

Chapter 9

Thermodynamic Decompression

It is one matter to describe the many aspects of decompression sickness and their theoretical implications but quite another to rationalize the deductions into a fundamental model from which to formulate practical decompression schedules. This process of synthesis is not aided by the need to recruit the whole spectrum of scientific disciplines from theoretical and mathematical physics to clinical diagnosis and blood chemistry in covering the multitude of facets of decompression for which explanations are needed. The foregoing chapters have shown that there is no obvious model or, at least, none which would gain universal approval in the field at this time. However, while there are still a few major issues the outcome of which is not yet certain, there is an adequate volume of data on which to make qualified deductions. Having synthesized such a universal model, it then needs rigorous analysis to test its viability.

To demonstrate the type of scrutiny to which any such model must be subjected, the 'thermodynamic' approach has been selected as a convenient example. This choice is based not only upon this writer's opinion that it still offers the most fundamental set of explanations but it was presented with the most attention paid to the type of detail which needs to be covered and yet is all too often omitted in the many calculation methods produced as short cuts to diving tables.

The Model

At the start of Chapter 3 a number of fundamental questions were listed and then discussed in the light of the factual evidence (Chapter 2)

to produce varying degrees of conclusion which can be synthesized into a model.

An opinion

These discussions lead this writer to the opinion that

(1) the primary event and the critical insult in producing symptoms of decompression sickness do not coincide (p. 75).

(2) The primary event is the activation of one or more of a 'reservoir of nuclei' normally present in tissue into growth and hence the inception of a stable gaseous phase. On the other hand, limb bends are determined by the local pressure differential tending to bend or otherwise distort a nerve ending beyond its pain-provoking threshold and hence their onset is more dependent upon the volume of gas separated from solution (p. 57).

(3) No more than one anatomical tissue type needs to be invoked in marginal cases of limb bends (p. 191). This is probably tendon or some other well-innervated connective tissue of minimal compliance (p. 60).

(4) For all except very rapid decompression, the gas separates from solution in extravascular sites (p. 147) and in any static blood when it can be regarded as effectively extravascular for computational purposes, deriving its gas from extravascular tissue.

(5) The much larger volumes of gas thus deposited in fatty tissues can be released into

the venous system as bubbles which remain asymptomatic unless they fail to be trapped by the lungs or are present in such numbers that they stimulate J-receptors in the pulmonary bed to invoke 'the chokes' (p. 68). Although a single decompression which deposits more gas in a fatty tissue is also more likely to deposit more gas in the critical tissue, any coincidence between the appearance of venous bubbles (e.g. as detected by Doppler) and the volume of gas 'dumped' in the critical tissue by a complex pressure history is likely to be largely fortuitous, i.e. the appearance of venous bubbles is not particularly relevant to limb bends (p. 146).

(6) Cerebral symptoms (p. 63) are caused by arterial bubbles which have permeated the lung under certain unusual conditions or can be formed by decompressions so rapid as to supersaturate arterial blood in its passage from lungs to brain, e.g. in submarine escape. However, in diving, they will not normally occur unless there is something unusual such as an extensive recompression or a pathological state in the lung impairing its bubble filtering action, e.g. after excessive exposure to oxygen (p. 68).

(7) Spinal 'hits' are less likely to be the result of arterial gas emboli (p. 64) and require rather more gas to be deposited in cortex than cause limb bends when 'dumped' in connective tissue. Hence it is probably safe to work on the basis that decompressions which avoid *marginal* 'limb bends' are unlikely to provoke any serious spinal injury and no further tissue needs to be invoked in calculations.

(8) However, another tissue *does* need to be considered to account for vestibular symptoms (category III, p. 70), particularly if diving deeper than 300–400 fsw or switching inert gases (p. 71). Symptoms are still determined by the local pressure differential analogous to limb bends (Equation 7) but any inert gas gradient between blood and the middle-ear cavity, or any counter-gradient of two gases, can contribute to this differential.

(9) This additional local differential pressure (δ_f) can be contributed by counterdiffusion, counterperfusion and gas-induced osmosis (Chapter 8) with no certainty at this time as to which, if any, predominates. This additional differential may also be a minor factor in limb bends but only from dives where there is a large amount of gas dissolved 'deep' in tissue (p. 58).

(10) Although haemoconcentration may impair the circulation, the primary cause of blood degradation in diving is intravascular bubbles. Thus blood factors need not influence decompression formulation provided the *primary event* is genuinely avoided in the critical tissue for limb bends. Preventing any gas separation in this tissue may not prevent it forming in fatty tissues but the volume should not be enough to cause the disruption of those tissues leading to lipid emboli and venous bubbles (p. 147) and hence haematological problems.

(11) The inception of the gaseous phase in any decompressed gas solution is a random process (Chapter 4) to which the critical tissue type for marginal limb bends is no exception.

(12) Limb bends are determined by the 'worst possible case'—the particular micro-region of the critical tissue where the activation of nuclei into a stable gas phase is so profuse that the tissue can only withstand minimal supersaturation before 'dumping' gas in excess of thermodynamic equilibrium into the gaseous phase (p. 100).

(13) In this densely nucleated zone, diffusion distances of any gas molecule to the nearest gas phase are so short that, when phase separation occurs, it is effectively complete within a few minutes of the decompression. Tissue regions other than this 'worst possible case' may retain their supersaturation or have a few nuclei which slowly grow into bubbles as they 'drain' the surrounding tissue of its dissolved gas in excess of saturation. Thus the behaviour of the vast majority which

determine overall gas elimination from a tissue is believed to be largely irrelevant.

(14) The onset of bends is delayed until the 'dumped' gas, which is finely dispersed, can coalesce or otherwise congregate within the extravascular tissue to produce a local pressure differential (mechanical stress) adequate to bend, or otherwise distort, a nerve ending beyond its pain-provoking threshold (p. 58).

(15) The gas dumped in the 'worst possible case' contains all gases present in tissue, the volumes contributed being proportional to their respective tissue tensions *after* separation. Thus water vapour is present at its vapour pressure, while the metabolic gases are contributed at their venous tensions or less (p. 102). Inert gas provides the difference between the total hydrostatic pressure and the total partial pressure of oxygen, carbon dioxide and water vapour needed to satisfy the thermodynamic criteria for local phase equilibration.

(16) The uptake of inert gas is limited by both blood perfusion and diffusion into the bulk of the cytoplasm (Chapter 7). The transport model envisaged is thus one of a fully stirred 'extended vascular' zone from which venous blood leaves in equilibrium with respect to all gases, while arterial blood either dilutes or replenishes the gases in that 'tank' as the case might be. From this effectively fully stirred 'extended vascular' zone the gases then diffuse into the 'cellular' zone by bulk diffusion, i.e. into a bulk of uniform permeability with no specific resistance offered by any particular membrane (fig. 65). *p. 182*

(17) Decompression should be optimized by preventing gas phase inception anywhere within that bulk, i.e. within the 'cellular' zone. This means applying the principle of phase equilibrium to each point in the tissue and hence involves determining the peak rather than the mean tissue tension (p. 125).

(18) If decompression proceeds to the point where the total pressure of gas in the nucleus is less than the total tension of all gases dissolved

at the peak then, in the 'worst possible case', gas is dumped until they are equal and allowance must be made for the corresponding fall in the driving force for inert gas elimination to blood (p. 138).

It is doubtful whether all of these points ultimately prove to be correct but they do represent a particularly comprehensive model which differs from others in one major respect. It details not only how to optimize a decompression for a particular exposure but how to predict quantitatively the course of events if the decompression were not optimal by these criteria. This writer takes the view that whereas you may disagree with the way the other fellow formulates his decompression, it is still necessary to predict *by your model* the result of the trials of *his tables*.

General criticism

The thermodynamic approach received quite hostile comments from some quarters when it was first introduced, since its net effect was to advocate much deeper stops and an overall deeper redistribution of decompression time. Moreover, it was introduced at a time when M values had reached a popularity peak and the U.S. Navy were decompressing as far as possible on their first long 'pull' in the belief that they were obtaining the greatest driving force for inert gas elimination in so doing. That is, they were exploiting to the maximum their belief that the greatest bends-free decompression ($P \downarrow \downarrow$) resulted in the greatest driving force ($\Delta P_{N_2} \uparrow \uparrow$) in Equation 51. Thus they were placing the maximum dependence upon their assumption that the critical tissue(s) of the bends-free diver were bubble-free, so that the 'thermodynamic' approach presented one of the first comprehensive challenges to most of their axioms of decompression formulation. Their formal assessment of this 'provocative' model (Hester, 1970) produced only one real criticism; that a dual set of equations were being employed throughout the quantitative analysis. Two sets of equations were indeed developed in the original publication of the

'thermodynamic' approach (Hills, 1966). However, what these critics had not appreciated was that this model clearly separates the *primary event* (gas phase inception) from the *critical level of insult* (local pressure differential) needed to provoke bends. Hence one set of equations refers to conditions which describes a critical volume of separated gas and hence stable bubbles (radius r_b), while the second refers to the tissue before the nucleus (radius r_n) is activated into growth. The net difference on a thermodynamic basis is minimal, actually amounting to a differential of about 2 fsw in the constant representing the 'mechanical' contribution to the total gas pressure.

To avoid any similar misunderstanding in this text, the mechanical contribution to hydrostatic pressure is designated B in the marginal bends-provoking bubble and B' in the nucleus (see fig. 29; also depicted in figs. 26 and 31). Hester (1970, 1971) also went on to point out several basic similarities in the decompression format produced by the 'thermodynamic' model and those produced essentially by empirical deduction by Bühlmann (p. 118).

Summary

The model can thus be summarized as a single tissue type (an avascular connective tissue) in which inert gas uptake is limited partly by the blood perfusion rate and partly by diffusion into the bulk of the cellular material of effectively uniform permeability (fig. 64(a)). Gaseous cavitation in this extravascular tissue is both temporally and spatially random but the imminence of limb bends is determined by events only in the 'worst possible case'. This may be only one in many million possible micro-regions but one where nuclei are activated for minimal degrees of supersaturation and where these are so profuse that any gas in supersaturated solution is rapidly 'dumped' into the gaseous phase. However, this gas separated from solution in extravascular sites will only produce limb bends after it has had time to coalesce and if, with any fluid accumulation, the total displacement can produce a local pressure differential which can bend, or

otherwise distort, a nerve ending beyond its pain-provoking threshold.

On the other hand, if the gas phase is not allowed to form, let alone reach its pain-provoking volume, then the volume of gas in separated fatty tissues should not become so large as to rupture fatty tissues and so produce a blood-gas interface causing all manner of haematological disorders. Moreover, prevention of the critical insult in this one critical connective tissue should also avoid spinal symptoms, while it is only after deeper exposures or for mixed inert gases that the 'vestibular tissue' can take over as rate-controlling.

This 'thermodynamic' model is not strictly 'zero-supersaturation' (Hills *et al.*, 1976) nor 'nil-supersaturation' (Bennett and Vann, 1975), since a minimal degree overpressure or additional gas tension is needed to balance the equation for phase equilibrium (Equation 21 and fig. 26). Thus the mechanical forces contributed by the curvature of the nucleus ($2\gamma/r_n$) and tissue compliance (δ_t) can be regarded as a minimal degree of tolerable supersaturation before gas starts to separate from solution. This underscores the two vital axioms on which the whole model is based:

- (a) it is really the local pressure differential and hence the volume of gas which can be 'dumped' which largely determines the imminence of limb bends;
- (b) since this volume of 'dumped' gas is only a maximum when the tissue and gas phases come to equilibrium, is there really any driving force then left for eliminating gas from tissue?

Test of the basic axioms

Prior to the 'thermodynamic' approach, many proponents of models and calculation methods had discussed the volume of gas separating from solution but only Nims and Bateman (p. 126) had actually incorporated this parameter into equations for predicting the imminence of clinical symptoms of decompression sickness. However, although the 'volume' concept was contrary to popular formulation, it is really fairly obvious. The passing 'niggles'

on moving to a shallower stop of a conventional decompression table and the minimum recompression needed to relieve most bends pains bear witness to this. Moreover, relief is usually instantaneous and, for marginal cases, can be effective if applied to the site itself, such as by an inflatable cuff or by simply immersing the limb in water. Deductions made from these simple observations are fully compatible with the abundant evidence for 'silent' bubbles within the body as a whole as shown by direct detection devices (e.g. ultrasonic, radiographic and conductometric techniques). Furthermore, experiments designed specifically to indicate asymptomatic gas separating from solution *in the critical tissue* leave little doubt that this is no exception.

While it does not prove that gas 'dumping' goes as far as establishing phase equilibrium, this point is so close on the decompression scale to those at which X-rays and conductance measurements show a phase change (p. 142) that it is highly likely that one out of the many million possible sites in each tissue will conform to this 'worst possible case'. Moreover, measurements of the mode of volume change on decompression of excised tissue indicates that the distribution of bubbles is so varied that at least one micro-region of the critical tissue is likely to come very close to this state on most decompressions exceeding equilibrium. This is the 'worst possible' because not only does it represent the maximum number of gas molecules separating from solution but there is then the minimum driving force for their elimination from the tissue.

The Inherent Unsaturations

The real test of the 'thermodynamic' approach arises when it comes to the second axiom. How can a gas phase in apparent local equilibrium with tissue eventually dissolve and disappear without any further change of pressure or of composition of the breathing mix; what driving force can there be? Such a driving force has been derived theoretically as ΔP_{N_2} in Equation 52—a concept termed the 'inherent unsaturation' by Hills (1966) and the 'oxygen

window' by Behnke (1967) in their independent approaches to prevention and treatment. The Buffalo group (Van Liew *et al.*, 1965) also deduced a similar expression from their observation of subcutaneous gas pockets which they did not name and surprisingly, perhaps, did not apply to the *prevention* of decompression sickness; although treatment was mentioned in their discussions. The significance of this driving force has recently been rediscovered by the U.S. Navy who term it the 'partial pressure vacancy' (Sass, 1976). However, the inherent unsaturation is such a vital factor in 'volume' approaches for quantifying the imminence of clinical symptoms that it really needs direct experimental verification *in vivo*. Moreover, the absence of such a driving force in his 'volume' model caused Bateman to abandon his approach (p. 126), while Nims would have been forced to do the same if he had followed Bateman's lead in attempting to formulate diving tables as opposed to predicting aerial bends.

Deduction of unsaturation

The inherent unsaturation has been largely implied from analyses of subcutaneous gas pockets in studies which have raised sporadic interest over many years. Campbell (1924) found that 500–1000 cc of air injected into rabbits changed in composition with time, the carbon dioxide rising to a steady 50 mm Hg within minutes while the oxygen fell from 150 to 50 mm Hg in 10 hours, reaching 20–30 mm Hg after 1½–3 days. The nitrogen then provided the additional partial pressure needed to compensate for the fall in $(P_{O_2} + P_{CO_2})$. These results were largely confirmed by Coryllos and Birnbaum (1932) using dogs and were in close agreement with values of 50 mm Hg (carbon dioxide) and 20 mm Hg (oxygen) recorded by Van Liew (1962) from the analysis of injected gas 'equilibrated' with rat liver.

The manner in which the nitrogen partial pressure in the cavity increases to accommodate the difference between the fall in oxygen (150 to 20 mm Hg) and rise in carbon dioxide (0 to 50 mm Hg) has been demonstrated by

Asknes and Rahn (1957) whose analyses show 90% nitrogen in resting dogs. This agrees well with a theoretically predicted dry-gas fraction of $100(760 - 20 - 50 - 47)/(760 - 47) = 90.2\%$ nitrogen. Moreover, for exercise, where P_{O_2} values should be lower, some of the early studies of bubbles in guinea pigs revealed 95% nitrogen (Harris *et al.*, 1945b). Van Liew *et al.* (1965) went on to point out how the nitrogen fraction could be estimated by using *venous* values for P_{O_2} and P_{CO_2} in the type of summation equation for Dalton's Law expressed by Equation 3 and depicted graphically in figs. 26 and 31. They then went on to deduce theoretically that this inferred an unsaturation in the tissue adjacent to the gas pocket.

Reaching the same conclusion quite independently of this study, the group in Adelaide

had considered that the inherent unsaturation was such a vital link in the 'thermodynamic' approach that it needed to be demonstrated directly (Hills, 1966; Hills and LeMessurier, 1969). They reasoned that the inherent unsaturation is basically a pressure difference and therefore needs to be demonstrated as such. This eliminates the use of gas pockets of the type studied by Campbell and later workers since these are essentially *constant-pressure* cavities.

This reasoning led to the use of *constant-volume* cavities in which a rigid membrane permeable to all gases and water vapour is used to restrain the tissue mechanically. Thus all gases will reach a true equilibrium between the adjacent tissue and the cavity in which the partial pressure of each will attain the tension of the

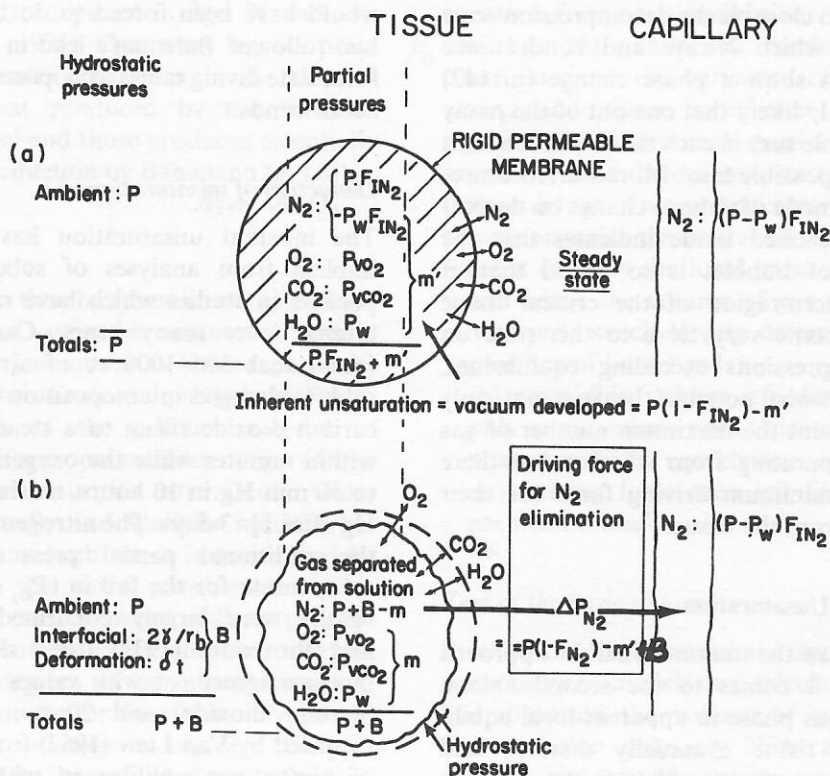


Fig. 81 (a) Depicting how a rigid cavity with a wall permeable to all gases reaches a steady state in which the total pressure of all gases ($P_{F_{N_2}} + m'$) is less than the ambient absolute pressure (P), the difference (or inherent unsaturation) being manifest as a partial vacuum. (b) Showing how this unsaturation becomes a driving force for eliminating nitrogen from the cavity if the rigid wall is removed and the tissue is allowed to compress the contained gas. B is an additional yet minor contribution arising from tissue compliance and surface curvature

same gas in tissue (fig. 81a). Hence the total tissue gas tension will now equal the sum of the partial pressures in the constant-volume cavity which, by Dalton's law, must equal the absolute pressure of gas in that cavity. Thus any inherent unsaturation in the tissue is manifest as a deficit in the cavity pressure relative to ambient, i.e. as the amount of vacuum developed in the probe after equilibrium has been established (fig. 81(a)). Rigid membranes not only provide a direct measurement of the inherent unsaturation but they provide a direct gas measurement divorced from mechanical uncertainties related to tissue compliance (p. 58), any negative hydrostatic pressure in tissue (Guyton, 1963), the curvature of a direct tissue-gas interface (p. 85) and any fluid influx (p. 57), all of which complicate any deductions made from constant-pressure cavities.

While these experiments on constant-volume cavities were in progress in Adelaide, Lategola (1964) published his findings using a cylindrical capsule with silicone-rubber membranes held rigid across the ends. However, his measured unsaturation of 41–48 mm Hg is open to alternative interpretation in view of his statement that the membranes had 'relatively negligible permeability to water vapour'. This implies that he could have been simply measuring the vapour pressure of water at body temperature—47 mm Hg!

The vital experiment

The Adelaide group implanted rigid PVC tubes subcutaneously in rabbits, having first proven that the walls of these probes were permeable to oxygen, carbon dioxide, nitrogen and water vapour. A partial vacuum was found to develop in these constant-volume cavities over 12 hours and not to change significantly over the next 24 hours. Moreover, the magnitude for air breathing at normal atmospheric pressures lay within the range 80–94 mm Hg (Hills, 1966; Hills and LeMessurier, 1969). This corresponds very closely to the failure of carbon dioxide (0 → 50 mm Hg) to replace oxygen (150 → 20 mm Hg) in gas pockets as measured by Campbell and later workers, i.e. inherent unsaturation = $150 - 20 - 50 = 80$ mm Hg.

This inherent unsaturation, as directly displayed by the partial vacuum, was interpreted as arising from two sources:

- (1) the fact that the solubility of carbon dioxide is some 25-fold greater than that of oxygen, so that metabolism is converting a *less* soluble gas (oxygen) into a comparable number of molecules, or slightly fewer ($RQ \approx 0.8$), of a *more* soluble gas (carbon dioxide) so that the total tension of local tissue oxygen + carbon dioxide tends to be reduced. Note that it is total gas *tension* and not concentration which determines the imminence of phase separation;
- (2) the shape of the oxyhaemoglobin dissociation curve where large elevations in arterial P_{O_2} may result in little change in venous P_{O_2} and hence in tissue oxygen levels. Compare the difference ($P'_{aO_2} - P'_{vO_2}$) for elevated oxygen with ($P_{aO_2} - P_{vO_2}$) for normal levels in fig. 10. p. 25

Varying pressure and composition

While such numerical agreement is reassuring, the most significant findings of the Adelaide group came when they varied the composition of the breathing mixture and the pressure. They then found that the inherent unsaturation

(a) increases linearly with absolute pressure for an inspired mix of constant composition (fig. 82(a));

(b) decreases linearly with mole fraction of inert gas in the inspired mix (fig. 82(b)).

This is in direct agreement with Equation 52 and, moreover, the gradients of the slopes found experimentally (figs. 82(a) and (b)) agree with those predicted in this expression. However, as it stands, the inherent unsaturation has been directly measured as a pressure difference, while Equation 52 refers to a driving force for nitrogen elimination (ΔP_{N_2}); so what is the connection?

Implications of the inherent unsaturation

It has been established experimentally that the ambient hydrostatic pressure will exceed the absolute pressure of all gases within a constant-

Bild
P. 138

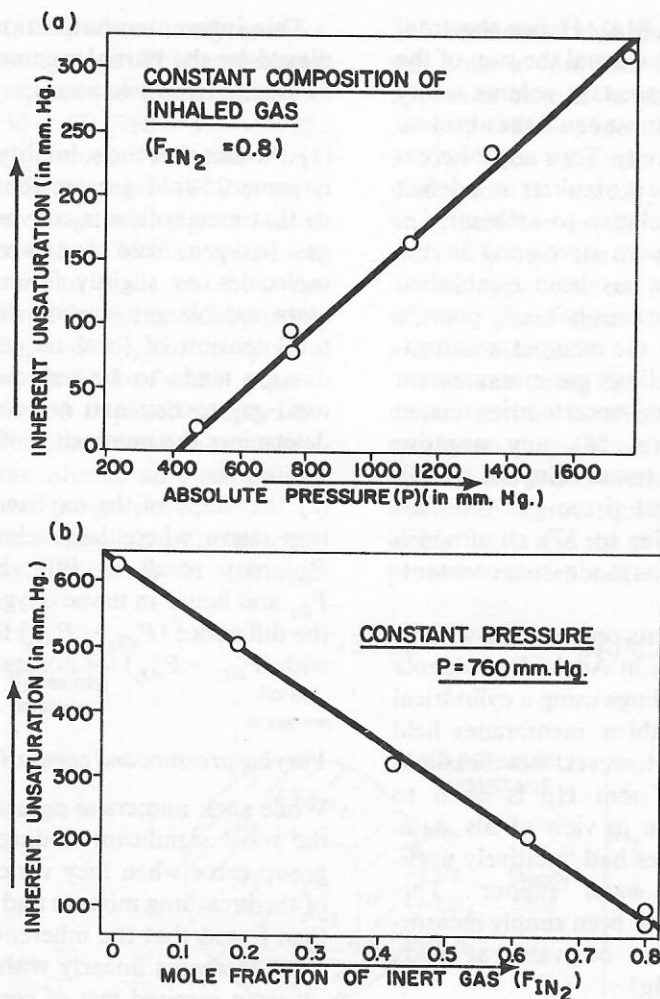


Fig. 82 Experimental measurements of the variation of the inherent unsaturation in the rabbit (a) with inert gas fraction in the breathing mix at normal pressure and (b) with pressure for a given breathing mix (air). Data from Hills and LeMessurier (1969)

volume cavity formed by a rigid membrane after the system has come to equilibrium. Thus the membrane is restraining the compliant tissue surrounding it by exerting a force equal to the developed vacuum, i.e. equal to the inherent unsaturation. If that rigid membrane is now removed, the gas is compressed by this amount. Thus the total partial pressure of all the enclosed gases will rise and exceed the total of the tensions which each strives to attain under those same steady-state conditions. Hence the equilibrium is destroyed and the degree of compression in letting the walls move

(fig. 81(b)) until cavity pressure is elevated to ambient now becomes a driving force for the walls to absorb the gas in what is now a constant-pressure cavity. The inherent unsaturation thus becomes the driving force for absorbing *all* gases previously equilibrated within the permeable rigid membrane. However, since carbon dioxide, oxygen and water vapour equilibrate so much faster than nitrogen as Campbell (1924) and later workers have shown, nitrogen is the gas controlling the rate of resolution of the gas phase in tissue and hence virtually the whole of the inherent unsaturation

becomes a driving force for nitrogen resolution. There will be an additional contribution to this compression provided by tissue compliance (δ_t) and a direct tissue-gas interface ($2\gamma/r_b$) formed by removing the rigid membrane. Together these contribute a mechanical term (B) to the driving force for nitrogen elimination (ΔP_{N_2} in Equation 52 and depicted in fig. 40).

While it is convenient to start from a rigid cavity and then remove the rigid permeable walls in understanding how the driving force arises for resolving the compliant cavity remaining, the same ΔP_{N_2} applies whatever means was used to create it—including decompression. Hence it is particularly significant that there is direct experimental evidence to support Equation 52, not only from the linear relationship between ΔP_{N_2} and both P and F_{IN_2} and the value of the constant (m) but from cases of constant-pressure cavities (bubbles) induced by decompression (Chapter 6).

Significance

The inherent unsaturation is important not only for determining the driving force for inert gas elimination once a stable gas phase is established but for setting the point of phase equilibrium. In other words, it also determines both the point of inception of gas separation (fig. 29) and the volume 'dumped' if this is exceeded (fig. 31).

The concept that the driving force (ΔP_{N_2}) increases with absolute pressure when the gas phase is present (Equation 52) was used as major evidence in Chapter 6 that conventional diving schedules—and U.S.N. tables in particular—were really *treatment* tables for an asymptomatic gas phase produced during their first long 'pull' towards the surface. Hence direct experimental justification of Equation 52 was essential before such a serious criticism of standard practice could be made.

The concept of the inherent unsaturation implies that the term 'saturation diving' is really a misnomer, since no diver can 'saturate' with gases in the true thermodynamic sense however long he may stay at one depth. He would need to have either zero metabolism or to breathe 100% inert gas—neither state to

be recommended. On the other hand, his inert gas would equilibrate at depth and so a more appropriate term might be 'steady-state' diving.

If a diver starts his ascent to the surface from a steady-state condition, then his whole decompression will be determined by the inherent unsaturation if phase equilibration dictates the state of the critical tissue. This is particularly compatible with the observation of many such exposures on men where the safe rate of ascent was found to be hyper-dependent on the inspired P_{O_2} and, at the tissue level, reflects more than simply the substitution of inert gas by oxygen (Vorosmarti *et al.*, 1975).

The final implication of the inherent unsaturation concerns oxygen breathing. Whereas it is obviously ideal for increasing ΔP_{N_2} in Equation 52 and hence superb for bends treatment (p. 232), oxygen is effective because it does not replace inert gas at the tissue level in the way that it does at the alveolar level. However, this vacancy which it leaves would not be welcome by the physician who may try to use hyperbaric oxygen to oxygenate peripheral tissues in certain disease conditions (see p. 15).

Quantitative Description

The model invokes two tissues; a relatively avascular connective tissue which determines limb bends and a 'vestibular' tissue needed for very great depths or when switching inert gases at an appreciable pressure. However, for normal diving, just one tissue and hence one equation is needed to describe the *level of insult* and hence the imminence of decompression sickness. By the same token, just one equation is needed to describe the *primary event*—the inception of a stable gas phase.

This leads to two basic equations which are needed.

(1) An expression for the condition that gas can separate from solution anywhere within the critical tissue, this event being random but possible whenever gas tensions have exceeded the point of thermodynamic equilibrium as

depicted in figs. 26 and 29. This equation for avoiding the primary event is particularly relevant to optimization when, if correctly formulated, it should also avoid haematological complications and those associated with other forms of decompression sickness—at least, unless the 'vestibular tissue' takes over as rate-determining.

(2) An expression for the imminence of symptoms and hence for the level of insult and its proximity to the pain threshold in the critical tissue. This is relevant to any decompression however it was formulated and applies where the condition for gas phase inception described by (1) has been far exceeded. Hence it is more relevant to the analysis of data and in checking the model rather than in optimizing a decompression.

If such an analysis is applied to a long history of gas phase presence, then ignoring haematological factors could introduce an error but, for the purpose of model testing, there are plenty of other cases where this should have been avoided. However, before the conditions described in (1) and (2) can be expressed comprehensively, it is necessary to know how to describe the distribution of gases *before* any inception of the gaseous phase.

Metabolic gases

Consider each of the gases likely to be present in the critical tissue and hence in any bubble. Water vapour will be present in any cavity at its vapour pressure for body temperature ($P_w = 47$ mm Hg) and will evaporate or condense on the surrounding tissue to maintain that value. Thus it will always contribute P_w towards the total tension tending to induce cavitation (fig. 29), or to grow a bubble.

The preceding discussion of the inherent unsaturation (pp. 239–243) leaves little doubt that oxygen and carbon dioxide are present at their *venous* tensions or, at least, the sum ($P_{O_2} + P_{CO_2}$) does not exceed ($P_{vO_2} + P_{vCO_2}$) and may be less. However, since this venous sum is small relative to diving pressures,

little error can be introduced by taking this value which, if anything, would err on the safe side. Thus, it is safe to say that

$$m = P_{O_2} + P_{CO_2} + P_w \leq 117 \text{ mm Hg} = 5.1 \text{ fsw} \quad (80)$$

Moreover, direct measurement of the inherent unsaturation indicates that this holds over wide ranges of arterial P_{O_2} (fig. 82).

Inert gas distribution

From a calculation standpoint, it is most unfortunate that the evidence on the perfusion versus diffusion controversy does not allow either to be ignored, since any hybrid model is far more difficult to describe mathematically than if one or other were to predominate. Moreover, resistance to gas transfer imparted by diffusion is not restricted to a simple membrane barrier but is imposed by the bulk of the 'cellular' phase as a whole. Thus the final model, although so simple to depict schematically (fig. 64), is most difficult to describe analytically. *p. 180*

This can be redrawn (fig. 83) to enable distance co-ordinates (r) to be assigned, when the effectively fully stirred 'extended vascular' zone has boundaries ($0 \leq r \leq a$) and the bulk 'cellular' phase has $a \leq r \leq b$. The 'extended vascular' zone comprises the intravascular plus that part of the interstitial space in motion or which is so permeable relative to cellular diffusivities (D_c) that it is also at the same tension as the efferent fluid (venous blood plus lymph). This is therefore effectively fully stirred and has a uniform venous tension (p_v).

The less permeable 'cellular' zone is assumed uniform but at first sight it cannot be determined whether it is spherical, cylindrical, flat, annular or even whether gas diffusion paths will converge or diverge. Thus the mode of curvature, if any, is unknown let alone the magnitude as expressed by the radius of a boundary (a or b in fig. 83). However, this has been circumvented by the use of a dimensionless 'shape factor' Hills (1969c) for the cell which must have a volume (V), surface area (A) and mean radius ($b - a$), when

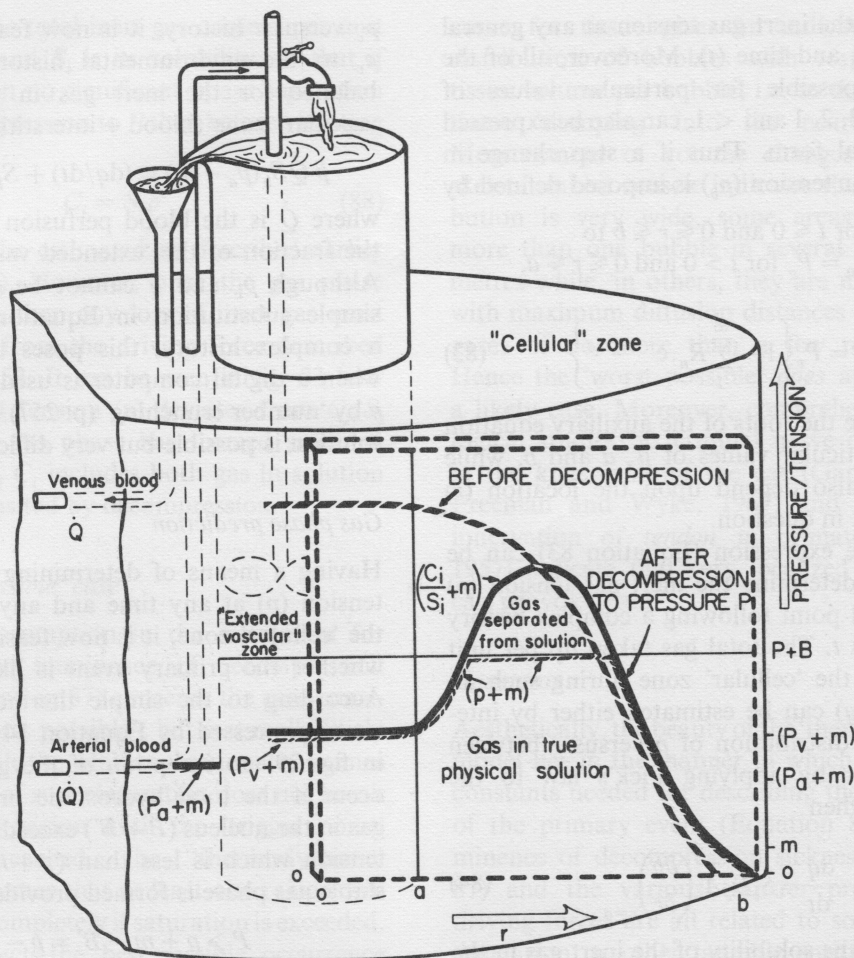


Fig. 83 The tissue model illustrated as a bulk 'cellular' zone of uniform permeability in which all transfer occurs by diffusion of gas supplied from the 'extended vascular' zone. This is depicted as the stirred tank in which arterial blood immediately mixes with the contents as it enters, while venous blood is effectively the overflow leaving in equilibrium with those contents in which there are no concentration gradients. The inset graph depicts the spatial distribution of gases through both zones for an arbitrary decompression, C_i being the inert gas content and p_i its tension. Where phase equilibrium has been exceeded, the excess gas has been 'dumped' in accordance with the 'thermodynamic' principle and the inert gas tension cut off at $P + B - m$ rather than following the contour of (C_i/S_i) as though it had remained in solution

$$\text{shape factor } (\mu) = A(b-a)/V \quad (81)$$

where $\mu = 1.0$ for a flat slab, 2.0 for a long cylinder, 3.0 for a sphere, etc. Taking the sphere as an example, $A = 4\pi(b-a)^2$ and $V = 4\pi(b-a)^3/3$, when substitution in Equation 81 gives $\mu = 3.0$

Moreover, it was pointed out (Hills, 1969c)

that, for all but cylindrical systems ($\mu = 2.0$), the Fick-Fourier equation (19) for bulk diffusion of an inert gas ($M = 0$) in a static phase ($\dot{Q} = 0$) can be expressed in the general form

$$\frac{1}{D_c} \frac{\partial p}{\partial t} = r^{(1-\mu)} \frac{\partial}{\partial r} \left[r^{(\mu-1)} \left(\frac{\partial p}{\partial r} \right) \right] \quad (82)$$

p. 13

where p is the inert gas tension at any general location (r) and time (t). Moreover, all of the solutions possible for particular values of μ , e.g. $\mu = 3, 2, 1$ and < 1 , can also be expressed in a general form. Thus if a step change in extracellular tension (p_v) is imposed defined by

$$p = 0 \text{ for } t \leq 0 \text{ and } 0 \leq r \leq b \text{ to} \\ p_v = P_v \text{ for } t > 0 \text{ and } 0 \leq r \leq a,$$

then

$$p = P_v \left\{ 1 - \sum_1^{\infty} R_n \cdot e^{-\alpha_n^2 D t} \right\} \quad (83)$$

where α_n are the roots of the auxiliary equation for the particular values of μ , a and b , while R_n values also depend upon the location (r) of the point in question.

The same expression (Equation 83) can be applied to determine the inert gas tension (p) at a general point following a complex history of p_v versus t . The total gas taken up per unit volume by the 'cellular' zone during such an operation (q) can be estimated either by integrating the distribution of p versus r between a and b or by applying Fick's law to the boundary when

$$\frac{dq}{dt} = S_c D_c A' \left(\frac{\partial p}{\partial r} \right)_{r=a} \quad (84)$$

where S_c is the solubility of the inert gas in the 'cellular' phase and A' is now the surface area per unit volume.

For a single step in p_v , the solution for q is exactly similar to Equation 83, where the roots (α_n) are the same but coefficients (R_n) are different.

The great advantage of the shape factor lies in the fact that α_n and R_n values lie on smooth curves when plotted against the values of μ , i.e. 3.0, 2.0, 1.0 and < 1.0 , for which mathematical solutions to Equation 82 are feasible (Hills, 1967b, 1974a). The shape factor is by no means comprehensive, nor offers a unique value for a particular cell, but it does give some 'handle' on the particularly difficult mathematics posed by the irregular nature of tissue 'geometry' and avoids the need to postulate ideal shapes.

Having related both p and (dq/dt) to the

p_v versus t history, it is now feasible to relate p_v to the environmental history by a mass balance for the inert gas in the 'extended vascular' zone (blood + interstitium), when

$$\beta \dot{Q} S_b (p_a - p_v) = (dq/dt) + S_b (dp_v/dt) \quad (85)$$

where \dot{Q} is the blood perfusion rate and β is the fraction of the 'extended vascular' region. Although p_v and q cannot be eliminated by simple substitution in Equations 83–85, for a complex history, this poses little difficulty when a digital computer is used to determine p by 'number crunching' (p. 257). An analytical solution is possible but very difficult to handle.

Gas phase prediction

Having a means of determining the inert gas tension (p) at any time and any point within the 'cellular' zone, it is now feasible to predict whether the primary event is likely to occur. According to the simple thermodynamic criterion expressed by Equation 14 and depicted in fig. 29, no inception of the gas phase can occur if the total hydrostatic pressure of the gas in the nucleus ($P + B'$) exceeds the total gas tension which is less than $(p + m)$. Hence no stable gas phase is formed provided

$$P > p + m - B' = p - c' \quad (86)$$

at all points within the tissue, where c is $(B' - m)$.

