method. This finding is compatible with the observation common in aviation that preoxygenation is much less effective in eliminating nitrogen if undertaken at altitude as opposed to ground level (ie.  $\Delta P'_{N_2} \downarrow$  as  $P \downarrow$  when the gas phase *is present*).

This point has been emphasized since it points to a very fundamental and significant inadequacy in conventional calculation methods used to formulate diving tables, ie. any separation of gas from solution can greatly reduce its rate of elimination from tissue.

### *Optimisation*

Well, it is easy to be critical, but does the concept of the 'worst possible case", ie. phase equilibrium and the inherent unsaturation, really offer any better alternative? This writer believes that there is a reservoir of nuclei in tissue with a spectrum of energies for their activation into stable bubbles capable of growth and, hence, the inception of bubbles is a somewhat random process. However, whereas most areas retain their supersaturation, it only requires one out of maybe many million micro regions to 'dump' its gas for limb bends to occur. Thus the "Thermodynamic" approach considers this 'worst possible" case to be the most relevant. This concept has subsequently been re-named "Nil Supersaturation", and "Zero Supersaturation" by other workers.

The criterion for optimisation is therefore one of avoiding any supersaturation and yet not wasting time in decompression, ie. keeping the one tissue for limb bends just on the point of true saturation. However, if bulk diffusion is one of the resistances to gas transfer, we must apply the phase equilibration rule to each point and not just to the value of total gas tension averaged over the whole tissue. Thus we must estimate the peak total gas tension and then decompress by reducing pressure until it coincides with this peak (Figure 12). Thus the decompression continues until the diver has the amount of gas in his tissue which he could tolerate on the surface. At this point (usually around 20-25 feet) he "drops out" and forms the gas phase but to just below the pain provoking volume as defined by equation 3.

### **Figure 12**

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#### HOW ANEMONEFISH SURVIVE SEA ANEMONE NEMATOCYSTS

Doug Wallin has reported (*Sea Frontiers*, 24(1), 1978) recent studies of this surprising survival of anemonefish in its chosen habitat. The mucus covering the skin of each fish contains an inhibitor chemical that prevents the nematocysts from discharging. The fish acquire this ability after birth, lacking this immunity when first settled from the plankton. This takes about an hour, during which time it repeatedly brushes briefly against the tentacles. The anemone tentacles themselves must obviously contain a similar chemical to avoid stinging each other into impotence.



