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REVIEW ARTICLE

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Extreme human breath-hold diving

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Abstract In this paper, the respiratory, circulatory and metabolic adjustments to human extreme breath-hold diving are reviewed. A survey of the literature reveals that in extreme divers, adaptive mechanisms take place that allow prolongation of apnoea beyond the limits attained by non-diving subjects, and preservation of oxygen stores during the dives. The occurrence of a diving response, including peripheral vasoconstriction, increased arterial blood pressure, bradycardia and lowered cardiac output, is strongly implicated. Some peripheral regions may be excluded from perfusion, with consequent reliance on anaerobic metabolism. In addition, extreme breath-hold divers show a blunted ventilatory response to carbon dioxide breathing, possibly as a consequence of frequent exposure to high carbon dioxide partial pressures during the dives. These mechanisms allow the attainment of particularly low alveolar oxygen (<30 mmHg) and high alveolar carbon dioxide (>50 mmHg) partial pressures at the end of maximal dry breath-holds, and reduce oxygen consumption during the dive at the expense of increased anaerobic glycolysis (rate of blood lactate accumulation >0.04 mM·s^{-1}). The current absolute world record for depth in breath-hold diving is 150 m. Its further improvement depends upon how far the equilibrium between starting oxygen stores, the overall rate of energy expenditure, the fraction of energy provided by anaerobic metabolism and the diving speed can be pushed, with consciousness upon emersion. The ultimate limit to breath-hold diving records may indeed be imposed by an energetic constraint.

Key words Alveolar gases · Anaerobic metabolism · Diving response · Gas stores · Ventilatory response

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Introduction

In 1988, Enzo Majorca established a remarkable record in breath-hold diving, reaching for the first time a depth of 100 m, under controlled conditions. His achievement attracted the interest of environmental physiologists, and stimulated new studies of breath-hold diving in humans. Extreme divers have been investigated, and some models have been proposed and hypotheses put forward to explain their performances. The time has come to review the respiratory, circulatory and metabolic adjustments to human extreme breath-hold diving.

Three questions are addressed in this paper:

1. What is the respiratory and cardiovascular response to maximal dry breath-holds and to deep breath-hold dives?
2. Does the energy expenditure during breath-hold diving limit the maximal diving depth?
3. Do specific adaptive mechanisms develop, facilitating the achievement of extreme depths?

Concerning the last point, most attractive is the hypothesis that a diving response, analogous to that demonstrated in diving mammals (Andersen 1966; Lin 1982; Blix and Folkow 1983; Hochachka 1986; Zapol 1996; Butler and Jones 1997; Kooyman and Ponganis 1998) may develop in breath-hold divers. The diving response combines bradycardia, reduced cardiac output, arterial hypertension and redistribution of regional blood flows, with consequent lactate accumulation in unperfused muscles. This contributes to minimised oxygen consumption during diving, and thus prolongs the diving duration. Indeed, the data discussed in this review support the hypothesis that the maximal diving depth could be set by the balance between the energy cost of diving, the speed of the dive and the minimal partial pressure of oxygen (PO_2) that is compatible with consciousness upon emersion.

Since this review is restricted to a discussion of the questions formulated here above, there will be no analytical overview of the subject of breath-hold diving.

Such an overview, however, can be found in several excellent review articles (Lin 1988, 1990; Manley 1990; Gooden 1994; Linér 1994; Lin and Hong 1996; Ferrigno and Lundgren 1999).

Historical remarks on breath-hold diving

Breath-hold diving has been practiced in Greece, Persia, India, Korea and Japan for more than 2,000 years, with the purpose of harvesting sponges, pearls and seafood. Deep dives were soon assisted by weights during descent, and by pulling the diver up with a rope during ascent. Breath-hold diving was also employed for military purposes, for example during the siege of Syracuse, Sicily (415 BC) and of Tyre, Lebanon (333 BC; Davis 1934). Herodotus was criticised by Aristotle for having stated that a Phoenician sailor could recover a column from a temple at a depth of 54 m: according to Aristotle, the maximal diving depth for humans was around 10 m. Underwater swimming raiders were also commonly employed by the Roman navy (*Urinatores*).

In Western civilization, breath-hold divers have accomplished the salvage of treasures: in 1680, Sir William Phipps is reckoned to have recovered about £200,000 in sterling silver from a Spanish galleon that had sunk in the Caribbean (Kindwall 1997). For the purpose of salvage operations, Hans von Treileben from Sweden developed a device, called the diving bell, which allowed the performance of dives with a greater oxygen supply than provided by the total lung capacity (TLC; Craig 1987a). In 1664, most of the cannons carried by a sunken warship in the Baltic Sea were recovered with the aid of this device. A remarkable performance was accomplished in 1913 by a Greek fisherman ashore the island of Skarpanthos, in the Aegean Sea: this man was able to rescue the anchor of an Italian ship, which was grounded at a depth of 70 m, by means of three consecutive breath-hold dives with a 15-kg counterweight on his belt. The ship physician reported that this diver suffered of emphysema and had no eardrums (please refer to the medical report in Appendix 1). The fisherman understated his achievement and claimed that he was used diving to 110 m (Molfino and Zannini 1964).

Although breath-hold swimmer raiders were still employed by the Royal Navy and by the United States and Italian Navies in World War II, and breath-hold diving is still practiced in the training of underwater demolition teams, the use of professional breath-hold divers is declining nowadays. It has been replaced in many respects by scuba diving. Much greater depths can even be reached by means of saturation diving. Sponge divers are disappearing from Greece, as are the pearl divers from the Persian Gulf and the Tuamotu Islands, Polynesia. However, breath-hold diving is still flourishing in Korea and Japan for seafood harvesting. There still exist some 20,000 divers in these countries, mainly women in Korea (Ama), and mainly men in Japan (Park et al. 1990).

The physiological study of diving populations was initiated by Teruoka (1932), who described the diving operations and patterns, their seasonal variations, and the diving equipment of the Ama, and estimated their gas exchange during diving. A late, but considerable interest in the physiology of breath-hold diving in humans was raised by Teruoka's report. Numerous studies on the Ama, as well as on other diving populations, have addressed potential features of physiological adaptations to diving. Diving patterns and techniques, alveolar gas exchange, lung volumes, heart rate, arterial blood gases, and temperature regulation have been studied. The results have been summarised in several books and review articles (Rahn and Yokoyama 1965; Hong and Rahn 1967; Hong et al. 1986; Rahn 1987; Park et al. 1990; Lin and Hong 1996).

In general, professional divers do not dive below depths of 10–20 m. Much greater depths are reached during competitive diving and record attempts. The modern history of competitive diving (free diving, assisted technique) started in 1949, when Raimondo Bucher dived to 30 m. A summary of the evolution of absolute world records in breath-hold diving is reported in Fig. 1. The present world record was established in 1999 by Umberto Pellizzari, who dived to a depth of 150 m. Figure 1 shows that after a slow record evolution in the pioneering years, a faster evolution occurred, which was interrupted in the mid-seventies; the more recent years have been characterised by dramatic record improvements, which were facilitated by the technical developments and the loose regulations applied to the pursuit of absolute depth records. The concurrent increase in the medical risks related to the practice of extreme breath-hold diving may explain why in 1991, the World Conference of Underwater Activities decided to stop recognising absolute world records and to restrict the rules of assisted breath-hold diving.

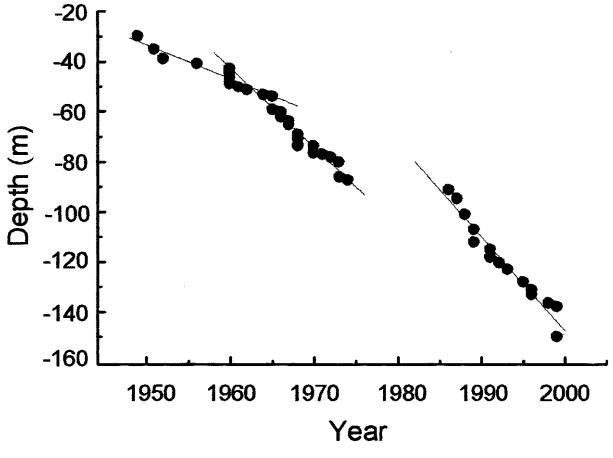

Fig. 1 Evolution of depth records in breath-hold diving. Regression lines describing three distinct periods of record evolution are also reported

Development of diving techniques

Counterweights have long been in use to accelerate the descent and preserve energy during the dives. The assisted divers from Japan (Funado) carried a 13- to 15-kg counterweight to pull them to the bottom. At the end of the dive, they were pulled up with a rope by a man on a boat above (Hong and Rahn 1967). This procedure reduces considerably the energy cost of diving with respect to the unassisted technique, so that the Funado can perform longer (60 s instead of 30 s) and deeper (20 m instead of 5 m) dives than unassisted divers (Cachido; Hong et al. 1963). The time spent at the bottom was some 25% of the total diving duration. The basic diving technique has undergone little change, although the introduction of fins and the use of wet suits instead of cotton suits have improved diving performance. The speed of descent and ascent has increased, so that the time spent at the bottom has grown to 40% of the diving duration (Park et al. 1983a). Both developments are energy-saving, the former by reducing the energy cost of diving, the latter by reducing the thermal stress. Indeed, one consequence of wearing wet suits has been the loss of adaptation to cold, which was a peculiarity of the Ama (Hong et al. 1986).

The pursuit of a faster speed and a reduction in the energy cost of diving has characterised the development of the assisted diving techniques for record attempts. Since the first records, counterweights were used for assisted dives. During ascent, however, the diver had to pull himself up along a rope. By the 1980s, this was no longer the case. On the occasion of his 100-m record dive, Enzo Majorca was pulled by a platform sliding along a rope, which was suspended vertically from a support boat via a pulley. At the expected bottom of the dive, he left the platform, which also acted as a counterweight, while a supporting scuba diver gave him a balloon inflated with air from a gas bottle. The balloon pulled him up to the surface and was released just before emersion (see Majorca 1987 for details). Inflatable suits are now in use, hydrodynamic postures are pursued, and every redundant movement is carefully avoided. As a consequence, the improvement in the dive record took place without significant changes in the duration of the dives, which remained steady at around 3.5 min.

Competitive dives are performed with an initial lung volume as close as possible to the diver's TLC and are generally preceded by voluntary hyperventilation manoeuvres. These can decrease alveolar carbon dioxide partial pressure (P_ACO_2) down to some 20 mmHg, and increase alveolar oxygen partial pressure (P_AO_2) up to 130 mmHg (Ferretti et al. 1991). Despite the increase in P_AO_2, hyperventilation does not enlarge the body oxygen stores, since the diver operates on the flat portion of the oxygen equilibrium curve. Instead, hyperventilation is effective at emptying the carbon dioxide stores, so that it takes longer to achieve the breath-hold breaking point. This is definitely useful for the experienced diver in a record attempt. However, the P_AO_2 can drop to dangerously low levels during ascent, because of inhibition of the carbon dioxide stimulus to breathing, carrying along the risk of blackout (Craig 1961, 1976; Rahn 1987). Indeed, almost all extreme breath-hold divers have experienced loss of consciousness upon emersion in their career. In fact, loss of consciousness under water is the cause of some 7,000 deaths per year in the United States, and is the primary cause of death among children and adolescents in several of the Union states (Neuman 1997).

Evolution of concepts on the maximal diving depth

The study of those factors that limit the maximal diving depth has evolved together with the record progression. The first widely accepted hypothesis relies on Boyle's law. The assumption was that the TLC could not be compressed below the residual volume (RV). Thus, the TLC:RV ratio would have indicated how much the lung volume could be compressed during a dive, and thus the depth limit. For instance, an RV of 1.14 l and a TLC of 4.58 l were measured in the Ama (Song et al. 1963), corresponding to a TLC:RV value of 4. This would indicate a maximal diving depth of 30 m, to be compared with actual diving depths of the Ama of around 20 m. Until the early 1960s, breath-hold diving records were compatible with this concept. Bob Croft's dive to 73 m in the late 1960s seemed to challenge this hypothesis, but his vital capacity was so high (7.8 l), that his TLC:RV ratio was above 7 (Schaefer et al. 1968). Since Bob Croft could exert particularly high expiratory pressures, which might have contributed to a significant reduction in his RV during the dives, it was concluded that at a depth of 70 m, his TLC was still larger than his RV.