Imminence of bends

While Equation 86 describes the primary event, the gas phase must grow considerably before it can cause the level of insult to reach its threshold for symptoms. The imminence of limb bends depends upon the local pressure differential to bend a nerve ending which, in turn, is largely determined by the maximum volume of gas (v) which can separate from solution in unit volume of tissue ($V = 1$), i.e. the 'worst possible' case. Substituting for v according to Equation 9, Equation 31 gives the condition that bends can occur if for one gas (i)

$$\frac{C_i - S_i(P + c)}{(P + c)} > \frac{S_c - \delta_f}{K} \quad (87)$$

(g) = p. 59
(31) = p. 104

where C_i is the total inert gas content per unit unit volume and S_i is its solubility, so that if there had been no significant phase separation before decompression to P then before violating Equation 86

$$C_i = S_i p \quad (88)$$

where p can be estimated as described earlier (pp. 244–246). However, once the gas phase is established, then any loss of inert gas must be estimated from the driving force described by Equation 52. It can be seen in fig. 83 how the actual gas tension (p) deviates from C_i/S_i wherever the gas phase has formed since, in those regions, C_i includes both gas in solution and that deposited by decompression.

The 'worst possible' case

The major assumption in the expression for estimating the imminence of decompression sickness (Equation 87) concerns the case considered the 'worst possible'; but is it really likely to occur? While the 'Haldane' rationale assumes that the state of supersaturation does not break down in any region until the 'trigger point' has been reached, the 'thermodynamic' approach assumes that at least one region will break down completely if saturation is exceeded. Thus one depicts the 'best possible' occurrence and the other the 'worst possible' while something in between may be more appropriate but much more difficult to describe mathematically.

To test the probability that the 'worst possible' is likely to occur, this writer has performed an experiment based on the following argument. It can be measured or predicted mathematically how a single bubble will grow as it drains the dissolved gas from a large volume of tissue which has been decompressed. Two bubbles far apart will give twice the volume change but otherwise follow the same profile. However, when two bubbles are near to each other, they will start by following the same profile but the rate of growth will reach a plateau sooner as they both start to drain gas dissolved in the same tissue zone. Thus the shape of the volume-versus-time

curve for tissue gives an indication of the distribution of bubbles with respect to the tissue volume per bubble, i.e. to the volume of tissue 'dumping' into the nearest bubble. Measurement of volume changes in excised skeletal rabbit muscle indicates that the distribution is very wide, some areas having no more than one bubble in several cubic millimetres while, in others, they are most profuse with maximum diffusion distances for dumped gases of no more than a few micrometers. Hence the 'worst possible' does appear to be a likely case. Moreover, comprehensive histological studies of terminal nerve distributions in the knee joint of the cat (Gardner, 1944; Freeman and Wyke, 1967) and the general innervation of tendon in primates (Stilwell, 1957) indicate that very localized stimulation can provoke pain.

Constants

Aesthetically, the beauty of the 'thermodynamic' model lies in the manner in which each of the constants needed for describing the occurrence of the primary event (Equation 86), the imminence of decompression sickness (Equation 87) and the various transfer processes and driving forces are all related to some physical dimension or other tangible entity. Surveying Equations 80–88, these include D_c , S_c , S_b , A , A' , μ , B , B' , m , a , b , δ_i , δ_f and K . Some of these are known without a doubt, such as $S_b = S_c$ = solubility of the inert gas in water at body temperature, while other constants which cannot be assigned values without positive identification of the critical tissue can be grouped. Thus the final list of constants reduces to

- (1) (D_c/a^2) with dimensions of $(\text{time})^{-1}$.
- (2) μ —the dimensionless shape factor.
- (3) b/a —dimensionless (unnecessary for $\mu = 1$).
- (4) \dot{Q} —with dimensions of $(\text{time})^{-1}$.
- (5) $(\delta_i - \delta_f)/K$ —index of pain sensitivity (Z)— $(\text{volume})(\text{pressure})^{-1}$ (unless δ_f varies).
- (6) c or c' related to mechanical factors (B or B') and the total venous tensions (m), while the other constants can be related to each other as

$$A = \beta A' / V = \beta \mu / (b - a) \quad (89)$$

and

$$\beta = (a/b)^\mu \quad (90)$$

thus reducing the effective number of constants to only six. Moreover, if a particular anatomical tissue is selected, then the geometric and perfusion terms are fixed to reduce the number of degrees of freedom in selecting constants to only two.

Having described the model quantitatively it is now feasible to assess its viability, not only in qualitative terms but to ensure that the equations are compatible with the words since *only the equations* are ultimately used to optimize a decompression.

Qualitative Assessment of the Model

Before any model is used to optimize the decompression procedure, it should first be tested for compatibility with the many general qualitative facts and then with basic quantitative data. Moreover, the derivation of certain fundamental constants from these quantitative analyses and any agreement with known physiological values offers the final test of fundamental adequacy.

From a quantitative standpoint, the comprehensive expression describing the imminence of decompression sickness (Equation 87) is essentially a comparison of two terms, the left-hand side describing the level of insult to the tissue, while the right-hand side relates the critical threshold to individual factors.

Level of insult

It can be seen that the condition for bends is more likely to occur if the total inert gas content of the tissue (C_i) is higher. This is compatible with the fact that longer and deeper exposures (p. 36) or those with a greater fraction of inert gas in the breathing mixture (p. 37) are more likely to produce bends—all for the same decompression.

A more soluble inert gas ($S_i \uparrow$) is more likely to violate the condition expressed by Equation 87 such as nitrogen tending to produce lower minimum bends depths than

helium for the same decompression and fraction of the breathing mix (p. 38). On the other hand, a faster diffusing gas ($D_c \uparrow$ in Equations 82–84) will increase the content of inert gas (C_i) before it asymptotes, so that air can take over from heliox as superior for deep 'bounce' dives (p. 188).

The comprehensive expression (Equation 87) shows that the critical condition for bends is more likely to be violated if there is greater decompression *per se* ($P \downarrow$). Moreover, the equation expresses the concept of a threshold, where reversal of the insult by elevating P (i.e. recompression) can reverse the development of symptoms (p. 41).

So far only the factors related to the degree of insult as expressed by the left-hand side of Equation 87 have been discussed—all equally well explained by other theories.

Individual factors

The right-hand side of Equation 87 refers more to the threshold itself and is therefore more closely related to individual factors. The distribution in susceptibility is determined largely by individual differences in threshold pressure differential (δ_i) for bending or otherwise distorting a nerve ending. Osmosis would act by increasing δ_i , while potentiating factors such as serotonin could decrease δ_i , where the threshold term is then more likely to be exceeded by the insult—i.e. by the left-hand term in Equation 86.

Obesity is reflected by an elevation of lipid inclusion in the critical tissue, if it is tendon (as observed in that tissue in guinea pigs—p. 60) which, in turn, elevates the solubility (S_i) and hence the likelihood of symptoms (p. 41).

Adaptation to decompression (p. 60) is expressed by the decrease in the modulus ($K \downarrow$) with successive exposures, thus elevating the threshold so that the same insult is less likely to exceed it. Similarly the increased susceptibility with age (p. 60) can be explained by the critical tissue becoming less compliant ($K \uparrow$), thus increasing the likelihood of exceeding the threshold.

Fluid shifts

Individuals with a higher natural fluid turnover rate are likely to have a lower tissue fluid pressure ($\delta_f \downarrow$) and hence higher ($\delta_t - \delta_f$) to explain their marginally lower susceptibility (p. 43). By the same token, the reduction of extravascular fluid pressure ($\delta_f \downarrow$) which should result from administering low molecular weight dextran is likely to decrease the threshold and even reverse a marginal bend—as observed clinically (p. 232).

Haematological disorders leading to trauma or oedema and a general exodus of fluid from the vascular system will elevate extravascular fluid pressure ($\delta_f \uparrow$), thus reducing the level of insult needed to exceed the bends condition (Equation 87). However, no claim is made in its presentation that the model can accommodate blood disorders, since this writer, at least, can find no simple means of quantifying them in terms of environmental history and hence relating them to δ_f .

On the other hand, it is an easier task to relate the fluid shift caused by any gas-induced osmosis to environmental parameters. Particularly during a 'saturation' decompression, where there is a large reservoir of inert gas deep in tissue, this gas could exert a significant osmotic pressure tending to 'pull' water out of blood and so increase δ_f . Hyperoxia would tend to augment this process (p. 222), as would a switch of inert gas, quite apart from any effect which this might have on the volume of any bubbles present. Since the shifting of fluid is a slow process, it is possible to spend too long decompressing when δ_f reduces the threshold (right-hand) term in Equation 87 to a level at which a minimal gas-mediated insult (left-hand term) is all that is needed to provoke symptoms. This could explain the low bends incidence (Bühlmann, 1969) in some short (8-day) decompressions from 1000 fsw compared with 21 days used by the U.S. Navy (Workman, 1969). However, these are exceptional exposures during which any one of a host of physiological factors could become critical.

The 'vestibular' tissue

For such long deep exposures, a second tissue with a high inherent threshold (δ_t) could take over as symptom-presenting if the combination of distensibility ($1/K$) and fluid shift (δ_f) were to reduce the threshold level for the critical insult $(\delta_t - \delta_f)/K$ to a value lower than needed to provoke marginal limb bends in the aqueous connective tissue. Moreover, δ_f is likely to be greatest if shifts due to a switch from helium to nitrogen as the inert gas are superimposed upon pre-existing pressure differentials. Thus the rate of fluid shift would be directly proportional to the osmotic driving force $\sigma S_c(p_i - P_{ai})$ i.e. $\delta_f \propto \sigma S_i(p_i - P_{ai})$ where P_{ai} is the arterial inert gas partial pressure. If, therefore, the proportionality constant is (K'), then for decompression from a steady state at an absolute pressure at P_1 to P_2 breathing the same mix (inert gas fraction F_i):

$$P_1 = P_2 + (\delta_f/K' \sigma S_c F_i) \quad (91)$$

Thus, on a plot of P_1 versus P_2 , such a tissue would have a lower gradient (1.0) yet an appreciably higher intercept ($\delta_f/K' \sigma S_c F_i$) than the connective tissue for limb bends. A physiological system particularly sensitive to internal fluid shift is the vestibular apparatus. Hence it is very interesting that Equation 91 gives a straight line which would intersect the limb bends line (Equation 27) in just about the same manner as found for P_1 versus P_2 plots for 'vestibular' and 'limb' bends in practice (fig. 20). Moreover, the gradient of unity is particularly compatible with the critical insult arising from an incompressible fluid—as seen in Equation 91.

If this explanation is correct, then it is tempting to speculate on the use of osmotic agents and mild diuretics during decompression. In fact, this could be a factor contributing to the successful prescription of alcohol to his divers by Krasberg (1976), particularly when given at the start of decompression from 1000 fsw and therefore most effective over the deeper stages where vestibular problems are more likely.

p. 103

p. 70

Kinetic factors

To return to normal exposure pressures, there are a number of features of the model not expressed mathematically yet which need appraisal. Most of these involve time.

The first is the random occurrence of symptoms (p. 32). For this to occur there must be a random physical or physiological mechanism—and what could be more appropriate than the random nature of nucleation (Chapter 4), particularly of the gas phase by decompression (p. 84). This is expressed quantitatively in Equations 86 and 87 that no gas phase or no bends will occur if the respective thresholds for nucleation or pain are not exceeded but this does not state that the converse need apply. If the right-hand sides of Equations 86 and 87 exceed the respective left-hand sides, then there are only certain probabilities that a nucleus will be activated into growth or that nucleation will be so profuse that the 'worst possible case' will actually occur on that occasion.

The random onset times of symptoms (p. 33) are highly compatible with the concept that the induction period represents the time needed for separated gas to coalesce until the combined mechanical stress of the congregated gas exceeds the local pain threshold. Moreover, exercise hastens onset (p. 45) and can be envisaged as the ideal motion for coalescing gas deposited in the sheaths of any connective tissue. Also, the more that the decompression exceeds marginal limits, the more gas separates and hence less coalescence is needed for the increasing level of insult to exceed the threshold. Thus the model can explain why severe cases have shorter onset times (p. 34).

Another action likely to promote coalescence is alternate expansion and contraction of bubbles. Hence the lower bends altitude found on repetitive aerial exposures (p. 39) can be explained by the need for less gas to separate from solution in the first instance, when the congregating mechanism is more effective. Similar arguments can explain apparent anomalies in decompression needed for repetitive dives (p. 162).

Coalescence can also explain why a delay in

recompressing a bends case can unduly prolong the treatment needed, while it also offers a simple explanation for the change in the X-ray of the subject's soft tissues. Postulated as the process delaying the onset of symptoms, coalescence could also be the factor rendering surface decompression/decanting feasible. The earlier appearance of symptoms on helium diving can be attributed to congregation by the faster rate at which the larger of two adjacent bubbles grows at the expense of the smaller (p. 96). A minor criticism of the model concerns the virtually instant 'dumping' of gas on decompression assumed for the sake of mathematical simplicity. It is quite likely that growth will prove to be a rate-contributing factor but probably not as important as coalescence (p. 96). However, the introduction of growth terms immensely complicates Equation 87 and has yet to be justified, since it is much more important to predict *whether* bends will occur rather than *when*.

Exercise and temperature

Exercise 'on the bottom' must increase the blood perfusion rate (\dot{Q}) and hence the gas taken up by the 'cellular' zone (q) and, in turn, C_i in Equation 87; so that bends become more likely on subsequent decompression.

Moreover, the increased gas uptake reflected by the decrease from 35 to 25 min in safe 'bottom' time at 150 fsw for no-stop air decompression (p. 46) is just about what would be expected from the increased perfusion and vasodilation of the *tendon* to which the exercising muscle is connected. The effect is much less than any anticipated for muscle itself. When it occurs, the bends pain is felt around the joint selectively exercised (p. 33) and in sites where there are tendons, among other connective tissues. Similarly, exercise during any pre-oxygenation will tend to accelerate nitrogen wash-out, reducing C_i and so tending to protect aviators against decompression sickness (p. 46).

By conventional reasoning, one might expect exercise during decompression to hasten inert gas wash-out and so reduce the likelihood of symptoms but the reverse is found to hold

in practice (p. 46)—at least using U.S. Navy tables. By the 'thermodynamic' approach exercise *during* decompression would act in two opposing ways: firstly to coalesce gas already separated from solution into a pain-provoking bubble; secondly, to increase blood flow ($\dot{Q} \uparrow$ in Equation 85) and vasodilation ($A' \uparrow$ in Equation 84) tending to eliminate what has remained in solution. This writer therefore contends that the outcome of the race between these processes will depend upon how much gas is 'dumped' and how much remains in solution. Thus a U.S. Navy profile with its characteristic long first 'pull' is likely to precipitate most of the tissue gas allowing coalescence to predominate in hastening eventual symptoms. On the other hand, during a decompression with more time spent deeper and hence more gas remaining in solution, increased wash-out of this gas should win and exercise should prove advantageous—as observed in caisson workers before the 'Haldane' rationale was introduced (p. 46).

Increasing the metabolic rate without the mechanical action of coalescence should decrease tissue P_{O_2} more than it elevates the P_{CO_2} , thus decreasing the value for m (Equation 86) and hence c in Equation 87. The small change in c with time of day is only likely to be significant relative to low values of P , when it can explain the small diurnal effect noticed in aerial decompressions (p. 44).

Temperature is as well explained by the 'thermodynamic' as by any other model. Apart from the obvious thermal effects on vasoconstriction and hence gas exchange in the peripheral tissues, there is the increased solubility of gases at lower temperatures. Thus at high pressures, where the thermal capacity of the inspired gas is significant relative to the pulmonary circulation, there could be slight cooling of blood in the lung resulting in a higher gas uptake. This could lead to a small amount of supersaturation as arterial blood returns to core temperature. This, in turn, would have the net effect of increasing the tissue inert gas content (C_i in Equation 87) and hence the likelihood of decompression sickness. This line of reasoning adds further

justification for warming the breathing mix at greater depths.

Critical features

So far, the 'thermodynamic' approach has been assessed in the light of practical experience (Chapter 2) rather than on the results of experiments specifically designed to test certain facets of the model. These have already been described in detail in Chapters 3, 6 and 7 and their compatibility requires no further discussion, since the same conclusions were used in deriving the model originally.

Many of these critical tests centred around the inherent unsaturation and its ability to provide a driving force for inert gas elimination. One of the more practical cases which the model can interpret is the lesser advantage to be gained by undertaking pre-oxygenation at altitudes which can precipitate sub-symptomatic bubbles (p. 37). It also explains the great advantage gained by the use of oxygen and high pressure ($P \uparrow$) in treatments ($\Delta P_{N_2} \uparrow \uparrow$ as $F_i \rightarrow 0$ or $P \uparrow$ in Equation 52).

p. 138

Quantitative Assessment

In addition to offering explanations for some basic facts, the qualitative assessment has indicated how the analytical description of the model is also compatible. In other words, the equations predict changes in the right direction. However, the next step is to determine whether those correctly predicted changes are of the right magnitude. Perhaps the simplest order for undertaking this more rigorous appraisal is to look first at the relationships between the key variables, then to see what agreement there is between various values derived for the constants and, finally, to compare the values which the decompression data would indicate for basic tissue parameters with known values.

Apparent decompression ratio

The first characteristic to explain is why a constant decompression ratio (P_1/P_2) appears to hold for simple air diving up to 300 fsw or,

p. 119 rather, a linear relationship between P_1 and P_2 (fig. 35). Just such a linear relationship was derived on p. 120 (Equation 43) on the basis that a constant volume fraction of separated gas (v in Equation 28) is responsible for marginal limb bends. This same equation (28) forms the basis for the comprehensive expression for the imminence of bends (Equation 87) after relating v to the critical pain threshold (δ_c). Moreover, the constants in these equations are compatible if the tissue is predominantly aqueous or, at least, contains up to 3% lipid (p. 194).

p. 103
p. 246 The derivation of these constants leads to the second set of data needing correlation by any comprehensive expression for the imminence of limb bends; the comparison of the minimum bends depth for air, the same for helium: oxygen and the minimum bends altitude for aerial decompression. It was shown on p. 193 how these can be correlated exactly if the volume fraction of separated gas ($v = 0.0045$) and the constant (c) has a value of 5.1 fsw for the marginal onset of bends. Thus, from Equations 64 and 80, a value for the combined mechanical contribution (B) of interfacial tension and tissue compliance can be derived:

$$B \leq 10.2 \text{ fsw} \quad (92)$$

This value will be related to more fundamental constants later (p. 253).

The third basic feature of decompression data concerns the remarkable adherence of bounce dives to the \sqrt{t} relationship (p. 122). Thus, for a single exposure, $C_i \propto \sqrt{t}$ in Equation 87. It was discussed earlier (p. 189) how uptake by diffusion into a bulk of almost any reasonable shape follows the \sqrt{t} relationship for small values of t . This is compatible with the predominantly bulk diffusion aspect of the 'thermodynamic' model.

Constants

p. 246 Another check of the analytical description of the model is provided by the point of inception of the gas phase in tissue. Mathematically, this is described by Equation 86 which gives the absolute pressure for aerial phase separation

as $0.8(760 - 47) - B' + m$. When compared with altitudes of 10,000–12,500 feet ($P = 474$ –533 mm Hg) for the first changes in the X-rays or in cerebrospinal fluid volumes, this gives $B' > 202$ mm Hg (8.8 fsw), a result compatible with the earlier estimate of B from minimum bends depths as 234 mm Hg (10.2 fsw).

Yet another crude estimate of B can be obtained from the analysis of bubbles formed by decompression (Harris *et al.*, 1945b). In exercising animals ($P_{O_2} \approx 0$; $P_{CO_2} \approx 50$ mm Hg) these contained 95% nitrogen on a dry gas basis at normal pressure:

$$\frac{(\text{nitrogen})}{(\text{total gas})} = \frac{(760 + B - 50 - 47)}{(760 + B - 47)} = 0.95 \quad (93)$$

which gives the mechanical 'overpressure' of the bubble (B) as 287 mm Hg. This estimate is crude, since a resting value of $P_{CO_2} = 46$ mm Hg gives $B = 207$ mm Hg but the range encompasses the earlier estimate of B .

Relation to fundamental parameters

It could be argued that B (or B' before a nucleus is activated) is simply part of a 'fiddle factor', c , needed to make the data fit the comprehensive expression for the imminence of symptoms (Equation 87) and its forerunners (Equations 26 and 28). Of the two terms m and B contributing to c , there can be little argument that 5.1 fsw (117 mm Hg) is a reasonable maximum value for total gas tensions of the metabolic gases (m in Equation 80). However, this leaves B for which the most comprehensive analysis (p. 194) gives $c = 5.1$ fsw (117 mm Hg) and hence $B = 234$ mm Hg. If B has the fundamental significance claimed, then it should give reasonable values for the fundamental physiological parameters δ_g and $2\gamma/r_b$ as defined in Equation 14. Taking δ_g as 11–26 mm Hg for no fluid shift ($\delta_f = 0$ in Equation 7), this gives a value of 208–223 mm Hg for $2\gamma/r_b$. Taking 50 dyne cm^{-1} for plasma as the value for γ , this gives a bubble radius (r_b) of 3.5 μm , i.e. a diameter of 7.0 μm , which would seem eminently reasonable by comparison with capillary diameters of 8.0 μm and intercapillary distances of the order of 20–40 μm .

Other mechanical factors

The next question in assessing whether the numbers are fundamentally consistent with the model concerns the stress which such a bubble would create. For no fluid shift ($\delta_f = 0$) and a pain-provoking stress of 11–26 mm Hg (Equation 7), together with a critical bends volume fraction of separated gas of $v = 0.0048$, Equation 9 gives a bulk modulus for tissue distension (K) of 3.7×10^4 dyne cm^{-2} . This should not be compared with Young's modulus for unidirectional stress in tendon, nor with values for compression which is largely measuring the compressibility of water but with those for bulk displacement. This is compatible with values of $2\text{--}11 \times 10^4$ dyne cm^{-2} found for connective tissue (Harkness and Harkness, 1965).

Diving data

So far the quantitative analysis has not involved time. Hence, in analysing data for practical dives which have actually been performed, it is necessary to select values for (D/a^2) and (b/a) . The computations are too lengthy to repeat here but thirteen sets of naval dives have been analysed by the 'thermodynamic' approach (Hills, 1966), to predict the bends cases out of hundreds of exposures involving at least fifty different decompression profiles. Most were formulated by conventional calculation methods (e.g. the data of Crocker, 1957). The constants which enabled such a successful analysis to be made were $(D_c/a^2) = 0.129 \text{ min}^{-1}$ and $b/a = 5.29$ for rest to 4.73 for exercise—all for an annular model ($\mu = 0.47$). The value of (D_c/a^2) agrees well with values for other 'transient' determinations (Table 12) while, taking $8.0 \mu\text{m}$ as the capillary diameter ($2a$), the (b/a) ratios give a fibre diameter $2(b-a)$ of the order $32 \mu\text{m}$, well within the range for tendon of $10\text{--}40 \mu\text{m}$ in skin (Bear, 1952; Gustavson, 1956) and sometimes up to $300 \mu\text{m}$ (Bear, 1952; Verzar, 1957).

Allowing the ratio (b/a) to vary provides a convenient mathematical means of accounting for vasodilatation and hence exercise.

As for shape factors, the geometric configura-

tion used in the foregoing analyses gave $\mu = 0.47$ but the first bulk diffusion model of Hempleman (p. 122) was planar ($\mu = 1.0$), while wash-out analysis from a different tissue (skeletal muscle) gave $\mu = 1.55$ (Hills, 1967b). This corresponds to roughly cylindrical fibres in which about 22% of their surface is in contact with their neighbours. Unfortunately this is about the only study which has paid much attention to the *mode* of curvature, if any, of the boundary between the 'cellular' and vascular or 'extended vascular' zones. However, if this value of 1.55 is applied to the resting and working ratios (5.29 and 4.73) in accordance with Equation 90, it gives a volume fraction for the 'extended vascular' zone (β) of 7–9%. This value coincides with 8.0% for the blood volume of man as a whole but because tendon is a relatively avascular tissue, it is greater than blood volume and indicates that 'extended vascular' is an appropriate term for the effectively fully-stirred zone.

Derivations such as these may be dull and arduous but the sensible values which they produce for the various physical and physiological parameters enhances confidence in the fundamental adequacy of the model. It is now feasible to contemplate using this model to *optimize* a decompression and to 'graduate' from testing its viability on data which often included decompressions whose formulation would appear far from optimal by 'thermodynamic' reasoning. However, although tedious, this is much more cost-effective than jumping straight into formulating a new book of tables only to discover any major errors at great expense in the field—a practice all too common.

Decompression Optimization

Perspective

Whatever model or calculation method one may adopt for optimizing decompression, the designer attempts to select the depth at which the rate of inert gas elimination from the critical area is a maximum at each particular time. It is inefficient to proceed towards the surface

too slowly and yet if one attempts to 'beat the system', then there is a penalty to be paid later. By conventional 'supersaturation' approaches, this penalty is simply that bends will occur if one violates a 'trigger point' for bubble formation. However, if the 'thermodynamic' model is correct and the primary event does not coincide with the critical level of insult then, simply by waiting for bends to occur, the designer can be fooled into paying the penalty of over-decompression without realizing it. Hence the U.S. Navy, among others, (Chapter 5) probably far exceed the conditions for initiating the primary event (Equation 86) and yet do not appreciate the fall in gas elimination rate from the critical area unless they exceed it by such a margin that the volume of separated gas reaches the pain threshold (Equation 87). Once again, there is no point in measuring overall gas elimination rates, even from one tissue, since these reflect the behaviour of the statistical mean and not the 'worst possible' case.

It is all too easy to criticize other approaches but how can the expressions for predicting the primary event (Equation 86) and for estimating the level of insult (Equation 87) be used for optimization, assuming them to be correct?

Sequence of gas elimination and 'dumping'

The no-stop decompression limits leave no doubt that, on return to the surface, man can tolerate a gas content of his critical tissue well in excess of normal and yet not experience symptoms of decompression sickness. Moreover, much of the gas taken up at depth can be 'dumped' into the gaseous phase and yet remain asymptomatic under normobaric conditions. Hence the gas assimilated at depth which exceeds the quantity which can be tolerated at the surface needs to be removed over the depth range affording the greatest driving force for its elimination. In this way part of the total pressure change in returning to the surface is used for 'dumping' gas and part for eliminating it by gradual decompression. This leads to three alternatives:

(a) 'dump' and then eliminate;

(b) eliminate and then 'dump' the rest;

(c) do both simultaneously.

The last of these is simply a compromise between the first two and will not be considered in the basic argument. Taking the first approach, a long first 'pull' towards the surface will 'dump' much gas initially and then bring about the position of needing to use Equation 52 (appropriate to a gaseous phase) to estimate the subsequent elimination over the remainder of the decompression at the graded rate. However, this equation (52) indicates that the driving force is greater at greater pressure ($\Delta P_{N_2} \uparrow$ as $P \uparrow$). Therefore it has always been the contention of this writer (Hills, 1966) that it is better to reverse the conventional procedure (1) and eliminate the excess gas at the greater depths, where there is more driving force, followed by a rapid decompression over the last 20–30 fsw to 'dump' the remainder. This procedure (2) still forms a gaseous phase but to an extent just below the dimensions predicted to give pain (Equation 87). According to 'thermodynamic' reasoning, the U.S.N. tables represent an extreme form of sequence (1) since, in molecular terms, they initially 'dump' more gas than could be tolerated at the surface. Hence much of the gas to be eliminated must be derived from the gas 'dump' and therefore needs to be first resorbed from the gaseous phase, thus adding a further potential resistance to the kinetics in addition to reducing the driving force. This becomes more acute if coalescence has occurred between 'dumping' and resorption.

Hence, by 'thermodynamic' criteria, it is better to follow sequence (2). However, in advocating deeper stops for the pressure range for gradual decompression, just how deep should these start?

Criterion for optimum

Consider a subject making an arbitrary first 'pull' towards the surface following completion of his exposure on the bottom. If he is decompressed so far as to form the gaseous phase, then the appropriate expression (Equation 52) reveals that he would have achieved a better elimination

p. 137
rate at the first stop if he had made that stop rather deeper ($\Delta P_{N_2} \uparrow$ as $P \uparrow$). On the other hand, if he decompresses only a short distance and does not form the gaseous phase, then the expression appropriate to this case (Equation 51) reveals that a better driving force would have been achieved by coming up further ($\Delta P_{N_2} \uparrow$ as $P \downarrow$).

By repeating this exercise for successively deeper first stops *with* the gas phase forming and successively shallower first stops *without* it forming, the two come together to give a maximum ΔP_{N_2} for the unique point where the gaseous phase is just on the brink of inception. Thus the optimal condition is expressed by Equation 86 describing the primary event. Hence the diver rapidly decompresses by the inherent unsaturation plus the mechanical factors (B'), compressing gas in the unactivated nucleus (fig. 31). He then continues to follow the critical condition for initiating the primary event (i.e. gas inception) as this gradient continues to provide the maximum driving force for eliminating the excess gas, all of it just remaining in solution if the calculations are correct.

Since the particular transport model considered relevant to the kinetics of the critical tissue involves a bulk 'cellular' phase, this optimum condition (Equation 86) needs to be applied at all points. Thus it is first necessary to determine the point of maximum gas content and then ensure that this peak does not exceed the critical condition for initiating the primary event. For a single exposure other than steady state, the highest total tension of all will be located in the 'extended vascular' pool and the immediately adjacent wall. Thus the first stop is based on venous gas tensions only. However, as gradual decompression proceeds, the peak will gradually recede from this wall deeper into the tissue. If the overall gas content has not been reduced to a level at which it is safe to jump to the surface sooner, then the peak will recede to the 'back wall' and stay there. This is the point at which the decompressions for short exposures ('bounce' dives) join the universal profile found for starting with a steady-state condition (the 'saturation' curve).

Procedure

This line of reasoning has led to the following procedure for decompression optimization according to the 'thermodynamic' approach.

(1) Calculate the pressure (P_1) to which the diver could be rapidly decompressed from his bottom pressure (P_b) without precipitating the primary event in venous blood and hence in the 'extended vascular' zone. Applying Equation 86 to this case:

$$P_1 > p_{vi} + m - B' = F_i P_b - c \quad (94)$$

where $c = B' - m' = B' - m + F_i P_w$ (Equations 27 and 64).

103, 134

(2) Start the decompression at a rate of 20 fsw min^{-1} from P_b to the value of P_1 calculated in Equation 93 or to the next deepest 10 fsw depth interval if $(P_1 - 33)$ does not prove to be a whole number of tens of feet. Call the pressure corresponding to this depth P'_1 .

(3) At one-minute intervals from the start of decompression, calculate the total gas tension ($p + m$) in venous blood and at each of twelve points in the bulk 'cellular' phase equally spaced between the interface ($r = a$) and the back-wall ($r = b$), using the kinetic equations (81–85) for the model (fig. 84). The input is the arterial inert gas tension (p_a in Equation 85) which will vary with the continuously decreasing absolute pressure according to Equation 2.

(4) At each of these one-minute intervals, determine which of these points has the highest inert gas tension (p_{\max}).

(5) Apply the critical gas inception equation (86) to this peak value, i.e. putting $p = p_{\max}$ to determine the new absolute pressure P to which it is safe to decompress (fig. 84).

(6) If this new value of P is greater than $(P'_1 - 10)$ by the time the diver reaches P'_1 , then he must stop at P'_1 and steps (3) to (5) must be repeated at one-minute intervals until the peak (p_{\max}) has subsided to the extent that

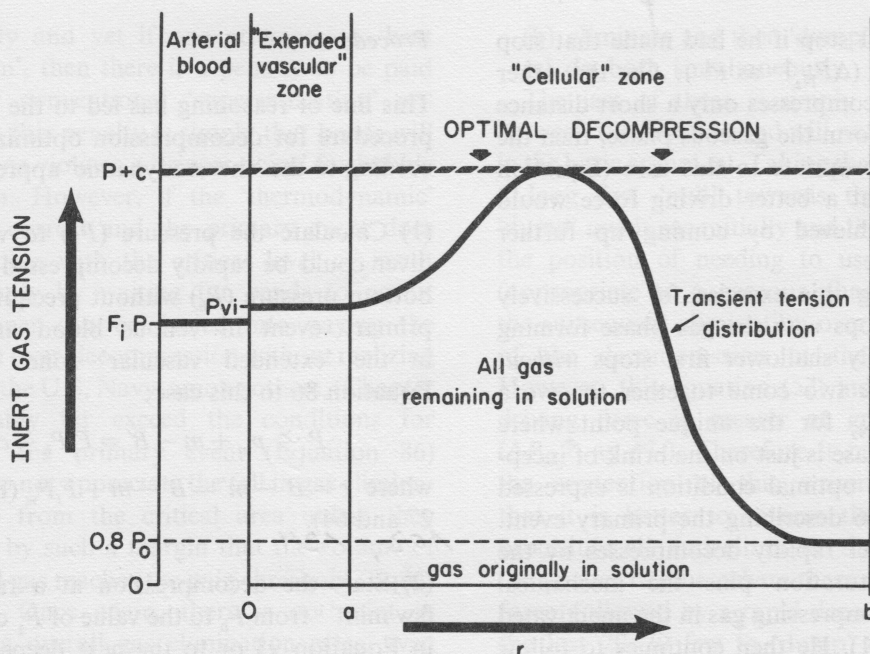


Fig. 84 Depicting the condition for optimal decompression according to the 'thermodynamic' method. At any instant the absolute pressure is selected which causes the peak gas tension to coincide with the point of phase equilibrium, i.e. just keeping all gas in true physical solution. It is convenient to work in terms of inert gas when other gases and mechanical factors can be 'lumped' into one minor constant (c), where $c = B - m' = \delta_g + (2\gamma/r_n) - P_{vO_2} - P_{vCO_2} - (1 - F_{IN_2})P_w$. Redrawn from Hills (1966)

this value enables Equation 94 to be satisfied for $P = P'_1 - 10$. The diver is then moved 10 fsw closer to the surface—or a greater number of 10-foot intervals, provided the peak has subsided sufficiently for the new absolute pressure (P) not to violate Equation 94.

(7) The change in arterial inert gas tension (p_a) resulting from this change in absolute pressure is fed into the 'kinetic' component of the computation via Equation 85.

(8) If P'_1 is greater than the $(p_{\max} - 10)$ value calculated from the peak by the time the diver reaches P'_1 , then he need not stop at P'_1 but can continue decompressing at the same rate until recalculations of the peak, in the light of his continuing change in arterial gas tension, show that he would violate Equation 94.

(9) When his depth becomes 40 fsw or less, then the same distribution data computed on

the whole dive history to estimate the peak is also used to calculate the imminence of decompression sickness as though that diver had been decompressed from that depth to the surface, i.e. Equation 87 is used for $P = 33$ fsw.

(10) Continue the gradual decompression by repeating steps (3) to (7) using Equation 94 as the criterion until an absolute pressure (P_s) is reached at which (9) shows that Equation 87 would not be violated if the diver decompressed from P_s to 33 fsw.

(11) When this occurs, decompress from this surfacing pressure (P_s) to the surface at 20 fsw min^{-1} .

Variations

Many variations on this theme are possible. One-minute intervals are used for updating

the computation because this is approximately the circulation time of blood in the body. The decompression rate of 20 fsw min^{-1} is selected for the two rapid phases of decompression ((2) and (11)) to avoid supersaturating arterial blood (see p. 65).

Since P_s usually lies in the region of 53–63 fsw (20–30 feet depth), it is convenient to put in stops at 35 feet and 25 feet to improve efficiency. This does not add any inconvenience because the very shallow stops at 10 and 20 feet found in conventional tables are now omitted.

Computer programs

The expressions describing gas transfer in the 'thermodynamic' model (Equation 82), together with the difficulty of eliminating such variables as q by simple substitution, must leave the initial impression of great complexity in any calculation involving them. Their usage would certainly be tedious if it were not for the ease with which they can be programmed for the digital computers so readily available today. Hence much of the old fear of the mathematics in invoking any transport system more complex than a simple 'Haldane tissue' is no longer justified.

However, it must still be remembered that no profile is better than the program fed into the computer and extreme care must be taken to ensure that the program is completely 'debugged'. In preparing this, the sequence of calculation steps ((1) to (11)) listed in the preceding section provides a convenient outline for the underlying logic diagram. There are basically two approaches to programming this model: the analytical approach and numerical methods involving finite difference techniques commonly used in solving many engineering problems concerned with heat transfer.

This technique has enabled the thermodynamic model to be simulated by a number of compartments in series, the first representing the 'extended vascular' zone and the remainder depicting segments of the bulk 'cellular' phase. Thus the first of the 'nodes', or boundaries between these 'tanks', is at arterial gas tension,

while the last represents the 'back wall'. By the finite difference method, the rate of transfer between any two compartments is directly proportional to the tension difference between them. The proportionality constant of the first is chosen to reflect the blood perfusion rate while the rest are determined by the shape of the 'cellular' zone. Each compartment is also assigned a capacity proportional to its ability to store dissolved gas. This program is therefore somewhat flexible in allowing the designer to select his blood perfusion rate (\dot{Q}), fraction of the 'extended vascular' zone (β), the mode of curvature (μ) and relative dimensions (b/a) of the bulk 'cellular' phase.

Confidence in the technique was gained from the agreement between the profiles produced by this program and one devised by an analytical method using Legendre quadrature to describe the particular case of a purely diffusion-limited radial system (Hills *et al.*, 1976). Although the finite-difference approach is particularly versatile, great care must be exercised in selecting the coarseness of the elements to prevent the programme from 'going critical'. This also applies to time intervals. However, the agreement between the profiles generated by this technique and those produced by a thermal analogue (fig. 85) and another by the analytical approach certainly allayed the inherent suspicions which this writer, at least, had of computers and their programmers.

Profiles

The type of profile which the 'thermodynamic' procedure produces is shown in fig. 85, where it is compared with a U.S. Navy air table in which total decompression time has been similarly titrated on both to give an equal bends incidence on large goats (Hills, 1966). The two major differences are the 'drop out' from 25 feet and the much deeper initial stops, together with an overall shift of time towards the deeper end of the decompression. Hence the calculation procedure is actually producing the type of format anticipated from the thermodynamic model, viz.

(a) more time spent deeper to avoid gas

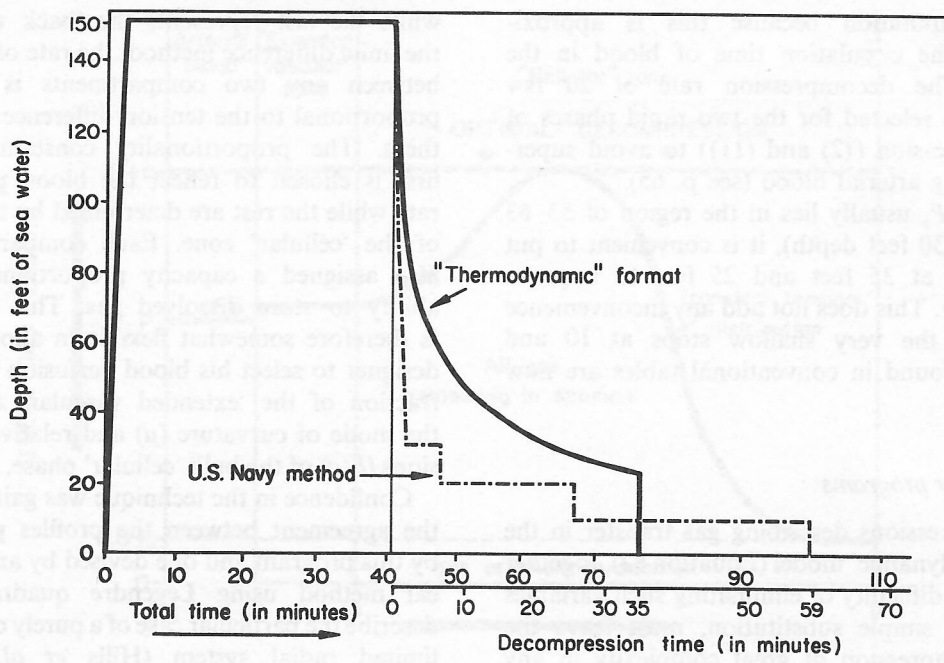


Fig. 85 A typical profile produced by the 'thermodynamic' approach and compared with a U.S. Navy profile for the same air exposure, both schedules being cut off at the total decompression time for equal bends incidence on the same goats. Redrawn from Hills (1966)

phase inception and hence giving a greater driving force to remove the excess gas, followed by

(b) a 'drop out' from 20–30 fsw to 'dump' the remainder of the gas as a sub-symptomatic gas phase.