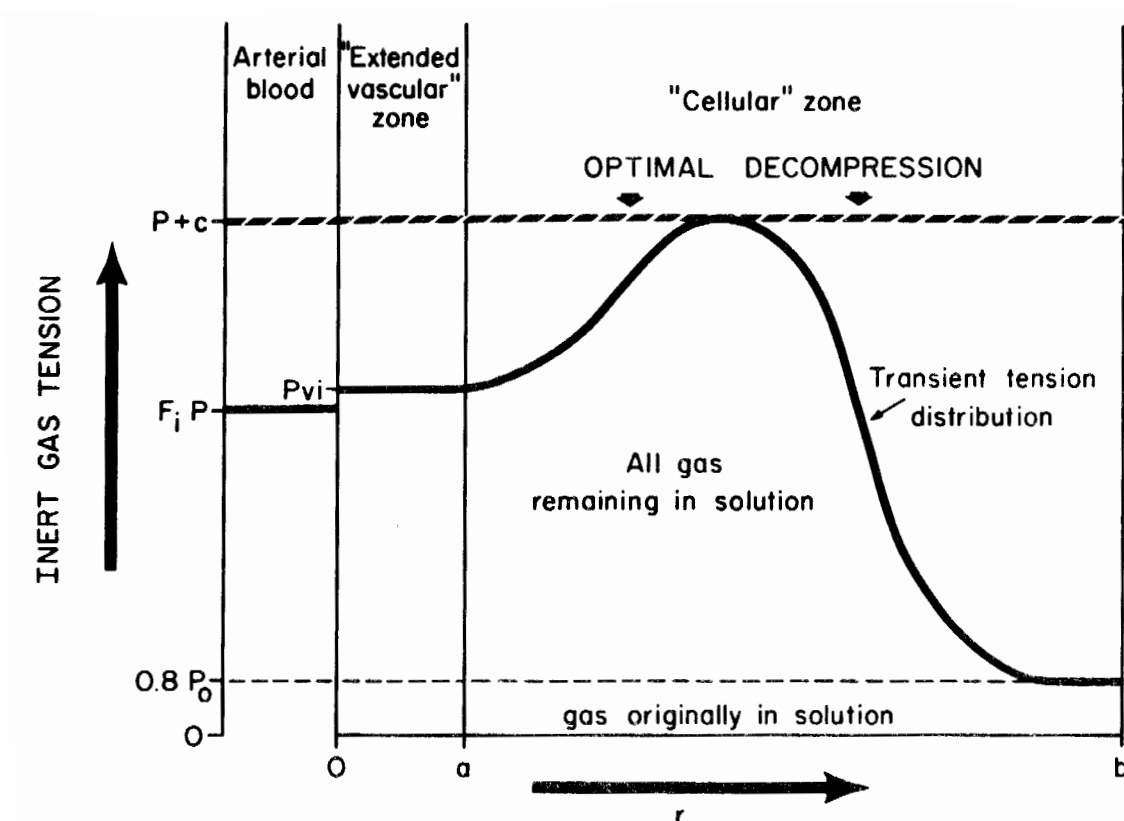


Figure 12

The Thermodynamic (Equilibrium) concept of decompression formulation.

The net effect is to introduce much deeper stops and redistribute decompression time towards the deep stages by comparison with conventional neo-Haldanian approaches - see Figure 13. Thus the Thermodynamic approach pioneered the concept of deep stops which have subsequently been introduced into most commercial tables by trial and error. At least, it provided a theoretical basis to justify the empirical modifications found necessary in order to reduce the high bends rate which those calculation methods were otherwise incurring. It also explains the much more efficient methods devised over years of trial and error by Okinawan pearl divers operating off the Northern coast of Australia.

### *Other Symptoms*

So far we have concentrated upon limb bends on the basis that their total avoidance also avoids 99% of other symptoms. However this is not entirely true, so we now need to look at the predisposing factors and then try to avoid them within the framework already outlined for programming the decompression based on limb bends.