The achievement of further records, however, soon led to the TLC:RV ratio hypothesis being refuted, and showed that lung volumes were not limiting the maximal breath-hold diving depth. Other factors were then considered that could prevent thorax breakdown at lung volumes lower than RV. Craig (1968) proposed that the negative pressures achieved in the thoracic cage at great depths could displace blood from the extremities to the thorax, thereby increasing the central blood volume and possibly reducing the RV. This hypothesis, often recalled in the ongoing years (see, for example, Odaglia 1985; Craig 1987b; Lin 1990), is supported by the estimate (Schaefer et al. 1968) of an increase in thoracic blood volume of about 1 l during open sea dives to 40 m, and by the observation that immersion in water is sufficient to displace some 700 ml of blood into the thorax (Arborelius et al. 1972). More recently, radiographic determinations on breath-hold divers who carried underwater radiological equipment (Morelli and Data, personal observations) have shown a reduction in lung volume, an elevation of the diaphragmatic dome, an engorgement of lung blood vessels and an increase in the transverse diameter of the heart. As a consequence of blood displacement, during a deep dive the circumference

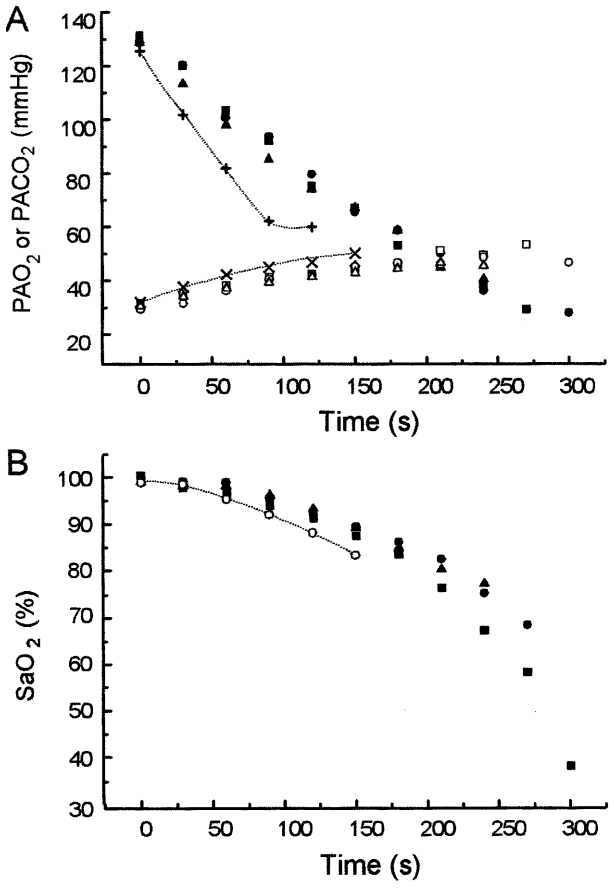

Fig. 2 Alveolar partial pressure of oxygen (P_AO_2, *filled symbols*) and alveolar partial pressure of carbon dioxide (P_ACO_2, *open symbols*) in extreme divers (**A**), and arterial oxygen saturation (S_aO_2, **B**) as a function of dry breath-hold time at rest. Symbols + and x refer to non-diver controls for P_AO_2 and P_ACO_2, respectively. Data from Ferretti et al. (1991). In panel **B**, which is reproduced after Ferretti et al. (1991), *filled symbols* refer to extreme divers, *open symbols* to non-diver controls. Data on extreme divers are reported individually

of the chest decreases less and more slowly than that of the abdomen (Warkander et al. 1996). The blood shift, the diaphragmatic shape changes, and the very high resistance of lung capillaries to mechanical stress (West et al. 1991; Bachofen et al. 1994) are likely to prevent lung disruption and alveolar haemorrhage (Lin and Hong 1996).

Alveolar gas exchange in maximal dry breath-holds

In Fig. 2, the time courses of P_AO_2 and P_ACO_2, and of arterial oxygen saturation (S_aO_2) in extreme divers during dry breath-holds are compared with those of non-diver controls. At any given time, the P_AO_2 and the S_aO_2 tend to be higher, and the P_ACO_2 to be lower in divers than in non-divers. The alveolar gas composition at the end of breath-holds of maximal duration is summarised in Table 1, together with the breath-hold time. After maximal breath-holds, the P_AO_2 and S_aO_2 of extreme divers are lower than, and the P_ACO_2 higher than, those of non-divers, but similar to those of non-extreme divers. Both groups of divers can perform longer breath-holds than non-divers. It is noteworthy that an arterial blood gas composition very close to the alveolar gas compositions reported in Table 1 was found in both the Ama and leisure divers (Qvist et al. 1993).

The volumes of oxygen taken up from (ΔVLO_2) and of carbon dioxide added to ($\Delta VLCO_2$) the lungs at the end of maximal dry breath-holds at rest are shown in Table 2. ΔVLO_2 and $\Delta VLCO_2$ are shown in Fig. 3 as a function of breath-holding time. The straight lines, indicating the resting oxygen consumption and carbon dioxide output ($\dot{V}O_{2r}$ and $\dot{V}CO_{2r}$, respectively), are also shown. In divers, ΔVLO_2 is less than the resting control volume of oxygen taken up over the same times (Ferretti et al. 1991). Indeed, ΔVLO_2 follows the same pattern as $\dot{V}O_{2r}$ only during the first 60 s of breath-holding.

Table 1 Alveolar gas composition at the end of maximal dry breath-holds at rest. In the study of Ferretti et al. (1991), *A*, *B* and *C* represent three elite divers, whose data are presented individually. In the study of Sterba and Lundgren (1988), each subject's face was flushed with water at 35°C. (P_AO_2 Alveolar oxygen partial pressure, P_ACO_2 alveolar carbon dioxide partial pressure)

Study	Subjects	Duration (s)	Duration of hyperventilation (s)	P_AO_2 (mmHg)	P_ACO_2 (mmHg)
Divers					
Ferretti et al. (1991)	A	270	60	29.3	53.3
	B	300	60	28.0	46.6
	C	240	60	40.6	45.6
Hong et al. (1971)	$n=5$	240		30.6	55.6
Non-divers					
Åstrand 1960	$n=1$	78	0	55.8	49.5
	$n=2$	125	60	49.6	41.5
Craig 1961	$n=12$	146	120	58.0	46.0
Tibes and Stegemann 1969	$n=6$	165	0	50.0	45.0
Lin et al. 1974	$n=5$	160	0	61.9	53.5
Sterba and Lundgren 1988	$n=5$	93	0	72.5	43.3
Ferretti et al. 1991	$n=9$	150	60	45.6	50.2
Feiner et al. 1995	$n=17$	107	0	60.6	45.9
		132	300	51.7	42.9

Table 2 Alveolar gas exchange at the end of maximal dry breath-holds at rest. In the study of Ferretti et al. (1991), *A*, *B* and *C* represent three elite divers, whose data are presented individually. (ΔVLO_2 Volume of O_2 taken up from the lungs during the breath-holds, $\Delta VLCO_2$ volume of oxygen added to the lungs during the breath-holds, *R* the apparent gas exchange ratio of $\Delta V\mathrm{LCO}_2$ to $\Delta V\mathrm{LO}_2$)

Study	Subject	Duration (s)	$\Delta V\mathrm{LO}_2$ (ml)	$\Delta V\mathrm{LCO}_2$ (ml)	*R*
Divers					
Ferretti et al. (1991)	A	270	693	109	0.157
	B	300	589	60	0.102
	C	240	555	60	0.108
Hong et al. (1971)	$n=5$	240	698	160	0.229
Non-divers					
Craig (1961)	$n=12$	146	639	268	0.419
Ferretti et al. (1991)	$n=9$	150	677	24	0.035

Thereafter, its rate of increase becomes slower than that of $\dot{V}\mathrm{O}_{2r}$, and tends to level off (Fig. 3). This is at variance with what is commonly observed in non-divers, who show identical $\Delta V\mathrm{LO}_2$ and $\dot{V}\mathrm{O}_{2r}$ values during dry breath-holding (Lanphier and Rahn 1963b; Craig and Harley 1968; Hong et al. 1971). Conversely, $\Delta V\mathrm{LCO}_2$ is almost equal to zero, and is sometimes even negative (reversal of carbon dioxide flow), in both divers and non-divers. As a consequence, the calculated pulmonary gas exchange ratio approaches zero, indicating carbon dioxide retention (Lanphier and Rahn 1963b; Paulev 1969; Tibes and Stegemann 1969; Hong et al. 1971).

Maximal dry breath-hold time is affected by initial lung volume, central blood volume, and temperature (Mithoefer 1959; Craig et al. 1962; Tibes and Stegemann 1969; Sebert and Sanchez 1981; Findley et al. 1983; Hayward et al. 1984; Sterba and Lundgren 1985, 1988; Feiner et al. 1995; Chang and Lundgren 1996). Most important in the present context is the effect of exercise: when dry breath-holding to the breaking point is performed at the steady-state of exercise from the same starting condition (subject's TLC, $P_\mathrm{A}\mathrm{O}_2$ 90–100 mmHg, $P_\mathrm{A}\mathrm{CO}_2$ 35–40 mmHg), the duration of breath-holding is inversely proportional to the steady-state oxygen uptake, suggesting that the volume of oxygen that can be consumed during a maximal dry apnoea is constant (Craig and Cummings 1958; Åstrand 1960; Craig and Medd 1968; Clarke and Godfrey 1969). By analogy, when hypoxic or hyperoxic mixtures are breathed prior to breath-holding at rest, the time of breath-holding is proportional to the starting volume of oxygen in the lungs (Otis et al. 1948; Klocke and Rahn 1959; Lin 1987). These findings suggest that the breaking point is attained around a given alveolar gas composition, independent of the exercise intensity and of the initial amount of oxygen in the lungs.