It is over this last stage that the diver on a 'thermodynamic' profile overtakes those following more conventional calculation methods to result in a table more economical in overall decompression time for the same apparent safety. However, before discussing the success or otherwise found by using this method in practice, nothing has been said of the breathing mix to use.

Dual optimization

At each time in a decompression, there are two parameters for which the designer is free to choose values, i.e. there are two degrees of freedom. These are the depth and the composition of the breathing mix—the fraction of

oxygen in particular. Consequently, to perform a simultaneous optimization of both depth and composition against time, two constraints are needed and these are provided by decompression sickness and oxygen toxicity. Thus the diver returning from the optimal decompression would be within known, yet safe, margins of both developing the bends and displaying the effects of oxygen poisoning.

If there is the facility to maintain a constant inspired P_{O_2} during the dive, then the highest value should be selected from the oxygen dose–time curve consistent with a reasonable margin of safety, e.g. using the U.S. Navy limits as shown in fig. 76. Normally this is not possible and it is seldom feasible to use more than four gas mixes during the entire exposure. However, it is quite practicable to alternate between any two mixes by filling the chamber with one and telling the diver when to breathe the other on BIBS. Thus in a dive to 500 fsw, the diver could breathe 7% oxygen mixed with predominantly helium on the

bottom, 14% oxygen from 300–350 fsw to 120–140 fsw, air to 40–60 fsw and then alternate between air and pure oxygen up to the drop-out depth.

In a particularly long dive, any sign of chronic oxygen toxicity should be used to switch to a subtoxic mixture. However, in shorter dives, the advantages to be gained from a significantly higher inspired P_{O_2} could result in convulsions arising too suddenly to prevent by evasive action. Hence the cumulative oxygen toxicity index (COTi on p. 226) has been programmed for continuous updating along with the total UPTDs on a separate computer program (Hills *et al.*, 1976).

This programme is used to compute the COTi for the thermodynamic program and at the same one-minute intervals. It is also used to compute another index: $CO(T + 10)i$. This is the index for the same history but ahead by 10 min, as though the diver were breathing the next more toxic mix for the whole of that period at that pressure. This continually up-

dated $CO(T + 10)i$ is used as a 'trigger' for the decompression program. It has no effect until its value falls below 1.0, which is the signal that it would be safe for the subject to switch to the mix with the next highest oxygen fraction for the next 10 min. In doing so, the new fraction of inert gas is fed into the decompression program. Upon return to the less toxic mix, the process is repeated so that the two programs, one for bends and the other acute oxygen toxicity, are effectively integrated. The interval 'on' the more toxic mix can be varied, but an arbitrary value of 10 min has been selected as a convenient time for the diver to be on BIBS without the annoyance of switching too often.

However plausible these optimizations may sound academically, are they doing what is intended?

Animal trials

Many trials on goats and kangaroo rats of the

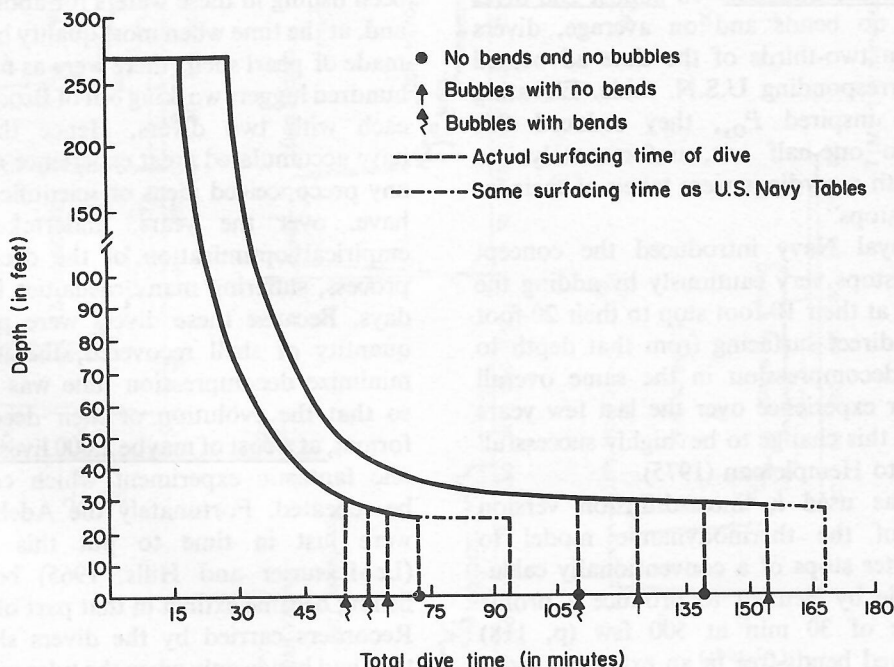


Fig. 86 Results of using the Doppler meter to monitor goats during decompressions following two thermodynamic profiles. Bubbles were never detected before surfacing while, on the U.S. Navy schedules for the same air exposures, asymptomatic bubbles were detected as early as the 40-foot stop. Data from Vann *et al.* (1973)

form shown in fig. 86 have shown that the 'thermodynamic' approach offers shorter decompression times; but is the body really conforming to the model? It is particularly difficult to detect bubbles *in vivo* for the reasons already outlined but if venous bubbles detected by the Doppler technique are at all meaningful, then it is interesting that none were detected in goats using a profile computed by Vann and Hills (Vann *et al.*, 1973) until after the drop-out (fig. 86). Moreover, those detected on the surface were asymptomatic, while U.S. Navy tables tested on the same animals *did* produce bubbles at depth.

Human experience

The greatest difference between thermodynamically and conventionally computed tables is seen in short, deep dives. Hence the first trials to test this method used tables computed on this model by Dr. Walter Starck and used by him and his crew for short deep air dives of up to 300 fsw off the Bahamas. In almost 200 dives they had no bends and on average, divers surfaced in two-thirds of the time advocated by the corresponding U.S.N. table. Elevating the mean inspired P_{O_2} , they reduced this fraction to one-half but, unfortunately, no actual depth recordings were taken of their in-the-water stops.

The Royal Navy introduced the concept of deeper stops very cautiously by adding the time spent at their 10-foot stop to their 20-foot stop with direct surfacing from that depth to complete decompression in the same overall time. Their experience over the last few years has shown this change to be 'highly successful' according to Hempleman (1975).

Vann has used a linear-diffusion version ($\mu = 1.0$) of the thermodynamic model to improve later stops of a conventionally calculated profile by Bennett to produce a profile for a dive of 30 min at 500 fsw (p. 118) which proved bends-free in an extensive series of human chamber trials at Duke University (Bennett and Vann, 1975). However, by far the most extensive series *in the ocean* must be those of Krasberg (comment at 6th Symposium,

Underwater Physiology) who, in over 800 dives for up to one hour and down to 600 feet, recorded only four bends (three Type I) of which several could be attributed to diver error. Moreover, using a linear-diffusion version of the model, his outstanding record of success has now been extended to exposures of up to 800 feet (Krasberg, 1976). Unfortunately, his final tables remain proprietary information.

Pearl divers

Returning to lesser depths and air diving, which is still the major part of the industry, by far the greatest experience of human exposure over the years must have been accumulated by the pearling fleets operating in the deep tidal waters off the northern coast of Australia. They employ many Okinawans who regularly dive to depths of up to 300 feet on air for as long as one hour, usually making two such dives per day, working six days per week and ten months per year. They have been fishing in these waters for about a century and, at the time when most quality buttons were made of pearl shell, there were as many as nine hundred luggers working out of Broome alone—each with two divers. Hence these people have accumulated great experience and without any preconceived ideas or scientific knowledge have, over the years, undertaken a truly empirical optimization of the decompression process, suffering many casualties in the early days. Because these divers were paid by the quantity of shell recovered, the incentive to minimize decompression time was very great, so that the evolution of their decompression format, at a cost of maybe 2,000 lives, represents one fantastic experiment which could never be repeated. Fortunately the Adelaide group were just in time to put this on record (LeMessurier and Hills, 1965) before pearl fishing became extinct in that part of the world. Records carried by the divers showed that they had bends only when the tides were running strongly and their simple use of the length of lifeline as their sole depth indication was badly in error. This brought the diver appreciably closer to the surface than intended

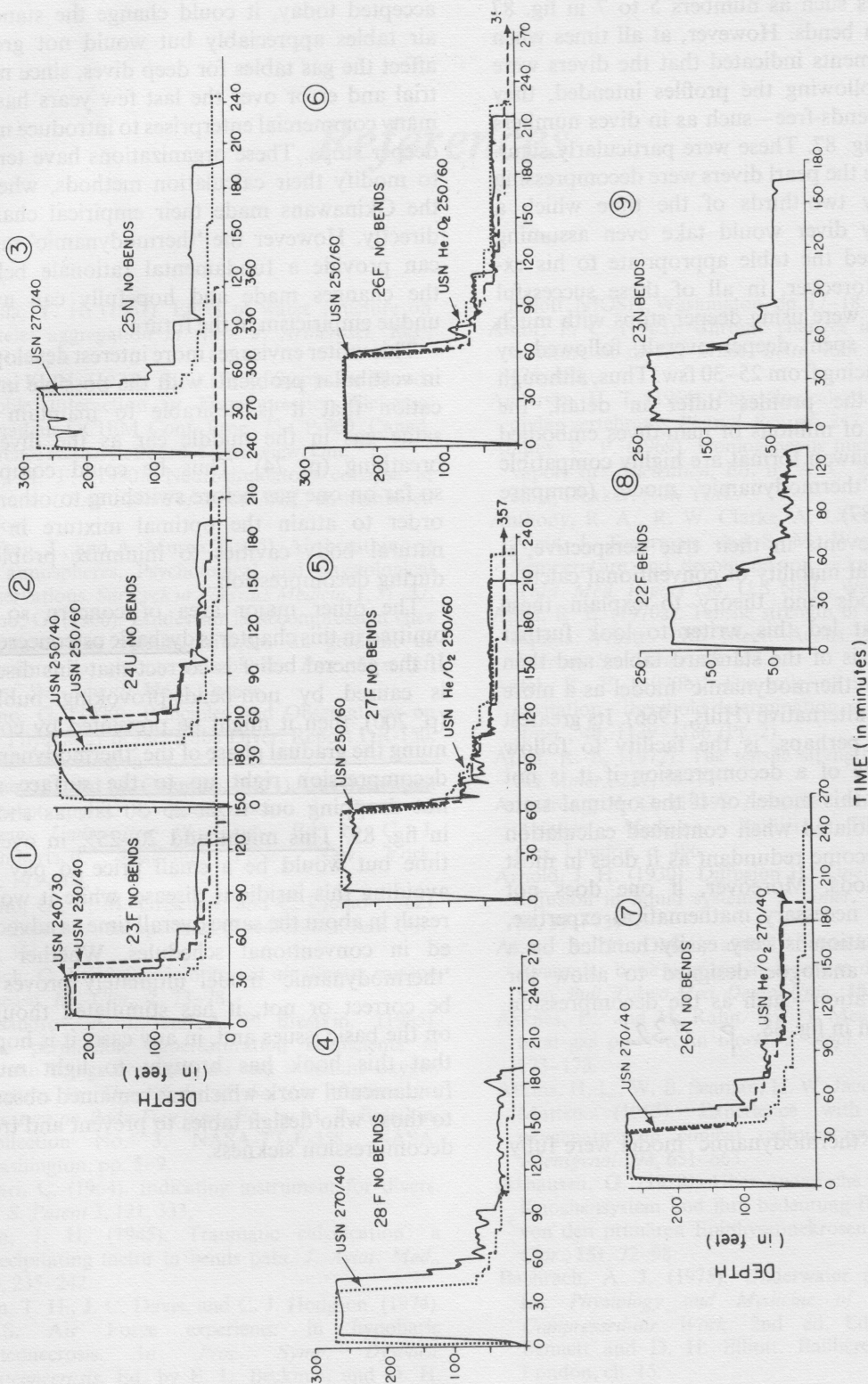


Fig. 87 Actual recordings of decompressions performed by Okinawan pearl divers operating in Australian coastal waters. They have devised these methods purely by trial and error over almost a century with the economic incentive of minimizing total ascent time. Data from LeMessurier and Hills (1965)

when dives such as numbers 5 to 7 in fig. 87 resulted in bends. However, at all times when the instruments indicated that the divers were actually following the profiles intended, they surfaced bends-free—such as in dives numbers 1 to 4 in fig. 87. These were particularly significant since the pearl divers were decompressing in roughly two-thirds of the time which a U.S. Navy diver would take even assuming that he used the table appropriate to his exposure. Moreover, in all of these successful dives, they were using deeper stops with much more time spent deeper overall, followed by direct surfacing from 25–30 fsw. Thus, although many of the profiles differ in detail, the experience of millions of man-dives embodied in the Okinawan format are highly compatible with the ‘thermodynamic’ model (compare figs. 85 to 87).

To put events in their true perspective, it was the total inability of conventional calculation methods and theory to explain these records that led this writer to look further into the basis of the standard tables and then to offer the ‘thermodynamic’ model as a more compatible alternative (Hills, 1966). Its greatest advantage, perhaps, is the facility to follow the outcome of a decompression if it is not optimal by this model or if the optimal state *has* been violated when continued calculation does not become redundant as it does in most other methods. Moreover, if one does not possess the necessary mathematical expertise, the computation is very easily handled by a mechanical analogue designed to allow for phase separation—such as the decompression meter shown in fig. 38.

p. 132

The future

Even if the ‘thermodynamic’ model were fully

accepted today, it could change the standard air tables appreciably but would not greatly affect the gas tables for deep dives, since much trial and error over the last few years has led many commercial enterprises to introduce much deeper stops. These organizations have tended to modify their calculation methods, whereas the Okinawans made their empirical changes directly. However the ‘thermodynamic’ model can provide a fundamental rationale behind the changes made and hopefully can avoid undue empiricism in the future.

This writer envisages more interest developing in vestibular problems with the possible implication that it is desirable to maintain the same gas in the middle ear as the diver is breathing (p. 74). Thus he could compress so far on one gas before switching to others in order to attain the optimal mixture in the natural body cavities to minimize problems during decompression.

The other major area of concern so far omitted in this chapter is dysbaric osteonecrosis. If the general belief is correct that this disease is caused by non-bends-provoking bubbles (p. 200), then it might be prevented by continuing the gradual phase of the ‘thermodynamic’ decompression right up to the surface and not dropping out from 25–30 fsw as shown in fig. 85. This might add 20–25% in overall time but would be a small price to pay for avoiding this insidious disease, while it would result in about the same overall time as advocated in conventional schedules. Whether the ‘thermodynamic’ model ultimately proves to be correct or not, it has stimulated thought on the basic issues and, in any case, it is hoped that this book has brought to light much fundamental work which has remained obscure to those who design tables to prevent and treat decompression sickness.

References

- Abdulla, Y. H. (1967). Effect of water structure on platelet aggregation *in vitro*. *J. Artheroscl. Res.*, **7**, 415-423.
- Ackles, K. N. (1973). (Ed.). Proc. Symp. on Blood-bubble Interaction in Decompression Sickness. Canadian DCIEM Conf. Proc. 73-CP-960. Canad. Defence Research Board, Downsview, Ont.
- Adler, H. F. (1950). Neurocirculatory collapse at altitude. *USAF School of Aviat. Med.*, un-numbered report.
- Adolfson, J., and A. Muren. (1965). Air breathing at 13 atmospheres. Psychological and physiological observations. *Sartryck ur Forsvars Medicin*, **1**, 31-37.
- Albano, G. (1960). Etudes sur la décompression chez l'homme. Les valeurs critiques du gradient de pression a la remonte sans paliers. *Proc. First Int. Conf. Sub-Aquatic Med., Cannes*.
- Albano, G. (1970). Principles and Observations on the Physiology of the Scuba Diver. Report DR-150. ONR, Washington.
- Albano, G., and M. Columba. (1971). Gas nucleation concept applied to decompression. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen, Academic Press, New York, pp. 193-204.
- Albano, G., P. M. Griscoli, and C. Ciulla. (1962). La sindrome neuropsichica di profondità. *Lav. Um.*, **14**, 351-358.
- Aldrich, C. J. (1900). Compressed air illness; caisson disease. *Int. Clin.*, **2**, 73-78.
- Aleksandrov, A. I., and A. P. Brestkin. (1965). The permissible supersaturation coefficients in human beings breathing air and a helium-oxygen mixture. In: *The Effects of the Gas Medium and Pressure on Body Functions*. Ed. by M. P. Brestkin, Collection No. 3, NASA-TT-F-358, NASA, Washington, pp. 5-9.
- Alinari, C. (1964). Indicating instrument for divers. *U. S. Patent* 3, 121, 333.
- Allan, J. H. (1945). Traumatic calcification: a precipitating factor in bends pain. *J. Aviat. Med.*, **16**, 235-242.
- Allen, T. H., J. C. Davis, and C. J. Hodgson. (1974). U.S. Air Force experience in hypobaric osteonecrosis. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 17-18.
- Alnor, P. C. (1963). Chronic changes in the bone structure of divers. *Bruns' Beitr. Klin. Chir.*, **207**, 475-485.
- Andersen, H. T. (1966). Physiological adaptations in diving vertebrates. *Physiol. Rev.*, **46**, 212-243.
- Andres, R. P. (1969). Homogeneous nucleation in a vapor. In: *Nucleation*. Ed. by A. C. Zettlemoyer, ch. 2, Dekker, New York.
- Anthony, R. A., R. W. Clarke, A. Liberman, L. F. Nims, J. Teperman, and S. M. Wesley. (1943). Temperature and decompression sickness. *Comm. Aviat. Med. Report 136*, U.S.NRC, Washington.
- Apfel, R. E. (1970a). Tensile strength of superheated liquids: Abstr. 79th Meet. Acous. Soc. Amer. *J. Acous. Soc. Amer.*, **48**, 115.
- Apfel, R. E. (1970b). The role of impurities in cavitation—threshold determination. *J. Acous. Soc. Amer.*, **48**, 1179-1186 (Pt. 2).
- Apfel, R. E. (1972). The tensile strength of liquids. *Sci. Amer.*, **227**, 58-71.
- Armstrong, H. G. (1939). *Principles and Practice of Aviation Medicine*. Baillière, Tindall and Cox, London, p. 496.
- Arnold, J. H. (1930). Diffusion II. Kinetic theory of diffusion in liquid systems. *J. Amer. Chem. Soc.*, **52**, 3937-3942.
- Asahi, S., H. Ohiwa, and I. Nashimoto. (1968). Avascular bone necrosis in Japanese diving fishermen. *Bull. Tokyo Med. Dental Univ.*, **15**, 247-257.
- Asknes, E., and H. Rahn. (1957). Measurement of total gas pressure in blood. *J. Appl. Physiol.*, **10**, 173-178.
- Atkins, H. L., W. B. Seaman, H. W. Jacox, and R. S. Matteo. (1965). Experience with hyperbaric oxygenation in clinical radiotherapy. *Amer. J. Roentgenol.*, **93**, 651-663.
- Axhausen, G. (1928). Über anamische infarkte am Knochensystem und ihre bedeutung für die Lehre von den primären Epiphyseonekrosen. *Arch. Klin. Chir.*, **151**, 72-98.
- Bachrach, A. J. (1975). Underwater performance. In: *Physiology and Medicine of Diving and Compressed-air Work*. 2nd ed. Ed. by P. B. Bennett and D. H. Elliott. Baillière & Tindall, London, ch. 15.

- Baldin, U. I. (1973). Effects of ambient temperature and body position on tissue nitrogen elimination in man. *Aerospace Med.*, **44**, 365-370.
- Baldin, U. I. (1976). The effects of body position and a vasodilator on xenon¹³³—elimination from human subcutaneous fat. In: *Undersea Biomedical Research*. Undersea Medical Society, Washington (in press).
- Barcroft, H. (1963). Circulation in skeletal muscle. In: *Handbook of Physiology*. Sect. II, vol. 2, Ed. by W. F. Hamilton. Amer. Physiol. Soc., Washington, pp. 1353-1438.
- Barcroft, J. (1914). *Respiratory Function of Blood*. Cambridge University Press, Cambridge.
- Barcroft, J. (1925). *Lessons from High Altitudes*, vol. 1, Cambridge University Press, Cambridge.
- Barlow, T. E., A. L. Haigh, and D. N. Walder. (1959). Dual circulation in skeletal muscle. *J. Physiol. (London)*, **149**, 18-19.
- Barlow, T. E., A. L. Haigh, and D. N. Walder. (1961). Evidence for two vascular pathways in skeletal muscle. *Clin. Sci.*, **20**, 367-385.
- Barnard, E. E. P. (1975). Fundamental studies in decompression from steady state exposures. In: *Proc. Vth Symp. Underwater Physiology*. Fed. Amer. Socs. Exp. Biol., Washington.
- Barnard, E. E. P., J. M. Hanson, M. A. Rawton-Lee, A. G. Morgan, A. Polak, and D. R. Tidy. (1966). Post decompression shock due to extravasation of plasma. *Br. Med. J.*, **2**, 154-155.
- Barnard, E. E. P., R. de G. Hanson, B. J. Reid, and J. Williams. (1973). Studies in nitrogen elimination. *Swedish J. Defence Med.*, **9**, 496-501.
- Barnes, R. (1967). Surgical treatment of bone lesions in compressed air workers. In: *Decompression of Compressed Air Workers in Civil Engineering*. Ed. by R. I. McCallum. Oriel Press, Newcastle upon Tyne.
- Bartels, H., R. Beer, E. Fleischer, H. J. Hoffheinz, J. Krall, G. Rodewald, J. Wenner, and I. Witt. (1955a). Bestimmung von Kurzschlussdurchblutung und Diffusionskapazität der Lunge bei Gesunden und Lungenkranken. *Pflügers Arch. ges. Physiol.*, **261**, 99.
- Bartels, H., R. Beer, H. P. Koepchen, J. Wenner, and I. Witt. (1955b). Messung der alveolar-arteriellen O₂-Druckdifferenz mit verschiedenen Methoden am Menschen bei Ruhe und Arbeit. *Pflügers Arch. ges. Physiol.*, **261**, 133.
- Barthelemy, L. (1963). Blood coagulation and chemistry during experimental dives and the treatment of diving accidents with heparin. In: *Proc. 2nd Symp. Underwater Physiol.* Ed. by C. J. Lambertsen and L. J. Greenbaum, Publ. 1181. U.S. Nat. Acad. Sci. NRC, Washington, pp. 46-56.
- Bateman, J. B. (1951). Review of data on value of pre-oxygenation in prevention of decompression sickness. In: *Decompression Sickness*. Ed. by J. F. Fulton, Saunders, Philadelphia, ch. 9 (Pt. I).
- Bateman, J. B., and J. Lang. (1945). Formation and growth of bubbles in aqueous solutions. *Canad. J. Res.*, **E23**, 22-31.
- Bean, J. W. (1950). Tensional changes of alveolar gas in reactions to rapid compression and decompression and question of nitrogen narcosis. *Amer. J. Physiol.*, **161**, 417-425.
- Bean, J. W. (1951). Adrenal alteration induced by oxygen at high pressure. *Proc. Fedn. Amer. Socs. Exp. Biol.*, **10**, 11.
- Bean, J. W. (1965). Factors influencing clinical oxygen toxicity. *Ann. New York Acad. Sci.*, **117**, 745-755.
- Bean, J. W., and N. E. Leatherman. (1969). Cerebral blood flow during convulsions alterations induced in animals by high pressure oxygen. *Arch. Neurol. (Chicago)*, **20**, 396-405.
- Bean, J. W., J. Lignell, and D. W. Burgess. (1972). Cerebral O₂, CO₂, regional cerebral vascular control, and hyperbaric oxygenation. *J. Appl. Physiol.*, **32**, 650-657.
- Bear, R. S. (1952). The structure of collagen fibrils. *Adv. Protein Chem.*, **7**, 69-160.
- Beard, S. E., T. H. Allen, R. G. McIver, and R. W. Bancroft. (1967). Comparison of helium of nitrogen in production of bends in simulated orbital flights. *Aerospace Med.*, **38**, 331-337.
- Behnke, A. R. (1937). The application of measurements of nitrogen elimination to the problem of decompressing divers. *U.S. Nav. Med. Bull.*, **35**, 219-240.
- Behnke, A. R. (1942). Investigations concerned with problems of high altitude flying and deep diving: application of certain findings pertaining to physical fitness to the General Military Science. *Milit. Surg.*, **90**, 9-29.
- Behnke, A. R. (1947). A Review of Physiological and Clinical Data Pertaining to Decompression Sickness. *Proj. X-443, Report No. 4*, Naval Medical Research Institute, Washington.
- Behnke, A. R. (1951). Decompression sickness following exposure to high pressures. In: *Decompression Sickness*. Ed. by J. F. Fulton, Saunders, Philadelphia, pp. 53-89.
- Behnke, A. R. (1967). The isobaric (oxygen window) principle of decompression. In: *Trans. Third Annual Conference of the Marine Technol. Soc.* Marine Technol. Soc., Washington, pp. 213-228.
- Behnke, A. R. (1969). Some early studies of decompression. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, ch. 11, pp. 226-251.
- Behnke, A. R. (1975). Early quantitative studies of gas dynamics in decompression. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. 2nd ed. Baillière & Tindall, London, pp. 392-416.
- Behnke, A. R., and P. J. Jones. (1974). Preliminary bart tunnel results. In: *Proc. Symp. on Dysbaric*

- Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 25-40.
- Behnke, A. R., and L. A. Shaw. (1937). The use of oxygen in the treatment of compressed air illness. *Nav. Med. Bull.*, **35**, 61-73.
- Behnke, A. R., and O. D. Yarbrough. (1938). Physiologic studies of helium. *Nav. Med. Bull.*, **36**, 542-558.
- Behnke, A. R., and O. D. Yarbrough. (1939). Respiratory resistance, oil-water solubility and mental effects of argon compared with helium and nitrogen. *Amer. J. Physiol.*, **126**, 409-415.
- Behnke, A. R., F. S. Johnson, J. R. Poppen, and E. P. Motley. (1935a). The effect of oxygen on man at pressures from 1 to 4 atmospheres. *Amer. J. Physiol.*, **111**, 565-572.
- Behnke, A. R., R. M. Thomson, and E. P. Motley. (1935b). The psychologic effects from breathing air to 4 atmospheres pressure. *Amer. J. Physiol.*, **112**, 554-558.
- Behnke, A. R., L. A. Shaw, A. C. Messer, R. M. Thomson, and E. P. Motley. (1936). The circulatory and respiratory disturbances of acute compressed-air illness and the administration of oxygen as a therapeutic measure. *Amer. J. Physiol.*, **114**, 526-533.
- Belkin, D. A. (1968). Aquatic respiration and underwater survival of two freshwater turtle species. *Resp. Physiol.*, **4**, 1-14.
- Bell, G. H., J. N. Davidson, and H. Scarborough. (1961). *Textbook of Physiology and Biochemistry*. Livingstone, London.
- Benjamin, R. B., G. E. Turbak, and F. J. Lewis. (1957). The effects of air embolism in the systemic circulation and its prevention during open cardiac surgery. *J. Thorac. Surg.*, **34**, 548-552.
- Bennet, R. A. (1976). Fine structure of decompression sickness. In: *Proc. 6th Symp. Underwater Physiology*. Abstr. UMS, Washington, p. 58.
- Bennett, P. B., and A. Glass. (1961). Electroencephalographic and other changes induced by high partial pressures of nitrogen. *Electroenceph. Clin. Neurophysiol.*, **13**, 91-98.
- Bennett, P. B., and A. J. Hayward. (1968). Relative decompression sickness hazards in rats of neon and other inert gases. *Aerospace Med.*, **39**, 301-302.
- Bennett, P. B., and R. D. Vann. (1975). Theory and development of sub-saturation decompression procedures for depths in excess of 400 feet. In: *Proc. Symp. on Development of Decompression Procedures for Depths in Excess of 400 feet*. Undersea Biomed. Soc., Washington.
- Bennett, P. B., J. C. Cromer, and S. Simon. (1975). Causes and mechanisms of high pressure nervous syndrome and inert gas narcosis. (Abstr.). Progress Report, Contract N00014-67-A-0251-0022. ONR, Washington, pp. 5-7.
- Berghage, T. E., J. M. Woolley, and L. J. Keating. (1974). The probabilistic nature of decompression sickness. *Undersea Biomed. Res.*, **1**, 189-196.
- Berry, C. A., and G. L. Hekhuis. (1960). X-ray survey for bone changes in low-pressure chamber operators. *Aerospace Med.*, **31**, 760-766.
- Bert, P. (1878). *La Pression Barometrique; Recherches de Physiologie Experimentale*. Masson, Paris. Translated by M. A. Hitchcock and F. A. Hitchcock, (1943), College Book Co., Columbus, Ohio.
- Berthelot, M. P. (1850). Sur Quelques Phenomenes de Dilatation Forces Des Liquids. *Annales de Chimie et de Physique*, **30**, 232-242.
- Beyer, D. L., B. G. D'Aoust, E. Casillas, and L. S. Smith. (1976). Decompression and isobaric supersaturation in fluid-breathing vertebrates. Timed response via bioassay, hematology and ultrasonic bubble detection. In: *Sixth Symp. Underwater Physiol.* Abstr. UMS, Washington, p. 53.
- Beyne, J. (1923). Sur l'Origine des accidents provoqués chez l'homme par les fortes dépressions atmosphériques et sur la protection de l'aviateur contre les troubles d'ordre anoxhemique. C. r. hebdom. Séanc. Acad. Sci., Paris, **176**, 1920-1923.
- Blackwood, F., and C. Edmonds. (1971). Otological investigations in diving. *Royal Australian Navy School of Underwater Medicine Project Report 2/71*.
- Blackwood, W. (1958). Discussion on vascular disease in the spinal cord. *Proc. Roy. Soc. Med.*, **51**, 543-547.
- Blake, F. G. (1949). The onset of cavitation in liquids I. Cavitation threshold sound pressures in water as a function of temperature and hydrostatic pressure. (Abstr.). Harvard University. *Acous. Res. Lab. Tech. Memo* 12.
- Blankenhorn, M. A., and E. B. Ferris. (1944). The nature of aviator's bends. *Trans. Ass. Amer. Phys.*, **58**, 86-91.
- Blankenhorn, M. A., E. B. Ferris, J. Romano, H. W. Ryder, G. L. Engel, J. P. Webb, I. A. Safer, and C. D. Stevens. (1942). *Decompression Sickness: (Exploratory Study)*, Report 6, U.S. NRC, Comm. Aviat. Med., Washington.
- Blankenship, J. E., R. Feinstein, and B. D. Butler. (1976). Effects of increased nitrogen tensions on electrical properties of nerve cells. In: *Proc. 6th Symp. Underwater Physiology*. Abstr. UMS, Washington, p. 26.
- Blenkarn, G. D., C. Aquadro, B. A. Hills, and H. A. Saltzman. (1971). Urticaria following sequential breathing of various inert gases at 7 ATA: A possible manifestation of gas-induced osmosis. *Aerospace Med.*, **42**, 141-146.
- Blinks, L. A., V. C. Twotly, and D. M. Whitaker. (1951). Bubble formation in frogs and rats. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, Philadelphia, ch. 5 (Pt. II).
- Boettcher, W. G. (1974). Epidemiological and etiological considerations in osteonecrosis. In: *Proc. Symp. on Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 87-90.

- Bohr, C. (1899). Definition und Methode zur Bestimmung der Invasions und Evasionscoefficienten bei der Auflösung von Gasen in Flüssigkeiten. *Annaln. Physik, Chemie.*, **68**, 500.
- Bonin, B., P. W. Straub, R. Schibli, and A. A. Bühlmann. (1973). Blood coagulation during critical decompression following diving experiments with oxygen/helium. *Aerospace Med.*, **44**, 508–512.
- Boothby, W. M., W. R. Lovelace, and O.O. Benson. (1940). High altitude and its effect on the human body. *J. Aero. Soc. Amer.*, **7**, 1.
- Bornmann, R. C. (1970). Decompression Schedule Development for Repetitive Saturation-excursion Helium-Oxygen Diving. Res. Report 1-70, Deep Submergence Systems Proj. Off., Chevy Chase, Maryland.
- Bornstein, A., and E. Plate. (1911). Über chronische Gelenkveränderungen, entstanden durch Pressluftkrankung. *Fortschr. Geb. Röntgenstrahl.*, **18**, 197–206.
- Borom, M. P., and L. A. Johnson. (1974). Decompression meter for scuba diving utilizing semipermeable membranes. *Aerospace Med.*, **45**, 135–142.
- Boucher, E. A. (1969). Nucleation in the atmosphere. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 10.
- Boycott, A. E., and G. C. C. Damant. (1908). Caisson disease: influence of fatness. *J. Hyg. London*, **8**, 445–456.
- Boycott, A. E., G. C. C. Damant, and J. S. Haldane. (1908). Prevention of compressed air illness. *J. Hyg. London*, **8**, 342–443.
- Bradley, M. E., and J. Vorosmarti. (1974). Hyperbaric arthralgia during helium-oxygen dives from 100 to 850 ft. *Undersea Biomed. Res.*, **1**, 151–168.
- Bradner, H., and R. S. Mackay. (1963). Biophysical limitations on deep diving: some limiting performance expectations. *Bull. Math. Biophys.*, **25**, 251–272.
- Brauer, R. W. (1968). Seeking man's depth level. *Ocean Ind.*, **3**, 28–33.
- Brauer, R. W. (1975). The high pressure nervous syndrome: animals. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. 2nd ed. Baillière and Tindall, London, ch. 13.
- Brauer, R. W., D. O. Johnsen, R. L. Pessotti, and R. W. Redding. (1966). Effects of hydrogen and helium to 67 atmospheres on mice and monkeys. *Fed. Proc.*, **25**, 202.
- Brestkin, A. P. (1965). Relationship between the supersaturation coefficient of gas-liquid systems and the tension of dissolved gas. In: *The Effect of the Gas Medium and Pressure on Body Functions*. Ed. by M. P. Brestkin, Collection No. 3, NASA-TT-F-358, Washington, pp. 10–17.
- Brestkin, A. P., P. M. Gamenitskiy, A. N. Mazin, P. V. Oblapenko, V. V. Ogleznev, and N. Rachov. (1958). Functions of the organism under conditions of an altered gas medium. *Akademiya Nauk SSSR*, **2**, 25–31.
- Brestkin, A. P., P. M. Gremenitskii, and N. Ya. Sidorov. (1965). Study of the safe supersaturation of the body with indifferent gases at different pressures. In: *The Effect of the Gas Medium and Pressure on Body Functions*. Ed. by M. P. Brestkin, Collection No. 3, NASA-TT-F-358, Washington, pp. 18–27.
- Brierley, J. B. (1963). Neuropathological findings in patients dying after open-heart surgery. *Thorax*, **18**, 291–304.
- Brierley, J. B. (1967). Brain damage complicating open-heart surgery: a neuropathological study of 46 patients. *Proc. R. Soc. Med.*, **60**, 34–35.
- Brierley, J. B., and A. N. Nicholson. (1969a). Neuropathological correlates neurological impairment following prolonged decompression. *Aerospace Med.*, **40**, 148–152.
- Brierley, J. B., and A. N. Nicholson. (1969b). Neurological study of simulated decompression in supersonic aircraft. *Aerospace Med.*, **40**, 830–833.
- Brierley, J. B., A. W. Brown, B. S. Meldrum, and D. Riche. (1970). The time course of ischaemic neuronal changes in the primate brain following profound arterial hypotension, air embolism and hypoglycaemia. *J. Physiol. (London)*, **207**, 59P–60P.
- Briggs, L. (1947). Possible explanation of Briggs's limiting negative pressure data. *J. Appl. Physics*, **23**, 931–935.
- Briggs, L. (1950). Limiting negative pressure of water. *J. Appl. Physics*, **21**, 721–722.
- Bromley, J., and W. Harvey. (1944). Radiology at simulated high altitudes. *FPRC Report 626, Flying Pers. Res. Comm.*, Air Ministry, London.
- Brown, H.H.S. (1965). The pressure cabin. In: *A textbook of Aviation Physiology*. Ed. by J. A. Gillies. Pergamon, Oxford, pp. 152–186.
- Buckles, R. G. (1968). The physics of bubble formation and growth. *Aerospace Med.*, **39**, 1062–1069.
- Buckles, R. G., and C. Knox. (1969). *In vivo* bubble detection by acoustic-optical imaging techniques. *Nature (London)*, **222**, 771–772.
- Bühlmann, A. A. (1969). The use of multiple inert gases in decompression. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, ch. 15.
- Bühlmann, A. A. (1974). Fatal fat embolism following decompression sickness in an experimental dive. In: *Proc. Symp. on Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 83–84.
- Bühlmann, A. A. (1975). Decompression theory: Swiss practice. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. 2nd ed. Baillière & Tindall, London, ch. 19.