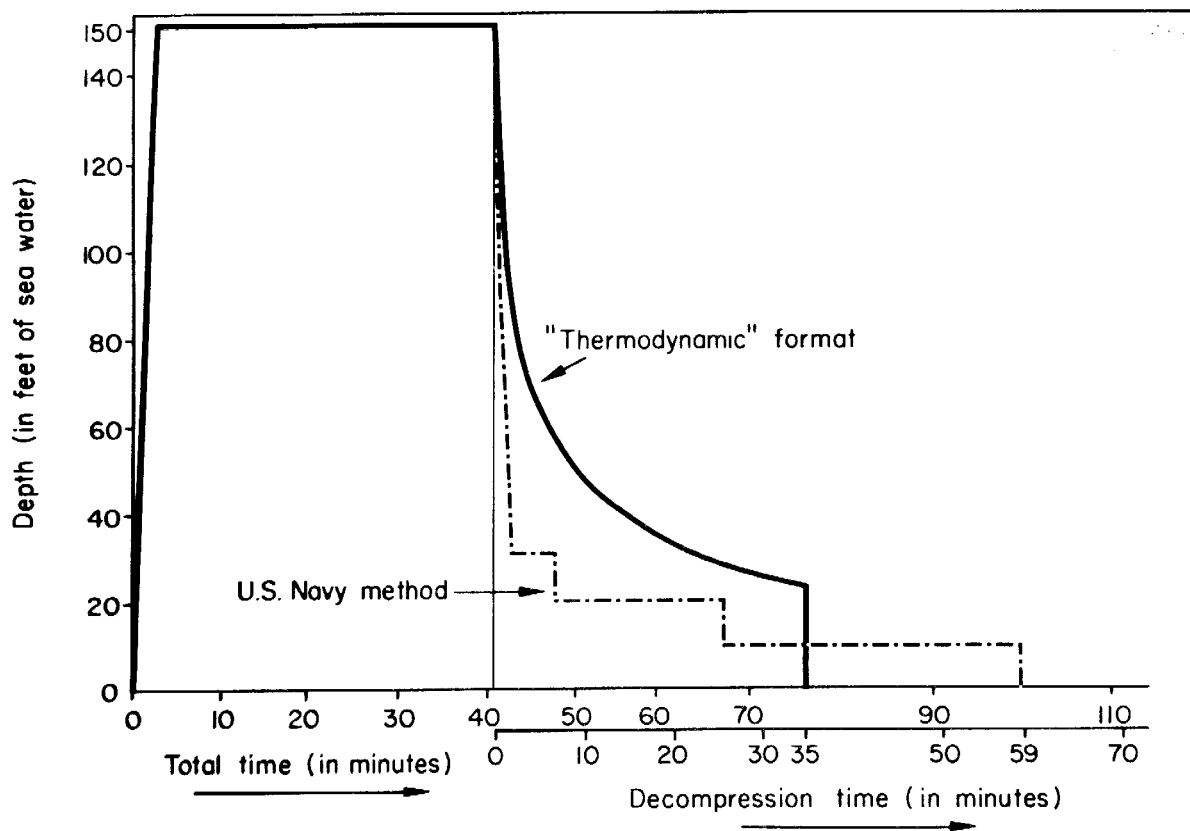


Figure 13

We cannot be sure of all the predisposing factors but two which seem to be emerging are:

1. Avoid arterial bubbles which can lead to cerebral and, possibly, spinal DCS, and
2. Avoid excessive tissue gradients of the heavier inert gases, eg. nitrogen, which tend to give vestibular problems.

The first of these relies upon maintaining the lung as an effective bubble trap for the mass of asymptomatic bubbles (and other emboli) which decompression can produce in the venous system - largely derived from fatty tissues. Our research on the lung is still at an early stage, but bubble filtering action seems to be impaired by:

1. Poisoning of the lung by excessive  $O_2$  for too long a period. This implies conservative use of oxygen for prevention and treatment.
2. Contaminants in the breathing mix.
3. Overload of the lungs by bubbles. This implies avoiding deep air diving where large amounts of nitrogen can be

liberated from fatty tissues in which this gas is most soluble.

4. Recompression. This suggests giving the minimum recompression to a diver with limb bends for fear of permitting trapped bubbles to enter the arterial system and produce a CNS "hit".

On the last point, there have been several cases of asymptomatic divers accompanying a colleague to pressure who has a limb bend, only to develop CNS symptoms themselves.

### *Vestibular problems*

Vestibular problems can occur *without* decompression if there are large gradients of the heavier gases. The mechanisms which have been proposed include:

1. counterdiffusion supersaturation,
2. gas-induced osmosis, and
3. counterperfusion supersaturation

The first is unlikely since the mechanism requires a lipid layer of appreciable thickness and there is no such diffusion barrier in the inner ear. However, whichever mechanism predominates, all are realities in some tissue and would act in the same sense in producing local pressure differentials to insult the vestibular apparatus. According to each, it would seem advisable to avoid excessive gradients of heavy gases by such means as:

1. Adding a little nitrogen to the diving mixes if the subject is going to switch to air upon transfer from the diving bell to the DDC.
2. In going to 500 feet for example, compressing part of the way, say to 100 feet, on air to force some nitrogen into the middle ear.
3. Slowly venting the bell with air before transferring the divers to the DDC.