Body gas stores during apnoea

The observation that in the divers $\Delta V\mathrm{LO}_2$ is less than $V\mathrm{O}_{2r}$ indicates the building of an oxygen deficit, which,

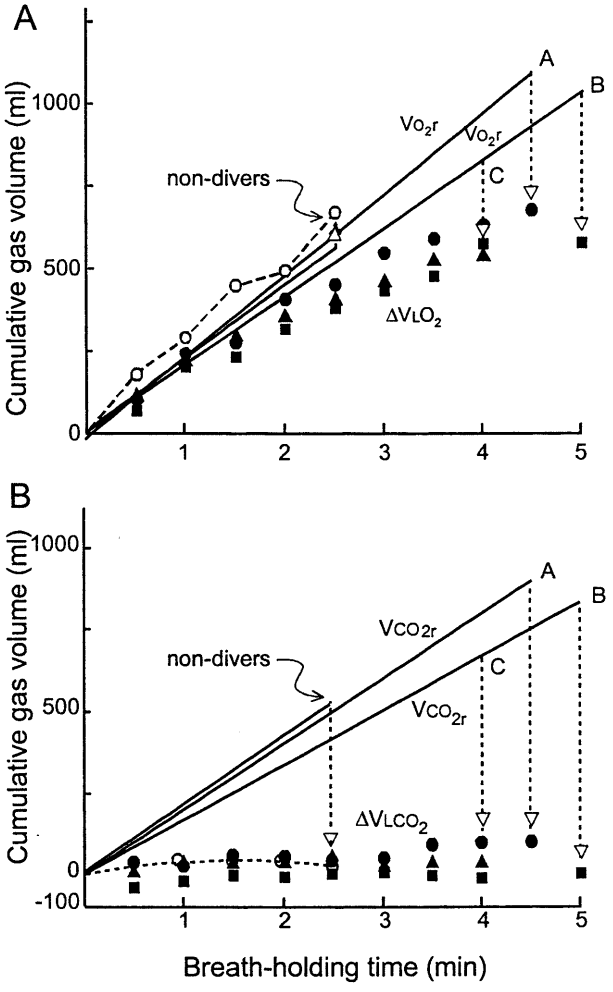

Fig. 3 Volume of oxygen (**A**) and carbon dioxide (**B**) exchanged in the lungs during resting dry breath-holds as a function of breath-hold time. *Filled symbols* refer to extreme divers, *open symbols* to non-diver controls. *Continuous straight lines* indicate the resting oxygen consumption (VO_{2r}, ml; **A**) or resting carbon dioxide production (VCO_{2r}, ml; **B**) of divers [subjects A (●), B (■) and C (▲) of Table 2, whose data are reported individually], and non-divers ( ). *Vertical dotted lines* indicate changes in oxygen or carbon dioxide stores (ΔSO_2 and ΔSCO_2, respectively) at the end of maximal breath-holds. These data are modified after Ferretti et al. (1991). (ΔVLO_2 Oxygen volume actually taken up from the lungs during breath-holds, $\Delta VLCO_2$ carbon dioxide volume actually added to the lungs during breath-holds)

in the absence of blood lactate ($[\mathrm{La}]_\mathrm{b}$) accumulation, can be sustained by the hydrolysis of high-energy phosphates and/or by the depletion of body oxygen stores. The maximum potential decrease in stored oxygen can be calculated as the difference between $V\mathrm{O}_{2r}$ and $\Delta V\mathrm{LO}_2$ (see Fig. 3), and in extreme divers it amounts to ≅500 ml (Ferretti et al. 1991). This deficit corresponds to approximately 40% of the overall initial lung oxygen stores (Cross et al. 1968), and is paid by an increased oxygen uptake in the early stages after apnoea (Linér and Linnarsson 1994).

The finding of a cumulative $\Delta V\mathrm{LCO}_2$ close to zero indicates the occurrence of tissue carbon dioxide

accumulation. The amount of stored carbon dioxide, calculated as the difference between $V\mathrm{CO}_{2r}$ and $\Delta V\mathrm{LCO}_2$ (see Table 2 and Fig. 3), approaches 800 ml in extreme divers. This value, together with the 200-mmHg increase in $P_\mathrm{A}\mathrm{CO}_2$, measured during maximal breath-holds in the same divers (Ferretti et al. 1991), indicates that the whole-body carbon dioxide storage capacity of divers is $\cong$0.7 ml·kg^{-1}·mmHg^{-1}, a figure twice as high as that ($\cong$0.3 ml·kg^{-1}·mmHg^{-1}) reported for non-divers (Klocke and Rahn 1959; Mithoefer 1959; Fowle and Campbell 1964; Hong et al. 1971).

The relatively short duration of the dives, associated with blood flow redistribution from peripheral to central vascular regions, suggests that during apnoea, most of the carbon dioxide storage occurs in fast-equilibrating tissues, such as the lungs and blood (Linér and Linnarsson 1994). The carbon dioxide storage capacity of lung tissue is 3 ml·mmHg^{-1}·l^{-1} of tissue (Farhi and Rahn 1960; Sackner et al. 1964; Plewes et al. 1976). Assuming an effective tissue volume of 1 l (Olszowka and Rahn 1987b), a lung carbon dioxide storage of 50–65 ml during maximal breath-holds can be estimated for the divers. Concerning blood, assume that: (1) the carbon dioxide dissociation curve is linear, with a slope of 4.5 ml·mmHg^{-1}·l^{-1} of blood in the investigated range of carbon dioxide partial pressures ($P\mathrm{CO}_2$; Farhi 1964), (2) the blood volume is 5 l in men and 4 l in women, and (3) disregarding the Haldane effect, the increase in mixed venous $P\mathrm{CO}_2$ is equivalent to that in $P_\mathrm{A}\mathrm{CO}_2$. On this basis, the carbon dioxide added to the blood during maximal breath-holds can be calculated as 488, 308 and 270 ml in subjects A, B and C of Ferretti et al. (1991). Thus, the amount of carbon dioxide stored in the lungs and in blood corresponds to some 70% of the overall added carbon dioxide. The remainder is probably stored in the slowly equilibrating tissues, including the muscles and the brain.

End point of apnoea and the ventilatory adaptation to diving

The $P_\mathrm{A}\mathrm{O}_2$ and $P_\mathrm{A}\mathrm{CO}_2$ values observed during dry breath-holds of increasing duration in divers and non-divers are plotted in Fig. 4 on an oxygen/carbon dioxide diagram (Rahn and Fenn 1955). The following curves are also shown: (1) the $S_\mathrm{a}\mathrm{O}_2$ isopleths for humans at sea level, (2) the normal alveolar air curve, (3) the breath-hold breaking point curve, and (4) the lines defining the regions of normal and impaired visual discrimination performance. A linear negative relationship between $P_\mathrm{A}\mathrm{O}_2$ and $P_\mathrm{A}\mathrm{CO}_2$ appears. This line is the same for divers and non-divers.

Interesting differences appear, however, when looking at the alveolar gas composition at the end of maximal breath-holds. The $P_\mathrm{A}\mathrm{O}_2$ and $P_\mathrm{A}\mathrm{CO}_2$ values for non-divers lie either on the breaking point curve, at the boundary of the severe hypoxia region, or to its right. Such an alveolar gas composition would exert a resting

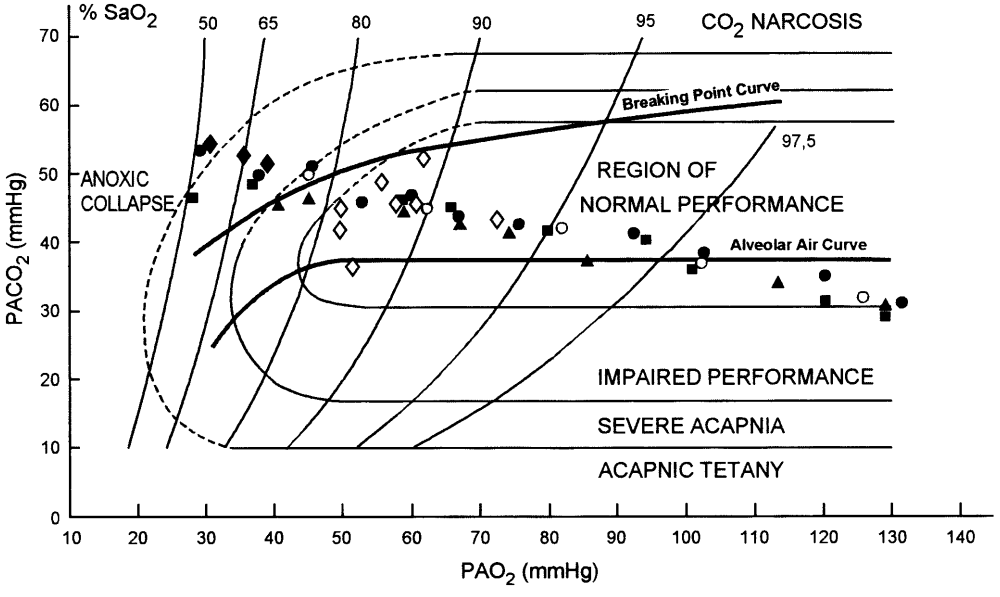

Fig. 4 Oxygen/carbon dioxide diagram describing alveolar air composition at the end of dry breath-holds up to maximal duration. The normal alveolar air curve (Rahn and Fenn 1955) and the apnoea breaking-point curve (Otis et al. 1948) are reported. Isopleths for $S_\mathrm{a}\mathrm{O}_2$ are also given (values on top of each isopleth), as well as the lines defining the regions on normal or impaired visual performance (Rahn and Fenn 1955). *Filled symbols* refer to extreme [subjects A (●), B (■), and C (▲) of Table 2, whose data are reported individually (Ferretti et al. 1991)] or leisure (◆, Hong et al. 1971) divers. The *open circle* refers to the non-diver control subjects from Ferretti et al. (1991). The *open diamond* refers to maximal breath-holds performed by non-divers, from several sources in the literature (Åstrand 1960; Craig 1961; Tibes and Stegemann 1969; Lin et al. 1974; Sterba and Lundgren 1988; Feiner et al. 1995). Lower-right values are below the normal alveolar air curve because of preceding hyperventilation. Upper-left values refer to maximal breath-holds: they occur at around about the apnoea breaking point curve for non-divers, and above it for divers

respiratory drive from combined hypoxia and hypercapnia, leading to some seven times greater ventilation than in normocapnic normoxia (Lloyd et al. 1958; Rebuck et al. 1977). In contrast, two extreme divers could prolong their apnoea until they reached P_AO_2 and P_ACO_2 values well beyond the breaking point line on the oxygen/carbon dioxide diagram, at the boundary of the expected anoxic unconsciousness region. The same is the case for some of the divers studied by Hong et al. (1971). The combination of P_AO_2 and P_ACO_2 at the breaking point of apnoea in divers would elicit, in normal individuals, a combined hypoxic and hypercapnic respiratory drive at rest, leading to some ten times greater ventilation than in normocapnic normoxia (Lloyd et al. 1958; Rebuck et al. 1977).

The finding that extreme divers are able to sustain apnoea until the attainment of P_AO_2 values lower than, and of P_ACO_2 values higher than, those on the breaking point line, revives the hypothesis that breath-hold divers may be characterised by a blunted ventilatory response to hypoxia, hypercapnia, or both. Indeed, unassisted-diving Ama were reported to have either normal ventilatory responses to both hypoxia and hypercapnia (Song et al. 1963), or a blunted ventilatory response to hypoxia (Masuda et al. 1981). The latter is also the case for synchronised swimmers (Bjurström and Schoene 1987). On the other hand, a blunted ventilatory response to hypercapnia was found in assisted-diving Ama (Masuda et al. 1982), in underwater hockey players (Davis et al. 1987), in submarine escape tower trainers (Schaefer 1965), and in Royal Navy divers (Florio et al. 1979).

The ventilatory response to breathing oxygen and carbon dioxide was determined by Grassi et al. (1994) for three extreme divers. Steady-state expired ventilation ($\dot{V}_E$), normalised per unit surface area, is plotted as a function of P_ACO_2 in Fig. 5. In all cases, except when breathing room air, the resulting steady-state $\dot{V}_E$ was significantly lower in the divers than in the controls, indicating a blunted ventilatory response to carbon dioxide in the former. Indeed, this response is not only lower than that of the non-divers, but also than that of individuals whom Lambertsen (1960) defined as having a low carbon dioxide sensitivity. In addition, the ventilatory response to oxygen was analysed by Grassi et al. (1994), who determined the time course of the changes in $\dot{V}_E$ following four breaths of pure oxygen administered from a baseline of steady-state ventilation in normoxia and in hypoxia. The peak $\dot{V}_E$ change following pure oxygen breathing was equal in divers and non-divers, indicating that the two groups had the same ventilatory responses to oxygen.