- Bühlmann, A. A., and W. Waldvogel. (1967). The treatment of decompression accidents. *Helv. Med. Acta*, **33**, 487-491.
- Burton, A. C. (1968). *Physiology and Biophysics of the Circulation*. Year Book Medical Publ., Chicago.
- Cabbarou, P., U. Finkeldey, H. D. Fust, H. Krekeler, K. G. Muller, and H. Oser. (1975). Development and tests of heliox dives in excess of 100 m (Pt. 1). Deutsche Luft und Raumfahrt, Bonn-Bad Godesberg, DLR-FB 75-48.
- Cain, C. C., and A. B. Otis. (1949). Some physiological effects resulting from added resistance to respiration. *J. Aviat. Med.*, **20**, 149-160.
- Campbell, J. (1968). The tribonucleation of bubbles. *Brit. J. Appl. Phys.*, Ser. 2, **1**, 1085-1088.
- Campbell, J. A. (1924). Changes in the tensions of CO₂ and O₂ in gases injected under the skin and into the abdominal cavity. *J. Physiol.*, **59**, 1-16.
- Campbell, J. A., and L. Hill. (1933). Studies in saturation of tissues with gaseous N₂. *Quant. J. Exp. Physiol.*, **23**, 219-227.
- Carlson, L. A. (1971). Nicotinic acid: its metabolism and its effects on plasma free fatty acids. In: *Metabolic Effects of Nicotinic Acid and its Derivatives*. Ed. by K. F. Gey and L. A. Carlson. Hans Huber, Bern, pp. 157-165.
- Carlsaw, H. S., and J. C. Jaeger. (1959). *Conduction of Heat Solids*. 2nd ed., Oxford University Press, Oxford.
- Caruso, V. G., P. E. Winkelmann, M. J. Correia, G. E. Miltenberger, and J. T. Love. (1977). Otologic and otoneurologic injuries in divers: Clinical studies on nine commercial and two sport divers. *Laryngoscope*, **87**, 508-521.
- Case, E. M., and J. B. S. Haldane. (1941). Human physiology under high pressure; effects of nitrogen, carbon dioxide and cold. *J. Hyg. London*, **41**, 225-249.
- Catsaras, M. (1890). Quoted by Gray (1951).
- Cattell, M. (1936). The physiological effects of pressure. *Biol. Rev.*, **11**, 441-475.
- Chang, K. S., and W. J. Yang. (1969). Survey of literature related to the problems of gas embolism in the human body. *J. Biomechanics*, **2**, 299-312.
- Chase, W. H. (1934). Anatomical experimental observations on air embolism. *Surg. Gyn. and Obst.*, **59**, 569-577.
- Chouteau, J. (1969). Saturation diving: the Conshelf experiments. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 491-504.
- Chouteau, J., J. M. Ocana de Sentuary, and L. Pironti. (1971). A comparative and theoretical study of compression rate in deep diving. In: *Abstr. 25th Congr. Physiol. Sciences Satellite Symp. Recent Progress in Fundamental Physiology of Diving* (Marseille), pp. 62-64.
- Chryssanthou, C. P. (1973). Studies on the mechanism and prevention of decompression sickness. *Abstr. Progress Report of the Physiol. Program*, ONR, Arlington, Virginia, pp. 7-8.
- Chryssanthou, C. P. (1974). Pathogenesis and treatment of decompression sickness. *N. Y. State J. Med.*, **74**, 808-812.
- Chryssanthou, C. P. (1975). Experimental dysbaric osteonecrosis: influence of various factors on its incidence and latency. In: *Proc 6th Symp. Underwater Physiology*. Abstr. UMS, Washington, p. 41.
- Chryssanthou, C. P., J. Kalberer, S. Koopestein, and W. Antopol. (1964). Studies of dysbarism. II: Influence of bradykinin and bradykinin-antagonists on decompression sickness in mice. *Aerospace Med.*, **35**, 741-746.
- Chryssanthou, C. P., F. Teichner, and W. Antopol. (1971). Studies on dysbarism. IV: Production and prevention of decompression sickness in "non-susceptible" animals. *Aerospace Med.*, **42**, 864.
- Chung, S. M. K., and E. L. Ralston. (1969). Necrosis of the femoral head in sickle cell anemia and its genetic variants. *J. Bone Joint Surg.*, **51A**, 33-58.
- Cissik, J. H., and R. E. Johnson. (1972). Myth of nitrogen equality in respiration. *Aerospace Med.*, **43**, 755-758.
- Cissik, J. H., R. E. Johnson, and D. K. Rokosch. (1972). Production of gaseous nitrogen in human steady-state conditions. *J. Appl. Physiol.*, **32**, 155-159.
- Clare, N. D. (1925). Supersaturation of gases in liquids. *Trans. Roy. Soc. Canada, Sec. III*, **19**, 32-33.
- Clark, J. M. (1976). Predictive studies. IV: Effects in man of rapid compression on He:O₂ to depths of 400-800-1200-1600 fsw. In: *Undersea Biomedical Research*. Abstr. 1, UMS, Washington.
- Clark, J. M., and C. J. Lambertsen. (1971). Pulmonary oxygen toxicity: a review. *Pharma. Rev.*, **23**, 37-133.
- Clark, L. C., and F. Gollan. (1966). Survival of mammals breathing organic liquids equilibrated with oxygen at atmospheric pressure. *Science*, **152**, 1755.
- Clarke, R. W., F. D. Humm, and L. F. Nims. (1945). The efficacy of preflight denitrogenation in the prevention of decompression sickness. Report 472, U. S. NRC, Comm. Aviat. Med., Washington.
- Clay, J. R. (1963). Histopathology of experimental decompression sickness. *Aerospace Med.*, **34**, 1107-1110.
- Clements, J. A. (1957). Surface tension of lung extracts. *Proc. Soc. Exptl. Biol. Med.*, **95**, 170-172.
- Coakley, W. T. (1971). Acoustic detection of single cavitation events in a focussed field in water at 1 MHz. *J. Acoust. Soc. Amer.*, **49**, 792-801.
- Cockett, A. T. K., and R. M. Nakamura. (1964). A new concept in the treatment of decompression sickness (dysbarism). *Lancet*, **1**, 1102.
- Cockett, A. T. K., S. M. Pauley, and A. P. Roberts. (1972a). Advances in treatment of decompression

- sickness: An evaluation of heparin. In: *3rd Int. Conf. on Hyperbaric Medicine and Underwater Physiology*. Ed. by X. Fructus. Doin, Paris, pp. 156-159.
- Cockett, A. T. K., S. M. Pauley, A. Pilmanis, and A. P. Roberts. (1972b). Formation of lipid emboli after significant decompression. In: *1972 Annual Meeting Aerospace Medical Assoc.* Bal Harbour, Florida, p. 219.
- Cohen, R., E. M. Overfield, and J. A. Kylstra. (1971). Diffusion component of alveolar-arterial oxygen pressure difference in man. *J. Appl. Physiol.*, **31**, 223-226.
- Colebatch, H. J. H. (1964). Responses of the lung to pulmonary embolism. Lect. Postgrad. Med. School, Hammersmith, reported by Fryer (1969).
- Coles, R. A. A., and J. J. Knight. (1961). Aural and audiometric survey of qualified divers and submarine escape training instructors. *Report RNPL 61/1011*, MRC, London.
- Committee on Hyperbaric Oxygenation. (1966). *Fundamentals of Hyperbaric Medicine*. NAS & NRC, Washington.
- Comroe, J. H. (1950). Blood oxygen tension. In: *Methods in Medical Research*. Year Book Med. Publ. Chicago, vol. 2, p. 162.
- Comroe, J. H. (1969). *Physiology of Respiration*. Year Book Med. Publ., Chicago.
- Cook, S. F. (1951). Role of exercise, temperature, drugs and water balance in decompression sickness. In: *Decompression Sickness*. Ed. by J. F. Fulton, Saunders, Philadelphia, ch. 8 (Pt. II).
- Cook, S. F., O. L. Williams, W. R. Lyons, and J. H. Lawrence. (1944). A comparison of altitude and exercise with respect to decompression sickness. *War Med.*, **6**, 182-187.
- Coryllos, P. N., and G. L. Birnbaum. (1932). Studies in pulmonary gas absorption in bronchial obstruction. II. The behaviour and absorption times of oxygen, carbon dioxide, nitrogen, hydrogen, helium, ethylene, nitrous oxide, ethyl chloride, and ether in the lung with some observations on pleural absorption of gases. *Amer. J. Med. Sci.*, **183**, 326-347.
- Costa, G. (1960). Hypothetical pathway of nitrogen metabolism. *Nature*, **188**, 549-552.
- Costa, G., L. Ullrich, F. Danter, and J. F. Holland. (1968). Production of elemental nitrogen by certain mammals including man. *Nature*, **218**, 546-551.
- Cotes, J. E. (1952). Notes on the incidence of cases of collapse in RAF decompression chambers. *FPRC Memo 33*, Air Ministry, London.
- Cotes, J. E., and D. G. C. Gronow. (1952). Further analysis of cases of descent in decompression chamber tests of 610 flying personnel. *FPRC Report 794*, Air Ministry, London.
- Coulson, J. M., and J. F. Richardson. (1965). *Chemical Engineering*. 2nd ed., vol. 2, Pergamon, Oxford.
- Cousteau, J. Y. (1953). *The Silent World*. Reprint Society, London.
- Cox, D. R. (1962). *Renewal Theory*. Methuen, London.
- Crank, J. (1956). *Mathematics of Diffusion*. Oxford University Press, Oxford.
- Crocker, W. E. (1957). Investigation into decompression tables, *Report No. IX, Revised Tables, RNPRC Report, U.P.S. 171*, MRC, London.
- Crocker, W. E., and H. J. Taylor. (1952). A method of calculating decompression stages and the formulation of new diving tables. Report III, Pt. B. Investigation into the decompression tables. *RNPRC Report, U.P.S. 131*, MRC, London.
- Crocker, W. E., F. C. Goodenough, and W. M. Davidson. (1951). Investigation into the Decompression Tables—Progress Report on the First Series of Human Exposures. *RNPRC Report, U.P.S. 118*, MRC, London.
- Crone, C., and N. A. Lassen. (1970). *Capillary Permeability*. Munksgaard, Copenhagen.
- Crump, S. F. (1949). Determination of Critical Pressures for the Inception of Cavitation in Fresh and Sea Water as Influenced by Air Content of the Water. *David Taylor Model Basin Report 575, NS 713-065*, U.S.N. PI-37.
- Cunnington, J. P. W., C. J. Lambertsen, and J. R. M. Cowley. (1975). The dynamics and composition of spontaneous, continuous gas embolism in the pig during isobaric gas counterdiffusion. In: *Proc. Sixth Symp. Underwater Physiology*. Abstr. UMS, Washington, p. 53.
- Curtillet, E. (1939). L'Emboli gazeuse arterielle. *J. Chir.*, **53**, 461-482.
- D'Aoust, B. G., K. H. Smith, and H. T. Swanson. (1976). Decompression-induced decrease in nitrogen elimination rate in awake dogs. *J. Appl. Physiol.*, **41**, 348-355.
- Davidson, J. K. (1964). Radiology in decompression sickness in the Clyde Tunnel. *Scot. Med. J.*, **9**, 1-9.
- Davidson, J. K., and P. D. Griffiths. (1970). Caisson disease of bone. *X-ray Focus*, **10**, 2-11.
- Davidson, W. M., B. M. Sutton, and H. J. Taylor. (1950). Decompression ratio for goats following long exposure and return to atmospheric pressure without stoppage. *RNPRC Report, U.P.S. 110*, MRC, London.
- Davis, J. C., R. Tager, H. P. Polkovitz, and R. D. Workman. (1971). Neurological decompression sickness: Report of two cases at minimal altitudes with subsequent seizures. *Aerospace Med.*, **42**, 85-88.
- Davis, R. H. (1947). *Breathing in Irrespirable Atmosphere*. Siebe-Gorman, London.
- Davis, R. W. (1962). *Deep Diving and Submarine Operations*. 7th ed. Siebe-Gorman, Chessington, Surrey.
- Davson, H. (1964). *A Textbook of General Physiology*. 3rd ed. Churchill, London.
- Dean, R. B. (1944). The formation of bubbles. *J. Appl. Physics*, **15**, 446-449.
- De Coppet, L. C. (1875). Théorie de la Surfusion et de la sursaturation, d'après les principes de la théorie

- mécanique de la chaleur. *Ann. Chim. Phys.*, **6**, 275–288.
- De Coppet, L. C. (1907). Recherches sur la surfusion et la sursaturation. *Ann. Chim. Phys.*, **10**, 457–527.
- Deiss, W. P. (1974). The metabolism of bone. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington.
- Deiss, W. P., L. B. Holmes, and C. C. Johnston. (1962). Bone matrix biosynthesis *in vitro*. I. Labeling of hexosamine and collagen of normal bone. *J. Biol. Chem.*, **237**, 3555–3559.
- De la Torre, E. J. Meredith, and M. G. Netsky. (1962). Cerebral air embolism in the dog. *Arch. Neurol.*, **6**, 307–316.
- Denton, E. J. (1964). The buoyancy of marine molluscs. In: *Physiology of Mollusca*. vol. I. Ed. by K. M. Wilbur and C. M. Yonge. Academic Press, New York, ch. 13.
- Des Granges, M. (1957). Repetitive diving decompression tables. *Research Report 6-57*, U.S. Navy Experimental Diving Unit, Washington.
- Despretz, C. (1837). Untersuchungen Über das Maximum der Dichtigkeit bei Flüssigkeiten. *Annalen der Physik.*, **41**, 58–71.
- Dick, D. A. T. (1959). The rate of diffusion of water into the protoplasm of living cells. *Exp. Cell. Res.*, **17**, 5–12.
- Dick, D. A. T. (1964). *Cell Water*. Butterworths, Washington.
- Dieter, E. (1954). Über das Vorkommen arteriovenöser Anastomosen im Skelettmuskel. *Pflügers Arch. Ges. Physiol.*, **258**, 470–474.
- Diggs, L. W., H. N. Pulliam, and J. C. King. (1937). Bone changes in sickle cell anemia. *Southern Med. J.*, **30**, 249.
- Dixon, H. H. (1914). *Transpiration and the Ascent of Sap in Trees*. Macmillan, London.
- Doll, R. E. (1965). Decompression sickness among U.S. Navy operational divers; an estimate of incidence using air decompression tables. *Research Report 4-64*, U.S. Navy Experimental Diving Unit, Washington.
- Doll, R. E., and T. E. Berghage. (1967). Interrelationships of several parameters of decompression sickness. *Research Report 7-65*, U.S. Navy Experimental Diving Unit, Washington.
- Donald, K. W. (1947). Oxygen poisoning in man. *Br. Med. J.*, **1**, 667–672.
- Donald, K. W. (1955). Oxygen bends. *J. Appl. Physiol.*, **7**, 639–644.
- Dudka, L. T., H. J. Inglis, R. E. Johnson, J. M. Pechinski, and S. Plowman. (1971). Inequality of inspired and expired gaseous nitrogen in man. *Nature*, **232**, 265–267.
- Duffner, G. J. (1958). Recent studies of helium exchange in diving. In: *Man's Dependence on the Earthly Atmosphere*. Ed. by K. E. Schaefer. Macmillan, New York.
- Duffner, G. J., O. E. Van der Aue, and A. R. Behnke. (1946). The treatment of decompression sickness. *Research Report 3*. Project X-443, U.S. Navy Experimental Diving Unit, Washington.
- Duffner, G. J., J. F. Snyder, and L. L. Smith. (1959). Adaptation of helium-oxygen to mixed gas scuba. *Research Report 3-59*, U.S. Navy Experimental Diving Unit, Washington.
- Dufour, L. (1863). Recherches sur la solidification et sur l'ébullition. *Ann. Chim. Phys.*, **68**, 370–393.
- Dunning, W. J. (1969). General and theoretical introduction. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 1.
- Durant, T. M., J. M. Long, and M. J. Oppenheimer. (1947). Pulmonary (venous) air embolism. *Amer. Heart J.*, **33**, 269–281.
- Durant, T. M., M. J. Oppenheimer, M. R. Webster, and J. Long. (1949). Arterial air embolism. *Amer. Heart J.*, **38**, 481–500.
- Dwyer, J. V. (1956). Calculation of repetitive diving decompression tables. *Research Report 1-57*, U.S. Navy Experimental Diving Unit, Washington.
- Ebel, W. J. (1969). Supersaturation of nitrogen in the Columbia River and its effect on salmon and steelhead trout. *Fish. Bull.*, **68**, 1–11.
- Edel, P. O. (1974). *Final Report on Project Hydrox*, ONR Contract N00014-73-C-0233. U.S. Navy, Arlington.
- Edel, P. O., J. M. Holland, C. L. Fischer, and W. P. Fife. (1972). Preliminary studies of hydrogen-oxygen breathing mixtures for deep sea diving. In: *Proc. Symp. on the Working Diver*. Marine Technol. Soc., Washington, pp. 257–270.
- Edmonds, C. (1973). Vertigo in diving. In: *The Use of Nystagmography in Aviation Medicine; ACARD Conference Proc.* No. 128. Ed. by F. E. Guedry. Naval Aerospace Med. Res. Inst., Pensacola.
- Eggleton, P., S. R. Eldsen, J. Fegler, and C. O. Hebb. (1945). A study of the effects of rapid decompression in certain animals. *J. Physiol.*, **104**, 129–150.
- Einstein, A. (1910). Theorie der Opaleszenz von homogenen Flüssigkeiten und Flüssigkeitsgemischen in der Nähe des kritischen Zustandes. *Ann. Physik*, **33**, 1275–1298.
- Elliott, D. H. (1967). The bends: current concepts in the treatment of decompression sickness. *J. Bone Jt. Surg.*, **49B**, 588–590.
- Elliott, D. H. (1969). The pathological processes of decompression sickness. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett, and D. H. Elliott. Baillière, Tindall & Cassell, London, ch. 17.
- Elliott, D. H. (1974). Discussion of experimental studies. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington.
- Elliott, D. H., and J. A. B. Harrison. (1970). Bone necrosis—An occupational hazard of diving. *J. Roy. Naval Med. Serv.*, **56**, 140–161.
- Elliott, D. H., and J. A. B. Harrison. (1971). Aseptic

- bone necrosis in Royal Navy divers. In: *Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York.
- Elliott, D. H., J. M. Hallenbeck, and A. A. Bove. (1974). Acute decompression sickness. *Lancet*, Occasional Survey, p. 1193.
- Elsner, R., D. L. Franklin, R. L. Van Citters, and D. W. Kenney. (1966). Cardiovascular defense against asphyxia. *Science*, **153**, 941-949.
- End, E. (1937). Rapid decompression following inhalation of helium-oxygen mixtures under pressure. *Amer. J. Physiol.*, **120**, 712-718.
- End, E. (1938). The use of new equipment and helium gas in a world record dive. *J. Ind. Hyg. Toxicol.*, **20**, 511-520.
- End, E. (1939). The physiologic effects of increased pressure. *Proc. 6th Pac. Sci. Congr.*, **6**, 91-97.
- End, E. (1971). Blood agglutination in decompression sickness. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York.
- End, E., and C. W. Long. (1942). Oxygen under pressure in carbon monoxide poisoning. *J. Ind. Hyg. Toxicol.*, **24**, 302.
- Enns, T., P. F. Scholander, and E. D. Bradstreet. (1965). Effect of hydrostatic pressure on gases dissolved in water. *J. Phys. Chem.*, **69**, 389-391.
- Epstein, P. S., and M. S. Plesset. (1950). On the stability of gas bubbles in liquid-gas solutions. *J. Chem. Phys.*, **18**, 1505-1509.
- Ernsting, J. (1965). The principles of pressure suit design. In: *A Textbook of Aviation Medicine*. Ed. by J. A. Gillies. Pergamon, Oxford.
- Evans, A. (1975). Ultrasonic surveillance of decompression. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott, 2nd ed. Baillière & Tindall, London, pp. 417-434.
- Evans, A., and D. N. Walder. (1969). Significance of gas macronuclei in the aetiology of decompression sickness. *Nature, London*, **222**, 251-252.
- Evans, A., and D. N. Walder. (1970). Detection of circulating bubbles in the intact mammal. *Ultrasonics*, **3**, 216-217.
- Evans, A., and D. N. Walder. (1974). Natural uranium and decompression sickness. *Nature*, **252**, 696.
- Evans, A., E. E. P. Barnard, and D. N. Walder. (1972). Detection of gas bubbles in man at decompression. *Aerospace Med.*, **43**, 1095-1096.
- Evans, A. L., A. Bustil, F. C. Gillespie, and J. Unsworth. (1974). The rate of clearance of xenon from rat liver sections *in vitro* and its significance in relation to intracellular diffusion rates. *Phys. Med. Biol.*, **19**, 303-316.
- Evans, N. T. S., and Naylor, P. F. D. (1963). The effect of oxygen breathing and radiotherapy upon the tissue oxygen tension of some human tumors. *Brit. J. Radiol.*, **36**, 418-425.
- Evelyn, K. (1941). The effects of low barometric pressure on the healthy adult male in the age group 19-32. RCAF un-numbered report.
- Fagan, C. J., E. L. Beckman, and J. B. Galletti. (1974). Sample survey of osteonecrosis in Gulf of Mexico commercial divers. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 9-15.
- Fahrenheit, R. S. S. (1724). Experimenta et observationes de congelatione aque in vacuo facte. *Roy. Soc. Lond. Phil. Trans.*, **33**, 78-84.
- Farmer, J. C., and W. G. Thomas. (1973). Vestibular injury during diving. In: *Proc. 1st Ann. Scient. Meeting Europ. Undersea Biomed. Soc.* Ed. by C. M. Hesser and D. Linnarsson. Försvarsmedicin, Stockholm, pp. 396-403.
- Farncombe, F. J. (1925). The initiation of bubbles in supersaturated solutions of gases. *Trans. Roy. Soc. Canada, Sec. III*, **19**, 32.
- Featherstone, R. M., C. A. Muehlbaeche, F. L. DeBon, and J. A. Forsaith. (1961). Interactions of inert anaesthetic gases with proteins. *Anesthesiology*, **22**, 977-981.
- Fenichel, I. R., and S. B. Horowitz. (1963). The transport of nonelectrolytes in muscle as a diffusional process in cytoplasm. *Acta Physiol. Scand.*, **60**, suppl. 221, 1-63.
- Fenn, W. O. (1969). The physiological effects of hydrostatic pressures. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London.
- Ferris, E. B., and G. L. Engel. (1951). The clinical nature of high altitude decompression sickness. In: *Decompression Sickness*. Ed. by J. F. Fulton, Saunders, Philadelphia, ch. 2.
- Ferris, E. B., J. P. Webb, H. W. Ryder, G. L. Engel, J. Romano, and M. A. Blankenhorn. (1943). The protective value of preflight oxygen inhalation at rest against decompression sickness. Report 132, U.S. NRC, Comm. Aviat. Med., Washington.
- Fife, W. P. (1974). Amazing decompression breakthrough. In: *Proc. 6th International Conf. Underwater Education*, pp. 159-167.
- Fillenz, M., and J. G. Widdicombe. (1971). Receptors of the lungs and airways. In: *Handbook of Sensory Physiology*. Ed. by E. Neil. Springer-Verlag, Heidelberg, vol. 3, p. 81.
- Fisher, J. C. (1948). The fracture of liquids. *J. Appl. Physics*, **19**, 1062-1067.
- Flynn, E. T., and C. J. Lambertsen. (1971). Calibration of inert gas exchange in the mouse. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 179-192.
- Fox, F. E., and K. E. Herzfeld. (1954). Gas bubbles with organic skin as cavitation nuclei. *J. Acous. Soc. Amer.*, **26**, 984-989.
- Fraser, A. M. (1942). A study of the possible relation of susceptibility to decompression sickness to the rate of blood denitrogenation and to corporeal specific gravity. *Proc. 15th Meetg. Exec. Assoc. Comm. Aviat. Med. Res.*, NRC, Ottawa.

- Fraser, A. M. (1943). Protection against decompression sickness by administration of oxygen immediately prior to ascent. *NRCC Assoc. Comm. Aviat. Med. Res. Report No. 2*, Clinical Investigation Unit, RCAF, Regina, Canada.
- Fraser, A. M., and E. T. Waters. (1942). The effect of hydrostatic pressure on the amelioration of symptoms of decompression sickness. *Proc. 15th Meetg. Exec. Assoc. Comm. Aviat. Med. Res.*, NRC, Ottawa.
- Fredrickson, P. (1956). The analysis and testing of Decomputer (Mark 1). Foxboro Company Res. Report MR-138.
- Freeman, P., and C. Edmonds. (1972). Inner ear barotrauma. *Archs. Otolar.*, **95**, 556-563.
- Frenkel, J. (1939). A general theory of heterophase fluctuations and pretransition phenomena. *J. Chem. Phys.*, **7**, 538-547.
- Fridovich, I. (1972). Superoxide radical and superoxide dismutase. *Acc. Chem. Res.*, **5**, 321-326.
- Fries, C. C., R. Levowitz, S. Adler, A. W. Cook, K. E. Karlson, and C. Dennis. (1957). Experimental cerebral gas embolism. *Ann. Surg.*, **145**, 461-470.
- Fructus, X. R., and J. P. Charpy. (1972). Étude psychométrique de 2 sujets lors d'une plongée fictive jusqu'à 52-42 ATA. Bulletin Medsubhyp. Service D'Hyperbare, Hopital Salvator, Marseille. Report No. 7.
- Fryer, D. I. (1962). Observations concerning the mechanism of subatmospheric decompression sickness. *NATO AIRCENT, 8th Ann. Med Conf.*, 24-36.
- Fryer, D. I. (1964). Decompression sickness at 18,500 feet. A case history with comment. *Aerospace Med.*, **35**, 479-481.
- Fryer, D. I. (1966). Preliminary physiological assessment of the FAE pressure/immersion suit (prototype form). *RAF Inst. Aviat. Med. Report 386*, Farnborough.
- Fryer, D. I. (1969). *Subatmospheric Decompression Sickness in Man*. Technivision Services, Slough, U.K.
- Fryer, D. I., and Roxburgh, H. L. (1966). Decompression sickness. In: *A Textbook of Aviation Physiology*. Ed. by J. A. Gillies. Pergamon, Oxford.
- Fürth, R. (1941). On the theory of the liquid state. *Proc. Cambridge Phil. Soc.*, **37**, 252.
- Fuxe, K., and G. Sedvall. (1965). The distribution of adrenergic nerve fibres to the blood vessels in skeletal muscle. *Acta Physiol. Scand.*, **64**, 75-86.
- Galletti, P. M., and G. A. Brecher. (1965). Artificial blood circulation. In: *Handbook of Physiology*. Sect. 2: Circulation. Ed. by W. F. Hamilton. Amer. Physiol. Soc., Washington, vol. III, ch. 60.
- Galloway, W. J. (1954). An experimental study of acoustically induced cavitation in liquids. *J. Acous. Soc. Amer.*, **26**, 849-857.
- Gardner, E. (1944). The distribution and termination of nerves in the knee joint of the cat. *J. Comp. Neurol.*, **80**, 11-32.
- Gay-Lussac, J. L. (1819). Premier Memoire sur la dissolubilité des sels dans l'eau. *Annales de Chimie et de Physique*, **11**, 296-315.
- Gehring, H., and A. A. Bühlmann. (1975). So-called vertigo bends after oxygen-helium dives (case reports). In: *Proc. Vth Symp. Underwater Physiology*. Fed. Amer. Socs. Exp. Biol., Washington.
- Gellhorn, A., M. Merrell, and R. M. Rankin. (1944). The rate of transcapillary exchange of sodium in normal and shocked dogs. *Amer. J. Physiol.*, **142**, 407-427.
- Gent, A. N., and D. A. Tompkins. (1969). Nucleation and growth of gas bubbles in elastomers. *J. Appl. Phys.*, **40**, 2520-2525.
- Geoghegan, T., and C. R. Lam. (1953). The mechanism of death from intracardiac air and its reversibility. *Ann. Surg.*, **138**, 351-359.
- Gerschman, R. (1964). Biological effects of oxygen. In: *Oxygen in the Animal Organism*. Ed. by F. Dickens and E. Neil. Macmillan, New York, pp. 475-492.
- Gersh, I. (1945). Gas bubbles in bone and associated structures, lung and spleen of guinea-pigs decompressed rapidly from high-pressure atmospheres. *J. Cell. Comp. Physiol.*, **26**, 101-117.
- Gersh, I. (1946). Correlation of X-ray and gross observations on gas bubbles in guinea pigs decompressed from high pressure atmospheres. *J. Cell. Comp. Physiol.*, **28**, 271-275.
- Gersh, I., and H. R. Catchpole. (1951). Decompression sickness: physical factors and pathologic consequences. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, Philadelphia, ch. 7.
- Gersh, I., and G. E. Hawkinson. (1944). The formation and appearance of tissue and vascular bubbles after rapid decompression of guinea pigs from high pressure atmospheres. Research Project X-284. *U.S. Navy Med. Res. Inst., Report No. 1*.
- Gersh, I., G. E. Hawkinson, and E. M. Rathbun. (1944). Tissue and vascular bubbles after decompression from high pressure atmospheres, correlation of specific gravity with morphological changes. *J. Cell. Comp. Physiol.*, **24**, 35-70.
- Gersh, I., G. E. Hawkinson, and E. H. Jenney. (1945). Comparison of vascular and extravascular bubbles following decompression from high-pressure atmospheres of oxygen, helium-oxygen, argon-oxygen and air. *J. Cell. Comp. Physiol.*, **26**, 63-74.
- Gesell, R. (1923). On the chemical regulation of respiration: Regulation of respiration with special reference to the metabolism of the respiratory center and the coordination of the dual function of hemoglobin. *Amer. J. Physiol.*, **66**, 5-49.
- Gibbs, J. W. (1906). *Scientific Papers*. Longmans Green, London, vol. I.
- Gillen, H. W. (1968). Symptomatology of cerebral gas embolism. *Neurology*, **18**, 507-512.
- Gillies, J. A. (Ed.). (1966). *A Textbook of Aviation Medicine*. Pergamon, Oxford.
- Gisborne, A. B., and J. B. Morrison. (1973). Preliminary report of DHB armoured suit. Royal

- Naval Physiological Laboratory, Report 8-73.
- Glasstone, S. (1953). *Textbook of Physical Chemistry*. 2nd ed. Macmillan, London, pp. 230 and 824.
- Glimcher, M. J., and S. M. Krane. (1968). The organization and structure of bone, and the mechanism of calcification. In: *Treatise on Collagen*. Ed. by B. S. Gould. Academic Press, London, vol 2 (Pt. B), ch. 2.
- Goggio, A. F. (1943). Survey of chamber reactions observed and studied at 33rd Altitude Training Unit prior to Oct. 1st. 1943 with related clinical studies. *Comm. Aviat. Med. U.S. NRC, un-numbered report*.
- Gold, H. J., and I. S. Longmuir. (1971). Application of experimental data to a new model of tissue oxygen transport. *Bull. Math. Biophys.* **33**, 295-297.
- Golding, F. C., P. Griffiths, W. D. M. Paton, D. N. Walder, and H. V. Hempleman. (1960). Decompression sickness during construction of the Dartford Tunnel. *Brit. J. Ind. Med.*, **17**, 167-180.
- Gomes, O. M., S. N. Pereira, R. C. Castagna, D. Bittencourt, R. V. G. Amaral, and E. J. Zerbini. (1973). The importance of the different sites of air injection in the tolerance of arterial air embolism. *J. Thor. Cardiovasc. Surg.*, **65**, 563-568.
- Gooden, B. A. (1973). The tadpole tail as a model for decompression sickness. *Austral. J. Exp. Biol. & Med. Sci.*, **51**, 109.
- Goodman, M. W., and R. D. Workman. (1965). Minimal recompression, oxygen breathing approach to the treatment of decompression sickness in divers and aviators. *Research report*, 5-65. U.S. Navy Experimental Diving Unit, Washington.
- Gordon, D. B., J. Flasher, and D. R. Drury. (1953). Size of the largest arterio-venous vessels in various organs. *Amer. J. Physiol.*, **173**, 275-281.
- Gouze, F. J. (1944). A method and study of surface decompression as a routine procedure. *U.S. Navy Med. Bull.*, **42**, 578-580.
- Gowdey, C. W., and R. B. Philp. (1965). Etiology and treatment of experimental decompression sickness with special reference to body lipids. *Milit. Med.*, **130**, 648-652.
- Graves, D. J., J. Idicula, C. J. Lambertsen, and J. A. Quinn. (1973). Bubble formation resulting from counter diffusion supersaturation; a possible explanation for isobaric inert gas 'urticaria' and vertigo. *Physic Med. Biol.*, **18**, 256-264.
- Gray, J. S. (1942). The prevention of aeroembolism by denitrogenation procedures. *Report 123, U.S. NRC, Comm. Aviat Med.*, Washington.
- Gray, J. S. (1943). The effect of exercise at altitudes on aeroembolism in cadets. *Report 169, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Gray, J. S. (1944a). Effect of denitrogenation at various altitudes on aeroembolism in cadets. *Report 258, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Gray, J. S. (1944b). Present status of the problem of decompression sickness. Review # 1, *U.S.A.F. Sch. Aviat. Med.* Randolph Field, Texas.
- Gray, J. S. (1944c). Quoted by Cook (1951).
- Gray, J. S. (1951). Constitutional factors affecting susceptibility to decompression sickness. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, London & Philadelphia, ch. 7.
- Gray, J. S., R. L. Masland, and S. C. F. Mahady. (1945). The effects of breathing carbon dioxide on altitude decompression sickness. Report 1, Project 409, *U.S.A.F. Sch. Aviat. Med.*
- Grenell, R., F. D. Humm, L. F. Nims, and H. M. Somberg. (1944). The reliability of the decompression subcommittee's 90 minute preselection test. *Report 355, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Griffiths, C. M., V. G. Caruso, and B. A. Hills. (1977). Nystagmus induced by ventilating middle ear with different gases at normal pressure (in press).
- Griffiths, H. B., K. W. Miller, W. D. M. Paton, and E. B. Smith. (1971). On the role of separated gas in decompression sickness. *Proc. Roy. Soc.*, **B178**, 389-406.
- Griffiths, P. D. (1969). Clinical manifestations and treatment of decompression sickness in compressed-air workers. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 451-463.
- Groves, G., and W. Munk. (1953). *A decompression gauge for divers*. Scripps Institution of Oceanography Reference, 53-64.
- Grulke, D. C. (1975). Experimental Cerebral Air Embolism: A Physical and Physiological Study using Uniform Microbubbles of Known Size. Thesis in Physiology, University of London.
- Grulke, D. C., and B. A. Hills. (1976). Experimental cerebral air embolism and its resolution. In: *Proc. Sixth Symp. Underwater Physiology* (in press).
- Grulke, D. C., N. A. Marsh, and B. A. Hills. (1973). Experimental air embolism: measurement of microbubbles using the Coulter counter. *Brit. J. Exp. Path.*, **54**, 684-691.
- Guest, M. M., C. H. Wells, and T. P. Bond. (1974). Changes in rheology of animals following various pressure exposures. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington.
- Gustavson, K. H. (1956). *The Chemistry and Reactivity of Collagen*. Academic Press, New York.
- Guyton, A. C. (1963). Concept of negative interstitial pressure based on pressures in implanted perforated capsules. *Circ. Res.*, **12**, 399-414.
- Halbouty, M. R., and D. R. Long. (1953). Neuro-circulatory collapse in aircraft flight. Report of a case. *J. Aviat. Med.*, **24**, 301-307.
- Haldane, J. S. (1895). Action of carbonic oxide on man. *J. Physiol.*, **18**, 430.
- Haldane, J. S. (1920). *Respiration*. 1st ed. Clarendon, Oxford, pp. 380-381.

- Haldane, J. S., and F. G. Meachem. (1898). Observations on the relation of underground temperature and spontaneous fires in the coal to oxidation and to the causes which favour it. *Trans. Inst. Mining Eng., London*, **16**, 467.
- Haldane, J. S., and J. G. Priestley. (1935). *Respiration*. Yale University Press, New Haven, p. 348.
- Hallenbeck, J. M., A. A. Bove, and D. H. Elliott. (1975). Mechanisms underlying spinal cord damage in decompression sickness. *Neurol.*, **25**, 308–316.
- Halsey, M. J., and E. I. Eger. (1973). Fluid shifts associated with gas-induced osmosis. *Science*, **179**, 1139–1140.
- Hamilton, R. W., and T. D. Langley. (1971). Neon as a diving gas: performance compared with nitrogen and helium at 7, 10 and 13 atmospheres. Abstr. 25th Congr. Physiol. Sciences Satellite Symp. Marseille, pp. 43–44.
- Hamilton, R. W., J. B. MacInnis, A. D. Noble, and H. R. Schreiner. (1966). Saturation Diving at 650 feet. *Ocean Systems Technical Memorandum B-411*. Tonawanda, New York.
- Hammel, J. J. (1969). Nucleation in glass-forming materials. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 9.
- Harkness, R. D., and M. L. R. Harkness. (1965). Some mechanical properties of collagenous framework and their functional significance. In: *Symp. on Biorheology*. John Wiley, New York, (Pt. 4) pp. 477–488.
- Harrelson, J. M., and B. A. Hills. (1970). Changes in bone marrow pressure in response to hyperbaric exposure. *Aerospace Med.*, **41**, 1018–1021.
- Harris, E. J., and G. P. Burn. (1949). The transfer of sodium and potassium ions between muscle and the surrounding medium. *Trans. Farad. Soc.*, **45**, 508–528.
- Harris, M., W. E. Berg, D. M. Whitaker, and V. C. Twitty. (1945a). The relation of exercise to bubble formation in animals decompressed to sea level from high barometric pressures. *J. Gen. Physiol.*, **24**, 241–251.
- Harris, M., W. E. Berg, D. M. Whitaker, V. C. Twitty, and L. R. Blinks. (1945b). Carbon dioxide as a facilitating agent in the initiation and growth of bubbles in animals decompressed to simulated altitudes. *J. Gen. Physiol.*, **28**, 225–240.
- Harrison, J. A. B. (1971). Aseptic bone necrosis in naval clearance divers: Radiographic findings. *Proc. Roy. Soc. Med.*, **64**, 1276–1278.
- Harrison, J. A. B. (1974). Radiological criteria in diagnosing dysbaric osteonecrosis. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 151–159.
- Harvey, C., and C. J. Lambertsen. (1976). Deep tissue isobaric inert gas exchange: predictions during normoxic helium, neon and nitrogen breathing by men at 1200 feet of sea water. In: *Proc. 6th Symp. Underwater Physiology*. Abstr. UMS, Washington, p. 44.
- Harvey, E. N. (1928). The oxygen consumption of luminous bacteria. *J. Gen. Physiol.*, **11**, 469–475.
- Harvey, E. N. (1945). Decompression sickness and bubble formation in blood and tissues. *Bull. N. Y. Acad. Med.*, **21**, 505–536.
- Harvey, E. N. (1951a). Physical factors in bubble formation. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, Philadelphia, pp. 90–114.
- Harvey, E. N. (1951b). Bubble formation in liquids. In: *Medical Physics*. Ed. by O. Glasser. Year Book Medical Publ., Chicago, vol. 2.
- Harvey, E. N., D. K. Barnes, W. D. McElroy, A. H. Whiteley, D. C. Pease, and K. W. Cooper. (1944a). Bubble formation in animals. I: Physical Factors. *J. Cell. Comp. Physiol.*, **24**, 1–22.
- Harvey, E. N., A. H. Whiteley, W. D. McElroy, D. C. Pease, and D. K. Barnes. (1944b). Bubble formation in animals. II: Gas nuclei and their distribution in blood and tissues. *J. Cell. Comp. Physiol.*, **24**, 23–34.
- Harvey, E. N., W. D. McElroy, A. H. Whiteley, G. H. Warren, and D. C. Pease. (1944c). Bubble formation in animals. III: An analysis of gas tension and hydrostatic pressure in cats. *J. Cell. Comp. Physiol.*, **24**, 117–132.
- Harvey, H. H., and S. B. Smith. (1961). Supersaturation of the water supply and occurrence of gas bubble disease at Cultus Lake Trout Hatchery. *Canad. Fish Culturist*, **30**, 39–47.
- Haugaard, N. (1968). Cellular mechanisms of oxygen toxicity. *Physiol. Rev.*, **48**, 311–373.
- Hawkins, J. A., C. W. Shilling, and R. A. Hanson. (1935). A suggested change in calculating decompression tables for diving. *Nav. Med. Bull.*, **33**, 327–338.
- Hawkins, T. L. (1974). Swimmer support equipment evaluated. *Faceplate*, **V5**, 24–25.
- Haxton, A. F., and H. E. Whyte. (1969). The compressed-air environment. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 1–16.
- Haymaker, W. (1957). Decompression sickness. In: *Handbuch des Speziellen Pathologischen Anatomie und Histologie*. Ed. by O. Lubarsch, F. Henke and R. Rossie. Springer-Verlag, Berlin, vol. XIII, (Pt. I), pp. 1600–1672.
- Hayward, A. T. J. (1967). Tribonucleation of bubbles. *Brit. J. Appl. Phys.*, **18**, 641–644.
- Heatwole, H., and S. A. Minton. (1975). Sea snakes from reefs of the Sahul Shelf. In: *The Biology of Sea Snakes*. Ed. by W. A. Dunson. University Park Press, Maryland, pp. 141–144.
- Hedgepeth, C. (1976). Comment at a Navywide Workshop on High Pressure Biomedical Research. USN, Panama City.
- Heimbecker, R. O., G. Lemire, C. H. Chen, I. Koven, D. Leask, and W. R. Drucker. (1968). Role of gas embolism in decompression sickness—

- A new look at "the bends". *Surgery*, **64**, 624.
- Heller, R., W. Marger, and H. von Schrotter. (1900). Luftdruckerkrankungen mit Besonderer Berücksichtigung der Sogenannten Caissonkrankheit. *Wien, Holder*, p. 1230.
- Hemmingsen, E. A. (1970). Supersaturation of gases in water: Absences of cavitation on decompression from high pressures. *Science*, **167**, 1493-1494.
- Hempleman, H. V. (1952). Investigation into the Decompression Tables, Report III, part A. A New Theoretical Basis for the Calculation of Decompression Tables. *RNPRC Report, UPS 131*, MRC, London.
- Hempleman, H. V. (1957). Investigation into the Decompression Tables. Further Basic Facts on Decompression Sickness. *RNPRC Report, UPS 168*, MRC, London.
- Hempleman, H. V. (1967). Decompression procedures for deep, open sea operations. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Williams & Wilkins, Baltimore, pp. 255-266.
- Hempleman, H. V. (1968). Bubble formation and decompression sickness. *Revue Physiol. Sub-aquatique*, **1**, 181-183.
- Hempleman, H. V. (1969). British decompression theory. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 291-318.
- Hempleman, H. V. (1975). Present state of the art. In: *Proc. Workshop on Decompression Procedures for Depths in Excess of 400 feet*. Ed. by M. Beckett. UMS, Washington.
- Hennessy, T. R. (1973a). New Mathematical Models of Inert Gas Transport through Biological Tissue in Hyperbaric Environments. Ph.D. Thesis, University of Cape Town.
- Hennessy, T. R. (1973b). The equivalent bulk diffusion model of the pneumatic decompression computer. *Med. Biol. Engng.*, **11**, 135-137.
- Hennessy, T. R., and H. V. Hempleman. (1977). An examination of the critical released gas volume concept in decompression sickness. *Proc. Roy. Soc. (B)*. (in press).
- Henry, F. M. (1943). The reliability of chamber classification for 'bends' susceptibility and the validity of the inert gas-exchange method. *Report 176, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Henry, F. M. (1945). The role of exercise in altitude pain. *Report 460, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Henry, F. M., and A. C. Ivy. (1951). Preselection tests. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, Philadelphia, ch. 10.
- Henry, F. M., H. B. Jones, J. B. Mohney, and C. A. Tobias. (1943). The reliability of decompression chamber classification using the step-up exercise, and the relation of inert gas exchange and other factors to bends-resistance. *Report 264, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Henry, F. M., S. F. Cook, E. Strajman, and D. W. Lund. (1944). Effectiveness of preflight oxygen breathing in preventing decompression sickness. *Report 384, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Herd, J. A., and H. M. Goodman. (1966). Blood flow rates through several adipose tissue depots of unanesthetized rats. *The Physiologist*, **9**, 202.
- Herron, J. M., H. A. Saltzman, B. A. Hills, and J. A. Kylstra. (1973). Differences between inspired and expired minute volumes of nitrogen in man. *J. Appl. Physiol.*, **35**, 546-551.
- Hesser, C. M. (1963). Measurement of inert gas narcosis in man. In: *Proc. 2nd Underwater Physiology Symp.* Ed. by C. J. Lambertsen and L. J. Greenbaum. NRC, NAS, Washington.
- Hester, R. (1970). The Hills Alternative to Naval Decompression Concepts. *Report 620, U.S. Nav. Sub. Med. Cen.*, Groton, Connecticut.
- Hester, R. (1971). Criteria for bubble growth. In: *Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 137-143.
- Hill, A. V. (1928). Diffusion of O₂ and lactic acid through tissues. *Proc. Roy. Soc., B*, **104**, 39-96.
- Hill, L. (1912). *Caisson Sickness and the Physiology of Work in Compressed Air*. Edward Arnold, London, p. 255.
- Hill, L., and J. J. McLeod. (1903). The influence of compressed air on respiratory exchange. *J. Physiol.*, **29**, 492-510.
- Hill, L., and A. F. Phillips. (1932). Deep sea diving. *J. Roy. Nav. Med. Serv. London*, **18**, 157-173.
- Hills, B. A. (1966). *A Thermodynamic and Kinetic Approach to Decompression Sickness; a 400 page monograph*. Libraries Board of South Australia, Adelaide.
- Hills, B. A. (1967a). Decompression sickness; A study of cavitation at the liquid-liquid interface. *Aerospace Med.*, **38**, 814-817.
- Hills, B. A. (1967b). Diffusion versus blood perfusion in limiting the rate of uptake of inert non-polar gases by skeletal rabbit muscle. *Clinical Science*, **33**, 67-87.
- Hills, B. A. (1967c). A thermal analogue for the optimal decompression of divers: Theory. *Phys. Med. Biol.*, **12**, 437-444.
- Hills, B. A. (1967d). A thermal analogue for the optimal decompression of divers: Construction and use. *Phys. Med. Biol.*, **12**, 445-454.
- Hills, B. A. (1967e). A pneumatic analogue for predicting the occurrence of decompression sickness. *Med. Biol. Eng.*, **5**, 421-433.
- Hills, B. A. (1968a). Variation in susceptibility to decompression sickness. *Internat. J. Biometeor.*, **12**, 343-349.
- Hills, B. A. (1968b). Relevant phase conditions for predicting occurrence of decompression sickness. *J. Appl. Physiol.*, **25**, 310-315.
- Hills, B. A. (1968c). Linear bulk diffusion into heterogeneous tissue. *Bull. Math. Biophys.*, **30**, 47-59.