### *Breathing mixture*

So far we have only considered the relationship between depth and time in programming the decompression - with decompression sickness as the sole constraint. We really need a simultaneous optimisation of depth vs time vs oxygen fraction in which there is a further constraint contributed by oxygen poisoning.

However this requires the limits for oxygen poisoning to be expressed quantitatively - not just for a constant inspired PSDO<sub>5</sub>(O<sub>2</sub>) as quoted in the USN manual, but for a complex oxygen history so common in diving. Two methods have been proposed:

1. That based upon the total UPTDs (unit of pulmonary toxicity dose) based upon changes in vital capacity of the lung. This gives some "handle" on the maximum oxygen which the lung can tolerate over long periods but is rather restrictive in optimising since it cannot allow for the known regression of the insult upon return to a sub-toxic mix.
2. A cumulative oxygen toxicity index (COTi) aimed primarily at predicting the onset of neurologic symptoms of  $O_2$  poisoning for which there are no reliable warning signals until it is too late to prevent. This index is based upon the principle of superposition (Figure 14) which seems to hold to within  $\pm 10\%$  in animals and men.

The second approach allows for regression of the oxygen insult upon reversion to a non-toxic breathing mixture and has been used for a simultaneous optimisation of depth vs time vs breathing mixture which is not so difficult to implement if a computer is available.

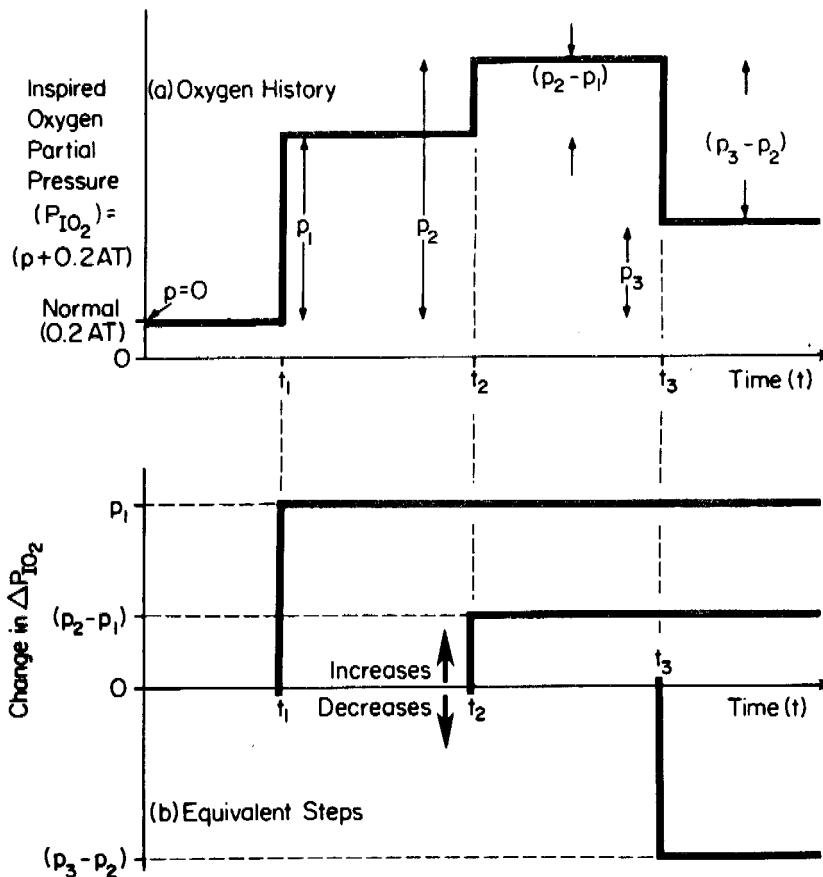


Figure 14

Simple arithmetic basis for the Cumulative Oxygen Toxicity Index - from Hills (1976).