The blunted ventilatory response to carbon dioxide breathing in divers is a clear index of adaptation to extreme breath-hold diving. During the dive, the hydrostatic pressure is transmitted to the alveolar gases, so that a condition of hypercapnic hyperoxia is maintained during most of the dive (Schaefer and Carey 1962; Lanphier and Rahn 1963a; Linér et al. 1993). This being the case, a diver would be exposed to hypoxia only in the last few metres of ascent, before emersion: the duration of such exposure is likely to be too short to induce a physiological adaptation to hypoxia in divers. On the other hand, the condition of hypercapnia that was maintained during most of the dive, which could even lead to a reversal of pulmonary carbon dioxide transfer, would compel the diver to resist the drive to breathe elicited by the stimulation of central and peripheral chemoreceptors. This opposition would be facilitated by the observed blunted ventilatory response to carbon dioxide. Carbon dioxide sensitivity could be a primary determinant of the breath-hold duration, at least in professional divers. It is noteworthy that also diving mammals, which are frequently exposed to high arterial PO_2 and PCO_2 values (Qvist et al. 1986), are characterised by blunted ventilatory responses to carbon dioxide compared with non-diving mammals of similar size (Dejours 1988).

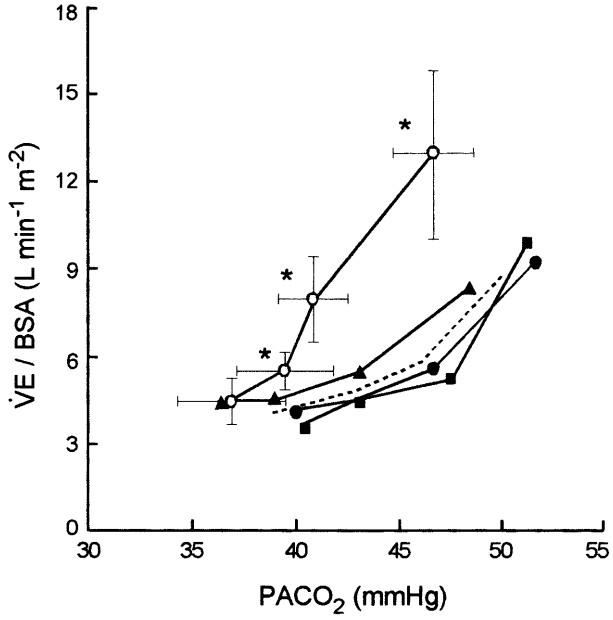

Fig. 5 Expired ventilation ($\dot{V}_E$, expressed per unit surface area; *BSA*) at the steady-state as a function of P_ACO_2. Symbols ●, ■ and ▲ refer to extreme divers A, B and C of Table 2, respectively, whose data are reported individually. The symbol ○ refers to non-diver controls. *Horizontal* and *vertical error bars*, indicating standard deviation, are also given for controls. *Significant difference from divers

Alveolar gas exchange during diving

The P_AO_2 and P_ACO_2 values observed in extreme divers at the end of the dives are shown in Table 3. Data obtained on non-professional divers as well as on Ama at shallow depths are presented for comparison. The calculated alveolar gas exchange during deep dives is shown in Table 4, together with the $[La]_b$ values. The duration of the dives is roughly proportional to the depth of the dive. Assuming an equal partition of time between

Table 3 Alveolar gas composition at the end of deep breath-hold dives. *A*, *B*, *C*, *D* and *E* represent five elite divers, whose data are presented individually

Study	Subject	Depth (m)	Time (s)	P_AO_2 (mmHg)	P_ACO_2 (mmHg)
Ferretti et al. 1991	A	70	144	39.4	47.0
		70	151	30.6	46.9
		60	115	39.2	44.1
	B	45	107	56.3	29.3
		50	131	33.5	42.8
		40	88	55.2	31.3
		50	91	46.9	30.2
	C	45	110	72.5	31.6
		50	88	93.6	26.5
Schaefer et al. 1968	D	70		40.0	35.0
Warkander et al. 1996	E	107		24.0	38.0
Caucasians					
Lanphier and Rahn 1963a	$n=7$	10	60	45.9	43.7
Schaefer 1965	$n=3$	27	90	27.0	32.0
	$n=10$	10	30	70.0	43.5
Craig and Harley 1968	$n=27$	10	60	65.0	44.3
	$n=27$	10	60	85.0	39.0
Craig and Medd 1968	$n=6$	10	60	79.0	35.0
Paulev and Naeraa 1968	$n=2$	18.5	45	60.0	40.0
	$n=1$	18.5	71	49.0	39.0
Linér et al. 1993	$n=5$	20	75	67.0	44.2
Linér and Linnarsson 1994	$n=11$	20	75	70.0	45.0
Ama					
Teruoka 1932	$n=19$	18	63	47.0	43.0
	$n=1$	21	65	30.0	39.0
Hong et al. 1963	$n=6$	11	45	41.0	42.0
	$n=1$	13	50	28.0	48.0

descent and ascent (Majorca 1987), the average diving speed for extreme divers turns out to be 0.9–1.1 $m \cdot s^{-1}$, i.e. much faster than observed for other diving categories during similar studies (0.15–0.25 $m \cdot s^{-1}$) yet slower than the speed attained by Umberto Pellizzari on the occasion of his current record dive (1.4 $m \cdot s^{-1}$). After the dives, P_AO_2 is as low as 30–40 mmHg, while P_ACO_2 can go up to 47 mmHg, independent of dive depth and duration. These figures are lower and higher, respectively, than those observed at the end of shallow dives (Table 3). Alveolar volume is lower at the end than at the start of the dive, because of a net oxygen uptake with little carbon dioxide addition (Table 4). In fact, as during dry breath-holds, the gas exchange ratio approaches zero. The peak $[La]_b$ at the end of the dives ranges between 1.71 and 6.46 mM. Within each subject, this figure is roughly related to the depth of the dive.

The P_AO_2 and P_ACO_2 values observed in the divers before and after the dives are reported in Fig. 6 in the form of an oxygen/carbon dioxide diagram (Rahn and Fenn 1955). The same curves reported in the diagram shown in Fig. 4 are also shown. A line representing the data from dry breath-holds is added. After deep dives, P_ACO_2 is linearly related to P_AO_2 (emersion curve), down to a P_AO_2 value of 45 mmHg. For lower P_AO_2 values, the line turns sharply upwards. P_AO_2 seems to level off at values around 30 mmHg, so that the breaking point curve is intercepted in the same region as for the dry breath-hold values.

The divers did not reach their limits during the deepest experimental dives. In fact, subjects E and F, on the occasion of their record attempts, dived some 30 m deeper than in the experimental dives, and still emerged conscious. The alveolar gas composition observed at the

Table 4 Alveolar gas exchange at the end of breath-hold dives. *A*, *B*, *C* and *D* represent four elite divers, whose data are presented individually. ($[La]_b$ Blood lactate concentration at the end of the dives)

Study	Subject	Depth (m)	Time (s)	ΔVLO_2 (ml)	$\Delta VLCO_2$ (ml)	R	$[La]_b$ (mM)
Extreme divers							
Ferretti et al. (1991)	A	70	144	819	42	0.05	4.41
		70	151	1,006	0	0.00	6.46
		60	115	800	–36	–0.05	3.25
	B	45	107	552	–23	–0.04	4.02
		50	131	627	55	0.09	5.68
		40	88	541	10	0.02	2.46
		50	91	603	34	0.06	3.85
	C	45	110	573	–23	–0.04	2.36
		50	88	455	39	0.09	1.71
Schaefer et al. (1968)	D	70	1,350	75	0.06		
Leisure divers							
Linér et al. (1993)	$n=5$	20	75	494	61	0.12	

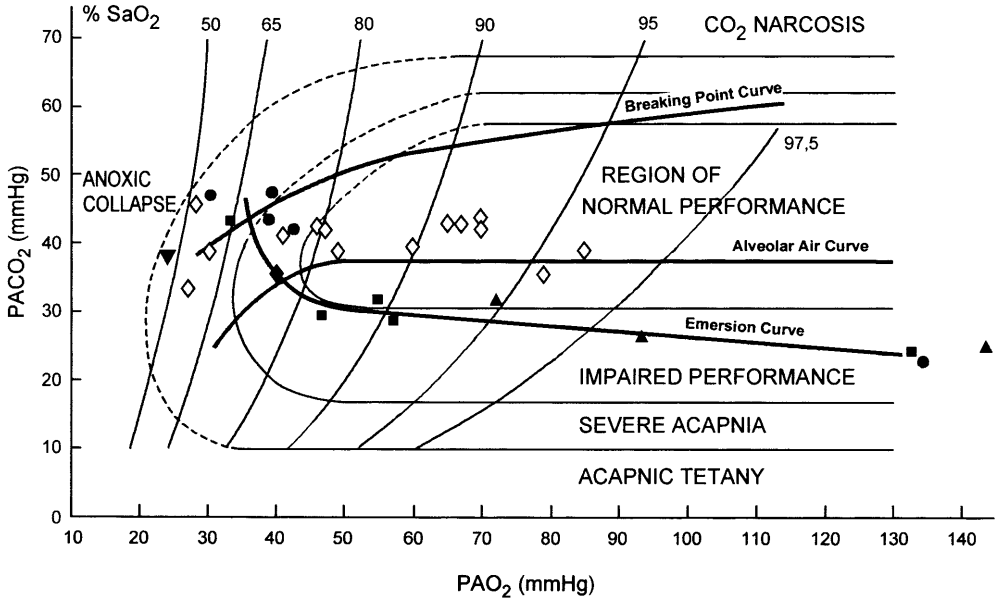

Fig. 6 Oxygen/carbon dioxide diagram describing alveolar air composition at the end of deep and shallow breath-hold dives. The normal alveolar air curve (Rahn and Fenn 1955), the apnoea breaking-point curve (Otis et al. 1948), and the deep diving emersion curve (Ferretti et al. 1991, dives preceded by hyperventilation) are reported. Isopleths for S_aO_2 are given (values on top of each isopleth), as well as the lines defining the regions on normal or impaired visual performance (Rahn and Fenn 1955). *Filled symbols* refer to extreme divers [subjects A (●), B (■), C (▲), D (◆) and E (▼) of Table 3, whose data are reported individually], after dives ranging from 40 to 107 m (Schaefer et al. 1968; Ferretti et al. 1991; Warkander et al. 1996). For subjects A, B and C, the alveolar gas composition at the end of the hyperventilation manoeuvres preceding a dive are also given (lower-right values). *Open symbols* refer to leisure or professional divers (Teruoka 1932; Hong et al. 1963; Lanphier et al. 1963a; Schaefer 1965; Craig and Harley 1968; Craig and Medd 1968; Paulev and Naeraa 1968; Linér et al. 1993; Linér and Linnarsson 1994). All subjects emerged conscious from the dives that the reported values refer to: indeed, all P_AO_2 values are higher than those that lead to anoxic collapse. It is of note that whereas the extreme divers performed prolonged hyperventilation manoeuvres before their dives, this was not the case for the leisure or professional divers

end of the deepest dives does not reach the limits attained after maximal dry breath-holds. Yet it is not far from these limits, which implies that if more energy is required to reach the record depths, most of it must come from anaerobic energy sources.

Energy expenditure during deep dives

The ΔVLO_2 values reported in Table 4 represent the overall amount of oxygen taken up from the lungs during the dives and correspond to an average rate of oxygen uptake between 287 and 417 $ml \cdot min^{-1}$. These values are independent of the diving depth, and are only slightly higher than the resting oxygen uptake. They do not represent the only energy source utilised during the dive.