- Hills, B. A. (1969a). A study of decompression sickness applied to the estimation of cellular oxygen tension and its elevation in tumour during clinical radiotherapy. *Rev. Subaqua. Physiol. & Hyperbar. Med.*, **1**, 151–156.
- Hills, B. A. (1969b). Radial bulk diffusion in heterogeneous tissue. *Bull. Math. Biophys.*, **31**, 25–34.
- Hills, B. A. (1969c). Acclimatization to decompression sickness: A study of passive relaxation in several tissues. *Clinical Science*, **37**, 109–124.
- Hills, B. A. (1969d). A quantitative correlation of conditions for the occurrence of decompression sickness for aerial and underwater exposures. *Rev. Subaqua. Physiol. & Hyperbar. Med.*, **1**, 249–254.
- Hills, B. A. (1969e). The time course for the uptake of inert gases by the tissue type responsible for marginal symptoms of decompression sickness. *Rev. Subaqua. Physiol. & Hyperbar. Med.*, **1**, 255–261.
- Hills, B. A. (1970a). Limited supersaturation versus phase equilibration in predicting the occurrence of decompression sickness. *Clinical Science*, **38**, 251–267.
- Hills, B. A. (1970b). Vital issues in computing decompression schedules from fundamentals. I—Critical supersaturation versus phase equilibration. *Internat. J. Biometeor.*, **14**, 111–131.
- Hills, B. A. (1970c). An assessment of the expression $C = \dot{Q} [1 - \exp(-PS/\dot{Q})]$ for estimating capillary permeabilities. *Phys. Med. Biol.*, **15**, 705–713.
- Hills, B. A. (1970d). Respiration of tissue as a medium of heterogeneous permeability. *Bull. Math. Biophys.*, **32**, 219–235.
- Hills, B. A. (1970e). Gas-induced osmosis as an aetiological agent for inert gas narcosis, gouty arthritis and aseptic bone necrosis induced by exposure to compressed air. *Rev. Subaqua. Physiol. & Hyperbar. Med.*, **2**, 3–7.
- Hills, B. A. (1971a). Decompression Sickness: A fundamental study of 'surface excursion' diving and the selection of limb bends versus CNS symptoms. *Aerospace Med.*, **42**, 833–836.
- Hills, B. A. (1971b). Gas-induced osmosis as a factor influencing the distribution of body water. *Clinical Science*, **40**, 175–191.
- Hills, B. A. (1971c). Analysis of contributions to (A-a) DO_2 by change of environment. *Bull. Math. Biophys.*, **33**, 259–280.
- Hills, B. A. (1971d). Concepts of inert gas exchange in tissues during decompression. In: *Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 115–122.
- Hills, B. A. (1971e). Osmosis induced by nitrogen. *Aerospace Med.*, **42**, 664–666.
- Hills, B. A. (1971f). Predicting the occurrence of oxygen convulsions, V—an osmotic mechanism for neurologic oxygen toxicity. *RNPL Report 15/71*, Department of Naval Physical Research, Ministry of Defence, London.
- Hills, B. A. (1972a). Clinical implications of gas-induced osmosis. *Arch. Int. Med.*, **129**, 356–362.
- Hills, B. A. (1972b). Neurologic oxygen toxicity: effects of switch of inert gas and change of pressure. *Aerospace Med.*, **43**, 716–723.
- Hills, B. A. (1972c). Gas-induced osmosis in the lung. *J. Appl. Physiol.*, **33**, 126–129.
- Hills, B. A. (1973a). Factors influencing respiration at increased ambient pressures. Progress Report. Physiology Programme of the Office of Navy Research. *ONR Rep. ACR 191*, ONR, Arlington, pp. 24–25.
- Hills, B. A. (1973b). Chemical facilitation of thermal conduction in physiological systems. *Science*, **182**, 823–825.
- Hills, B. A. (1973c). Biophysical aspects of decompression sickness (Abstr.). Progress Report. Physiology Programme of the Office of Navy Research. *ONR Rep. ACR 191*, ONR, Arlington, pp. 22–23.
- Hills, B. A. (1974). Cumulative oxygen toxicity index allowing for regression of effects at low inspired O_2 partial pressures. *NSMRL Report 805*, U.S. Navy Sub. Base, Groton, Conn.
- Hills, B. A. (1974a). *Gas Transfer in the Lung*. Cambridge University Press, Cambridge.
- Hills, B. A. (1974b). Air embolism: fission of microbubbles upon collision in plasma. *Clinical Science & Molec. Med.*, **46**, 629–634.
- Hills, B. A. (1974c). Effect of DPL at the mercury-water interface and total lung surface area. *J. Appl. Physiol.*, **36**, 41–44.
- Hills, B. A. (1975a). Decompression after breathing various inert gases either simultaneously or sequentially. *RNPL Report*, Ministry of Defence (in press).
- Hills, B. A. (1975b). A decompression meter based upon the zero-supersaturation principle. Abstr. In: *Proc. Sixth Symp. Underwater Physiology*. (in press).
- Hills, B. A. (1976a). Supersaturation by counter-perfusion and diffusion of gases. *J. Appl. Physiol.* (in press).
- Hills, B. A. (1976b). A cumulative oxygen toxicity index allowing for the regression of effects at low inspired oxygen partial pressures. *RNPL Report 4/76*, Ministry of Defence, London.
- Hills, B. A. (1976c). A cumulative oxygen toxicity index which can decrease with subtoxic breathing mixtures. *Undersea Biomed. Res.*, **3**, A22.
- Hills, B. A., and B. D. Butler. (1976). The kangaroo rat as a model for Type I decompression sickness. *Undersea Biomed. Res.*, **3**, A34.
- Hills, B. A., and A. N. Dossett. (1968). Predicting the occurrence of oxygen convulsions. 1—A test of the principle of superposition, *Report UPS 276*, MRC, (U.K.).
- Hills, B. A., and D. C. Grulke. (1975). Evaluation of ultrasonic bubble detectors *in vitro* using calibrated microbubbles at selected velocities. *Ultrasonics*, **13**, 181–184.

- Hills, B. A., and E. Kuonen. (1973). Longitudinal dispersion of composition differences in the lung. *Math. Biosciences*, **18**, 351-364.
- Hills, B. A., and D. H. LeMessurier. (1969). Unsaturation in living tissue relative to the pressure and composition of inhaled gas and its significance in decompression theory. *Clinical Science*, **36**, 185-195.
- Hills, B. A., and Y. L. Ng. (1974). Significance of the contact angle in studies of lung surfactant. *J. Physiol.*, **241**, 52-53P.
- Hills, B. A., and R. Straley. (1972). Aseptic osteonecrosis: a study of tibial blood flow under various environmental conditions. *Aerospace Med.*, **43**, 724-728.
- Hills, B. A., E. L. Beckman, and J. A. Moore. (1976). The zero-supersaturation approach to decompression. In: *Development of Decompression Procedures for Depths in Excess of 400 Feet*. Ed. by R. W. Hamilton. UMS, Washington, pp. 100-118.
- Hilton, J. G., and C. H. Wells. (1976). Effects of nicotinic acid on plasma volume loss of experimental dysbarism. In: *Undersea Biomedical Research*, Undersea Medical Society, Washington, vol. 3, pp. 157-161.
- Hlastala, M. P., and L. E. Fahri. (1973). Absorption of gas bubbles in flowing blood. *J. Appl. Physiol.*, **35**, 311-316.
- Hochachka, P. W., and K. B. Storey. (1975). Metabolic consequences of diving in animals and man. *Science*, **187**, 613-621.
- Hoff, C. A. (1948). *A Bibliographic Sourcebook of Compressed Air, Diving and Submarine Medicine*. Govt. Printing Office, Washington.
- Holland, J. A. (1969). Discussion of disseminated intravascular coagulation in decompression sickness. *Report 585*, U.S. Naval Sub. Med. Center, Groton, Conn.
- Holmgren, A. (1966). The oxygen conduction line of the human body. In: *Proc. Int. Symp. Cardiovasc. Respir. Effects of Hypoxia*. Ed. by D. Hatcher and D. B. Jennings. Hafner, New York, pp. 391-400.
- Hong, S. K., J. R. Claybaugh, V. Frattali, R. Johnson, F. Kurata, M. Matsuda, A. McDonough, C. V. Paganelli, R. M. Smith, and P. Webb. (1976). Body fluid balance during a 24 day dry heliox saturation dive to 18.6 ATA (Hana Kai II). *Undersea Biomed. Res.*, **3**, A25.
- Hoppe-Seyler, F. (1857). Ueber den Einfluss, welchen der Wechsel des Luftdruckes auf das Blut ausubt. *Arch. Anat. Physiol.*, **24**, 63-73.
- Horton, J. W., and C. H. Wells. (1973). Resonance measurements of microscopic gas bubbles. *Internat. Symp. on Decompression Gas Bubbles*, Seattle.
- Houston, C. S. (1947). Occurrence of bends, scotomata and hemianopsia at altitudes below 20,000 feet. *J. Aviat. Med.*, **18**, 165-168.
- Howard, R. S., and H. Bradner. (1976). Theory and evaluation of the single pneumatic resistor decompression computer. *Scripps Institute Report*, La Jolla.
- Hueter, T. F. (1951). On the biological effects produced by ultrasound. *Chem. Eng. Progr.*, **47**, 57-68.
- Hughes, G. M., and G. A. Kerkut. (1965). Electrical activity in a slug ganglion in relation to the concentration of Locke solution. *J. Exp. Biol.*, **33**, 282-291.
- Hunsaker, J. C. (1935). Cavitation research. *Mech. Engng. N.Y.*, **57**, 211-216.
- Huntec. (1964). *Application of Ultrasonics to the Aetiology of Decompression Sickness: A Feasibility Study*. R & D Div. Huntec Limited, Toronto.
- Hunter, W. L., and P. B. Bennett. (1974). The causes, mechanisms and prevention of the high pressure nervous syndrome. *Undersea Biomed. Res.*, **1**, 1-28.
- Hyman, C., R. L. Paidino, and E. Zimmermann. (1963). Local regulation of effective blood flow in muscle. *Circulation Res.*, **12**, 176-181.
- Idicula, J., D. J. Graves, and J. A. Quinn. (1975). Bubble formation resulting from the steady counter-diffusion of two inert gases. In: *Proc. Vth Symp. Underwater Physiology*. Fed. Amer. Socs. Exp. Biol., Washington.
- Ikels, K. G. (1969). Physical-Chemical Aspects of Bubble Formation, "USAF" Task No. 7758-02.
- Ikels, K. G. (1970). Production of gas bubbles in fluids by tribonucleation. *J. Appl. Physiol.*, **28**, 524-527.
- Immelman, E. J., S. Bank, H. Krige, and I. N. Marks. (1964). Roentgenologic and clinical features of intramedullary fat necrosis in bones in acute and chronic pancreatitis. *Amer. J. Med.*, **36**, 96-105.
- Ingvar, D. H., and N. A. Lassen. (1962). Regional blood flow of the cerebral cortex determined by krypton. *Acta Physiol. Scand.*, **54**, 325.
- Ingvar, D. H., J. Adolfson, and C. Lindemark. (1973). Cerebral air embolism during training of submarine personnel in free escape: an encephalographic study. *Aerospace Med.*, **44**, 628-635.
- Inman, V. T., and J. B. Saunders. (1944). Referred pain from skeletal structures. *J. Nerv. Ment. Dis.*, **99**, 660-667.
- Iyengar, K. S., and E. G. Richardson. (1958). Measurement on the air nuclei in natural water which give rise to cavitation. *Brit. J. Appl. Physics*, **9**, 154-158.
- Jacobs, M. H., and D. R. Stewart. (1942). Observations on the blood of albino rats following rapid decompression. In: *Committee of Aviation Medicine Report 76*. U.S. National Research Council, Washington, pp. 1-5.
- James, C. C. C. (1945). Late bone lesions in caisson disease. Three cases in submarine personnel. *Lancet*, **ii**, 6.
- Japp, H. (1909). Caisson disease and its prevention. *Trans. Amer. Soc. Civil Engrs.*, **65**, 1-3.
- Johnson, F. H., and E. A. Flagler. (1950). Hydrostatic pressure reversal of narcosis in tadpoles. *Science*, **112**, 91-92.
- Johnson, J. A., H. M. Cavert, and N. Lifson. (1952).

- Kinetics concerned with distribution of isotopic water in isolated perfused dog heart and skeletal muscle. *Amer. J. Physiol.*, **171**, 687-693.
- Jones, H. B. (1951). Gas exchange and blood-tissue perfusion factors in various body tissues. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, Philadelphia, pp. 278-321.
- Jones, H. B., C. Tobias, W. F. Loomis, J. B. Mohney, W. N. Sears, J. C. Larkin, J. G. Hamilton, and J. H. Lawrence. (1942). An objective method for the study of the physiology of aeroemphysema and for the selection of high altitude aircrew using the radioactive isotopes of inert gases. *Report 81*, U.S. NRC, *Comm. Aviat. Med.*, Washington.
- Jones, J. P. (1974). Orthopedic management and treatment of osteonecrosis. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington.
- Jones, J. P., E. P. Engleman, and J. S. Najarian. (1965). Systemic fat embolism after renal homotransplantation and treatment with corticosteroids. *New Engl. J. Med.*, **273**, 1453-1458.
- Jones, P. J., L. Sakovich, and C. E. Anderson. (1974). Experimentally produced osteonecrosis as a result of fat embolism. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 117-132 (and discussion p. 143).
- Kahlstrom, S. C., C. C. Burton, and D. B. Phemister. (1939). Aseptic necrosis of bone: II. Infarction of bones of undetermined etiology resulting in encapsulated and calcified areas in diaphyses and in arthritis deformans. *Surg. Gynec. Obstet.*, **68**, 631-641.
- Kalser, M. H., H. K. Ivy, L. Pevsner, J. P. Marbarger, and A. C. Ivy. (1951). Changes in bone marrow pressure during exposure to simulated altitude. *J. Aviat. Med.*, **22**, 286-294.
- Karpovich, P. V. (1944). Relation between bends and physical fitness. *Air Surg. Bull.*, **1**, 5.
- Keays, F. L. (1909). Compressed air illness, with a report of 3,692 cases. Researches from the Department of Medicine, Cornell University Medical College, Ithaca, New York.
- Keidel, W. D., and W. D. Neff. (1974). (Ed.). *Handbook of Sensory Physiology. VI: Auditory System, Anatomy, Physiology (Ear)*. Springer-Verlag, Berlin, p. 653.
- Keller, H., and A. A. Bühlmann. (1965). Deep diving and short decompression by breathing mixed gases. *J. Appl. Physiol.*, **20**, 1267.
- Kennedy, R. S. (1974). General history of vestibular disorders in diving. *Undersea Biomed. Res.*, **1**, 73-82.
- Kennedy, R. S., and J. A. Dachenko. (1975). Incidence of vestibular symptomatology in 2,500 U.S. Navy diving accidents (1933-1970). *Aviat. & Space Environ. Med.*, **46**, 432-435.
- Kenrick, F. B., C. S. Gilbert, and K. L. Wismer. (1924a). The superheating of liquids. *J. Phys. Chem.*, **28**, 1297-1307.
- Kenrick, F. B., K. L. Wismer, and K. S. Wyatt. (1924b). Supersaturation of gases in liquids. *J. Phys. Chem.*, **28**, 1308-1315.
- Kent, E. M., and B. Blades. (1942). Experimental observations upon certain intracranial complications of particular interest to the thoracic surgeon. *J. Thor. Surg.*, **11**, 434-445.
- Kent, H. (1965). Abuses and uses of ultrasound. *Oklahoma State Med. Assoc. J.*, **58**, 13.
- Kessler, J., and R. H. Patterson. (1970). The production of microemboli by various blood oxygenators. *Ann. Thorac. Surg.*, **9**, 221-228.
- Kety, S. S. (1951). Theory and applications of exchange of inert gases at lungs and tissue. *Pharmac. Rev.*, **3**, 1-41.
- Kety, S. S. (1960). The cerebral circulation. In: *Handbook of Physiology* Section 1: Neurophysiology. Ed. by J. Field, H. W. Magoun and V. E. Hall. Williams & Wilkins, Baltimore, pp. 1751-1960.
- Kety, S. S. and C. F. Schmidt. (1945). The determination of cerebral blood flow in man by the use of nitrous oxide in low concentrations. *Amer. J. Physiol.*, **143**, 53-56.
- Kidd, D. J. (1969). Use of the pneumatic analogue computer for divers. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 386-413.
- Kidd, D. J., and D. H. Elliott. (1969). Clinical manifestations and treatment of decompression sickness in divers. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 464-490.
- Kidd, D. J., and D. H. Elliott. (1975). Decompression disorders in divers. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. 2nd ed. Baillière & Tindall, London, pp. 471-495.
- Kidd, D. J., R. A. Stubbs, and R. S. Weaver. (1972). Comparative approaches to prophylactic decompression. In: *Proc 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 167-178.
- Kimble, S. T. (1961). Fatal nontraumatic fat embolism in an alcoholic. *Med. Ann. D. C.*, **30**, 283-287, 314.
- Kindwall, E. P. (1973). *Hyperbaric Medicine Procedures*. St. Luke's Hospital, Milwaukee.
- Kindwall, E. P. (1974). Milwaukee sewerage project. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, pp. 41-46.
- Kindwall, E. P. (1975). Measurement of helium elimination from man during decompression breathing air or oxygen. In: *Undersea Biomedical Research*. Undersea Medical Society, Washington, vol. 2, pp. 277-284.

- Kindwall, E. P., A. Baz, E. N. Lightfoot, E. H. Lanphier, and A. Seireg. (1975). Nitrogen elimination in man during decompression. In: *Undersea Biomedical Research*. Undersea Medical Society, Washington, vol. 2, pp. 285–297.
- Kleinfeld, M., and J. T. Wilson. (1956). Decompression sickness (compressed air illness) in a tunnelling operation. *A.M.A. Arch. Ind. Hlth.*, **14**, 539–542.
- Knapp, R. T. (1952). Cavitation mechanics and its relation to the design of hydraulic equipment. *Proc. Inst. Mech. Eng.*, **166**, 150–163.
- Kooyman, G. L. (1966). Maximum diving capacities of the Weddell seal, *Leptonychotes Weddelli*. *Science*, **151**, 1553–1554.
- Kooyman, G. L. (1973). Respiratory adaptations in marine mammals. *Amer. Zool.*, **13**, 457–468.
- Krasberg, A. (1966). Saturation diving techniques. In: *Proc. Fourth Int. Congress Biometeorology*. Rutgers University, New Brunswick, New Jersey.
- Krasberg, A. (1976). Use of alcohol and nitrogen to prevent high pressure nervous syndrome. In: *Proc. Int. Diving Symp. Assoc. Diving Contractors*, New Orleans.
- Krogh, A. (1907). Some new methods for the tonometric determination of gas tensions in fluids. *Skand. Arch. Physiol.*, **20**, 259.
- Krogh, A. (1918). The rate of diffusion of gases through animal tissues, with some remarks on the coefficient of invasion. *J. Physiol.*, **52**, 391.
- Krogh, A. (1941). *Comparative Physiology of Respiratory Mechanisms*. Pennsylvania University Press, Philadelphia.
- Krueger, P. G., and E. J. Reed. (1976). Biological impact of small air ions. *Science*, **193**, 1209–1213.
- Kuehn, L. A., and R. Y. Nishi. (1975). The use of decompression computers in diving. In: *Proc. Symp. Chemistry & Physics of Aqueous Gas Solutions, Electrochemical Soc.*, pp. 486–497.
- Kylstra, J. A. (1968). Experiments in water breathing. *Scient. Amer.*, **219**, 66–74.
- Kylstra, J. A., C. V. Paganelli, and E. H. Lanphier. (1966). Pulmonary gas exchange in dogs ventilated with hyperbarically oxygenated liquid. *J. Appl. Physiol.*, **27**, 177–184.
- Kylstra, J. A., R. Nantz, J. Crowe, W. Wagner, and H. A. Saltzman. (1967). Hydraulic compression of mice to 166 atmospheres. *Science, N.Y.*, **158**, 793–794.
- Kylstra, J. A., I. S. Longmuir, and M. Grace. (1968). Dysbarism; a study of gas osmosis. *Science, N.Y.*, **161**, 289.
- Lambertsen, C. J. (1955). Respiratory and circulatory actions of high pressure. In: *Proc. 1st Symp. Underwater Physiology*. Ed. by L. G. Goff. NAS, NRC, Washington, pp. 25–38.
- Lambertsen, C. J. (1965). Effects of oxygen at high partial pressure. In: *Handbook of Physiology*. Section 3. Ed. by W. O. Fenn and H. Rahn. Amer. Physiol. Soc., Washington, vol. 2, pp. 1027–1046.
- Lambertsen, C. J. (1966). Physiological effects of oxygen inhalation at high partial pressures. In: *Fundamentals of Hyperbaric Medicine*. NAS, NRC, Washington, pp. 12–20.
- Lambertsen, C. J. (1967). Basic requirements for improving diving depth and decompression tolerance. In: *Proc. 3rd Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Williams & Wilkins, Baltimore, pp. 223–240.
- Lambertsen, C. J. (1970). *A Quantitative Method for Calculating Cumulative Pulmonary Oxygen Toxicity Use of Unit Pulmonary Toxicity Dose (UPTD)*. Pennsylvania University Press, Philadelphia.
- Lambertsen, C. J. (1975). Collaborative investigation of limits of human tolerance to pressurization with helium, neon and nitrogen. Simulation of density equivalent to helium–oxygen respiration at depths to 2,000, 3,000, 4,000, and 5,000 feet of sea water. In: *Proc. 5th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Federation of American Societies of Experimental Biology, Washington, pp. 35–48.
- Lambertsen, C. J., and S. G. Owen. (1960). Continuous rate sampling modification of nitrous oxide method for cerebral blood flow in man. In: *Methods in Medical Research*. Ed. by H. B. Bruner. Year Book Medical Publ., Chicago.
- Landis, E., and J. R. Pappenheimer. (1963). Exchange of substances through the capillary walls. In: *Handbook of Physiology*. Section 2, Amer. Physiol. Soc., Washington, vol. 2, ch. 29.
- Lanphier, E. H. (1969). Pulmonary function. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, pp. 58–112.
- Lanphier, E. H., and I. W. Brown. (1966). The physiological basis of hyperbaric therapy. In: *Fundamentals of Hyperbaric Medicine*. NAS, NRC, Washington, pp. 33–55.
- Lansing, A. I. (1944). Treatment of aeroembolism. *Air Surg. Bull.*, **1**, 5.
- Larson, H. E. (1959). A history of self-contained diving and underwater swimming. *Publ. 469*. NAS, NRC, Washington.
- Lassen, N. A., and A. Klee. (1965). Cerebral blood flow determined by saturation and desaturation with krypton: evaluation of the inert gas method of Kety and Schmidt. *Circ. Res.*, **16**, 26–32.
- Lassen, N. A., and O. Munck. (1954). The cerebral blood flow in man determined by the use of radioactive krypton. *Acta Physiol. Scand.*, **33**, 30–49.
- Lategola, M. T. (1964). Measurement of total pressure of dissolved gases in mammalian tissue *in vivo*. *J. Appl. Physiol.*, **19**, 322–324.
- Lederer, F. L., and A. R. Hollender. (1951). *Textbook of Ear, Nose and Throat*. 3rd ed. Davis, Philadelphia, p. 282.
- Ledingham, M. C. A., D. G. McDowall, and A. M. Harper. (1966). Cerebral cortical blood flow under hyperbaric conditions. In: *Proc. 3rd Internat. Conf. on Hyperbaric Med.* Ed. by I.W. Brown and B. W.

- Cox. Publ. 1404, NAS, Washington, pp. 243–248.
- Lee, W. H., and P. Hairston. (1971). Structural effects on blood proteins at the gas-blood interface. *Fed. Proc.*, **30**, 1615–1620.
- LeMessurier, D. H. (1972). Supersaturation and performed nuclei in the etiology of decompression sickness. Paper presented at the 2nd International Meeting on Aerospace Medicine, Melbourne.
- LeMessurier, D. H., and B. A. Hills. (1965). Decompression sickness: A study of diving techniques in the Torres Strait. *Hvaldradets Skrifter*, **48**, 54–84.
- Lever, M. J., K. W. Miller, W. D. M. Paton, and E. B. Smith. (1966). Experiments on the genesis of bubbles as a result of rapid decompression. *J. Physiol. London.*, **184**, 964–969.
- Lever, M. J., W. D. M. Paton, and E. B. Smith. (1971a). Decompression characteristics of inert gases. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 123–136.
- Lever, M. J., K. W. Miller, W. D. M. Paton, and E. B. Smith. (1971b). Pressure reversal of anaesthesia. *Nature*, **231**, 368–371.
- Levy, E. (1922). Compressed-air illness and its engineering importance, with a report of cases at the East River Tunnels. *Tech. Pap. Bur. Mines*, **285**, Washington, p. 47.
- Liebermann, L. (1957). Air bubbles in water. *J. Appl. Physics*, **28**, 205–211.
- Light, L. H. (1972). Ultrasonic Doppler techniques in blood velocity measurement. In: *Fluid Dynamic Measurements in the Industrial and Medical Environments*. Ed. by D. J. Cockrell. Leicester University Press, Leicester, vol. 1.
- Longmuir, I. S. (1966). Tissue oxygen transport. In: *Hyperbaric Medicine*. Ed. by I. W. Brown and B. G. Cox. NRC, Washington, pp. 46–51.
- Longmuir, I. S., and M. Grace. (1969). Physiological effect of the osmotic pressure of dissolved gas. *Fedn. Proc. Fedn. Amer. Soc. Exp. Biol.*, **28**, 720.
- Lowry, O. H., D. R. Gilligan, and E. M. Katersky. (1941). The determination of collagen and elastin in tissues, with results obtained in various normal tissues from different species, *J. Biol. Chem.*, **139**, 795–804.
- Lynch, M. J. G., S. S. Raphael, and T. P. Dixon. (1959). Fat embolism in chronic alcoholism: Control study of incidence of fat embolism. *Arch. Path.*, **67**, 68–80.
- Lyttleton, A. (1855). Quoted by Hoff (1948).
- McCallum, R. I. (1975). Dysbaric Osteonecrosis: aseptic necrosis of bone. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. 2nd ed. Baillière & Tindall, London, ch. 27.
- McCallum, R. I., D. N. Walder, and V. B. Thickett. (1976). Bone necrosis in commercial divers. *Undersea Biomed. Res.*, **3**, A41.
- McCollum, D. E., R. S. Mathews, and M. T. O'Neil. (1971). Gout, hyperuricemia and aseptic necrosis of the femoral head. In: *Idiopathic Ischemic Necrosis of the Femoral Head in Adults*. Ed. by W. M. Zinn. Thieme, Stuttgart, Germany, ch. 9, pp. 133–139.
- McCormick, J. G. (1976). Slide shown at a Navy-wide Workshop in High Pressure Biomedical Research. Panama City, Florida.
- McDowall, D. G. (1966). Hyperbaric oxygen in relation to acute trauma. *Brit. J. Anaesth.*, **38**, 308–316.
- McElroy, W. D., A. H. Whiteley, G. H. Warren, and E. N. Harvey. (1944a). Bubble formation in animals. IV. The relative importance of CO₂ concentration and mechanical tension during muscle contraction. *J. Cell. Comp. Physiol.*, **24**, 133–146.
- McElroy, W. D., A. H. Whiteley, K. W. Cooper, D. C. Pease, G. H. Warren, and E. N. Harvey. (1944b). Bubble formation in animals. VI. Physiological factors: the role of circulation and respiration. *J. Cell. Comp. Physiol.*, **24**, 273–290.
- McIver, R. C., W. P. Fife, and K. G. Ikels. (1965). Experimental Decompression Sickness from Hyperbaric Nitrous Oxide Anaesthesia. *Report SAM-TR-65-47*. USAF School of Aerospace Med.
- Maio, D. A., and L. E. Farhi. (1967). Effect of gas density on mechanics of breathing. *J. Appl. Physiol.*, **23**, 687–693.
- Mandlebaum, I., and H. King. (1963). Pulmonary air embolism. *Surg. Forum*, **14**, 236–238.
- Manley, D. M. J. P. (1969). Ultrasonic detection of gas bubbles in blood. *Ultrasonics*, **7**, 102–105.
- Mapleson, W. W. (1963). An electric analogue for uptake and exchange of inert gases and other agents. *J. Appl. Physiol.*, **18**, 197–204.
- Marchand, P., H. van Hasselt, and C. H. Luntz. (1964). Massive venous air embolism. *S. Afr. Med. J.*, **38**, 202–208.
- Marsh, M. C., and F. P. Gorham. (1904). The Gas Disease in Fishes. U.S. Government Report of the Commissioner of Fisheries.
- Marsland, D. (1970). Pressure temperature studies on the mechanism of cell division. In: *High Pressure Effects on Cellular Processes*. Ed. by A. M. Zimmerman. Academic Press, New York, pp. 260–306.
- Martin, K. J. (1972). Observations on haematological and biochemical parameters. In: *The Association of Clinical Pathologists: 89th General Meeting. Symp. II. Decompression Sickness*. *J. Clin. Pathol.*, **25**, 1004–1005.
- Martin, K. J., and G. Nichols. (1971). Changes in platelets in man after simulated diving. *RNPL Report 5/71*. Ministry of Defence, London.
- Martin, K. J., and G. Nichols. (1972). Further studies on platelet changes in man after simulated diving. *RNPL Report*, Ministry of Defence, London.
- Masland, R. L. (1943). Review of cases of collapse occurring in altitude chambers. *USAF Report 179*,

- NRC, *Comm. Aviat. Med.*, Washington.
- Matthews, B. H. C. (1939). Quoted by Fryer (1969).
- Matthews, W. H., and J. A. Kylstra. (1976). Investigation of a new breathing liquid. In: *Proc. Sixth Symp. Underwater Physiology*. UMS, Washington.
- Mauro, A. (1960). Some properties of ionic and nonionic semipermeable membranes. *Circulation*, **21**, 845–858.
- Mauvoisin, F., J. Bernard, and J. Germain. (1955). Aspect tomographic des hanches chez un Goutteux. *Rev. Rhum.*, **22**, 336.
- Mayer, J. E. (1937). The statistical mechanics of condensing systems 1. *J. Chem. Physics*, **5**, 67–83.
- Mediflor. (1975). *Fluorocarbon Liquid M-6015 (FC-80)*. 3M Company, St. Paul.
- Meldrum, B. S., J. J. Papy, and R. A. Vigouroux. (1971). Intracarotid air embolism in the baboon; effects on cerebral blood flow and the electroencephalogram. *Brain Res.*, **25**, 301–315.
- Meyer-Breslav, J. (1911). Zur Kenntnis des Negativen Druckes in Flüssigkeiten. *Z. Electrochem.*, **17**, 743–745.
- Miers, H. A., and F. Isaac. (1908). The spontaneous crystallisation of substances which form a continuous series of mixed crystals: Mixtures of naphthalene and β naphthol. *J. Chem. Soc.*, **93**, 927–936.
- Miles, S. (1962). *Underwater Medicine*. Staples, London.
- Miles, S., and D. E. Mackay. (1959). The nitrogen narcosis hazard and the self-contained diver. *UPS Report 184*, R. N. Pers. Res. Comm., MRC, London.
- Milhorn, H. T. (1966). *Application of Control Theory to Physiological Systems*. Saunders, Philadelphia.
- Miller, K. W., and E. B. Smith. (1973). Intermolecular forces and the pharmacology of simple molecules. In: *A Guide to Molecular Pharmacology-Toxicology*. Ed. by R. M. Featherstone, Dekker, New York, Pt. II.
- Miller, K. W., W. D. M. Paton, and E. B. Smith. (1967). The anesthetic pressures of certain fluorine-containing gases. *Brit. J. Anaesthesia*, **39**, 910–918.
- Miller, S. L. (1961). A theory of gaseous anaesthetics. *Proc. Nat. Acad. Sci.*, **47**, 1515–1524.
- Milsum, J. H. (1966). *Biological Control Systems Analysis*. McGraw-Hill, New York.
- Mochizuki, M., and H. Bartels. (1955). Electrochemische Methoden zur Messung des O_2 -Druckes im Vollblut. *Monogr. Res. Inst. Applied Electr.*, **5**, 53.
- Moir, E. W. (1896). Tunnelling by compressed air. *J. Roy. Soc. Arts.*, **45**, 15.
- Molumphy, C. G. (1950). Computation of helium-oxygen decompression tables. *Research Report 7–50*. U.S. Navy Experimental Diving Unit, Washington.
- Momsen, C. B., and K. R. Wheland. (1939). *Report on use of Helium-oxygen Mixtures for Diving (Revised 1942)*. U.S. Navy Experimental Diving Unit, Washington.
- Money, K. (1976). Presentation at a Navy-wide workshop on High Pressure Biomedical Research. USN, Panama City.
- Moses, H. (1964). Casualties in individual submarine escape. *USN Submarine Med. Center Report 438*, Proj. MR005. 14–3002–4. 17. U.S.N. Bureau Med. & Surg., Washington.
- Motley, H. L., H. I. Chinn, and F. A. Odell. (1945). Studies on bends. *J. Aviat. Med.*, **16**, 210–234.
- MRC. (1966). Bone lesions in compressed air workers with special reference to men who worked on the Clyde Tunnels (1958–1963). Decompression Sickness Panel Report. *J. Bone & Jt. Surg.*, **48B**, 207–235.
- MRC. (1971). Decompression sickness and aseptic necrosis of bone. Investigations carried out during and after the construction of the Tyne Road Tunnel (1962–1966). Decompression Sickness Panel Report. *Brit. J. Ind. Med.*, **28**, 1–21.
- MRC. (1974). Construction Industry Research and Information Association. Decompression Sickness Panel Report, London.
- MRC. (1975). Minutes of meeting of the Decompression Sickness Panel 30th September 1975. MRC, London.
- Muysers, K. (1970). Gibt es eine Stickstoffabgabe über die Menschliche Lunge. *Pflügers Arch. Ges. Physiol.*, **317**, 157–172.
- Muysers, K., U. Smidt, G. von Nieding, H. Krekeler, and K. E. Schaefer. (1974). Diffusional and metabolic components of nitrogen elimination through the lungs. *J. Appl. Physiol.*, **37**, 32–37.
- Nachemson, A. L., and J. H. Evans. (1968). Some mechanical properties of the third human lumbar interlaminar ligament. (LIGAMENTUM FLAVUM). *J. Biomechanics*, **1**, 211–220.
- Naquet, R. (1966). Epileptic activity evoked by air embolism in cat, monkey, and man. In: *Proc. Int. Symp. Comp. Cell, Pathophysiol.* Ed. by Servit. Epilepsy, Liblice, Excerpta Medica, Amsterdam, pp. 89–102.
- Nashimoto, I., and Y. Gotoh. (1976). Relationships between precordial Doppler ultrasound records and decompression sickness. *Sixth Symp. Underwater Physiology* (in press).
- Nellen, J. R., and E. P. Kindwall. (1972). Aseptic necrosis of bone secondary to occupational exposure to compressed air. Roentgenologic findings in 59 cases. *Amer. J. Roentg. Radium Ther. Nucl. Med.*, **115**, 512–524.
- Nelson, G. E., P. J. Kelly, L. F. Peterson, and J. M. Jones. (1960). Blood supply of the human tibia. *J. Bone & Joint Surg.*, **42A**, 625–636.
- Neuman, W. F., and M. W. Neuman. (1957). Emerging concepts of the structure and metabolic functions of bone. *Amer. J. Med.*, **22**, 123–131.
- Neuman, W. F., and M. W. Neuman. (1958). *The Chemical Dynamics of Bone Mineral*. Chicago University Press, Chicago.
- Nicholas, D. J. D. (1963a). Biochemistry of nitrogen

- fixation. *Proc. Symp. Soc. Gen. Microbiol.*, pp. 92–124.
- Nicholas, D. J. D. (1963b). The metabolism of inorganic nitrogen and its compounds in micro-organisms. *Biol. Rev. Cambridge Phil. Soc.*, **38**, 530–568.
- Niden, A. H., and D. M. Aviado. (1956). Effects of pulmonary embolism on the pulmonary circulation with special reference to arteriovenous shunts in the lung. *Circulation Res.*, **4**, 67–73.
- Nims, L. F. (1951). Environmental factors affecting decompression sickness. In: *Decompression Sickness*. Ed. by J. F. Fulton. Saunders, Philadelphia, ch. 8.
- Nishi, R. Y., and S. D. Livingstone. (1973). Intravascular changes associated with hyperbaric decompression: theoretical considerations using ultrasound. *Aerospace Med.*, **44**, 179–183.
- NOAA. (1975). *The NOAA Diving Manual*. Govt. Printing Office, Washington.
- Noltingk, B. E., and E. A. Neppiras. (1950). Cavitation produced by ultrasonics. *Proc. Roy. Soc. London*, **63B**, 674–685.
- Noltingk, B. E., and Neppiras, E. A. (1951). Theoretical conditions for the onset of cavitation. *Proc. Roy. Soc. London*, **64B**, 1032–1038.
- Ogston, A. G. (1956). *Proc. Internat. Wood Textile Research Conference* (C.S.I.R.O., Melbourne), vol. B, p. 92.
- Ohta, Y., and O. Shigeto. (1969). Quoted by Elliott and Harrison (1970).
- O'Neill, W. J. (1970). A study of the breathing hydrostatics of various bag-type diving apparatuses and description of the two-valve toroidal and abalone back-mounted design. In: *Proc. Symp. Equipment for the Working Diver*. Marine Technol. Soc., Washington, pp. 183–228.
- Oser, H. (1975). Current work at D.F.V.L.R. *Proc. Symp. on Development of Decompression Procedures for Depths in Excess of 400 feet*. UMS, Washington.
- Ostwald, W. (1897). Studien über die Bildung und Umwandlung fester Körper. *Z. Phys. Chem.*, **22**, 289–330.
- Ostwald, W. (1900). Über die vermeintliche isomerie des roten und gelben Quecksilberoxyds und die Oberflächen-spannung fester Körper. *Z. Phys. Chem.*, **34**, 493–503.
- Overfield, E. M., and J. A. Kylstra. (1971). Change in (A-a)DO₂ as a function of F_{IO₂} at a constant P_{IO₂} in normal man. *J. Appl. Physiol.*, **31**, 581–585.
- Oyama, Y., and M. P. Spencer. (1971). Cardiopulmonary effects of intravenous gas embolism; with special reference to fate of intravascular gas bubbles. *Jap. Circ. J.*, **35**, 1541–1549.
- Paintal, A. S. (1969). Mechanisms of stimulation of type-J receptors. *J. Physiol. London*, **203**, 511.
- Pask, E. A. (1942). Preliminary note on a method of investigating the site of origin of pain in decompression sickness. *FPRC Report 484*, Air Ministry, London.
- Patel, D. J., R. N. Vaishnav, B. S. Gow, and P. A. Kot. (1974). Hemodynamics. *Ann. Rev. Physiol.*, **36**, 125–134.
- Paton, W. D. M. (1967). Experiments on the convulsant and anaesthetic effects of oxygen. *Brit. J. Pharm.*, **29**, 350–366.
- Paton, W. D. M., and D. N. Walder. (1954). Compressed air illness—an investigation during the construction of the Tyne Tunnel, 1948–1950. *Spec. Report Ser. No. 281*, MRC, London.
- Pattle, E. R. (1966). Surface tension and the lining of the lung alveoli. In: *Advances in Respiratory Physiology*. Ed. by C. G. Caro. Edward Arnold Limited, London.
- Pauling, L. (1961). A molecular theory of general anaesthesia. *Science*, **134**, 15–21.
- Pease, D. C., and L. R. Blinks. (1947). Cavitation from solid surfaces in the absence of gas nuclei. *J. Phys. Chem., Ithaca*, **51**, 556–567.
- Peck, A. S. (1974). Discussion session. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, p. 8.
- Pellegrini, L. (1963). Working at depth without decompression. *Proc. 2nd World Conf. Underwater Activities*. Brit. Sub-Aqua Club, London, pp. 133–138.
- Perl, W., H. Rackow, E. Salanitro, G. L. Wolf, and R. M. Epstein. (1965). Intertissue diffusion effect for inert fat-soluble gases. *J. Appl. Physiol.*, **20**, 621–627.
- Perry, J. H. (1950). *Chemical Engineers' Handbook*. 3rd ed. McGraw-Hill, New York, pp. 540–541.
- Peterson, R. E. (1976). An unscheduled presentation at: *International Diving Symposium*. New Orleans.
- Philp, R. B. (1964). The ameliorative effects of heparin and depolymerized hyaluronate on decompression sickness in rats. *Can. J. Physiol. Pharmacol.*, **42**, 819–829.
- Philp, R. B. (1974). A review of blood changes associated with compression-decompression: relationship to decompression sickness. *Undersea Biomed. Res.*, **1**, 117–150.
- Philp, R. B., and C. W. Gowdey. (1964). Experimental analysis of the relation between body fat and susceptibility to decompression sickness. *Aerospace Med.*, **35**, 351–356.
- Philp, R. B., C. W. Gowdey, and M. Prasad. (1967). Changes in blood lipid concentration and cell counts following decompression sickness in rats and the influence of dietary lipid. *Can. J. Physiol. Pharmacol.*, **45**, 1047–1059.
- Philp, R. B., P. Schacham, and C. W. Gowdey. (1971). Involvement of platelets and microthrombi in experimental decompression sickness: similarities with disseminated intravascular coagulation. *Aerospace Med.*, **42**, 494–502.
- Philp, R. B., M. J. Inwood, and B. A. Warren. (1972). Interactions between gas bubbles and components of the blood. Implications in decompression

- sickness. *Aerospace Med.*, **43**, 946-953.
- Philp, R. B., M. J. Inwood, K. N. Ackles, and M. W. Radomski. (1974). Effects of decompression on platelets and hemostasis in man and the influence of antiplatelet drugs. (RA233 and VK744). *Aerospace Med.*, **45**, 231-240.
- Piccard, J. (1941). Aero-emphysema and the birth of gas bubbles. *Proc. Mayo Clin.*, **16**, 700-704.
- Pierce, E. C. (1967). II: The membrane lung. Its excuse, present status and promise. *J. Mt. Sinai Hosp.*, **34**, 437-468.
- Pietrogrande, V., and R. Mastromarino. (1957). Osteopatia da prolungato trattamento cortisonico. *Ortop. Traumatol.*, **25**, 791-810.
- Piiper, J., R. E. Canfield, and H. Rahn. (1962). Absorption of various inert gases from subcutaneous gas pockets in rats. *J. Appl. Physiol.*, **17**, 268-274.
- Pilmanis, A. A. (1976). Intravenous gas emboli in man after compressed air ocean diving. *ONR Contract Report N00014-67-A-0269-0026*. Office of Naval Research, Washington.
- Pittinger, C. B., R. M. Featherstone, E. Stickley, L. Levy, and S. C. Cullen. (1956). Observations on the kinetics of transfer of xenon and chloroform between blood and brain in the dog. *Anesthesiology*, **17**, 523-530.
- Plesset, M. S. (1963). The pulsation method for generating cavitation damage. *J. Basic Eng.*, **85**, 360-364.
- Pol, B., and T. J. J. Watelle. (1854). Mémoire sur les effets de la compression de l'air, appliquées au creusement des puits a houille. *Ann. Hyg. Publ.* 2° Ser., **1**, 241-279.
- Poulton, E. C., M. J. Catton, and A. Carpenter. (1964). Efficiency at sorting cards in compressed air. *Brit. J. Industr. Med.*, **21**, 242-245.
- Powell, C. F. (1928). Condensation phenomena at different temperatures. *Proc. Roy. Soc., London*, **A119**, 553-577.
- Powell, M. R. (1972a). Leg pain and gas bubbles in the rat following decompression from pressure: monitoring by ultrasound. *Aerospace Med.*, **43**, 168-172.
- Powell, M. R. (1972b). Gas phase separation following decompression in asymptomatic rats: visual and ultrasonic monitoring. *Aerospace Med.*, **43**, 1240-1244.
- Poyart, C. F., E. Bursaux, and A. Fréminet. (1975). The bone CO₂ compartment: evidence for a bicarbonate pool. *Resp. Physiol.*, **25**, 89-99.
- Poynting, J. H. (1896a). Osmotic pressure. *Phil. Mag.*, **42**, 289-299.
- Poynting, J. H. (1896b). Change of state; solid-liquid. *Phil. Med.*, **5**, 12-48.
- Price, F. P. (1969). Nucleation in polymer crystallisation. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 8.
- Pudenz, R. H. (1945). Quoted by Wagner (1945).
- Rackow, H., E. Salanitro, R. M. Epstein, G. L. Wolf, and W. Perl. (1965). Simultaneous uptake of N₂O and cyclopropane in man as a test of the compartmental model. *J. Appl. Physiol.*, **20**, 611-620.
- Rahn, H. (1967). Gas transport from the external environment to the cell. In: *Development of the Lung*. Ed. by A. V. S. de Reuck and R. Porter. Little, Brown, Boston, pp. 3-29.
- Rahn, H., and W. O. Fenn. (1955). *A Graphical Analysis of the Respiratory Gas Exchange*. Amer. Physiol. Soc., Washington.
- Raisz, L. G. (1970). Physiologic and pharmacologic regulation of bone resorption. *New Engl. J. Med.*, **282**, 909-916.
- Rait, W. L. (1959). The aetiology of post-decompression shock in aircrewmembers. *U.S. Arm. Forces Med.*, **10**, 790-804.
- Randel, H. W. (1971). *Aerospace Medicine*. 2nd ed. William & Wilkins, Baltimore, p. 5.
- Rangell, L. (1942). Cerebral air embolism. The question of arterialization of intravenous air across the barrier of the pulmonary capillaries; report of a case following assumption of the knee-chest position post partum with recovery. *J. Nerv. Ment. Dis.*, **96**, 542-555.
- Rashbass, C. (1954). Investigation into the decompression tables; a consideration of basic theories of decompression sickness. *UPS Report 139*, RNPRC, MRC, London.
- Rashbass, C. (1955). Investigation into the decompression tables. *Report VI, New Tables, UPS Report 151*, RNPRC, MRC, London.
- Rashbass, C. (1956). The aetiology of itching on decompression. *UPS Report 167*, RNPRC, MRC, London.
- Raslins, J. S. P. (1959). Quoted by Edmonds (1973).
- Renfro, W. C. (1963). Gas-bubble mortality of fishes in Galveston Bay, Texas. *Trans. Amer. Fish. Soc.*, **92**, 320-322.
- Renkin, E. M. (1955). Effects of blood flow on diffusion kinetics in isolated, perfused hindlegs of cats. A double circulation hypothesis. *Amer. J. Physiol.*, **183**, 125-136.
- Renkin, E. M. (1959). Transport of potassium-42 from blood to tissue in isolated mammalian skeletal muscles. *Amer. J. Physiol.*, **197**, 1205-1210.
- Renkin, E. M. (1967). Blood flow and transcappillary exchange in skeletal and cardiac muscle. In: *Proc. Internat. Symp. Coronary Circ. and Energetics of the Myocardium*. Ed. by G. Marchetti and B. Taccardi. Karger, Basel, pp. 18-29.
- Reynolds, O. (1878). On the internal cohesion of liquids and the suspension of a column of mercury to a height more than double that of the barometer. *Mem. Manch. Lit. Phil. Soc.*, **17**, 159-175.
- Ridgway, S. H., B. L. Sconce, and J. Kanwisher. (1969). Respiration and deep diving in the bottlenose porpoise. *Science*, **166**, 1651-1654.
- Ring, G. C., A. S. Blum, T. Kurbatov, W. D. Moss, and W. Smith. (1961). Size of microspheres passing

- through the pulmonary circuit in the dog. *Amer. J. Physiol.*, **200**, 1191–1196.
- Rivera, J. C. (1964). Decompression sickness among divers: an analysis of 935 cases. *Milit. Med.*, **129**, 314–334.
- Robertson, J. D. (1959). The ultrastructure of cell membranes and their derivatives. *Biochem. Soc. Symp.*, **16**, 3.
- Robertson, J. S. (1962). Mathematical treatment of uptake and release of indicator substances in relation to flow analysis in tissues and organs. In: *Handbook of Physiology*. Sect. 2., Circulation. Ed. by W. F. Hamilton and P. Dow. Amer. Physiol. Soc., Washington, vol. 1, ch. 19.
- Robinson, T. W. (1943). High altitude tolerance tests. *U.S. NRC Report 205*, Comm. Aviat. Med., Washington.
- Rohsenow, W. M., and H. Y. Choi. (1961). *Heat, Mass and Momentum Transfer*. Prentice-Hall, Englewood Cliffs, N. J.
- Rollhäuser, H. (1950). Konstruktions—und Altersunterscheide in Festigkeit killagemer Fibrillen. *Morph. Fb.*, **90**, 157–179.
- Rose, R. J. (1962). Survey of work in compressed air during the construction of the Auckland Harbour Bridge. *Special Report No. 6*. Medical Statistics Branch, Dept. of Health, Wellington, New Zealand.
- Ross, E. T. (1938). Crystallization. *Pacific Chem. Met. Inds.*, **2**, 9–20.
- Ross, H. C. (1976). Serotonin as a mediating factor in the development of decompression sickness. In: *Proc. Sixth Symp. Underwater Physiol.* (in press).
- Roth, S. H., and P. Seeman. (1972). The membrane concentrations of neutral and positive anaesthetics (alcohols, chlorpromazine, morphine) fit the Meyer-Overton rule of anaesthesia; negative narcotics do not. *Bioch. Biophys. Acta*, **255**, 207–219.
- Roth, S. H., R. A. Smith, and W. D. M. Paton. (1975). Pressure reversal of nitrous oxide conduction failure in peripheral nerve. In: *Proc. Vth Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Fedn. Amer. Socs. Exp. Biol., Washington.
- Roughton, F. J. W. (1952). Diffusion and chemical reaction velocity in cylindrical and spherical systems of physiological interest. *Proc. R. Soc.*, **B140**, 203–221.
- Roughton, F. J. W. (1964). Transport of oxygen and carbon dioxide. In: *Handbook of Physiology*. Sect. 3, Respiration. Ed. by W. O. Fenn and H. Rahn. Amer. Physiol. Soc., Washington, vol. II, ch. 31.
- Rozsahegyi, I. (1956). Die chronische Osteoarthropathie der Caissonarbeiter. *Arch. Gewerbepath. Gewerbehyg.*, **14**, 483–510.
- Rozsahegyi, I. (1959). The late consequences of the neurological forms of decompression sickness. *Brit. J. Ind. Med.*, **16**, 311–317.
- Rubenstein, C. J., and J. K. Summitt. (1971). Vestibular derangement in decompression. In: *Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York.
- Rubissow, G. J., and R. S. Mackay. (1973). Bends studies and control using ultrasonic imaging of body bubbles. In: *Proc. Ultrasonic Symp.* New York. Inst. Electrical & Electronic Engineers.
- Russell, D. C. (1943). Clinical observations on selection tests carried out in the decompression chamber. *FRPC Report 519*, Air Ministry, London.
- Ryder, H. W., C. D. Stevens, J. P. Webb, and M. A. Blankenhorn. (1945). The Measurement of Decompression Sickness. *Report 412*, U.S. NRC, Comm. Aviat. Med., Washington.
- Saltzman, H. A., L. Hart, B. Anderson, E. Duffy, and H. O. Sieker. (1964). The response of the retinal circulation to hyperbaric oxygenation. *J. Clin. Invest.*, **43**, 1283.
- Saltzman, H. A., J. V. Salzano, G. D. Blenkarn, and J. A. Kylstra. (1971). Effects of pressure on ventilation and gas exchange in man. *J. Appl. Physiol.*, **30**, 443–449.
- Samuel, E. P. (1952). The autonomic and somatic innervation of the articular capsule. *Anat. Rec.*, **113**, 53–70.
- Sanders, J. H., and I. M. Isoc. (1947). Intravenous oxygen and pulmonary embolism. *Ann. Surg.*, **126**, 208–214.
- Sass, D. J. (1976). Minimum ΔP for bubble formation in pulmonary vasculature. *Undersea Biomed. Res.*, **3**, A28.
- Saumarex, R. C., J. F. Bolt, and R. J. Gregory. (1973). Neurological decompression sickness treated without recompression. *Brit. Med. J.*, **1**, 151–152.
- Saunders, J. B., and V. T. Inman. (1943). Preliminary Observations on the Qualitative and Quantitative Characteristics of Pain Elicited by Stimulation of Somatic Structures. Unpublished results quoted by Nims (1951).
- Sayers, R. R., and W. P. Yant. (1926). The value of helium-oxygen atmosphere in diving and caisson operations. *Anesth. Analg.*, **5**, 127–138.
- Sayers, R. R., W. P. Yant, and J. Hildebrand. (1925). Quoted by Workman (1969).
- Schaefer, K. E. (1965). *Handbook of Physiology*. Sect. 2. Ed. by W. F. Hamilton and P. Dow. Amer. Physiol. Soc., Washington, vol. III, ch. 51, pp. 1843–1873.
- Schaefer, K. E. (1969). Carbon dioxide effects. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, ch. 6.
- Schaefer, K. E., R. D. Allison, I. H. Dougherty, C. R. Carey, R. Walker, F. Yost, and D. Parker. (1968). Pulmonary and circulatory adjustments determining the limits of depths in breath-hold diving. *Science*, **162**, 1020–1023.
- Schoenborn, B. P., H. C. Watson, and J. C. Kendrew. (1965). Binding of xenon to sperm whale myoglobin. *Nature, London*, **207**, 28–30.

- Schoenfish, W. H., G. D. Blenkarn, B. A. Hills, and J. A. Kylstra. (1975). Liquid Breathing: Expiratory flow and CO₂ elimination using fluorocarbon and aqueous solutions. In: *Proc. Vth Symp. Underwater Physiol.* Fedn. Amer. Socs. Exp. Biol., Washington.
- Scholander, P. F. (1940). Experimental investigations on the respiratory function in diving mammals and birds. *Hvalradets Skrifter Norske videnskaps-Akad, Oslo*, 22.
- Scholander, P. F. (1958). Counter-current exchange—a principle in biology. *Hvalradets Skrifter, Oslo*, 44.
- Scholander, P. F. (1960). O₂ transport through hemoglobin solutions. *Science*, 131, 585–590.
- Scholander, P. F., H. T. Hammel, E. A. Hemmingsen, and E. D. Bradstreet. (1964). Hydrostatic pressure and osmotic potential in leaves of mangroves and some other plants. *Proc. Nat. Acad. Sci.*, 52, 119–125.
- Schreiner, H. R., and P. L. Kelley. (1967). Computation methods for decompression from deep dives. In: *Proc. 3rd Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Williams & Wilkins, Baltimore, pp. 277–299.
- Schreiner, H. R., and P. L. Kelley. (1971). A pragmatic view of decompression. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 205–219.
- Schreiner, H. R., R. W. Hamilton, and T. D. Langley. (1972). Neon: an attractive new commercial diving gas. In: *Proc. Offshore Technol. Conf.* Houston, 1, 501–516.
- Schröder, H., and Th. von Dusch. (1854). Ueber Filtration der Luft in Beziehung auf Fäulniss und Gährung. *Justus Leib. Annal. Chem.*, 89, 232–243.
- Schrotter, H. von. (1906). *Der Sauerstoff in der Prophylaxe und Therapie der Luftdruckerkrankungen*. Hirschwald, Berlin, p. 278.
- Scriven, L. E. (1959). Dynamics of phase growth. *Chem. Engng. Sci.*, 10, 1–13.
- Sealey, J. L. (1974). Seattle tunnel follow-up report. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, p. 23.
- Searle, J. R. (1975). Gas Phase in Tissue: Its Origin, Growth and Measurement during Decompression. Ph.D. Thesis in Biomedical Engineering, Duke University, Durham, N. C.
- Searle, J. R., and B. A. Hills. (1976). A device for the remote detection of gas phase in tissue. In: *Proc. Vth Symp. Underwater Physiology*. Fedn. Amer. Socs. Exp. Biol., Washington.
- Seiple, R. L., K. E. Jennings, and J. R. Losee. (1973). Advanced technology for a diver depth gage and decompression computer. Naval Undersea Centre, Tech. Note 949.
- Selman, M. W., W. A. McAlpine, and R. S. Ratan. (1967) The effectiveness of various heart-lung machines in the elimination of microbubbles from the circulation. *J. Thorac., Cardiovasc. Surg.*, 53, 613.
- Seusing, J., and H. Drube. (1960). The importance of hypercapnia in depth intoxication. *Klin. Wschr.*, 38, 1088–1090.
- Seymour, R. S. (1974). How sea snakes may avoid the bends. *Nature*, 250, 489–490.
- Shewmon, P. (1963). *Diffusion in Solids*. McGraw-Hill, New York, pp. 164–170.
- Shilling, C. W. (1941). Compressed air illness. *U.S. Nav. Med. Bull.*, 39, 367–376.
- Shilling, C. W., and W. W. Willgrube. (1937). Quantitative study of mental and neuromuscular reactions as influenced by increased air pressure. *U.S. Nav. Med. Bull.*, 35, 373–380.
- Shim, S. S., F. P. Patterson, and M. J. Kendall. (1967). Hyperbaric chamber and decompression sickness. *Canad. Med. Ass. J.*, 97, 1263–1272.
- Shoup, C. S. (1929). The respiration of luminous bacteria and the effect of oxygen tension upon oxygen consumption. *J. Gen Physiol.*, 13, 27–45.
- Sicardi, F. (1971). La Coagulation au cours de la plongée profonde. *Maroc. Medical.*, 545, 244–245.
- Sigsbee, R. A. (1969). Vapor to condensed-phase heterogeneous nucleation. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 4.
- Simms, N. M., D. M. Long, and L. A. French. (1971). Cerebral arterial air embolism. *Minn. Med.*, 54, 589–592.
- Simpson, A. (1857). *Compressed Air as a Therapeutic Agent in the Treatment of Consumption, Asthma, Chronic Bronchitis and Other Diseases*. Sutherland & Knox, Edinburgh.
- Slark, A. G. (1962). Treatment of 137 cases of decompression sickness. *RNPRC Report 63/1030*, MRC, London.
- Sloan, H., J. D. Morris, J. Mackenzie, and A. Stem. (1962). Open-heart surgery; results in 600 cases. *Thorax*, 17, 128.
- Sluyter, M. E. (1963). The treatment of carbon monoxide poisoning by administration of oxygen at high atmospheric pressure. *Proc. Roy. Soc. Med.*, 56, 1002.
- Smith, A. H. (1873). *The Effects of High Atmospheric Pressure, including the Caisson Disease*. Prize Essay Alumn. Ass. Coll. Physic. Surg. N. Y. Eagle Book & Job Printing Dept., Brooklyn, p. 43.
- Smith, J. L. (1899). The pathological effects due to increase of oxygen tension in the air breathed. *J. Physiol.*, 24, 19–35.
- Smith, K. H. (1974). Discussion. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, p. 143.
- Smith, K. H. (1975). Current work at Virginia Mason. In: *Proc. Symp. on Development of Decompression Procedures for Depths in Excess of 400 feet*. Undersea Biomed. Soc., Washington.
- Snell, E. H. (1896). *Compressed-air Illness or So-called Caisson Disease*. Lewis, London, p. 251.
- Sobell, H. (1971). Effect of Hyperbaric Oxygen–Nitrogen Mixtures. ONR Program on Underwater

- Physiology Report ACR-175. Dept. of the Navy, Arlington.
- Sorensen, S. C., and J. W. Severinghaus. (1968a). Respiratory sensitivity to acute hypoxia in man born at sea level living at high altitudes. *J. Appl. Physiol.*, **25**, 211–216.
- Sorensen, S. C., and J. W. Severinghaus. (1968b). Irreversible respiratory insensitivity to acute hypoxia in man born at high altitudes. *J. Appl. Physiol.*, **25**, 217–220.
- Sorrentino, L., F. Capasso, and M. DiRosa. (1972). Indomethacin and prostaglandins. *Eur. J. Pharmacol.*, **17**, 306–308.
- Special Regulations*. (1958). Work in Compressed Air, Ministry of Labour and National Service S. I No. 61. H. M. Stationery Office, London.
- Spencer, F. C., P. Bosomworth, and W. Ritcher. (1966). Fatal pulmonary injury from prolonged inhalation of oxygen in high concentrations. In: *Hyperbaric Medicine*. Ed. by I. W. Brown and B. G. Cox. Nat. Acad. Sci., Washington, pp. 189–199.
- Spencer, M. P., and S. D. Campbell. (1968). The development of bubbles in the venous and arterial blood during hyperbaric decompression. *Bull. Mason Clin.*, **22**, 26–32.
- Spencer, M. P., and D. C. Johanson. (1974). Investigation of new principles for human decompression schedules using the Doppler Ultrasonic blood bubble detector. *Final Technical Report: contract N00014-73-C-0094*. ONR, Washington.
- Spencer, M. P., and H. Okino. (1972). Venous gas emboli following repeated breathhold dives. *Fed. Proc.*, **31**, 355.
- Spencer, M. P., G. A. Lawrence, G. I. Thomas, and L. R. Sauvage. (1969). The use of ultrasonics in the determination of arterial air embolism during open-heart surgery. *Ann. Thor. Surg.*, **8**, 489.
- Spencer, M. P., D. C. Johanson, and H. F. Clarke. (1973). Precordial blood bubble detector. *Final Report; contract N00014-72-C-0095*. ONR, Washington.
- Spyropoulos, C. S. (1957). The effects of hydrostatic pressure upon the normal and narcotized nerve fiber. *J. Gen. Physiol.*, **40**, 849–857.
- Stekhoven, J. H. S., and F. Kreuzer. (1967). Shunt component of alveolar-arterial oxygen pressure difference and atelectasis. *Respirat. Physiol.*, **3**, 192–202.
- Stern, S. A., and H. L. Frisch. (1973). Dependence of inert gas narcosis on lipid 'free volume'. *J. Appl. Physiol.*, **34**, 366–373.
- Stevens, C. D., M. Inatome, H. W. Ryder, E. B. Ferris, and M. A. Blankenhorn. (1943). The rate of nitrogen elimination from the lungs and its relation to individual susceptibility to decompression sickness. *Report 456, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Stewart, C. B., G. L. Bateman, and D. J. Milne. (1943). A comparison of two procedures designed to select airmen resistant to decompression sickness. *FPMS Report D-6*. Can. NRC.
- Stewart, W. J. (1933). Aseptic necrosis of the head of the femur following traumatic dislocation of the hip joint. Case report and experimental studies. *J. Bone & Joint Surg.*, **15**, 413–438.
- Stilwell, D. L. (1957). The innervation of tendons and aponeuroses. *Amer. J. Anat.*, **100**, 289–318.
- Stransky, A., M. Szereda-Przestaszewska, and J. G. Widdicombe. (1973). The effect of lung reflexes on laryngeal resistance and motoneurone discharge. *J. Physiol.*, **231**, 417.
- Strasberg, M. (1956). Undissolved air cavities as cavitation nuclei. In: *Proc. Symp. on Cavitation in Hydrodynamics*. London.
- Strates, B., and W. F. Neuman. (1958). On the mechanism of calcification. *Proc. Soc. Exptl. Biol. Med.*, **97**, 688–689.
- Straub, P. W., and A. A. Bühlmann. (1970). Reduction of blood volume by voluntary hyperventilation. *J. Appl. Physiol.*, **29**, 816–817.
- Strauss, R. H. (1974). Bubble formation in gelatin: Implications for prevention of decompression sickness. *Undersea Biomed. Res.*, **1**, 169–174.
- Strauss, R. H., and T. D. Kunkle. (1974). Isobaric bubble growth: a consequence of altering atmospheric gas. *Science*, **186**, 443–444.
- Stubbs, R. A., and D. J. Kidd. (1965a) A pneumatic analogue decompression computer. *Report 65-RD-1*. Institute of Aviat. Med., Canadian Forces Medical Service.
- Stubbs, R. A., and D. J. Kidd. (1965b). Control of decompression by analogue computer. *Report 65-RD-8*. Institute of Aviat. Med., Canadian Forces Medical Service.
- Stubbs, R. A., and D. J. Kidd. (1967). Computer analogues for decompression. In: *Proc. 3rd Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Williams & Wilkins, Baltimore.
- Summitt, J. K., and J. D. Reimers. (1971). Noise: a hazard to divers and hyperbaric chamber personnel. *Aerospace Med.*, **42**, 1173–1177.
- Sundmaker, W. K. H. (1974). Vestibular function. *Special Summary Program—Predictive Studies III*, University Pennsylvania.
- Sutphen, J. H. (1968). The feasibility of using pulsed ultrasound to detect the presence of *in vivo* tissue gas bubbles. *Report 508*. U.S. Nav. Sub. Med. Center, Groton, Conn.
- Swain, V. A. J. (1942). Caisson disease (compressed air illness) of bone with a report of a case. *Brit. J. Surg.*, **29**, 365–370.
- Swann, H. G., and T. B. Rosenthal. (1944). A survey of the incidence of decompression sickness with reference to some constitutional and environmental variants. *Report 32*. USAAF 31st Alt. Trng. Unit. Kingman, Arizona.
- Swindle, P. F. (1937). Occlusion of blood vessels by

- agglutinated red cells, mainly as seen in tadpoles and very young kangaroos. *Amer. J. Physiol.*, **120**, 59–74.
- Szekely, J., G. P. Martin, and S. D. Fang. (1972). Bubble growth by diffusion: the effect of viscosity, inertia and surface tension. *V. D. I. Berichte*, **182**, 13–22.
- Tai, R. C., and H. K. Chang. (1974). Oxygen transport in heterogeneous tissue. *J. Theor. Biol.*, **43**, 265–276.
- Tammann, G. (1898). Veber die Abhängigkeit der Zahl der Kerne. Welche Sich in Verschiedenen Unterkühlten Flüssigkeiten bilden von der temperatur. *Z. Physik. Chem. (Leipzig)*, **25**, 441–479.
- Tammann, G. (1926). *States of Aggregation*. Van Nostrand, New York.
- Tanasawa, I., R. Echigo, and D. R. Wotton. (1971). Measurements of mass diffusivity of gases in plasma and reaction velocity constant in bloods. *J. Biomech.*, **4**, 265–273.
- Taylor, E. W. (1965). Brownian and saltatory movements of cytoplasmic granules and the movement of anaphase chromosomes. In: *Proc. Symp. on Biorheology*. John Wiley, New York, Pt. 4, pp. 175–191.
- Thomas, S., and O. L. Williams. (1944). High altitude joint pains; their radiographic aspects. *Report No. 395, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Thompson, A. M., H. M. Cavert, and N. Lifson. (1958). Kinetics of D₂O and antipyrine in isolated perfused rat liver. *Amer. J. Physiol.*, **192**, 531–537.
- Thompson, J. W., C. B. Stewart, O. H. Warwick, G. L. Bateman, D. J. Milne, and D. E. Gray. (1944). *Flying Personnel Medical Section Report D-3 to the NRC*, Canada.
- Thomson, J. J. (1859). On recent theories and experiments regarding ice at or near its melting point. *Proc. Roy. Soc.*, **10**, 152–160.
- Thorne, I. J. (1941). Caisson Disease: A study based on three hundred cases observed at the Queens-Midtown Tunnel, Project 1938. *J.A.M.A.*, **8**, 585–589.
- Thorpe, W. H., and D. J. Crisp. (1947). Studies on plastron respiration I. The biology of Aphelocheirus and the mechanism of plastron retention. *J. Exp. Biol.*, **24**, 227–269.
- Tobias, C. A., W. F. Loomis, F. C. Henry, W. R. Lyons, H. B. Jones, W. N. Sears, W. N. Cook, J. B. Mohney, J. G. Hamilton, and J. H. Lawrence. (1943). Circulation and Decompression Sickness. *Report 144, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Tobin, C. E., and M. O. Zariquiey. (1950). Arteriovenous shunts in the human lung. *Proc. Soc. Exp. Biol. Med.*, **75**, 827–829.
- Todd, G. P. (1969). Decompression patterns developed by an interdependent electric analog. *Report SMRL 580, 10. U.S. Nav. Sub. Med. Center*.
- Triger, A. G. (1845). Lettre à M. Arago, C.r. held. *Séanc. Acad. Sci., Paris*, **20**, 445–449.
- Trimble, V. H. (1974). *The Uncertain Miracle—Hyperbaric Oxygenation*. Doubleday, New York.
- Truex, R. C., and M. B. Carpenter. (1969). Origin and composition of the nervous system. In: *Human Neuroanatomy*. Williams & Wilkins, Baltimore.
- Tureen, L. L., and J. B. Divine. (1936). The pathology of air embolism: report of two cases. *J. Mo. St. Med. Ass.*, **33**, 141–144.
- Turnbull, D. (1949). The subcooling of liquid metals. *J. Appl. Physics*, **20**, 817.
- U.S. Navy. (1954). *Diving Manual*. U.S. Government Printing Office, Washington.
- U.S. Navy. (1974a). *Diving Manual*. (NAVSHIPS 0994–001–9010). U.S. Government Printing Office, Washington.
- U.S. Navy. (1974b). *Handbook of U.S. Navy Diving Operations*. U.S. Government Printing Office, Washington.
- Van Allen, C. M., L. S. Hordina, and J. Clark. (1929). Air embolism from the pulmonary vein: a clinical and experimental study. *Arch. Surg.*, **19**, 568.
- Van der Aue, O. E., E. S. Brinton, and R. J. Keller. (1945). Surface decompression and testing of decompression tables with safety limits for certain depths and exposures. *Res. Proj. X-476, Report 1*. U.S. Nav. Exp. Diving Unit, Washington.
- Van der Aue, O. E., R. J. Keller, and E. S. Brinton. (1949). The effect of exercise during decompression from increased barometric pressures on the incidence of decompression sickness in man. *Res. Report 8–49*. U.S. Nav. Exp. Diving Unit, Washington.
- Van der Aue, O. E., R. J. Keller, E. S. Brinton, H. D. Gilliam, and R. J. Jones. (1951). Calculation and testing of decompression tables for air dives. *EDI Report MM 002.007*. U.S. Navy, Washington.
- Vanhook, A. (1969). Graining in sugar boiling. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 11.
- Van Liew, H. D. (1962). Tissue pO₂ and pCO₂ estimation with rat subcutaneous gas pockets. *J. Appl. Physiol.*, **17**, 851–855.
- Van Liew, H. D. (1971). Dissolved gas washout and bubble absorption in routine decompression. In: *Proc. 4th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Academic Press, New York, pp. 145–150.
- Van Liew, H. D. (1976). Variability of the inert gas partial pressure in bubbles and tissues. In: *Undersea Biomedical Research*. Abstr. 31. UMS, Washington.
- Van Liew, H. D., B. Bishop, P. D. Walder, and H. Rahn. (1965). Effects of compression on composition and absorption of tissue gas pockets. *J. Appl. Physiol.*, **20**, 927–933.
- Vann, R. D., and H. G. Clark. (1975). Bubble growth and mechanical properties of tissue in decompression. *Undersea Biomed. Res.*, **2**, 185–194.
- Vann, R. D., P. J. Widell, D. A. Youngblood, and B. A. Hills. (1973). Decompression of widely

- differing profile monitored by the ultrasonic bubble detector. In: *Proc. Symp. Blood Bubble Detection*. Seattle.
- Vann, R. D., W. N. Duran, and E. M. Renkin. (1976). *Slow components of washout: flow or diffusion limited?* Duke University Hyperbaric Unit, Durham, N.C.
- Vernon, H. M. (1907). The solubility of air in fats and its relation to caisson disease. *Lancet*, (ii), 691–693.
- Vervloet, A. F. C., M. J. Edwards, and M. L. Edwards. (1967). Minimal apparent blood damage in Lande-Edwards membrane oxygenator at physiologic gas tensions. *J. Thor. Cardiovas. Surg.*, 7, 415–423.
- Verzar, F. (1957). The ageing of collagen. In: *Connective tissue: a symposium*. Ed. by R. E. Tunbridge. Blackwell, Oxford, pp. 208–221.
- Vignon, G., J. Duquesné, M. Drogue, and Y. Vezat. (1960). Les Nécroses primitives de la tête fémorale chez l'adulte (à propos de 9 observations). *Rev. Lyon Med.*, 9, 1177–1183.
- Vigreux, J. (1970). Contribution to the Study of the Neurological and Mental Reactions of the Organism of the Higher Mammal to Gaseous Mixtures Under Pressure. M. D. Thesis. Toulouse University.
- Villaret, M., and R. Cachera. (1939). Les embolies cérébrales: étude de pathologie expérimentale sur les embolies solides et gazeuse du cerveau. Masson, Paris.
- Vincent, R. S. (1941). Measurement of tension in liquids by means of a metal bellows. *Proc. Physics Soc.*, 53, 126–129.
- Volmer, M., and A. Weber. (1926). Keimbildung in Übersättigten Gebilden. *Zeit. Physik. Chem.*, 119, 277–301.
- Vonnegut, B. (1947). The nucleation of ice formation by silver iodide. *N. Appl. Physics*, 18, 593–595.
- Vorosmarti, J., R. de G. Hanson, and E. E. P. Barnard. (1975). Further studies in decompression from steady-state exposures to 250 metres depth. In: *Proc. Sixth Symp. Underwater Physiology*. Abstr. UMS, Washington.
- Wagner, C. E. (1945). Observation of gas bubbles in pial vessels of cats following rapid decompression from high pressure atmospheres. *J. Neurophysiol.*, 8, 29–32.
- Waite, C. L., W. F. Mazzone, M. E. Greenwood, and R. T. Larsen. (1967). Cerebral air embolism. I. Basic Studies. *Report 493*, U.S. Nav. Sub. Med. Center, Groton, Conn.
- Walder, D. N. (1948). Serum surface tension and its relation to the decompression sickness of aviators. *J. Physiol. London*, 107, 43P.
- Walder, D. N. (1963). A probable explanation for some cases of severe decompression sickness in compressed air workers. In: *The Regulation of Human Respiration*. Ed. by D. J. C. Cunningham and B. B. Lloyd. Blackwell, Oxford.
- Walder, D. N. (1965). Blood viscosity and Reynaud's disease. *Lancet*, 1, 1086.
- Walder, D. N. (1966). Some problems of working in an hyperbaric environment. *Ann. R. Coll. Surg.*, 38, 288–307.
- Walder, D. N. (1967). Ultrasonics in the detection of intravascular bubbles. *Can. Med. Ass. J.*, 96, 1233–1234.
- Walder, D. N. (1968). Adaptation to decompression sickness in caisson work. *Biometeor.*, 11, 350–359.
- Walder, D. N. (1969). The prevention of decompression sickness in compressed-air workers. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London, ch. 18.
- Walder, D. N. (1974). Management and treatment of osteonecrosis in divers and caisson workers. In: *Proc. Symp. Dysbaric Osteonecrosis*. Ed. by E. L. Beckman and D. H. Elliott. NIOSH, Washington, and discussion p. 147.
- Walder, D. N., A. Evans, and H. V. Hempleman. (1968). Ultrasonic monitoring of decompression. *Lancet*, 1, 897–898.
- Walsh, M. N. (1941). Changes in intracranial volume in ascent to high altitudes and descent as in diving. *Proc. Mayo Clin.*, 16, 220–221.
- Walton, D. (1969). Condensation of metal vapors on substrates. In: *Nucleation*. Ed. by A. C. Zettlemoyer. Dekker, New York, ch. 7.
- Warwick, O. H. (1943). Further studies on the relationship of fluid intake and output to the incidence of decompression sickness. *Flying Personnel Medical Section Report I*, 'Y' Depot RCAF Halifax NRC, Canada.
- Weaver, R. S. (1967). Pneumatic analogue computer control of decompression. In: *Decompression of Compressed-air Workers in Civil Engineering*. Ed. by R. I. McCallum. Oriel, London.
- Webb, J. P., H. W. Ryder, G. L. Engel, J. Romano, M. A. Blankenhorn, and E. B. Ferris. (1943). The effect on susceptibility to decompression sickness of preflight oxygen inhalation at rest as compared to oxygen inhalation during strenuous exercise. *Report 134*, U.S. NRC, *Comm. Aviat. Med.*, Washington.
- Webb, J. P., G. L. Engel, J. Romano, H. W. Ryder, C. D. Stevens, M. A. Blankenhorn, and E. B. Ferris. (1944a). The mechanism of pain in aviators' 'Bends'. *J. Clin. Invest.*, 23, 934–935.
- Webb, J. P., E. B. Ferris, G. L. Engel, J. Romano, H. W. Ryder, C. D. Stevens, and M. A. Blankenhorn. (1944b). Radiographic studies of the knee during bends. *Report 305*, U.S. NRC, *Comm. Aviat. Med.*, Washington.
- Wegener, P. P., and L. M. Mack. (1958). *Advances in Applied Mechanics*. Academic Press, New York.
- Weibel, E. R. (1964). Morphometrics of the lung. In: *Handbook of Physiology*. Sect. 3, Respiration. Ed. by W. O. Fenn and H. Rahn. Amer. Physiol. Soc., Washington, vol. 1.
- Welham, W., J. J. Blanch, and A. R. Behnke. (1944).