Changes in tissue oxygen stores, which are essentially related to the oxygen bound to myoglobin, were estimated to correspond to 200 ml (Olszowka and Rahn 1987a, 1987b), independent of dive depths of between 20 and 100 m. This figure was calculated assuming, as a first approximation, (1) an equilibrium between tissue and end-capillary PO_2 in muscle, (2) a myoglobin concentration of 5 $mg \cdot g^{-1}$ (0.3 mM) (Jansson et al. 1982), and (3) a P_{50} for myoglobin of 5 mmHg (Gayeski and Honig 1983). The energy provided by anaerobic glycolysis can be calculated from the $[La]_b$ values given in Table 4, assuming an oxygen equivalent for lactic acid of 3 $ml \cdot mM^{-1} \cdot kg^{-1}$ (di Prampero 1981; di Prampero and Ferretti 1999). On these bases, an estimate of the energy balance in extreme divers during deep breath-hold dives is presented in Table 5 (from Ferretti et al. 1991). The average resulting metabolic power during the dives ranges between 0.5 and 0.9 $l \cdot min^{-1}$, corresponding to some 20–30% of the maximum oxygen uptake of the divers. These figures somewhat underestimate the actual energy expenditure because they do not account for the changes in blood oxygen stores, which may represent some 200–500 ml of oxygen, depending upon the final P_AO_2. Nevertheless, they indicate that the rate of energy expenditure is low during breath-hold diving (Ferretti et al. 1991), and in no case, even considering the changes in blood oxygen stores, exceeds 50% of the maximum oxygen uptake.

Such metabolic powers do not lead to $[La]_b$ accumulation in normally breathing subjects who perform light dynamic exercise. In addition, the observed $[La]_b$ accumulation is in apparent contrast with the lack of increase in $[La]_b$ during maximal resting dry breath-holds. The $[La]_b$ increase that occurs during diving in the absence of an elevated metabolic rate could be the consequence of a marked peripheral vasoconstriction. This would imply a decrease in blood flow and oxygen delivery, most likely to the muscles and skin, at the

Table 5 Energy balance of deep breath-hold dives. These data are taken from Ferretti et al. (1991). *A*, *B* and *C* represent three elite divers, whose data are presented individually. (ΔVSO_2 Changes in tissue oxygen stores during the dives, E_{La} energy – in oxygen equivalents – derived from anaerobic sources, E total energy expenditure – equal to the sum of ΔVLO_2 plus ΔVSO_2 plus E_{La}, $\dot{E}$ average rate of energy expenditure – in oxygen equivalents)

Subjects	Depth (m)	Time (s)	ΔVLO_2 (ml)	ΔVSO_2 (ml)	E_{La} (ml)	E (ml)	$\dot{E}$ (ml·min^{-1})
A	70	144	819	200	668	1,687	703
	70	151	1006	200	1,133	2,339	929
	60	115	800	200	405	1,405	733
B	45	107	552	200	474	1,226	688
	50	131	627	200	738	1,565	717
	40	88	541	200	226	967	659
	50	91	603	200	447	1,250	824
C	45	110	573	200	221	994	542
	50	88	455	200	97	752	513

expense of an increased reliance on anaerobic energy sources, thus providing indirect support to the hypothesis that a diving response takes place in extreme divers during deep breath-hold dives.

The hypothesis of a diving response in humans

The occurrence of a diving response in freely diving mammals was postulated long ago (Scholander 1940), and widely demonstrated afterwards. It consists of bradycardia, decreased cardiac output, increased arterial blood pressure, and redistribution of regional blood flows. Bradycardia may have a vagal origin, and is associated with an extreme peripheral vasoconstriction. The latter predominates on the former, so that arterial blood pressure increases. Cardiac output is reduced, reflecting the inability by the heart to achieve a compensatory increase in stroke volume, as a consequence of a reduced venous return. Blood flow is redistributed, preferentially to the vital organs, and lactate may be accumulated in unperfused muscles (Andersen 1966; Lin 1982; Blix and Folkow 1983; Hochachka 1986; Zapol 1996; Butler and Jones 1997; Kooyman and Ponganis 1998).

The hypothesis that a diving response may also occur in humans stems essentially from observations made during face-immersion experiments. A decrease in heart rate, perhaps due to increased vagal activity (Hayashi et al. 1997), was found in every study on subjects who held their breath at the water surface (Manley 1990; Gooden 1994). The occurrence of bradycardia was also observed during breath-holding while exercising (Strømme et al. 1970; Butler and Woakes 1987; Sundblad and Linnarsson 1996; Lindholm et al. 1999). Arterial blood pressure is also elevated (Kawakami et al. 1967; Heistad et al. 1968; Hong et al. 1971; Lin et al. 1983; Bjertnaes et al. 1984; Paulev et al. 1988). These effects are more pronounced when the starting lung volume is lower than the TLC (Andersson and Schagatay 1998), and when the subjects have undergone a specific breath-hold training protocol (Schagatay et al. 2000). Cardiac output appears to be either unchanged (Heistad et al. 1968; Hong et al. 1971; Paulev et al. 1988), increased (Hong 1988) or decreased (Paulev and Wetterqvist 1968; Lin et al. 1983; Bjertnaes et al. 1984). The ensuing increase in peripheral vascular resistance suggests strong peripheral vasoconstriction (Lin et al. 1983; Bjertnaes et al. 1984). In fact, peripheral blood flow is reduced during breath-holding (Sterba and Lundgren 1988; Andersson and Schagatay 1998), but carotid artery blood flow is increased (Jiang et al. 1994; Pan et al. 1997), in agreement with the hypothesis of blood flow redistribution towards the brain.

During breath-hold diving, the main hint for a diving response is provided by bradycardia, which occurs in a variety of diving populations (Olsen et al. 1962; Scholander et al. 1962; Irving 1963; Hong et al. 1967; Paulev 1968; Sterba and Lundgren 1985; Ferrigno et al. 1991). In contrast, cardiac output was found to increase during diving to 20 m in thermoneutral water (Ferrigno et al. 1987), an observation in apparent contradiction with the diving response hypothesis.

Heart rate and rhythm during deep breath-hold diving

Ferrigno et al. (1991) recorded the evolution of the heart rate of extreme divers during sea dives, and the diving pattern of sea dives lasting 100–130 s (Fig. 7). After an increase during the hyperventilation period, heart rate dropped dramatically after the beginning of the dive, so that values as low as 20 beats·min^{-1} (subjects B and C) and 24 beats·min^{-1} (subject A) were observed at a depth of 40 m. A slow but progressive increase in heart rate took place thereafter, especially after emersion. Similarly, in subject E, during a dive to 107 m, heart rate decreased to a minimum of 40 beats·min^{-1}, attained at a depth of 60 m. This level was maintained constant until the bottom of the dive and during the first phase of the subsequent ascent (Warkander et al. 1996). The bradycardia observed in extreme divers during deep dives confirms previous observations on humans diving at shallower depths (Craig 1963; Hong et al. 1967; Song et al. 1969; Sterba and Lundgren 1985; Ferrigno et al. 1987), and concurs with the observations made on diving mammals, although the latter, in contrast with

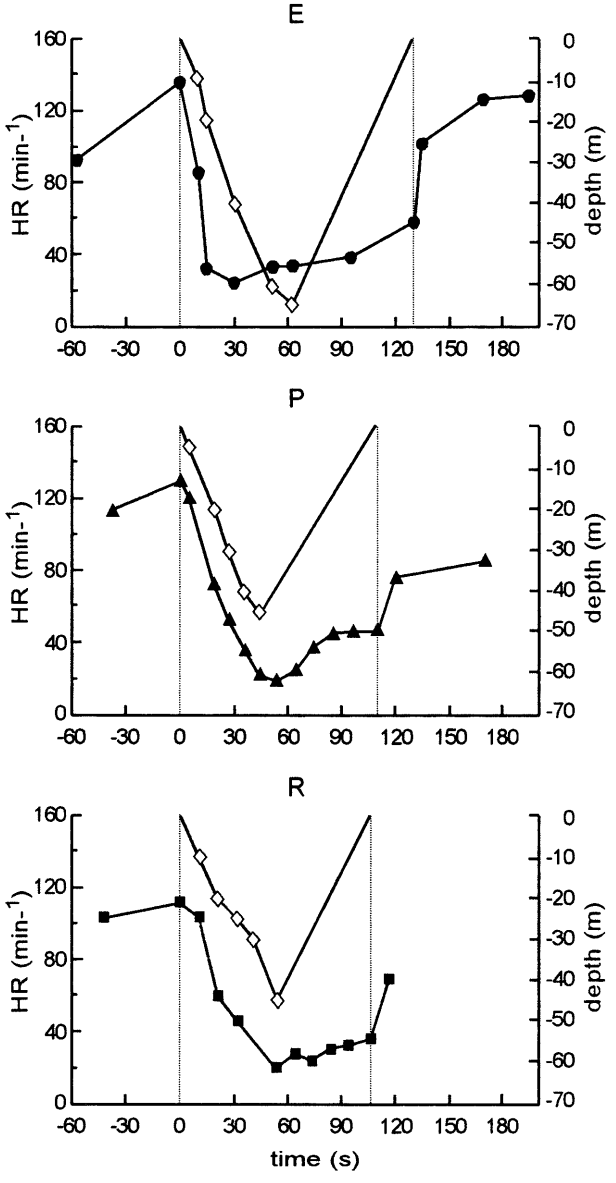

Fig. 7 Heart rate (*HR*) changes and diving patterns as a function of time during deep breath-hold dives in the sea. *Filled symbols* refer to HR, *open symbols* to depth for, from top to bottom, divers A, C and B. Modified after Ferrigno et al. (1991)

human divers, do not show dysrhythmic beats (Elsner and Gooden 1983). Similarly, Arnold (1985) reported some cases of extreme asymptomatic bradycardia elicited by apnoeic face immersion in iced water. The great decrease in heart rate induced a continuous shift of the site of impulse generation from the sino-atrial to the atrio-ventricular node during descent in subjects B and C. Junctional rhythm was maintained during ascent. A ventricular bigeminal rhythm, lasting 20 s, was observed in subject C during ascent. When the same subjects were investigated in a pressure chamber at a water temperature similar to that of the sea dives, heart rate values of between 20 and 30 beats·min^{-1} were recorded near the bottom. The longest R-R interval, observed at 30 m during descent, lasted 7.2 s, which corresponds to an instantaneous heart rate of 8 beats·min^{-1}. Numerous arrhythmic beats were observed, especially during ascent. Again, junctional rhythm was often observed in conjunction with extreme bradycardia. Subject B had a dive in which no one single sinus beat was observed for 45 s. Ventricular and supra-ventricular extrasystoles were observed frequently. No specific type of arrhythmia predominated (Ferrigno et al. 1997).

Dysrhythmic beats are a common finding during breath-hold diving, even at shallow depths (Olsen et al. 1962; Scholander et al. 1962; Sasamoto 1965; Hong et al. 1967; Paulev 1968; McDonough et al. 1987; Bonneau et al. 1989; Tipton et al. 1994). Their frequent occurrence during actual and simulated dives, however, is not associated with either other clinical signs or with symptoms reported by the divers, even though a drowning syndrome may occur in patients after a period of 5–10 s without a heart contraction. The occurrence of a syncope may be prevented, at least in part, if all premature beats are haemodynamically effective, as was the case for subjects A, B and C, from whom simultaneous recordings of heart rate and arterial blood pressure were obtained. This may have contributed to the maintenance of a blood pressure level that was sufficient to keep cardiac output as high as required for the perfusion of vital organs, particularly the brain.

Haemodynamics of deep breath-hold diving

In spite of extreme bradycardia, the stroke volume of the heart, determined by impedance cardiography during dives in a hyperbaric chamber, does not change appreciably in extreme divers (Ferrigno et al. 1997). As a consequence, at the beginning of the dive, cardiac output is higher than the resting control value, then it progressively decreases during descent, to reach values that are lower than resting values. In two divers from the study of Ferrigno et al. (1997), cardiac output values below 3 l·min^{-1} were found toward the end of descent!

Invasive beat-by-beat arterial blood pressure during dives was obtained for two divers in the study of Ferrigno et al. (1997). Blood pressure, which tends to increase during hyperventilation, reaches particularly high values during descent (280/200 mmHg in one case, 290/150 mmHg in the other, with occasional systolic peaks above 300 mmHg). It stays elevated during ascent until emersion, to be progressively brought back towards control values during recovery. Although elevated, arterial blood pressure during face immersion or breath-holding just below the water surface does not undergo as intense and dramatic changes as observed in extreme divers during deep dives (Olsen et al. 1962; Scholander et al. 1962; Kawakami et al. 1967; Heistad et al. 1968; Campbell et al. 1969; Gross et al. 1976; Bjertnaes et al. 1984; Sterba and Lundgren 1988).