- A procedure for selection of diving and aviation personnel resistant to decompression sickness based on tests in a low pressure chamber. *Report 282, U.S. NRC, Comm. Aviat. Med.*, Washington.
- Wells, C. H. (1975). Personal communication.
- Wells, C. H., T. P. Bond, M. M. Guest, and C. C. Barnhart. (1971). Rheologic impairment of the microcirculation during decompression sickness. *Microvascular Res.*, **3**, 163–169.
- Welsby, V. G. (1967). Acoustic Non-Linearity Due to Microbubbles in Water. Dept. Memo 311, Dept. of Electronic & Electrical Engineering, University of Birmingham, England.
- West, J. B. (1965). *Ventilation/Blood Flow and Gas Exchange*. Blackwell, Oxford.
- Westwater, J. W. (1964). Measurements of bubble growth during mass transfer. In: *Cavitation Real Liquids*. pp. 34–54.
- Whitaker, D. M., L. R. Blinks, W. E. Berg, V. C. Twitty, and M. Harris. (1945). Muscular activity and bubble formation in animals decompressed to simulated altitudes. *J. Gen. Physiol.*, **28**, 213–223.
- Whiteley, A. H., W. D. McElroy, G. H. Warren, and E. N. Harvey. (1944). Bubble formation in animals. V. Dentrigenation. *J. Cell. Comp. Physiol.*, **24**, 257–272.
- Whitman, W. G. (1923). The two-film theory of absorption. *Chem. Met. Engng.*, **29**, 147.
- Willard, G. (1953). Ultrasonically induced cavitation in water. *J. Acoust. Soc. Amer.*, **25**, 669–686.
- Willmon, T. L., and A. R. Behnke. (1948). Residual lung volume determinations by the methods of helium substitution and volume expansion. *Amer. J. Physiol.*, **153**, 138–142.
- Wilson, C. T. R. (1897). Condensation of water vapour in the presence of dust-free air and other gases. *Phil. Trans. Roy. Soc., London*, **A189**, 265–307.
- Wilton-Davies, C. C. (1970). Computer-assisted monitoring of ECG and heart sounds. I: Method. *Report 7/70. RNPL*, London.
- Winkelmann, P. E., V. G. Caruso, M. J. Correia, J. T. Love, and G. E. Miltenberger. (1975). Clinical studies on commercial and sports divers: Otoneurologic findings. In: *Proc. Internat. Symp. on Man in the Sea*. Ed. by S. K. Hong. University Hawaii, Honolulu, pp. VI, 213–226.
- Wintrobe, M. M. (1968). *Clinical Hematology*. Lea & Febiger, Philadelphia.
- Wise, D. A. (1963). The constitutional factors in decompression sickness. *Report 2–63. U.S. Nav. Exp. Diving Unit*, Washington.
- Wisner, K. (1922). Supersaturated solutions of gases. *Trans. Roy. Soc. Canada*, **16**, 217–227.
- Wittenberg, J. B. (1970). Myoglobin in muscle. *Physiol. Rev.*, **50**, 559–636.
- Wittenborn, A. F. (1963). An analytical development of a decompression computer. In: *Proc. 2nd Symp. Underwater Physiol.* Nat. Acad. Sci., Washington, pp. 82–90.
- Wolman, M. (1963). In: *The Selective Vulnerability of the Brain in Hypoxemia*. Ed. by J. P. Schade and W. H. McMenemy. Blackwell, Oxford, p. 349.
- Wood, J. D. (1975). Oxygen toxicity. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière & Tindall, London.
- Wood, W. B. (1963). Ventilatory dynamics under hyperbaric states. In: *Proc. 2nd Symp. Underwater Physiology*. Ed. by C. J. Lambertsen and L. J. Greenbaum. Nat. Acad. Sci., NRC, Washington, pp. 108–123. (Publ. 1181).
- Workman, R. D. (1957). Calculation of air saturation decompression tables. *Research Report 11–57. U.S. Nav. Exp. Diving Unit*, Washington.
- Workman, R. D. (1963a). Evaluation of a decompression computer developed for divers. *USN EDU Evaluation Report 1–63*.
- Workman, R. D. (1963b). Studies of decompression and inert gas-oxygen mixtures in the U.S. Navy. In: *Proc. 2nd Symp. Underwater Physiology*. Ed. by C. J. Lambertsen and L. J. Greenbaum. Nat. Acad. Sci., NRC, Washington, pp. 22–28. (Publ. 1181).
- Workman, R. D. (1965). Calculation of decompression schedules for nitrogen-oxygen and helium-oxygen dives. *Res. Report 6–65. U.S. Nav. Exp. Diving Unit*, Washington.
- Workman, R. D. (1969). American decompression theory and practice. In: *The Physiology and Medicine of Diving and Compressed-air Work*. Ed. by P. B. Bennett and D. H. Elliott. Baillière, Tindall & Cassell, London. ch. 12.
- Wyman, J., P. F. Scholander, G. A. Edwards, and L. Irving. (1952). On the stability of gas bubbles in sea water. *J. Marine Res.*, **XI**, 1, 47–62.
- Yang, W. J., and C. Y. Liang. (1972). Dynamics of dissolution of gas bubbles or pockets in tissues. *J. Biochem.*, **5**, 321–322.
- Yang, W. J., R. Echigo, D. R. Wotton, J. W. Ou, and J. B. Hwang. (1972). Mass transfer from gas bubbles to impinging flow of biological fluids with chemical reaction. *Biophys. J.*, **12**, 1391–1404.
- Yarbrough, O. D. (1937). Calculation of decompression tables. *Research Report. U.S. Nav. Exp. Diving Unit*, Washington.
- Yarbrough, O. D., and A. R. Behnke. (1939). The treatment of compressed-air illness utilizing oxygen. *J. Ind. Hyg. Tox.*, **21**, 213–218.
- Young, S. W. (1911). Mechanical stimulus to crystallization in supercooled liquids. *S. Amer. Chem. Soc.*, **33**, 148–166.
- Young, S. W., and W. J. van Sicklen. (1913). The mechanical stimulus to crystallization. *J. Amer. Chem. Soc.*, **35**, 1067–1078.
- Youngblood, D., P. B. Bennett, R. W. Smith, W. G. Thomas, and J. C. Farmer. (1975). Operational management of vestibular decompression sickness.

- In: *Abstr. Sixth Symp. Underwater Physiology*. UMS, Washington.
- Yunkin, I. P. (1970). Correlation of animal resistance to hypoxia and caisson disease. *Patol. Fiziol. eksp. Terap.*, **14**, 71-73.
- Zal'tsman, G. L., and I. D. Zinov'eva. (1965). Comparative determinations of the permissible supersaturation value of the human body with indifferent gases under different conditions. In: *The Effect of the Gas Medium and Pressure on Body Functions*. Ed. by M. P. Brestkin. Collection No. 3, NASA-TT-F-358, Washington, pp. 28-33.
- Zannini, D., G. Odaglia, and G. Sperati. (1976). Auditory changes in professional divers. In: *Proc. 5th Symp. Underwater Physiology*. Ed. by C. J. Lambertsen. Fed. Amer. Soc. Exp. Biol., Washington, pp. 675-684.
- Zimmerman, A. M. (1970). *High Pressure Effects on Cellular Processes*. Academic Press, New York.
- Zirkle, L. G., C. E. Mengel, B. D. Horton, and E. J. Duffy. (1965). Studies of oxygen toxicity in the central nervous system. *Aerospace Med.*, **36**, 1027-1032.
- Zuntz, N. (1897). Zur Pathogenese und Therapie der durch rasche Luftdruckänderungen erzeugten Krankheiten. *Fortschr. d. Med.*, **15**, 632-639.
- Zweifach, B. (1949). Basic mechanisms in peripheral vascular homeostasis. In: *Trans. 3rd Conf. on Factors Influencing Blood Pressure*. Ed. by B. W. Zweifach and E. Shorr. Josiah Macy Foundation, New York.

Index

- abdominal pain, 30, 32
- abdominal veins, 67
- accidents, surgical, 61
- acclimatization, 39, 40, 43, 60, 87, 206, 208
- acidosis, 206, 220
- acoustic intensity, 83
- acoustic-optical imaging, 144
- actinomyces, 15
- action potential, 208
- activation energies, spectrum of, 100–101
- activation energy
 - for bubbles, 78
 - for nucleation, 85
- activation of nucleus, 238, 250
- active secretion of O_2 , 140
- active transport, 27
- adaptation to decompression, 40
 - see also* acclimatization
- adipose tissue, 142, 146
 - dimensions for, 184
 - perfusion of, 189
- adrenal cortex, 142, 146
- aerial incidence, pre-oxygenation on, 38
- aerobic fat catabolism, 107
- aeroembolism, 54
- age, 41, 42, 197
- agglutination, 49, 50, 51, 145, 201
- agitation, 30
- air bottles, 6
- air columns in vessels, 99
- aircraft, cabin pressure, 17, 18
- air cyst, 66
- air embolism, 233
 - burst lung, 14
 - coronary, 62
 - mortality rate, 62
 - open-heart surgery, 61
- air/gas emboli, 54, 60
 - arterial, 61
 - circulating, 54, 55
 - iatrogenic, 61
- air pumps, 4
- air supply
 - continuous, 6
 - from surface, 6
- albumin, 56
- alcohol/alcoholism, 72, 197, 199, 203, 207, 220, 249
- aldosterone, 56
- alkaline phosphatase, 56
- alternating patency, 190
- altitude
 - adaptation to, 15
 - early record attempts, 17
 - means of ascent, 15
 - threshold for bends, *see also* minimum bends altitude, 35
- alveolar–arterial difference, 28
- alveolar gas, 213
- alveolar walls, swelling of, 220
- amateur divers, number of, 1
- amnesia, 204
- anaerobic metabolic capacity, 107
- anaesthetic gases, 200, 204, 208
 - wash-out of, 176
- anaesthetics, pressure effect, 208
 - uptake kinetics, 169
- analgesics, 58, 234
- analogue–digital conversion, 133
- analogue systems, 128
 - geometric similarity of, 128
 - hydraulic, 128
 - temporal similarity of, 128
- analytical time function, 189
- anatomical tissues, 191
- animal models, 141
- annular model, 253
- anoxia, relief by HBO, 15
- anoxic pain, 38
- anticoagulants, 56, 134
- antithrombin III, 56
- anxiety, 219
- aphasia, 30
- aplysia*, 217
- apnoea, 62
- aqua-lung, 6
- aqueous tissue, 121
 - as critical, 149
 - versus lipid tissue, 192–194
- arcella*, 140
- armaghs, diving, 1, 3
- armour-plated suits, 107
- arterial blood, supersaturation of, 65
- arterial emboli, 38, 67, 99, 200
 - after surgery, 65
 - circulating, 64, 65, 66
 - steady-stage, 213
- arterial occlusion, 65
- arteries
 - blood velocities in, 22
 - sizes of, 22
- arterio-venous differences, 170–172, 217
- arthritis, 196, 199, 206
- articular cartilage, 216
- atrial–ventricular septal defects, 67
- aseptic necrosis of bone, 195–204
 - after kidney transplant, 199, 203
 - idiopathic, 199, 203
 - in alcoholics, 197, 199, 200, 203
 - surgery, 32
 - with Gaucher's disease, 199
 - with gout, 199
 - with pancreatitis, 199
 - with sickle-cell anaemia, 199
 - see also* dysbaric osteonecrosis
- aspartate aminotransferase, 56
- asymptomatic bubbles, 146, 236, 259
 - see also* silent bubbles
- asymptotes for wash-out, 176
- astronauts, 18
- atelectasis, 66
- attenuation of ultrasound, 143
- autochthonous bubble, 75
- automatic optimization, 127
- A–V anastomoses, 177
- aviators, 41
 - bone lesions in, 199
 - onset time in, 33
 - symptoms in, 33

- Avogadro's law, 104
- azobacters, 27
- azygos system, 66
- back-peak technique, 143
- back wall, 255, 257
- backward projection, 175
- bacteria, diffusion in, 187
- balloon ascents, 15
- barotrauma, 69, 206–207
 - pulmonary, 61
- Batson plexus, 67
- bends
 - at depth, 232
 - bends pain, 55
 - criterion for, 142
 - effect of massage, 60
 - mechanism of, 52
 - models, tail-biting, 141
 - origin of word, 12
- bends rates, reporting of, 116
- bends thresholds, in animals, 141, 142
- benzene, 82
- BIBS, 9, 219, 221, 258, 259
- bicarbonate, 26, 47, 217
- bifurcations, 67, 98
- biochemical parameters, general, 56
- bladder, 31
- blindness, 219
- blood
 - as convection medium, 24
 - bubble inception in, 139
 - bubble interface, 126
 - cortisol in, 56
 - degradation of, 50, 55, 229
 - denaturation of, 58
 - enzymes, 44
 - factors, 236
 - Hb content, 56
 - kinetics of O₂ uptake, 21, 26
 - lipids, 55, 192, 198
 - overall description, 24
 - oxygen uptake by, 24
 - pigments, 24, 25
 - pressures, 22, 56, 62, 65–66, 143
 - residence time, 171, 172
 - saturation of, 28
 - sedimentation rate, 56
 - specific gravity of, 56
 - velocity in vessels, 22
 - viscosity of, 234
 - volume of, 56, 253
- blood–air barrier, 209, 213
- blood flow
 - direct measurement of, 171, 172
 - diversion of, 106, 107
 - extracorporeal control of, 178
 - velocity, 144
- blood–gas diagrams, 26
- blood perfusion, *see* perfusion
- blood:tissue exchange, 169–181
- blubber, role of, 106–107
- body fat, 60
- body orientation, 99
- body temperature, 41
 - see also* temperature
- body weight, 56
- Bohr effect, 26, 221
- bolus of air
 - fracture of, 99
 - injection of, 62
- bone
 - aseptic necrosis of, *see also* dysbaric osteonecrosis, 195–204
 - lesions, *see also* dysbaric osteonecrosis, 31, 55
 - marrow, 146
 - marrow, half-time for, 174
 - mineral deposition, 196, 202, 203
 - mineralization process, 201, 202
 - physiology of, 195, 198, 199
 - prostheses, 196
 - vessel walls in, 200
 - X-rays of, 195, 201
- 'bone rot', 31
- bottom time
 - effect of, 36
 - influence of exercise, 46
- bounce dives, 248, 252, 255
 - no-stop limits for, 36, 37, 39, 122, 123, 124, 125, 176, 189, 193
 - N₂ versus He, 188
 - significance of, 109
- bounce recompression, 230
- boundary conditions, 93, 94
- boundary layer, 91, 92
- Boyle's law, 127
- bradykinin, 135
- brain, 62, 177
 - effect of temperature, 222
 - frontal areas, 63
 - grey matter, 64
 - half-time for, 174
 - higher centres, 97
 - parietal areas, 63
 - pathology, 63
 - perfusion rate, 186
 - uptake by, 171, 172, 173
 - white matter, 64
- breath-hold diving, 1–3, 23
- breath-hold excursions, 4
- breath-hold limits, 3
- breathing apparatus, ancient, 7
- breathing mix, 9
 - computer for, 133
 - heating of, 10, 251
 - optimum composition, 108, 134, 223–228, 258
- breathing underwater, mechanics of, 4
- broncho-constriction, 135
- bronchodilators, 134
- bubble amplifier, 151, 152
- bubble composition
 - analysis of, 252
 - variability, 190
- bubble detection
 - by conductivity, 149, 150
 - indirect methods, 151–166
 - non-invasive, 65
 - ultrasonically, 143–146
- bubble inception, 81–90
 - activation energy, 78
 - in lungs, 213
 - interfacial factors, 89
 - mechanical factors, 87
 - radius at, 86
 - trigger point to, 77
 - see also* trigger points
- bubble regression factor, 121, 123
- bubbles, *see also* silent bubbles
 - analysis of, 252
 - in animals, 126
 - circulating, 50
 - collision of, 96
 - critical radius, 252
 - dimensions, 93
 - direct detection of, 139–151
 - distribution of, 247
 - early implication, 108
 - extravascular, 65
 - filtration of, 67
 - fracture of, 67, 100
 - heat transfer in, 91
 - in juxtaposition, 96, 98
 - in small animals, 140
 - mass transfer to, 91
 - mechanical 'overpressure', 252
 - metabolic gases in, 102
 - origin of, 54
 - overload of lung, 67
 - pain-provoking, 93, 250
 - 'popping' together of, 99
 - pressure in, 126
 - resonance in, 145
 - resorption of, 254
 - sub-symptomatic, 137, 139, 157, 162
 - target organ for, 62
- Bühlmann approach, 110

- Bühlmann calculation method, 118, 238
- bulk diffusion, 123–124, 125, 168, 176, 252
- equations for, 245, 246
- models for, 125
- simulation of, 128, 129
- thermal simulation, 131
- bulk modulus, 58, 143, 248
- bullfrogs, 141, 142
- buoyancy, 3, 6
- burns, 15
- burst lung, 13
- bursts of elimination, 190
- cabin pressure, 17, 18
- caissons, 10
- caisson workers, 195, 196, 197
- selection of, 41
- symptoms in, 33, 108–119
- calculation methods, 52, 126
- air, 136
- justification for, 108, 119
- calculator for oxygen exposure, 133
- Canadian pneumatic meter, 129, 184
- capillaries
- alternating patency of, 94, 190
- density of, 184
- capillary wall, 179, 213
- diffusion in, 169
- permeability of, 178
- surface area of, 184
- carbonate, 26
- carbon dioxide, 241, 242
- circulation control, 23
- diffusion of, 209
- from diggings, 47
- in dysbaric osteonecrosis, 202, 203
- effect on incidence, 46–47
- effect on lung, 68
- effect on vestibular DCS, 72, 74
- in gas pockets, 239
- in liquid breathing, 106
- in narcosis, 204
- in tunnels, 11
- on oxygen poisoning, 220, 221
- osmotic potency, 217
- retainers of, 39, 206
- role in agglutination, 49, 51
- role in respiration, 206
- scrubber for, 8, 13
- supersaturation by, 84
- transport of, 26
- vestibular symptoms, 69
- carbon monoxide
- carbon monoxide poisoning, 14
- effect on vestibular DCS, 72, 74
- cardiac stimulants, 134
- cardioid valve, 5
- cardio-respiratory system, 20, 47
- carotid air injections, 63
- catecholamines, 56
- cats, bubbles in, 142
- caucasians, 43
- cavitation, 49
- at will, 140
- in blood, 65
- potential, 84
- see also gaseous cavitation
- cell
- convection in, 168
- diameter of, 183
- environment around, 19
- membrane, 183, 213
- sap in trees, 82
- shrinkage of, 63
- wall, permeability of, 185
- water, 186
- cellular diffusion, 169, 180
- coefficient for, 183–184, 188
- cellular phase, 244, 245, 246, 258, 255, 256, 257
- permeability for O₂, 187
- permeability of, 180
- cellular zone, 237, 253
- cerebellar cortex, 71
- cerebellum, 31
- dimensions for, 184
- cerebral air embolism, 61, 62, 71, 99
- cerebral arteries, bubbles in, 63
- cerebral blood flow, 61, 62, 172
- cerebral circulation, 98
- cerebral compensation, 71
- cerebral cortex, dimensions for, 184
- cerebral symptoms, 31, 68, 75, 191, 192, 236
- cerebral vasoconstriction, 221, 222
- cerebration, 204
- cerebrospinal fluid, 64, 146
- volume of, 143, 252
- chemical affinity, 92
- for gas, 24
- chemical reaction in blood, 22
- chemoreceptors, 23, 205, 206
- chloride, 217
- chokes, the, 30, 32, 68, 146, 236
- cholesterol, 56
- cholinesterase activity, 55
- Circle of Willis, 63
- circulation
- control of, 22
- details of, 22
- evolution of the, 19
- restitution of, 57
- circulation-limited uptake, 122
- see also perfusion-controlling transfer
- civil engineering
- projects, 198
- use of compressed air, 10, 12
- classification of symptoms, 31
- 'classical' theory of nucleation, 80, 85
- clathrates, 204
- clearance
- definition of, 178, 179
- versus blood perfusion, 179
- closed-circuit rebreathing, 8
- closed circuit SCUBA, 219
- clouds, supercooled, 77
- Clyde Tunnel, 198
- CNS, 64, 65, 69, 106, 206, 207, 213, 227
- depressants, 134
- involvement, 33, 36
- mechanism of, 60–68
- oxygen exposure limits, 218
- oxygen on the, 221
- relief of, 229
- symptoms, 31, 71, 97, 193, 234
- tissue for, 192
- treatment of, 230–231
- coagulation, 51, 57
- coalescence, 54, 79, 95–100, 161, 188, 237, 238, 250, 251
- by muscular action, 142
- coarseness of elements, 94
- coarse tremors, 207
- cocaine, 208
- cochlear mechanism, 31, 70–71
- cochlear membrane, 69
- cold, 50
- collagen
- biosynthesis of, 203
- oxidation of, 201, 202
- collapse, 30, 31, 32
- collateral circulation, 65
- collision fission, 100
- colloid osmotic pressure, 216, 234
- coma, 14
- commercial calculation methods, 116
- comparison of inert gases, 193–194
- compartmental analysis, 183
- competing gas pathways, 97
- compliance, tissue, 91, 238, 243, 248
- composition and the inherent unsaturation, 241
- composition of critical tissues, 191–194

- compressed-air workers, registry of, 12
- compressible fluids, flow of, 128, 129
- compression, 106
 - rate of, 203, 207
- computer for oxygen history, 228
- computer programs, 116, 257
- condensation, theory of, 81
- confusion, 219
- congregating mechanism, 250
- connective tissues, 57, 97, 235, 238, 250, 253
- consecutive exposures, 153–154
- constant-pressure cavities, 240, 241
- constants
 - evaluation of, 247
 - in analysis, 252
- constant-volume vacuities, 240, 241
- contact angle, 66, 83, 209
- container walls, 88
- continental shelf, 208
- controlled blood flow, 178, 179
- convection, 91
- convective currents, 168
- convective gas transfer, 19
- convulsions, 13, 30, 62, 208, 217, 219–220, 225, 226, 227, 228
- corporeal density, 42
- cortex, 236
- cortical atrophy, 63
- cortical passages, 55
- corticosteroid therapy, 197, 199
- cosmic radiation, 85
- COTi, 134, 223, 225–228, 259
- coughing, 32
- counterdiffusion, 92, 236
- counterdiffusion supersaturation, 71, 75, 210–212, 215, 217
- counter-gradient of gases, 236
- counterperfusion, 236
- counterperfusion supersaturation, 72, 75, 212, 217
- cranial window, 230
- cranial window preparation, 98
- creatinine phosphokinase, 55, 56
- criterion for bubble inception, 125
- criterion for optimum, 254, 256
- critical inception pressure, 83
- critical insult, 48, 52–54, 124
- critical level of insult, 53, 125, 134
- critical size of nucleus, 85, 127
- critical stress for bends, 253
- critical supersaturation, 101, 150, 152, 153, 162, 165
- critical tissue(s), 191–194
 - aqueous versus fatty, 189, 252
 - identity of, 60
- lipid content, 194
- crushing, in promoting bubbles, 142
- crush injuries, treatment of, 15
- crystallites, diffusion in, 186
- crystallization, 77, 79–80, 202
- cuffs, 239
- cumulative effect of O_2 , 223–228
- cumulative incidence, 34, 45
- cumulative oxygen toxicity index, 134, 223, 225–228, 259
- curvature of interface, 86
- curve fitting, 109
- cutaneous vasodilatation, 31
- cuttlefish, 105
- cyanide poisoning, 15
- cyanosis, 220
- cylinder, 245
- cylindrical interaction, 184
- cyproheptadine, 135
- cytoplasm, 88
 - consistency of, 208
 - diffusion in, 168, 237
 - diffusion coefficient, 185
 - nature of water in, 186
 - O_2 diffusion, 222
 - see also cellular phase
- Dalton's law, 23, 27, 28, 86, 100–101, 210, 241
- Dartford Tunnel, 198
- DDC, 9, 220
 - transfer to, 70, 213
- deactivation of superoxide radicals, 221
- deafness, 30, 31
- death, 29, 31, 220, 230
 - from oxygen, 220
- decanting, 11, 161, 162, 250
- decompression formulation, 109–127, 253–260
 - basic philosophy of, 97
 - Haldane's philosophy, 112
- decompression meters, 127–133
- Decompression Panel (MRC), 31
- decompression *per se*, 30, 35, 49, 221, 248
 - effect on elimination, 155–157
- decompression ratio, 84, 89, 109–119, 125
 - and Doppler sounds, 192
 - apparent value, 251
 - depth dependent, 117–119
 - for caissons, 117
 - simulation of, 128
 - spectrum of, 122
 - validity of, 113–114, 119–122
 - variable, 117
 - versus bubbles, 140
- volume basis for, 104
- deeper stops, 159, 160, 165, 254, 257–258
- deformation of tissue, 54, 58
- degree of insult, see level of insult
- degrees of freedom, 116, 123, 124, 126, 258
- dehydration, 217
- delayed symptoms, 34
- delays imposed by lungs, 167
- demand regular, 6
- density measurements, 142
- denucleated solutions, 88
- deuterated water, uptake of, 117
- dextran, 55, 58, 134, 228, 234, 249
- differential fluid pressure, local, 74
- differential pressure in tissue, 58, 237, 249
- diffusion
 - distances, 184, 236, 247
 - in muscle fibres, 183
 - interbubble, 96
 - non-gaseous solutes, 185
 - of oxygen, 186–187
- diffusion coefficient, 110, 123, 124, 128, 174
 - cellular, 182, 185
 - for CO_2 , 209
 - Krogh values, 181–184
 - steady-state, 212, 213
 - values for, 184
- diffusion-controlling, 110, 237, 238
 - models for, 168
- diffusion in crystallites, 186
- diffusion times, 181–187
- diffusion versus blood perfusion, 167–187
- digital computer, 246
- digital decompression calculator, 130
- dilatation of arteries, 99
- dilatometry, 126, 180
- dimethothiazine, 135
- dipole moment, 186
- direct ascent, 36, 37
- direct microscopic analysis, 177
- dislocations, as nuclei, 78
- diuretics, 249
- diurnal effect, 44, 251
- dive history, assimilation of, 127
- divers, selection of, 41
- diversity of symptoms, 31
- diving
 - analysis of data, 7
 - breath-hold, 1
 - closed-suit, 6
 - half-suit, 6
 - oxygen reserve/availability, 2, 3
 - surface supply, 4

- diving bells, 3, 4, 107
 - ancient, 3
 - modern, 9
 - transfer from, 70
 - use of, 10
- diving helmet, 6
- diving insects, 3
- diving mammals, 3, 106–107
- diving reptiles, 106
- diving suits, normobaric, 14
- diving tables, format for, 107, 108
- dizziness, 61, 72, 207, 219
- dogs as a model, 141
- dolphins, 106
- Doppler meter, 69, 144, 236
- Doppler meter, monitoring position, 20
- Doppler monitoring, 192–193, 259, 260
- Doppler sounds and decompression ratio, 192
- dose–time curves for O₂, 219
- downward excursions, 39
- driving force
 - for gas elimination, 24, 111, 113, 121, 124, 125, 126, 130, 132, 137, 139, 155, 162, 165, 230–232, 237–239, 241, 242, 243, 247, 249, 251, 254, 255
 - with O₂, 219
- droplet formation, 77, 78
- drop-out depth, 125, 256, 257, 260, 262
- drowning, 6
- drugs, 234
 - for O₂ poisoning, 221
- dual optimization, 258
- dumping of gas, 236, 250, 254
 - significance of, 138–139, 157
 - simulation of, 133
 - see also* gas separation
- 'dumps' of gas, wash-out from, 157
- duodenum, dimensions for, 184
- dysbaric osteonecrosis, 30, 31, 52, 75, 137
 - aetiology of, 47, 49
 - age, 197
 - bilateral occurrence, 198
 - bilateral symmetry, 197, 200, 203, 217
 - blood clots, role of, 200, 201
 - carbon dioxide role of, 202, 203
 - case histories, 196
 - comparison with bends, 198, 199, 203
 - compression rate, 203
 - delayed onset, 196, 201
 - diving fisherman, 198
 - in experimental diving, 199
 - exposure pressure, 197
 - exposure time, 197
 - extravascular gas, role of, 200
 - fat emboli, role of, 200
 - features of, 196
 - gas emboli in, 200
 - gas-induced osmosis in, 202
 - in aviators, 199, 203
 - in submarine escape, 197, 203
 - incidence of, 196, 197, 199
 - increased blood viscosity, 201
 - infarction mechanisms for, 198
 - lung disorders, 202
 - management of, 196
 - mechanisms, 199–203
 - oxidation of collagen, 201
 - pathogenesis of, 195, 199, 200
 - radiography, 195–197, 199
 - sites of, 197
 - sludging, role of, 201
 - statistical analysis, 199
 - surveys for, 196
 - symptomatology, 195–196
 - thrombi, role of, 200–201
 - time course of, 201
 - with hypercortisonism, 197, 199
 - see also* aseptic necrosis of bone
- ECG/EKG, 150
- EEG, 62
- egg albumin, 88
- eighth nerve syndrome, 70
- elastic factors, 88, 91
- elastic forces, 85
- elastic modulus, 59, 60
- elastomers, 86–87
- electrical conductance, 149, 150, 239
- electrical conduction
 - analogue, 128
 - pressure on, 208
- electrical simulations, 130, 131
- electrocution, 15
- electrolyte shifts, 55
- electromagnetic blood flow meter, 145
- electronic meters, 131
- electron microscopy, 148
- electro-physiological measuring, 217
- elimination, *see* gas elimination
- emotional stability, 204
- empirical functions for uptake, 189
- empirical optimization, 260–262
- end-artery type circulation, 71
- end-expired air, 28
- endocrine system, 222
- endolymph, 72, 73
- endoplasmic reticulum, 183
- endothelial cells, 55, 59, 148
- endothelial wall, 22, 66, 147
- energy threshold, 151
- enumeration of time constants, 175, 176
- environmental parameters, 34, 77
- eosinophil, 56
- epidural vertebral venous system, 64
- epileptic seizures, 219
- epinephrine, 56
- epiphyseal–metaphyseal plate, 198
- epithelium, 211
- equilibration times, 35
- equi-narcotic partial pressures, 204
- enzymes, membrane-associated, 221
- erythrocytes, diffusion in, 21, 26
- erythrocytes, swelling by gases, 215
- ether, 82
- euglobulin lysis time, 56
- euphoria, 204
- Eustachian tube, 206
- excessive decompression, simulation of, 131–133
- excised films, 181
- excised skeletal muscle, 180
- exercise, 45–47, 60, 250
 - during decompression, 250, 251
 - effect on calculation, 117
 - effect on pre-oxygenation, 38
 - effect on wash-out, 176, 177
 - in 'dry' runs, 41
 - mathematical allowance for, 253
 - oxygen poisoning, 220
 - vestibular symptoms, 70
- expansion ratio, 79
- explosive decompression, 65, 66, 88
- exponential function, 122
 - derivation of, 111
- exposure pressure, effect of, 36
- exposure time, effect of, 36
- extended vascular zone, 237, 244, 245, 246, 253, 255, 256, 257, 258
- extensive parameters, 128
- extracellular medium, permeability of, 180
- extraction of time constants, 175, 176
- extrapolation, limitations of, 109
- extravascular bubbles, 147, 165, 229
- extravascular cell, bursting of, 148–149
- extravascular fluid, pressure, 249
- extravascular gas, 200

- extravascular sites, 234, 235
- extravascular tissue, 237
- facilitated diffusion, 92, 209
 - of oxygen, 187
- factor 5, 56
- factor 8, 56
- faintness, 61
- fascia, 57, 59
- fascial planes, 150
- fat, cavitation in, 89
- fat emboli, *see* lipid emboli
- fatigue, 32, 219
- fatty acids, 52
- fatty inclusions, 192
- intracellular, 146
- fatty tissues, 147, 193, 235, 238
 - bubbles in, 59
 - see also* adipose tissue
- ferment, 197, 199, 200, 201
- bre diameter, 253
- brin-fibrinogen, 56
- brinolysis, 55, 56
- bula, 199
- Fourier equation, 93, 245
- Fick's law, 93, 182, 183, 210, 212, 246
- filter for venous bubbles, 66
 - impaired in lung, 192
 - limits by lungs, 67
- filtration by capillary wall, 169
- filtration of microspheres, 177
- finger, perfusion rate of, 186
- first 'pull', 165, 251, 254
- fish, 105
- fixed charges, 186
- fixed ΔP , 117, 120, 123
- fat slab, 245
- flickering of capillaries, 94, 190
- flow of gases, 127
- fluid accumulations, 238
- fluid cuffs, 200
- fluid dynamics, 91
- fluid flow analogue, 128
- fluid homeostasis, 179
- fluid loss, 55
- fluid pressure, 57
- fluid shifts, 51, 57–58, 59, 120, 213–217, 234, 241, 249
- fluorescein, 63
- fluorocarbons, 209
- local signs, 61
- optical blood-flow detector, 144
- forced descents/recompressions, 34, 44, 46
- free fatty acids, 56
- free radicals, 221
- French G.E.R.S., 116
- frequency shift in ultrasound, 144
- frictional heating, 83
- frog sartorius muscle, 183
- fully-stirred concept, 126, 168, 169, 170, 177, 212, 237, 244, 245, 253
- fundamental parameters, 252
- ganglionic blockers, 134
- gas balance in tissue, 169
- gas content, effect on cavitation, 83
- gas density, effect on ventilation, 205
- gas elimination, 113, 156, 241, 254
 - at fixed P_{O_2} , 156–157
 - at surface, 160, 161
 - delay due to lungs, 167
 - driving force for, *see also* driving force, 111
 - during decompression, 154–157
 - exercise on, 250, 251
 - implications, 159
 - in surface interval, 164
 - whole body, 175–177, 237
- gas emboli, *see also* air/gas emboli *de novo*, 53
- gaseous anaesthesia, 51
- gaseous cavitation, 52, 83–84, 139, 146
 - mechanism of, 61
- gas-induced osmosis, 51, 52, 58, 72, 75, 201, 202, 203, 205, 207, 213–217, 249
 - by oxygen, 222
- gas nuclei, 54
- gas pockets, 102, 230
 - subcutaneous, 94
- gas separation, *see* separation from solution
- gas solutions, expansion of, 126
- gastrocnemius muscle, 178
- Gaucher's disease, 199
- gelatin, 92, 99
 - solutions of, 86, 87, 88
- gels, 87, 90
- geometric models, 94
- geometric similarity, 128
- Gibbs free energy, 78
- gill, 209
 - membrane of, 105
- glasses, nucleation in, 77
- glass walls, bubbles on, 88
- globulin, 56
- glottis, closure of, 13, 61
- glucose, 56
- glycogen-based fermentation, 107
- goats
 - as a model, 141
 - CSF of, 143
 - susceptibility of, 35
- gout, 57, 199
- gradual decompression, 29
- Graham's law, 169, 174, 175
- Grecian bend, 2, 12
- growth, 80, 90–94, 97, 161, 188, 238, 244
 - conditions for, 100
 - definition of, 76
 - models based upon, 126, 127
- guinea pigs as a model, 141
- haematocrit, 56
- haematological disorders, 238, 249
- haematological factors, 56, 244
- haematologic problems, 236
- haematology, 25
- haemoconcentration, 51, 55, 56, 201, 234, 236
- haemodynamic studies, 139
- haemoglobin, 24
 - dissociation curve, 241
 - facilitating diffusion, 92
 - heat of oxidation, 222
 - in O_2 poisoning, 221
- haemoptysis, 30, 220
- haemorrhage, 71
 - acute perivascular, 63
 - bubble-induced, 147, 148
- Haldane approach/rationale, 79, 80, 95, 104, 110–119, 124, 125, 137, 138, 146, 151, 152, 159, 160, 161, 163, 164, 165, 166, 247, 251
 - simulation of, 128–130
- Haldane effect, 26, 221
- Haldane gas samples, 28
- Haldane philosophy, 125
- Haldane tissues, 111–117, 170, 173, 177, 180, 189, 257
- half-saturation times, 111, 174, 186
 - effect of exercise, 176
 - spectrum of, 191
- hamster cheek pouch, 144, 147
- hand, perfusion of, 186
- hand pumps, 5
- hard-hat diver, 5
- HBO, 12, 13, 14, 15
 - physiological aspects, 15
- headache, 30, 31
- head injuries, 15
- head-tent, use of with O_2 , 13
- hearing disorders, 69
- heart, 59, 142
 - blood pressures in, 22
 - perfusion rate of, 186
 - pictorial, 20
 - rate, 56
 - sounds, 139