To my knowledge, peripheral blood flow has not been measured in extreme divers during deep dives. However,

very high arterial blood pressure values, associated with very low cardiac output values, indicate a high total peripheral resistance during the dive. Moreover, blood pressure decreases slowly during diastole, thereby preventing the attainment of very low diastolic pressure values, in spite of the extreme bradycardia attained early during descent. These findings are strongly suggestive of extreme peripheral vasoconstriction. Since there is a net accumulation of lactic acid in blood during the dives, as shown in Table 4, indicating the occurrence of anaerobic metabolism, it is likely that some peripheral region could even be excluded from blood circulation during deep dives.

Of a diving response in extreme divers

The cardiovascular adjustments during deep breath-hold diving described in the preceding paragraphs represent, to the author's knowledge, the first comprehensive clear-cut demonstration of a diving response in humans. In fact, a strong peripheral vasoconstriction is associated with a marked increase in blood pressure. Activation of the baroreceptors results in an extreme drop in heart rate, which is not compensated for by a higher stroke volume, with a consequent decrease in cardiac output and increase in total peripheral resistance. Preservation of the stroke volume, which might play a key role in the prevention of a drowning syndrome, may be due to several mechanisms, including water immersion, leg squeezing during compression, and the efficacy of dysrhythmic heart beats. During breath-holding, the cardiac index is higher during water submersion than when in air (Ferrigno et al. 1987; Linér and Linnarsson 1995), probably as a consequence of increased heart filling. An analogous mechanism may contribute to the maintenance of stroke volume in deep dives. Leg compression induces a spontaneous reduction in limb blood volume associated with an increase in venous return. Finally, the haemodynamic efficacy of premature beats keeps arterial blood pressure elevated and opposes an excessive drop in cardiac output.

The diving response observed in humans, although analogous to that occurring in diving mammals, is nonetheless characterised by some differences with respect to the latter. Diving mammals do not show dysrhythmic beats (Elsner and Gooden 1983). Moreover, an extreme increase in arterial blood pressure, as observed in human divers, is prevented in other mammals (e.g. the seal) by an extreme reduction in both cardiac output and regional blood flows (Zapol et al. 1979).

Oxygen-preserving mechanisms

The diving response and the ensuing lactate accumulation at low metabolic levels can be viewed as an oxygen-preserving mechanism, which postpones the attainment of the breaking point of apnoea and of an alveolar gas composition that is incompatible with consciousness. Unperfused muscles do not consume oxygen, except for the amount bound to myoglobin, so that oxygen stores are reduced at a slower rate than predicted from the expected metabolic rate. That the diving response acts as an oxygen-preserving mechanism has indeed been demonstrated in diving mammals (Butler and Jones 1997). In humans, Lindholm et al. (1999) have shown that the greater the bradycardia and the increase in arterial blood pressure, the slower the reduction in S_aO_2 during breath-holding with exercise.

Oxygen-conserving mechanisms can also be related to an increase in the size of oxygen stores. For instance, the Weddel seal is able to increase its haematocrit and haemoglobin concentration, thereby enhancing its body's capacity to store oxygen (Qvist et al. 1986). This finding was possibly attributed to spleen contraction during the dive. No equivalent determinations on extreme divers have been performed, yet Hurford et al. (1990) demonstrated, using ultrasonography, that splenic contraction actually occurs during breath-hold diving in the Korean Ama.

Finally, it should be pointed out that the Ama, who perform frequent breath-hold dives every working day, were characterised by reduced basal metabolic rates and increased body thermal insulation (Rennie et al. 1962; Kang et al. 1963, Kang et al. 1965), which can also be viewed as oxygen-preserving mechanisms. These features, however, have been progressively lost since the introduction of wet suits instead of cotton suits (Kang et al. 1983; Park et al. 1983b; Hong et al. 1986).

Of a 150-m dive

The evolution of alveolar gases during a dive to 100 m lasting 220 s (Enzo Majorca's record) was modelled by Olszowka and Rahn (1987b). Although based on the erroneous assumptions of a resting oxygen consumption and an absence of lactate accumulation during the dive, the computation showed a P_AO_2 value at the end of the dive of some 35 mmHg, to be compared with the 24 mmHg figure observed after a 107-m dive (Warkander et al. 1996).

I have tried to perform a similar analysis for a simulated current-record dive to 150 m performed at TLC and preceded by hyperventilation manoeuvres like those described by Ferretti et al. (1991). Details on this simulation procedure are reported in Appendix 2. Within the context of this simulation, at emersion from a 150-m dive like that performed by Umberto Pellizzari, Enzo Majorca (TLC = 7.2 l)[1] and Bob Croft (TLC = 7.8 l) would exhibit a P_AO_2 of 26.1 and 34.6 mmHg, and a P_ACO_2 of 41.1 and 39.7 mmHg, respectively. These figures are still compatible with consciousness. The

[1]Contemporary divers may have an even higher TLC: values above 9 l were indeed measured on two divers from Cuba (unpublished observations).

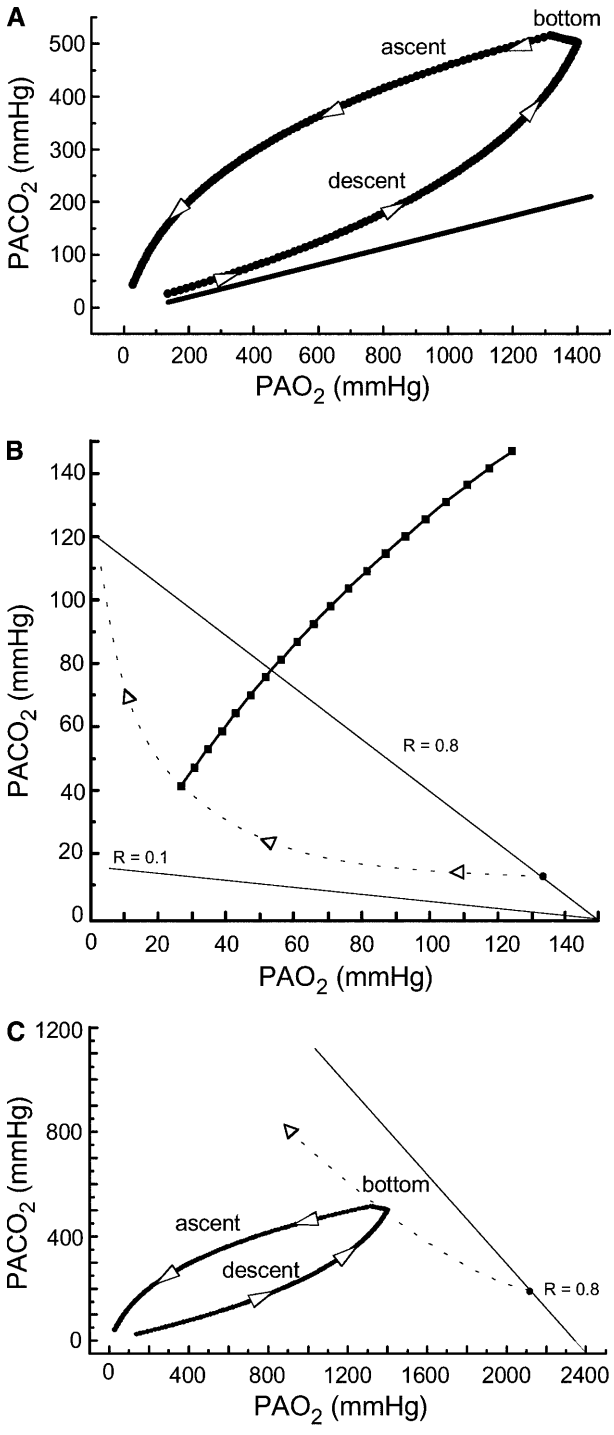

Fig. 8A–C Evolution of alveolar gas composition during a simulated breath-hold dive to 150 m. **A** The estimated alveolar gas composition during the entire dive. *Arrows* indicate the pattern followed by alveolar gases during descent, at the bottom of the dive, and during the subsequent ascent. The *straight line* indicates the oxygen and carbon dioxide pressures (PO_2 and PCO_2, respectively) in a balloon filled with a gas whose composition was the same as that of the alveolar air at the start of the dive, describing the alveolar gas patterns in the absence of metabolism. The upward and leftward displacements of the actual diving pattern with respect to the balloon line describe the effects of metabolism in the diver. **B** Magnification of the data from the final part of the ascent, describing the unsteady-state condition on emersion. Steady-state gas exchange ratio (R) isopleths for $R=0.8$ and $R=0.1$ at the inspired PO_2 of 150 mmHg (surface) are also reported. The *dashed curve* describes a hypothetical pathway of alveolar hypoventilation upon which the diver may operate; the *arrowhead* indicates the direction followed along the pathway. **C** The unsteady-state condition at the bottom of the dive. The curve simulating alveolar gas composition during a 150-m dive is reported in its entirety, as in **A**. The isopleth for $R=0.8$ at the inspired PO_2 of 2400 mmHg (bottom) is also reported. The *dashed curve* describes the pathway of alveolar hypoventilation upon which the diver may operate; the *arrowhead* indicates the direction followed along the pathway. The alveolar pathways reported in **B** and **C** cannot be completed: indeed the new steady-state for an alveolar ventilation of 0 $l \cdot min^{-1}$ would occur at $P_AO_2=0$ mmHg, and a P_ACO_2 of either 120 mmHg at the surface or 1920 mmHg at the bottom of the dive

evolution of alveolar gas composition during a computer-simulated dive to 150 m is shown in Fig. 8. The effects of changing TLC, the rate of energy expenditure and the rate of $[La]_b$ accumulation on the P_AO_2 at the end of a 150-m dive are shown in Fig. 9. P_AO_2 values are higher with increasing values of TLC, the lower the rate of energy expenditure, and the greater the rate of $[La]_b$ accumulation. Curiously enough, the simulation predicts that Enzo Majorca or Bob Croft cannot emerge conscious from a 150-m dive if the rate of lactate accumulation is 40 $\mu M \cdot s^{-1}$ instead of 45 $\mu M \cdot s^{-1}$, nor can they if the rate of energy expenditure increases from 1.00 to 1.05 $l \cdot min^{-1}$! The occurrence of a marked diving response, leading to a substantial contribution of anaerobic energy sources to the overall energy expenditure during the dive, is a *condicio sine qua non* for a record dive. Finally, it would be impossible to dive to 150 m and emerge conscious (or even alive) if the dive is carried out at a speed of 0.9 $m \cdot s^{-1}$, as was the case for Enzo Majorca on the occasion of his 100-m record dive. Such a speed would in fact prolong the breath-hold duration to times that are incompatible with consciousness upon emersion.

Conclusions

The results obtained from studies on extreme breath-hold divers during resting dry breath-holds and deep dives in the sea show that: (1) at the end of maximal dry breath-holds, the P_AO_2 is lower and the P_ACO_2 higher than at the breaking point of apnoea for non-diving controls, (2) the alveolar gas composition at emersion from dives to 40–107 m is compatible with consciousness, and (3) a remarkable accumulation of lactate occurs during deep dives, despite the low rate of energy expenditure. P_AO_2 values higher than, and P_ACO_2 values lower than those observed after maximal dry breath-holds have been shown to occur after deep dives. A significant reserve is still available for longer and deeper dives than those that have been performed for the sake of the studies reviewed herein.

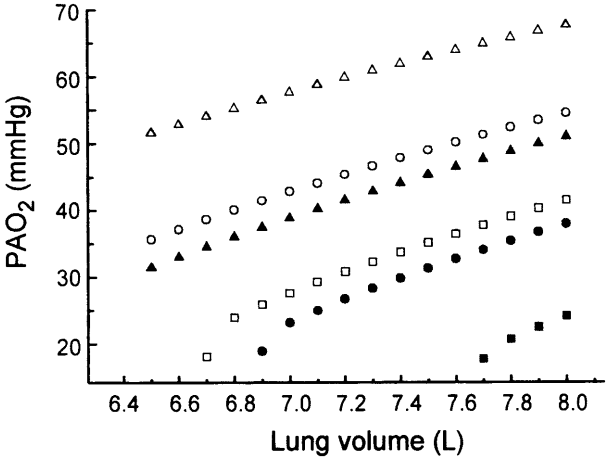

Fig. 9 Effects of changing starting lung volume on P_AO_2 upon emersion from a 150-m dive, at different rates of energy expenditure and blood lactate accumulation. The rate of blood lactate accumulation is either 45 $\mu M \cdot l^{-1}$ (*filled symbols*) or 50 $\mu M \cdot l^{-1}$ (*open symbols*). Three values of rate of energy expenditure were tested: 0.95 $l \cdot min^{-1}$ (*triangles*), 1.00 $l \cdot min^{-1}$ (*circles*) and 1.05 $l \cdot min^{-1}$ (*squares*). Increasing the rate of blood lactate accumulation displaces the curves upwards (e.g. from *triangle* to *open triangle*). Increasing the rate of energy expenditure displaces the curves downward (e.g. from *filled triangle* to *filled circle* or *filled square*)

These results suggest the occurrence of some adaptive mechanisms that allow the prolongation of apnoea beyond the limits attained by non-diving controls, and the preservation of oxygen stores during the dives. The occurrence of a consistent diving response has been demonstrated, which includes peripheral vasoconstriction, increased arterial blood pressure, bradycardia and lowered cardiac output. This phenomenon may lead to the exclusion of some peripheral regions from perfusion, with consequent reliance on anaerobic metabolism, thereby explaining the observation of $[La]_b$ accumulation during dives. As a consequence, oxygen could be preserved and redistributed towards the vital organs, particularly the brain. In addition, extreme breath-hold divers appear to have a blunted ventilatory response to carbon dioxide breathing, possibly as a consequence of frequent exposure to high PCO_2 levels during the dives. This would allow the attainment of the particularly low P_AO_2 and high P_ACO_2 values that are observed at the end of maximal dry breath-holds.

The reviewed literature suggests that an energetic constraint may indeed impose the ultimate limit to deep breath-hold diving. $[La]_b$ accumulation cannot exceed the limits imposed by the maximal hydrogen ion concentration that can be tolerated by the muscles and by the cerebrospinal fluid, and oxygen stores cannot decrease to levels that are incompatible with consciousness. This being the case, there still exist appreciable margins for improving the depth records in breath-hold diving. The ultimate aim of a diver is to dive deeper while still emerging conscious. This implies an increase in the diving speed, in order to avoid prolongation of breath-hold time and excessive lactate accumulation. The diving speed (v) is directly proportional to the rate of energy expenditure:

$$v = E/C \quad (1)$$

where C is the energy cost of diving. The term C in turn can be described by two other terms, as follows:

$$C = D/\eta \quad (2)$$

where D is the drag (i.e. the force opposing the movement of a body in water), which depends upon the water resistance and upon the buoyancy of the body, and η is the mechanical efficiency of movement. There are no margins to increase E, or to enhance the body's oxygen stores, unless dangerous and unethical practices are carried out. Thus, an increase in v can be achieved only through a reduction of C, which requires either a reduction in D, or an improvement in η, or both. Hydrodynamic research may help to lower D, especially through a decrease in the drag coefficient (Pendergast 1987), the pursuit of better diving techniques, limiting useless movements and reducing internal work, all of which may help to improve η.

Three of the divers from whom the data reported in this review were obtained are members of the same family. Indeed, all of the reported cardiovascular data were obtained from them. Thus, it cannot be excluded that at least some of the observed phenomena represent genetically inherited characteristics, rather than real adaptations to breath-hold diving.

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Appendix 1

On 16 July 1913, Mr. Giorgios Haggi Statti rescued the anchor of the Italian ship "Regina Margherita", which was grounded at a depth of 70 m, during three consecutive breath-hold dives. The following is his medical report, which was established by the ship's physician after the dives. This medical report was translated by the author, and the original text is reported in Molfino and Zannini (1964).

Haggi Statti Giorgios, born in Simi, sponge diver, 35-year-old, married, four children, all alive and healthy. He is 1.70 m tall and weighs 65 kg. His resting thoracic perimeter is 0.92 m, being 0.98 m after a maximal inspiration, and 0.90 m after a maximal expiration. Dark-skinned, slim, he has an ordinary muscle mass. Although an examination of the thorax reveals a remarkable lung emphysema, the upper part of the thorax

has not yet reached a large size, even if it is somewhat convex and rigid. The heart tones are far, but regular. The pulse rate is 80–90, and the respiratory rate is 20–22. Nothing abnormal in the nervous system, nor in the eyes. He has impaired auditory function because of the complete lack of the eardrum in one ear, and only the remnants of one in the other. He suffered from no illness, except for a trachoma, healed after surgery. He reports only pain in his back, which he tolerates resignedly. When asked to hold his breath in the ordinary ambient, he first refused, claiming that the test had no value because he could resist much more under water. Then he accepted, and it resulted that his capacity under these conditions is only 40 s. Yet in the rescue operations he dived to depths varying from 40 to 60 m, and even to 80 m, staying under water for 1.30–3.35 min. He claims that he has reached 110 m, and that he can stay at 30 m for up to 7 min. Statti emerged from all dives in good shape and vigour, as demonstrated by the way he jumped into the boat and released the water that had entered his nose and ears. When questioned on the phenomena he feels during the dives, he says he perceives none. Probably accustomed since childhood, he does not perceive them. He only says he feels all pressure on his shoulders. Nothing on his eyes. He also claims that at 80 m, despite the weakening of light, one can see enough to work, if the water is clear.

Appendix 2

A simulation of the evolution of alveolar gases during a 150-m dive was carried out along the following lines. Imagine a diver who dives at a constant speed of 1.43 $m \cdot s^{-1}$, with an average rate of energy expenditure of 1 $l \cdot min^{-1}$ STPD. At any time t, he travels a given distance, so that the changes of total pressure in the lungs can be computed by consuming a given constant amount of energy. This energy (E_{tot}) comes from lung, blood and tissue oxygen stores, and from anaerobic lactic and alactic energy stores. Let us now subtract a constant amount of energy derived from anaerobic lactic metabolism (E_{La}), and from tissue oxygen stores. E_{La} is calculated on the assumption of a constant rate of lactate accumulation in blood of 0.045 $mM \cdot s^{-1}$, as can be computed for subject A from the data given in Table 4, and of an oxygen equivalent for blood lactate accumulation of 3 $mlO_2 \cdot mM^{-1} \cdot kg^{-1}$ (di Prampero and Ferretti 1999). For the sake of simplicity, an overall change in tissue oxygen stores of 200 ml (Olszowka and Rahn 1987b) is also assumed to take place at a constant rate. The subtraction of E_{La} and tissue oxygen stores from E_{tot}, yields an amount of energy – derived from aerobic and anaerobic alactic energy sources – that is consumed during time t at a rate corresponding to the rate of oxygen consumption that one would expect after the attainment of a virtual metabolic steady state (let's call this $\dot{V}O_{2ss}$). After the subtraction of resting oxygen consumption, oxygen consumption at time t ($\dot{V}O_{2(t)}$) can be calculated from $\dot{V}O_{2ss}$, on the assumption that the kinetics of oxygen consumption can be described by a mono-exponential function, with a velocity constant of 0.027 s^{-1} (di Prampero and Ferretti 1999). The knowledge of $\dot{V}O_{2(t)}$ allows the calculation of the energy derived from anaerobic alactic sources at time t as the difference between $\dot{V}O_{2ss}$ and ($\dot{V}O_{2(t)}$, corrected for the appropriate time scale ($VO_{2(t)}$). The term $VO_{2(t)}$, which is made of two components, the oxygen derived from the lungs and the oxygen derived from blood stores, is partitioned between the lungs and blood as follows. At any time t, let a given volume of oxygen move from the lungs to the blood, and another volume of oxygen move from the blood to the tissues: the difference between these two volumes would be equal to the change in blood oxygen stores. The changes in blood oxygen stores at time t–1 are added to the $VO_{2(t)}$ to obtain the amount of oxygen taken up from the lungs, expressed in STPD. This allows the calculation of the STPD volume of oxygen inside the lungs at time t, which, after correction for the pressure acting at the depth attained at time t, allows the computation of P_AO_2. Assuming (1) a constant cardiac output of 4 $l \cdot min^{-1}$, (2) equilibrium between alveolar and end-capillary gases, (3) 2% venous admixture, and (4) a blood volume of 5 l, 20% of which is located on the arterial side, the new level attained by blood oxygen stores at the end of time t, and thus the changes in oxygen stores occurring at time t, can be calculated. The latter will then be used for the calculation of lung oxygen uptake at time $t+1$. Finally, the volume of carbon dioxide added to the lungs can be calculated by assuming a gas exchange ratio of 0.1, and the corresponding P_ACO_2 values obtained.

This procedure was iterated from the start to the end of a 150-m dive, performed at a speed of 1.43 $m \cdot s^{-1}$, with a starting lung volume equal to TLC, and starting P_AO_2 and P_ACO_2 levels equal to 134.7 and 24.0 mmHg, respectively (data from Ferretti et al. 1991; Table 2, average of subjects EM and RM). The simulation results reported in Figs. 8 and 9 were obtained for a diver who weighs 70 kg, and has a blood haemoglobin concentration of 150 $g \cdot l^{-1}$, a TLC of 7.2 l and a mixed venous oxygen concentration of 125 $mlO_2 \cdot l^{-1}$. The time basis for each iteration was 1 s, the total duration of the dive was 220 s, with 10 s spent at the bottom.

It is of note that the assumption of constant rates of energy expenditures from various sources, although adding simplicity to the simulation, is not correct. For instance, lactate is likely to accumulate at a greater rate when cardiac output attains its minimum, whereas tissue oxygen stores are likely to be depleted essentially in the final part of ascent, when the P_AO_2 has returned to values below 100 mmHg. Although this introduces a certain degree of error in the data shown in Fig. 8, in so far as the P_AO_2 at 150 m may be somewhat underestimated, this should not affect the alveolar gas composition at the end of the dive.