- heat loss from diver, 10
- height, 42
- heliox, onset time, 33
- heliox diving, 7
- helium versus nitrogen, 39
- helium window, 152
- hemiplegia, 30
- Henry's law, 23, 24, 27, 76, 133
- heparin, 56, 134, 232
- heterogeneous nucleation, 78, 81, 105, 181
 - in bone, 202
- heterogeneous perfusion, 113, 174, 177
- heterogeneous systems, 88
- histamine, 148
- HMS *Poseidon*, 197, 202
- homeostatic forces, 74, 215, 216
- homogeneous nucleation, 77–80, 202
- Hook's law, 133
- HPNS, 29, 204, 206, 207, 208, 217
- humerus, 197, 199, 200
- humoral factor, 52, 55, 58
 - controlling breathing, 4
 - evidence for, 135
- hydration, 134
- hydrodynamic flow, 169, 216
- hydrogen, 39
- hydrophobic surface, 90, 105
- hydrostatic pressure, 86
 - effect on elimination, 155–157
 - negative, 83
- hydrostatic pressure *per se*, 84
- hydroxyapatite, 201
- hyperbaric arthralgia, 29, 204, 206–207, 216
- hyperbaric hotel, 13
- hyperbaric oxygen, on joints, 207
- hyperbaric phenomena, 208–217
- hyperbaric radiotherapy, 15
- hyperbaric therapy, 12, 13, 14, 15
- hyperbaric urticaria, 211
- hypercapnia, 2, 7, 202, 203, 206, 209
- hyperconfidence, 204
- hyperexcitability, 207
- hyperoxia, 57, 157, 204, 249
- hyperoxia *per se*, 38
- hyperthermia, 222
 - surgical, 61
- hyperventilation, 206
- hypobaric chambers, 15
- hypothetical tissues, 111–117, 164
 - number of, 153–155
- hypoxia, 2, 8, 17, 22, 38, 55, 57, 67, 68, 143, 204, 206, 220
 - altitude, 15
 - limitations of, 3
- iceberg formation, 204
- ice nuclei, 77
- identity of bends tissue, 151
- identity of critical tissues, 191
- illegal abortion, treatment of, 14
- imbibed fluid, 43
- immersion, 41
- imminence of DCS, 53, 126, 134, 246–247, 248
 - effect of exercise, 45
- imperfections, effect on diffusion, 186
- inadequate decompression, 30
- incidence
 - binomial distribution, 44–45
 - nature of inert gas, 152
- incoordination, 30
- indirect detection of bubbles, 151–166
- individual factors, 248
- individual variability, O₂ poisoning, 228
- indomethacin blockade, 52
- inductive conductivity, 150
- inert gas
 - balance, 102
 - distribution, 244, 245
 - kinetic versus solubility, 39
 - molecular weight, 174
 - narcosis, 29, 204–205, 206, 207, 217
 - nature of, 38–39
 - selection of, 38
 - simultaneous uptake of, 174
 - solubility considerations, 188
 - wash-out of, *see also* wash-out of inert gas, 211
- infarction, 54
- inherent susceptibility, 35
 - index of, 45
- inherent unsaturation, 23, 102, 103, 105, 127, 150, 154, 211, 232, 239–243, 244, 251, 254, 255
- initial staging/stops, 125
- injection pressure, 57
- injury, 41
- innear ear, 31, 37, 71
- inspiratory effort, 4
- inspired volume, 8
- intensity of pain, 34
- intensive parameters, 128
- intercapillary distance, 124, 183, 184, 252
- interface, 90, 211
 - artificial, 149
 - blood–air, 55, 238
 - bubble formation at, 78
 - tissue–gas, 241, 243
- interfacial factor, 86, 252
- intermittent oxygen breathing, 224, 259
- interstitium, 168, 169, 244, 246
- inter-tissue diffusion, 176
- intestinal pain, 30
- intestine, perfusion rate of, 186
- intoxication, 204, 220
- intracapillary bubbles, 147
- intracellular gas, 99
- intramedullary pressure, 198, 200
- intravascular bubbles, 165, 230
 - coalescence of, 98
 - origin of, 147–148
 - relevance of, 146–147
 - steady-state, 213
 - velocity of, 99
 - wash-out as, 156
- ion-exchange block, 205
- ionogram of vestibular fluids, 73
- ions as nuclei, 78
- irregular boundaries, 180
- irregular exposures, 137
- irritability, 30
- irritant level for prolonged O₂, 223
- irritant receptors, 68
- ischaemia, 49, 54, 67, 230
 - pain of, 50
- ischaemic cell damage, 63
- ischaemic episode in osteonecrosis, 200–203
- ischaemic insult versus mechanical, 64
- isobaric decompression sickness, 69, 213
- isoenzymes, 55
- isolated muscle fibres, 183
- isometric compression, 29
- isotopes
 - clearance of, 62, 99
 - nucleation by, 85
 - studies of, 172–174
- itching, 30, 31
- jet engine, impact of, 17
- JIM, 14, 107
- joints
 - capsule of, 57
 - gas in, 142, 143
 - pains in, 29, 207
- Jones's data, 175–177
- J-receptors, 68, 236
- jugular venous blood, 174
- juxta-articular lesions, 32, 196
- kangaroo rat, 141
- Kety and Schmidt's experiment, 171, 173

- kidney, 135
 - perfusion rate of, 186
 - transplant of, 199
- kinetic approach to nucleation, 85
- kinetics of coalescence, 96–97
- kinetics of gas separation, 94
- kinins, 51
- Knudsen flow range, 129
- krypton clearance, 172, 175, 181, 204
- labile solutions, 79
- labyrinthine system, 71–74
- lack of coordination, 219
- larynx, 68
- last-stop, titration of, 158, 162
- LDH, 56
- Legendre quadrature, 257
- lethal air dose, 62
- level/degree of insult, 52, 109, 244, 248, 254
- level of recompression, 229, 230
- ligament, 57, 59
- limb appearance in bends, 41
- limb bends, 235, 248, 249, 252, 253
 - description of, 31, 32
 - effect of cuffs, 58
 - effect of injury, 41
 - imminence of, 256
 - incidence of, 30
 - mechanism of, 54–60, 75, 147, 236
 - onset time, 33
 - potentiating agents, 134
 - propagation of, 33
 - random nature of, 33
 - relief of, 229
 - site of, 58–59
 - tissue type, 191
 - versus vestibular, 70–71
- limb pain, 32, 199
 - incidence of, *see also* limb bends, 32
 - intensity of, 32
- limited supersaturation, 104
- linear density, 42
- linear diffusion, 95, 184, 260
- linear interaction, 184
- linear relationship for decompression, 119–121, 252
- lipid–aqueous interface, 89, 211
- lipid emboli, 64, 65, 148, 236
 - in bone, 200, 203
- lipid inclusions, 90, 248
 - see also* fatty inclusions
- lipid layer, 89, 210, 211
- lipid peroxides, 221
- lipids
 - coalesced, 145
 - content of critical tissue, 194
 - lipid tissue, 121
 - lip-twitching, 219
 - liquid breathing, 21, 90, 106, 209–210
 - liquid–liquid interface, 78
 - liquid–liquid systems, 89
 - liquids, fracture of, 81
 - liver, 59, 142, 146, 177
 - half-time for, 174
 - O₂ diffusion in, 187
 - localized pain, 30
 - local reactions, 31
 - lock
 - horizontal, 11
 - medical, 9, 10, 12
 - locomotor system, 32, 60, 97
 - long bones, 31
 - surveys of, 195
 - loquacity, 204
 - Lorraine Smith effect, 220
 - lucite calvarium, 142
 - lumbar puncture, 64
 - lunar modules, 18
 - lungs
 - active secretion by, 140
 - as bubble trap/filter, 20, 47, 54, 66–67, 192, 211
 - airway branching, 20
 - blood–air barrier, 19
 - blood shunt in, 21, 26
 - bubble inception in, 213
 - bubbles trapped by, 236
 - centre of pressure in, 4, 5, 6
 - dead space of, 4
 - delay to elimination, 167
 - diaphragm of, 5
 - diffusion barrier in, 209
 - disorders and bone lesions, 202
 - filtration limits, 67
 - fluid accumulation in, 201
 - gas-induced osmosis in, 217
 - honeycomb concept, 22
 - hydration of, 217, 220
 - hypothermia in, 251
 - mixing in, 4
 - 'overload' of filter, 67, 68
 - receptors in, 68
 - residual volume of, 107
 - resistances to gas transfer, 21
 - respiratory zone of, 4
 - surfactant in, 66, 98
 - terminal airways of, 107
 - tidal volume of, 4
 - upper airways of, 107
 - vasomotor tone of, 67
 - lymph, 169
 - lymphatic occlusion, 30, 31
 - lymphatic system, 22, 146
 - lymphatic vessels, 55
 - lymphocytes, 56
 - mangrove tree, 83, 84
 - marrow pressure, 198
 - mask-off effect, 221
 - mass balance, 169, 171, 172
 - mass spectrometer, 155
 - mathematical function, 109
 - mathematical models, 108, 122–127
 - for diffusion, 184
 - of tissue slab, 180
 - mathematical symmetry, 113–114, 121, 153–157
 - mathematics, allowance for silent bubbles, 125
 - maximum voluntary ventilation, 205
 - mean tissue gas tension, 170, 171
 - measurement of blood flow, 171, 172
 - mechanical analogue, 262
 - mechanical approaches, 57–58, 75
 - mechanical balance, 100
 - mechanical contribution, 252
 - mechanical factors, 238, 247, 253, 256
 - mechanical injury, 107
 - mechanical insult versus ischaemia, 64
 - mechanical simulation, 127
 - mechanical stimulation, 68
 - mechanical stress, 54, 142, 237
 - mechanisms for DCS, 142
 - bubble-free, 49
 - for O₂ poisoning, 221
 - infarction-based, 49
 - rheological approach, 50
 - medical lock, 10, 12
 - melts, supercooled, 77
 - membranes
 - compression of, 205
 - flow across, 216
 - oxygenator, 51
 - permeability of, 221
 - memory for hydrostatic pressure, 84, 87, 89
 - Menière-type complex, 69
 - meningeal veins, 63
 - mental function, 204
 - mesentery, 52, 229
 - metabolic gases, 122, 150, 207, 217, 240, 244, 252
 - assimilation of, 93
 - contribution of, 102
 - osmotic effects, 215, 217
 - production of, 93

- metabolic heat, 222
- metabolic nitrogen, 27
- metabolic pathway, 221
- metabolic rate, 44, 220, 251
- metabolism, 187, 241, 243
- metastable limit, 79, 80, 89, 114, 123, 139, 151, 152
- metastable state, 77, 86
- metastable zone, 80–81
- meters
 - effect of temperature, 133
 - electrical design, 127
 - mechanical design, 127
 - miniaturization, 131
- mice as a model, 141
- microbubbles, 68
 - venous infusion of, 62
- micro-cavities, natural, 139
- micro-circulation, 57, 147, 229
 - bubbles in, 65
 - Zweifach concept, 177–178
- micro-disseminated coagulation, 51, 55
- microscopic examination, 99, 139, 140
- microthrombi, 201, 203
- middle ear, 29, 71, 236
 - gas in, 262
 - ideal gas mix for, 74
 - liquid fill, 106
- mineral oil, 82
- minerals, salting out, 203
- minimum bends altitude, 35, 36, 39, 45, 162, 193, 252
 - distribution of, 45
- minimum bends depth, 36, 39, 45, 49, 120, 151, 188
 - He versus N₂, 193
 - distribution of, 44
 - effect of oxygen, 37
- mitochondria, 63
- mixed efferent blood, 174
- mixed inert gases, 152, 208, 210–213
 - versus single, 153
- models
 - aim of, 136
 - based upon growth, 126, 127
 - computer programming, 183
 - for gas transfer, 168
 - fundamental adequacy, 248–253
 - mixed perfusion: diffusion, 125
 - parallel-faced slab, 126
 - planar, 253
 - quantitative assessment, 251
 - radial diffusion, 126
 - sodium diffusion, 183
 - synthesis of, 167
 - tests of, 123
- thermodynamic, 235
- tissue slab, 122, 123
- mode of curvature, 257
- mode of diffusion, 183
- molecular radius, 215
- monitoring by ultrasound, 143–146
- mountaineering, 15
- moving boundary, 94
- moving bubbles, 91, 146
- MRC, 195, 196, 197, 198, 201, 203
- multiple inert gases, 92, 103, 116, 118, 152
- muscular action, 142
- muscular twitching, 30
- muscular weakness, 30
- mutual diffusion coefficient, 92
- M* values, 109–119, 122, 139, 142, 144, 146, 151, 152, 158, 159, 161, 163, 164, 165, 166
- myelin sheath, 65, 142
- myocardial infarction, treatment of, 15
- myocardium, 67
- myoglobin, 187
- narcosis, 29
 - see also* inert gas
- narcotic potency
 - of oxygen, 221
 - of various gases, 39, 204, 205
- natural gas cavities, 29
- nausea, 30, 31, 37, 69, 70, 72, 207, 219
- negative pressure, 100
- negroes, 43
- neon, 39
- nerve endings, 54, 58, 192, 235, 237, 238, 248
 - preponderance of, 59, 148, 149
- nerve fibres, examination of, 142
- fenestration of, 58
- nervous system, effect of gases, 217
- neurologic oxygen toxicity, 219–220
- neurone firing, osmolality on, 222
- neutrophils, 56
- newts, 205
- nicotinic acid, 52
- niggles, 161, 238
- nil-supersaturation concept, 238
- nitrogen
 - metabolic consumption, 27
 - metabolic production, 27
 - narcosis, 220
 - resolution of, 243
- nitrogen elimination, effect of CO₂, 47
- nitrogen wash-out, and space flight, 18
- nitrous oxide, osmosis induced by, 215
- nodes, 55
- non-invasive probes, 144, 145
- non-linear gas resistor, 184
- non-polar, 186, 187
- normobaric diving suits, 107
- no-stop decompression limits, 36, 39, 122, 160
 - see also* bounce dives
- nucleation, 76–81
 - by isotope decay, 85
 - definition of, 76
 - in gels, 87
 - kinetics of, 80
 - mechanical means of, 81
 - probability of, 80
 - rate of, 77
 - statistical approaches, 81
 - statistical mechanics of, 80–81
 - thresholds for, 95
 - volume change on, 80
- nuclei
 - activation into growth, 86
 - critical radius of, 86
 - varying size of, 127
- number of critical tissues, 153, 191
- numb fingers, 219
- numbness, 30
- nutrients, depletion by vasoconstriction, 222
- nystagmus, 30, 31, 70, 74
- obese animals, 135
- obesity, 41–43, 49, 192
- occupations, pressure-related, 1
 - effect on symptoms, 33
- ocean trials, 41
- oedema in lungs, 220
- Okinawans, 260–262
- omental vessels, 50
- onset of symptoms
 - hastening of, 34
 - prediction of, 126
 - random nature of, 95–97
 - reason for delay, 96
 - times of, 33, 46, 52, 237, 250, 251
- opacities in X-rays, 143
- optimal elimination, 163
- optimal mix, 258
- optimization procedure, thermodynamic, 253, 255, 256
- orange-peel appearance, 31
- organic skin, 86

- orientation, 99
 - effect on respiration, 5
 - stable, 6
- orifices, flow through, 129, 130
- osteocytes, 203
 - death of, 198
- osteomyelitis, 15
- osmolality, effect on neurones, 222
- osmosis by gases *in vivo*, 215
- osmotic potency, 207, 216
- osmotic pressure, 214
- osmotic selectivity, 72, 74
- otologic disorders, 31, 69–74
- overload of lung, 67, 68
- oxidation of enzymes, 221
- oxidative metabolism, 19, 221
- oxygen, 241
 - active secretion of, 28
 - analogue, 133
 - as a narcotic, 221
 - breathing, 37, 39
 - capacity of blood, 24
 - chemical equilibrium, 13
 - computer, 228
 - conduction line for, 21, 26
 - debt, 177
 - diffusion of, 186–187
 - drugs, 134, 221
 - 'dump' system, 13
 - effect on coalescence, 99
 - effect on vestibular DCS, 72, 74
 - exposure, optimization of, 219
 - exposure limits, 222–227
 - hazards of using, 13
 - history, assimilation of, 223–228
 - ignition potential, 13
 - increase of bottom time, 37
 - infusion of, 68
 - in gas pockets, 239
 - mask, 13
 - mask-off effect, 69, 72
 - metabolic usage, 25, 49
 - programming, 259
 - storage, 107
 - substitution of, 38, 152
 - supply, electronic control, 8
 - therapy, 12, 13, 14
 - treatment, 231
 - uptake by bubble, 92
- oxygenation, 66
 - in lungs, 20
 - of peripheral tissues, 243
- oxygenators, 51, 55, 61
- oxygen bends, 37, 64
- oxygen toxicity, 134, 204, 217–228, 258
 - effect of decompression, 69
- pulmonary, 213
- simulation, 133
- oxygen window, 239
- oxyhaemoglobin, 24, 25
- pain
 - effect of compression, 58
 - gas volume for, 125
 - intensity of, 34
 - mechanism of, 54
 - referred, 58, 60
 - threshold for, 58, 235, 237, 248, 250
- pancreas, dimensions for, 184
- pancreatitis, 199
- papillary muscle, dimensions for, 184
- paraffin surface, 89
- parallel-sided slab, muscle, 180
- paralysis, 30, 31, 208, 230
- parenchymal cell, 18, 19
- paresthesia, 30
- partial pressure vacancy, 239
- partition coefficient, 23, 113, 172
- passive relaxation, 60
- pathological studies, 59, 62–63
- peak fluid shift, 216, 217
- peak gas tension, 237, 255, 256
 - steady-state, 211, 212
- peak of insult, 53
- pearl divers, 6, 34, 43, 44, 108, 229, 260–262
- pedal ganglion, 217
- pelvic veins, 67
- perfusion
 - impaired by emboli, 72
 - model for, 95
- perfusion-controlling transfer, 110, 113, 237, 238
 - model for, 168
 - simulation of, 128, 130
- perfusion:diffusion
 - confusion, 94, 124, 167–187
 - controversy, 137, 153, 155, 244
 - parallel interpretations, 173
- perfusion rates, 93, 257
 - allowance for, 168–170
 - clinical significance of, 170
 - effect of, 246, 251
 - of fat, 189
 - spectrum of, 117
 - values for, 186
- periarticular tissue, gas in, 143
- pericapillary filtrate, 22, 169
- perilymph, 71–72, 73
- permanent disablement, 29
- permeability
 - homogeneous, 168
 - of cell wall, 185
 - of tissue, 182–184
- personality changes, 30
- personnel transfer capsule, 9
- personnel transfer lock, 9, 12
- phantom limb, 59
- pharmacological approaches, 134
- phase equilibration, 85, 95, 100–104, 125, 137–166, 236, 237, 238, 239, 243, 245
- phase interface, 90
- phase separation
 - critical limit for, 136
 - simulation of, 131, 132
 - thermodynamic criteria, 131
 - thermodynamics of, 77, 78, 80, 86, 90
 - wash-out after, 157
- philosophy of decompression formulation, 97
- phospholipid, 56
- phosphorus, transitions in, 77
- physical fitness, 43
- physically dissolved O₂, 24
- physical systems simulating transfer, 128
- physiological changes, 62
- physiological conditions, 180
- physiological parameters, general, 56
- physiological state, 168
- pial arteries, 63
- piezoelectric crystals, 144
- plasma
 - colloids, 25
 - diffusion in, 21, 26
 - expanders, 134, 234
 - lactate, 56
 - lipids, 56
 - loss of, 51, 52
 - osmotic pressure of, 56
 - proteins, denaturation of, 51, 55
 - total, 56
- plastron respiration, 3
- platelet
 - aggregates, 51, 145
 - count, 55, 56
 - role in healing, 25
- pneumatic meters, 128–130
- pneumatic simulation, 127
- pneumatic systems, sources of error, 133
- polymorphic forms, 77
- ponderal index, 42
- popliteal fossa, bubbles in, 143
- popping joints, 57, 206
- pores in membranes, 214
- porous plugs, 129, 130
- positive-pressure suits, 17, 18
- post-fracture avascular necrosis, 198, 201
- post-mortem findings, 99

- posture, 99
 PPCH, 135
 precordial position, 20
 pregnancy, 43
 pre-oxygenation, 38, 126, 251
 effect of altitude, 37, 162
 effect of exercise, 37
 with exercise, 46
 pressure
 antagonistic action of, 205
 on the unsaturation, 241
 pressure memory of solutions, 84, 87, 89
 pressure *per se*, 13, 204, 205, 207, 208–209
 pressurized cabins, 17
 pretreatment for Type II DCS, 230–231
 prevention, pharmacological approaches, 134
 primary event, 48–54, 95, 124, 125, 134, 135, 136, 235, 236, 244, 254–255
 primary insult, physical versus chemical, 134
 primitive organisms, 139, 140
 principle of superposition, 224, 225, 227
 probability
 distribution of cavitation, 83
 of nucleation, 86, 89, 90
 professional divers, number of, 1
 programming, 108
 stability for, 257
 prostaglandins, 51, 52
 prothrombin, 56
 protozoa, 140
 pruritus, 30, 31, 41
 psychological stress, 43, 44
 psychosomatic disturbances, 32
 psychosomatic effects, 41, 44
 pulmonary capillary bed, 67
 pulmonary circulation, 20, 22
 pulmonary gas embolism, 69, 211, 213
 pulmonary haemorrhage, from the squeeze, 14
 pulmonary membrane, 14, 19, 21, 217
 rupture of, 66
 pulmonary oxygen exposure limits, 218
 pulmonary oxygen toxicity, 220
 pulmonary toxicity dose, 223–225, 227, 228
 pulse-echo techniques, 144
 pulse pressure, 62
 pulse rate, 56, 176
 pumping, cavitation in, 84
 RQ, 241
 race, 43
 radial diffusion, 123, 128, 181, 257, 284
 model for, 95, 126
 radio-rubidium, wash-out of, 179
 radius of nucleus, 86
 random bubble inception, 157, 236, 238
 random incidence, 250
 random nucleation/activation, 81, 83, 86, 89, 90, 95
 random perfusion, 190
 rapid compression, effect on seizures, 222
 rash, 31
 rate-limiting process, 169
 rats, bubbles in, 141, 142
 Raynaud's disease, 50
 RBC count, 56
 reactive gases, 92
 reactive hyperaemia, 177, 178
 rebreathing, closed-circuit, 8
 recompression, 41, 52, 53, 54, 60, 228, 229–231, 236, 239, 248
 effect on conductivity, 149
 relevance of, 147
 time of, 229, 231
 vestibular response, 74
 rectified diffusion, 83
 rectum, 31
 recurrence of symptoms, 147, 231
 reflection coefficient, 214, 215
 Reissner's membrane, 72–73, 213
 reliability physics, 45
 relief of pain, 230
 renal cortex, dimensions for, 184
 renal medulla, dimensions for, 184
 renewal theory, 45
 repetitive aerial exposures, 250
 repetitive decompression, 39
 repetitive diving, 161, 162, 165
 repetitive exposures, 160, 161
 repetitive group, 162
 repetitive tables, 162
 reservoir of nuclei, 85–89, 100, 235
 residual nitrogen time, 161, 162
 residual symptoms, 229
 resistances to gas transfer, 26
 resolution of 'dumped' gas, 232
 resonance in bubbles, 145
 respiration, control of, 19, 205
 respiratory fatigue, 5
 resting potential, 208
 restitution of circulation, 229
 restlessness, 30
 reticulocytes, 56
 retinal damage, 219
 reversibility of insult, 53
 reversible affinity for a gas, 92
 Reynold's cavitation, 83, 139
 Reynold's number, 91
 rheological hypothesis, 51
 rheological mechanism, 201, 203
 rigid particles, filtration of, 67
 round window, 69, 71, 72
 Royal Navy/RNPL, 116, 123, 153, 159, 224, 260
 Royal Navy tables, 122
 rubbing facilitating cavitation, 83
 Russian work, 116
 saturation curve, 255
 'saturation' decompression, 249
 'saturation' diving, 103, 220, 243
 'saturation' exposures, 35
 'saturation system', 9
 scotomata, 61
 SCUBA, 1, 5, 6, 10
 sea, gas concentration in, 106
 sea-lion, 3
 seals, 106
 sea snakes, 106
 second harmonic in ultrasound, 143
 secretion of O₂, 28
 seeding
 crystals, 202
 of clouds, 77
 supersaturated solutions, 81
 seek the peak, 125, 126
 seizures, hypoxia, 220
 selectivity of membranes, 215
 separated/dumped gas, *see also*
 silent bubbles, 76, 95, 127, 150, 152, 158, 160, 161, 238
 clearance of, 251
 effect of elimination, 162
 intracellular, 99
 overall, 94
 simulation of, 133
 volume of 58
 separation from solution/dumping, *see also* dumping of gas, 49, 125
 in bone, 203
 in gels, 87–88
 sequestration of bone mineral, 196
 serotonin, 56, 134, 148, 248
 serum, 88
 serum ions, 56
 serum phosphorus, 56
 sex, 43
 shape factor, 181, 244, 245, 246, 247, 248, 253
 shear rate, 51
 shrimp, bubbles in, 87, 140, 142

- shock, 32
- shunts, 181
 - in dividing mammals, 106
 - in skeletal muscle, 177–179
 - morphological evidence for, 66, 177
 - total, 67
- sickle cell anaemia, 15, 199
- silent bubbles, 97, 121, 125, 133, 134, 137, 146, 150, 151, 152, 153, 155, 157, 160, 162
 - subjective evidence for, 161
- siloxane membranes, 130
- simulation of decompression, 127–133
 - by fluid flow, 128
 - by thermal conduction, 128
 - electrical conduction, 128
- simultaneous inert gases, 152
 - dumping of, 104
 - uptake of, 174
- single-stop titration, 159
- single-tissue model, 122
- single-tissue wash-out, 177
- sinus cavities, 29, 106
- skeletal muscle, 59, 142, 181
 - dimensions for, 184
 - exercise of, 177
 - perfusion rate of, 186
 - wash-out from, 175, 179
- skin, 106, 148, 156, 253
 - immersion of, 41
- skin 'bends', 49, 69
 - description of, 31
 - incidence of, 30
 - no decompression, 213
- skin contact, 41
- skin-fold thickness, 42
- skin grafts, 15
- sludging, 49, 55, 57, 201
- slugs of gas in vessels, 99
- SMAF, 135
- small animals, bubbles in, 140
- smoking, blood CO, 14
- smooth muscle
 - activity factor, 135
 - antagonists, 134
 - fibres, O₂ on, 221
 - stimulants, 135
- snorkel, 4
- sojourn of blood, 171, 172
- sol-gel transitions, 208
- solubility considerations, 193–194
- solubility of gas
 - conductivity, 149
 - in lipid, 59
 - metabolic gases, 241
 - osmotic effect of, 214
- solute transfer, analytical
 - approaches, 91
 - solutions
 - criterion for stability, 81
 - seeding of, 81
 - solvents for gases, 23, 88
 - somatotyping, 41
 - SOS meter, 128
 - sound, velocity of, 144
 - space docking, 18
 - space suits, 18
 - spasms, 208
 - spatial distribution of gas, 92–93
 - spectrum of activation energies, 100–101
 - spectrum of half-times, 111, 191
 - spectrum of *M* values, 117, 120, 122
 - spectrum of tissues, 113
 - speech, 8, 204, 205, 208
 - sphere, 245
 - sphincter control, 31
 - spikes, 217
 - spinal cord, 64, 65, 146
 - involvement of, 75
 - pathology of, 64–65
 - spinal DCS, 31, 39, 192, 236
 - incidence of, 30
 - symptoms of, 31
 - spino-vertebral veins, 67
 - spino-vestibular tract, 31
 - spleen, perfusion rate of, 186
 - sponge divers, 2, 34, 42, 43, 229
 - squeeze, the, 13
 - squid giant axon, 208
 - 'squishy' joints, 216
 - stabilizing microbubbles, 86
 - staggers, the, 30, 31
 - stagnation flow, 97
 - stapes, 71
 - status epilepticus, 62
 - steady state, decompression from, 36, 103–104
 - steady-state decompression limits, 119, 120
 - steady-state methods for *D*, 181–183
 - steam generation, 91
 - steam nozzles, droplet formation at, 77
 - step changes, superposition of, 225, 226
 - sterno-clavicular joint, 32
 - stimulation, promotion of bubbles, 140, 142
 - stirred fluids, 92
 - stirred-tank concept, 174
 - see also* fully stirred concept
 - stochastic theory, 94, 190
 - stomach, perfusion rate of, 186
 - stretch receptors, 68
 - stroke, 15
 - structure of liquids, 82
 - subcutaneous emphysema, 30
 - subcutaneous gas pockets, 102, 239, 241, 242
 - submarines, 1, 4
 - escape, 62, 196–197, 236
 - escape training, 14, 65
 - submersible decompression chamber, 8
 - submersibles, 1, 4
 - substernal pain, 220
 - sub-symptomatic decompression, 124
 - sub-symptomatic gas phase, 97, 158, 160, 161, 251, 258
 - see also* silent bubbles
 - supercooling, 77, 81, 82
 - superoxide dismutase, 221
 - superposition, principle of, 224, 225, 227
 - supersaturation, 76–81
 - chemical generation of, 84
 - definition of, 76
 - Haldane's conception of, 110
 - limit to, 77, 81, 89, 90, 95
 - metastable limit to, 79
 - of inspired water, 105
 - quantification of, 86
 - ratio, 77
 - threshold, 161
 - vessel size on, 81
 - without decompression, 210–213
 - supersonic aircraft, 18
 - superstition, 44
 - supply of nutrients, 169
 - suppressed transformation, 77, 78, 80, 81
 - surface coalescence, 99
 - surface decompression, 9, 161, 162, 165, 250
 - surface interval, 11, 160, 161, 162, 163
 - surface tension, 85, 91, 96, 100, 252
 - surfacing
 - conditions for, 254
 - ratio for, 117
 - surfactant, 66, 98, 134
 - survival times on high O₂, 222
 - susceptibility, 56
 - age, effect of, 41–43
 - distribution of, 44
 - individual, 41
 - physical fitness on, 43
 - obesity, 42
 - of animals, 141

- susceptibility (contd)
 race, 43
 sex, 43
 water balance, 43
 suspended transformation, 77, 124
 suspended transitions
 liquid-in-gas, 77
 liquid:liquid, 77
 solid-in-gas, 77
 solid:liquid, 78
 solid:solid, 77
 Swedish Royal Navy, 116
 swim-bladder mechanism, 140
 switching blood flow, 180
 switching gases, 116, 180
 inert gases, 69, 74, 75, 92,
 103–104, 151, 152, 206, 213,
 222, 232
 inert gas simulation, 130
 on narcosis, 205
 to air, 37
 sympathetic stimulation, 178
 symptomatology, 30
 symptoms, simultaneous occur-
 rence, 32
 syncope, 30, 32
 synthesis of a model, 235–237
 systemic circulation, 20, 22
 systemic embolization, 64–67
 see also arterial emboli

 tabulating dives, 162
 tachypnoea, 32, 62
 tadpole, 87, 208
 narcosis in, 205
 tail-biting, as bends model, 141
 tap-water, supersaturation of, 106
 target organ, 64, 72
 temperament, 44
 temperature, 41, 88, 214, 250, 251
 effect on elimination, 157
 of brain, 222
 oral, 56
 temporal similarity, 128
 temporo-mandibular joint, 32
 tendon, 57, 59, 60, 142, 149, 190,
 192, 235, 247, 248, 253
 tensile strength of liquids, 81–83
 tension, versus partial pressure, 23
 terminal airways, 21, 107
 terminal rise velocity, 67
 testicular damage, 220
 Thebesian veins, 67
 therapeutics, supplementary, 234
 thermal analogue, 128, 131, 182,
 257
 thermal protection, 6
 thermodynamic approach, 95,
 124–126, 131–133, 235–262
 criteria for, 254
 human experience, 260–262
 program for, 259
 thermodynamics, 77, 78, 80, 86,
 90, 113, 137, 151, 236, 237,
 243
 of coalescence, 96
 thixotropic fluids, 87
 threshold
 current, 208
 for bends, 53, 131
 for bubble inception, 151
 for pain, 57
 insult, 134
 potential, 208
 thrombosed erythrocytes, 148
 thrombosis, 49
 thyroid, perfusion rate of, 186
 tibia, 197, 199
 tidal volume, 21
 time constants, 111
 enumeration of, 175, 176
 extraction of, 175, 176
 for bulk diffusion, 124
 interpretation of, 113
 time function, 111
 by empirical deduction, 189
 exponential derivation, 112
 for blood perfusion, 113
 time of day, 44
 time of year, 44
 tingling fingers, 219
 tinnitus, 30, 31, 37, 69
 tissue, statistical mean area, 254
 tissue analogue, 120–121
 tissue analysis for gas, 174
 tissue averaging of gas, 95
 tissue compliance, 235
 tissue type(s), 59, 151
 'Haldane'/hypothetical, 116
 number of, 137, 153–154
 tolerance
 individual, 32, 36
 to intravascular air, 61
 topside computer, 127
 tourniquet, 41
 traffic-light indicator, 133
 transcutaneous gas transfer, 106,
 156, 157, 211
 transfer sphere, 9
 transient gas gradients, 215
 transient methods for D , 181, 184
 transition point in P_1 versus P_2 ,
 191
 transport processes, 18, 168
 development of, 19
 trapped bubbles, release of, 231
 trauma, 58
 treatment, 53, 228–234, 250
 delay in seeking, 97
 liquid-breathing for, 106, 209,
 210
 lumbar puncture as, 64
 of fish, 105
 of underlying cause, 134
 oxygen, effect of, 99
 prescribing O_2 , 228
 requests for, 34
 tables for, 231, 233
 use of O_2 , 219
 trebeculae, dead, 196
 trees, 83
 \sqrt{t} relationship, 122, 123, 176,
 190, 191, 252
 trial-and-error methods, 108
 tribonucleation, 57, 83
 trigger points, 77, 81, 89, 90, 95,
 117, 123, 124, 131, 134, 137,
 139, 142, 151, 152, 163, 164,
 247, 254
 triglyceride, 56
 trimix, 8, 207
 tris-buffer (THAM), 209
 true saturation, 125
 true supersaturation, 152
 tumours, oxygen tension in, 15
 tunnel construction, 110
 tunnel vision, 219
 tunnel work, 10
 air supply, 11
 British regulations, 12
 tunnel workers, 195, 196
 adaptation of, 40
 number of, 1
 turbulent flow, 83, 139
 turgor, 83
 turtles, 106
 two-phase concept, 178, 180, 186
 model for, 181, 187
 systems, 99
 two-phase decompression meter,
 132
 Type I DCS, 31
 see also limb bends
 Type II DCS, 31
 see also CNS symptoms

 ulcers, 15
 ulna, 199
 ultrasonics
 automatic decompression by,
 144
 detection of bubbles, 61, 81,
 143–146
 Doppler meter, 69
 inducing cavitation by, 82, 143
 irradiation with, 88, 151
 umbilical supply, 8

- unconsciousness, 8, 30, 31, 61, 206
- unicellular organisms, 19
- unintentional decompression, 6, 107
- unperfused areas, 190
- unsaturated liquids, 29
- upper airways, 107
- uptake: elimination asymmetry, 153–157
- uptake of inert gases
 - empirical functions for, 189
 - mixed perfusion/diffusion, 170
 - rate of, 113
- UPTDs, 223–225, 227, 228
- upward excursion, 39, 164, 192
- urea, wash-out of, 179
- urinary disturbances, 30
- urine
 - catecholamines, 56
 - cations, 56
 - corticosteroids, 56
 - creatinine, 56
 - phosphorus, 56
 - volume, 56
- urticaria, 30, 31
- USAF records, 42
- U.S. Navy, 116, 220, 237, 239, 243, 249, 254, 257, 258, 259, 260, 262
- U.S.N. tables, 39, 46, 97, 122, 130, 139, 158, 159, 161, 162, 163, 164, 165, 227, 228, 230, 231, 233, 251
- history of, 114–116
- oxygen exposure limits, 218
- vacuoles, 140
- valsalva manoeuvre, 74
- vaporisation, 81–82
- vascular congestion, 147
- vascular haemorrhage, 53
- vascular occlusion, 54
 - in bone, 199
- vasoconstriction, 251
 - effect on clearance, 180
 - effect on elimination, 156
- vasodilatation, 250
 - mathematical allowance for, 253
- veins
 - blood velocities in, 22
 - sizes of, 22
- vena cava, 22
- venous admixture, 67
- venous blood, monitoring of, 146
- venous sinuses, 63
- venous tensions, significance of, 37
- ventilation at pressure, 205–206
 - with liquid, 209, 210
- ventilation/perfusion inequalities, 21, 26, 66
- ventricular tissue, dimensions for, 184
- vertebral column, 32
- vertigo, 30, 31, 37, 69, 70, 71
 - treatment of, 15
- vessel rupture, 142
- vessel size, on superheating, 82
- vessel walls, 22
 - in bone, 200
- vestibular DCS, 30, 31, 32, 37, 49, 120, 191, 232
 - effect of gas switch, 37
 - mechanism of, 69–71
 - no-decompression, 69, 71–72, 74, 213
 - potentiating factors, 69–71, 74
 - prevention of, 74–75
 - steady-state, 211
 - symptoms, 69, 72, 236
 - 'tissue' the, 238, 243, 244, 249
- vestibular limitations, 70, 75
- vestibular membrane, 72–73
- vestibulo-spinal tract, 71
- viscosity
 - effect on nucleation, 83
 - pressure on, 208
- viscosity of blood, 50, 51
- visual disorders, 31
- visual disturbances, 30
- vital capacity, 56
 - O₂ on, 223, 224
- vital issue, 137–166
- volume of bubbles, 147
- volume of separated/dumped gas, 100, 102–104, 113–114, 120, 121, 124, 125, 126, 160, 230, 238, 239, 246
 - calculation of, 125
 - critical, 253
 - estimate of, 194
- volume of solutions, 205
- voluntary hyperventilation, 23, 217
- vomiting, 30, 31, 37, 69, 207
- vortex formation, 139
- wash-out of inert gas
 - at altitude, 162
 - automatic integration of, 172
 - bursts of, 190
 - during decompression, 154–157
 - effect of exercise, 176
 - effect of sympathetic stimulation, 178, 179
 - from a single tissue, 177
 - from excised tissue, 180
 - stopped blood flow, 178
 - whole body, 122
 - see also gas elimination
- water
 - clathrate structure of, 51
 - compressibility of, 253
 - displacement of gases, 216
 - tensile strength of, 81–82
- water balance, 43
- watershed zones, 63
- water vapour, supercooling of, 77–79
- WBC count, 56
- Weibull distribution, 44, 45
- weight, 42
- West German approach, 116
- West German NMI, 116
- wet suits
 - effect of, 41
 - pulmonary effects of, 5
 - thermal protection by, 10
- wet versus dry exposure, 41
- wheel, 30, 31
- white matter, dimensions for, 184
- Wilson cloud chamber, 77, 78, 87
- work of breathing, 4, 7, 205
- worst possible case, 95, 101, 124, 237, 238, 239, 247, 250, 254
- wound healing, 54, 56
- xenon, clearance of, 172, 175, 204
- X-rays, 54, 239, 250
 - interpretations of, 252
 - studies, 57, 142–143, 147, 149, 150
- Young's modulus, 253
- zero-supersaturation, 90, 131, 238



PADI
INSTRUCTOR 33913
Albrecht Salm

61/2008