References

Andersen HT (1966) Physiological adaptations in diving vertebrates. Physiol Rev 46:212–243

Andersson J, Schagatay E (1998) Effects of lung volume and involuntary breathing movements on the human diving response. Eur J Appl Physiol 77:19–24

Arborelius M Jr, Balldin UI, Lilja B, Lundgren CEG (1972) Hemodynamic changes in man during immersion with the head above water. Aerospace Med 43:592–598

Arnold RW (1985) Extremes in human breath-hold, facial immersion bradycardia. Undersea Biomed Res 12:183–190

Åstrand PO (1960) Breath holding during and after muscular exercise. J Appl Physiol 15:220–224

Bachofen H, Schurch S, Possmayer F (1994) Disturbance of alveolar lining layer: effects on alveolar microstructure. J Appl Physiol 76:1983–1992

Bjertnaes L, Hange A, Kjekshus J, Soyland E (1984) Cardiovascular responses to face immersion and apnea during steady-state muscle exercise. Acta Physiol Scand 120:605–612

Bjurström RL, Schoene RB (1987) Control of ventilation in elite synchronized swimmers. J Appl Physiol 63:1019–1024

Blix AS, Folkow B (1983) Cardiovascular adjustments to diving in mammals and birds. In: Shephard JT, Abboud FM (eds) Handbook of physiology. The cardiovascular system III. American Physiological Society, Bethesda, Md., pp 917–945

Bonneau A, Friemel F, Lapierre D (1989) Electrocardiographic aspects of skin diving. Eur J Appl Physiol 58:487–493

Butler PJ, Jones DR (1997) Physiology of diving of birds and mammals. Physiol Rev 77:837–899

Butler PJ, Woakes AJ (1987) Heart rate in humans during underwater swimming with and without breath-hold. Respir Physiol 69:387–399

Campbell LB, Gooden BA, Horowith JD (1969) Cardiovascular responses to partial and total immersion in man. J Physiol (Lond) 202:239–250

Chang LP, Lundgren CEG (1996) Maximal breath-holding time and immediate tissue CO_2 storage capacity during head-out immersion in humans. Eur J Appl Physiol 73:210–218

Clark TJ, Godfrey S (1969) The effect of CO_2 on ventilation and breath-holding during exercise and while breathing through an added resistance. J Physiol (Lond) 201:551–566

Craig AB Jr (1961) Causes of loss of consciousness during underwater swimming. J Appl Physiol 16:583–586

Craig AB Jr (1963) Heart rate responses to apneic underwater diving and to breath-holding in man. J Appl Physiol 18:854–862

Craig AB Jr (1968) Depth limits of breath hold diving (an example of Fennology). Respir Physiol 5:14–22

Craig AB Jr (1976) Summary of 58 cases of loss of consciousness during underwater swimming and diving. Med Sci Sports 8:171–175

Craig AB Jr (1987a) Von Trieleben's diving bell. In: Lundgren CEG, Ferrigno M (eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 4–8

Craig AB Jr (1987b) Depth limits of breath-hold diving. In: Lundgren CEG, Ferrigno M (eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 9–11

Craig FN, Cummings EG (1958) Breath holding during exercise. J Appl Physiol 13:30–34

Craig AB Jr, Harley AD (1968) Alveolar gas exchanges during breath-hold dives. J Appl Physiol 24:182–189

Craig AB Jr, Medd WL (1968) Oxygen consumption and carbon dioxide production during breath-hold diving. J Appl Physiol 24:190–202

Craig AB Jr, Halstead LS, Schmidt GH, Schnier BR (1962) Influences of exercise and O_2 on breath-holding. J Appl Physiol 17:225–227

Cross CE, Packer BS, Altman M, Gee JBL, Murdaugh HV, Robin EE (1968) The determination of total body exchangeable O_2 stores. J Clin Invest 47:2402–2410

Davis FM, Graves MP, Guy HJB, Prisk GK, Tanner TE (1987) Carbon dioxide response and breath-hold times in underwater hockey players. Undersea Biomed Res 14:527–534

Davis RH (1934) Deep diving and underwater rescue. J R Soc Arts 82:1032–1047

Dejours P (1988) Responses to ambient oxygen and carbon dioxide variations. In: Respiration in water and air. Elsevier, Amsterdam, pp 49–69

di Prampero PE (1981) Energetics of muscular exercise. Rev Physiol Biochem Pharmacol 89:143–222

di Prampero PE, Ferretti G (1999) The energetics of anaerobic muscle metabolism: a reappraisal of older and recent concepts. Respir Physiol 118:103–115

Elsner R, Gooden B (1983) Diving and asphyxia. Cambridge University Press, New York

Farhi LE (1964) Gas stores in the body. In: Fenn WO, Rahn H (eds) Handbook of physiology. Respiration. American Physiological Society, Washington, D.C., pp 873–885

Farhi LE, Rahn H (1960) Dynamics of changes in carbon dioxide stores. Anesthesiology 21:604–614

Feiner JR, Bickler PE, Severinghaus JW (1995) Hypoxic ventilatory response predicts the extent of maximal breath-holds in man. Respir Physiol 100:213–222

Ferretti G, Costa M, Ferrigno M, Grassi B, Marconi C, Lundgren CEG, Cerretelli P (1991) Alveolar gas composition and exchange during deep breath-hold diving and dry breath holds in elite divers. J Appl Physiol 70:794–802

Ferrigno M, Lundgren CEG (1999) Human breath-hold diving. In: Lundgren CEG, Miller J (eds) The lung at depth. Dekker, New York, pp 529–585

Ferrigno M, Hickey DD, Linér MH, Lundgren CEG (1987) Simulated breath-hold diving to 20 meters: cardiac performance in humans. J Appl Physiol 62:2160–2167

Ferrigno M, Grassi B, Ferretti G, Costa M, Marconi C, Cerretelli P, Lundgren CEG (1991) Electrocardiogram during deep breath-hold dives by elite divers. Undersea Biomed Res 18:81–91

Ferrigno M, Ferretti G, Ellis A, Warkander D, Costa M, Cerretelli P, Lundgren CEG (1997) Cardiovascular changes during deep breath-hold dives in a pressure chamber. J Appl Physiol 83:1282–1290

Findley LJ, Ries AL, Tisi GM, Wagner PD (1983) Hypoxemia during apnea in normal subjects: mechanisms and impact of lung volume. J Appl Physiol 55:1777–1783

Florio JT, Morrison JB, Butt WS (1979) Breathing pattern and ventilatory response to carbon dioxide in divers. J Appl Physiol 46:1076–1080

Fowle ASE, Campbell EJM (1964) The immediate carbon dioxide storage capacity of man. Clin Sci 27:41–49

Gayeski TEJ, Honig CR (1983) Direct measurement of intracellular O_2 gradients: role of convection and myoglobin. Adv Exp Med Biol 159:613–621

Gooden B (1994) Mechanism of the human diving response. Integr Physiol Behav Sci 29:6–16

Grassi B, Ferretti G, Costa M, Ferrigno M, Panzacchi A, Lundgren CEG, Marconi C, Cerretelli P (1994) Ventilatory responses to hypercapnia and hypoxia in elite breath-hold divers. Respir Physiol 97:323–332

Gross PM, Terjung RL, Lohman TG (1976) Left ventricular performance in man during breath-holding and simulated diving. Undersea Biomed Res 3:351–360

Hayashi N, Ishihara M, Tanaka A, Osumi T, Yoshida T (1997) Face immersion increases vagal activity as assessed by heart rate variability. Eur J Appl Physiol 76:394–399

Hayward JS, Hay C, Mathews BR, Overweel CH, Radford DD (1984) Temperature effect on the human dive response in relation to cold water near drowning. J Appl Physiol 56:202–206

Heistad DD, Abboud FM, Eckstein JW (1968) Vasoconstrictor response to simulated diving in men. J Appl Physiol 25:542–549

Hochachka PW (1986) Balancing conflicting metabolic demands of exercise and diving. Fed Proc 45:2948–2952

Hong SK (1988) Man as a breath-hold diver. Can J Zool 66:70–74

Hong SK, Rahn H (1967) The diving women of Korea and Japan. Sci Am 216:34–43
Hong SK, Rahn H, Kang DH, Song SH, Kang BS (1963) Diving pattern, lung volumes, and alveolar gas of the Korean diving women (Ama). J Appl Physiol 18:457–465
Hong SK, Song SH, Kim PK, Suh CS (1967) Seasonal observations on the cardiac rhythm during diving in the Korean Ama. J Appl Physiol 23:18–22
Hong SK, Lin YC, Lally DA, Yim BJB, Kominami N, Hong PW, Moore TO (1971) Alveolar gas exchanges and cardiovascular functions during breath-holding with air. J Appl Physiol 30:540–547
Hong SK, Rennie DW, Park YS (1986) Cold acclimatization and deacclimatization in Korean women divers. Exerc Sports Sci Rev 14:231–268
Hurford WE, Hong SK, Park YS, Ahn DW, Shiraki K, Mohri M, Zapol WM (1990) Splenic contraction during breath-hold diving in the Korean Ama. J Appl Physiol 69:932–936
Irving L (1963) Bradycardia in human divers. J Appl Physiol 18:489–491
Jansson E, Sylvén C, Nordevang E (1982) Myoglobin in the quadriceps femoris muscle of competitive cyclists and untrained men. Acta Physiol Scand 114:627–629
Jiang ZL, He J, Yamaguchi H, Tanaka H, Miyamoto H (1994) Blood flow velocity in common carotid artery in humans during breath-holding and face immersion. Aviat Space Environ Med 65:936–943
Kang BS, Song SH, Suh CS, Hong SK (1963) Changes in body temperature and basal metabolic rate of the Ama. J Appl Physiol 18:483–488
Kang DH, Kim PK, Kang BS, Song SH, Hong SK (1965) Energy metabolism and body temperature in the Ama. J Appl Physiol 20:46–50
Kang DH, Park YS, Park YD, Lee IS, Yang DS, Lee SH, Hong SY, Rennie DW, Hong SK (1983) Energetics of wet-suit diving in Korean women breath-hold divers. J Appl Physiol 54:1702–1707
Kawakami Y, Natelson BH, DuBois AB (1967) Cardiovascular effects of face immersion and factors affecting diving reflex in man. J Appl Physiol 23:964–970
Kindwall EP (1997) A short history of diving and diving medicine. In: Bove AA (ed) Diving medicine. Saunders, Philadelphia, pp 3–11
Klocke FJ, Rahn H (1959) Breath holding after breathing of oxygen. J Appl Physiol 14:689–693
Kooyman GL, Ponganis PJ (1998) The physiological basis of diving to depth: birds and mammals. Annu Rev Physiol 60:19–32
Lambertsen CJ (1960) Carbon dioxide and respiration in acid base homeostasis. Anesthesiology 21:642–651
Lanphier EH, Rahn H (1963a) Alveolar gas exchange during breath-hold diving. J Appl Physiol 18:471–477
Lanphier EH, Rahn H (1963b) Alveolar gas exchange during breath-holding with air. J Appl Physiol 18:478–482
Lin YC (1982) Breath-hold diving in terrestrial mammals. Exerc Sports Sci Rev 10:70–307
Lin YC (1987) Effect of O_2 and CO_2 on breath-hold breaking point. In: Lundgren CEG, Ferrigno M (eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 75–86
Lin YC (1988) Applied physiology of diving. Sports Med 5:41–56
Lin YC (1990) Physiological limitations of humans as breath-hold divers. In: Lin YC, Shida KK (eds) Man in the sea. Best, San Pedro, Calif., pp 33–56
Lin YC Hong SK (1996) Hyperbaria: breath-hold diving. In: Fregly MJ, Blatteis CM (eds) Handbook of physiology. Environmental physiology. Oxford University Press, New York, pp 979–995
Lin YC, Lally DA, Moore TO, Hong SK (1974) Physiological and conventional breath-hold breaking points. J Appl Physiol 37:291–296
Lin YC, Shida KK, Hong SK (1983) Effects of hypercapnia, hypoxia and rebreathing on circulatory response to apnea. J Appl Physiol 54:172–177
Lindholm P, Sundblad P, Linnarsson D (1999) Oxygen-conserving effects of apnea in exercising men. J Appl Physiol 87:2122–2127
Linér MH (1994) Cardiovascular and pulmonary responses to breath-hold diving in humans. Acta Physiol Scand [Suppl] 620:1–32
Linér MH, Linnarsson D (1994) Tissue oxygen and carbon dioxide stores and breath-hold diving in humans. J Appl Physiol 77:542–547
Linér MH, Linnarsson D (1995) Intrapulmonary distribution of alveolar gas exchange during breath-hold diving in humans. J Appl Physiol 78:410–416
Linér MH, Ferrigno M, Lundgren CEG (1993) Alveolar gas exchange during simulated breath-hold diving to 20 m. Undersea Hyperbaric Med 20:27–38
Lloyd BB, Jukes MGM, Cunningham DJC (1958) The relation between the alveolar oxygen pressure and the respiratory response to carbon dioxide in man. Q J Exp Physiol 43:214–227
Majorca E (1987) Depth records: practical considerations. In: Lundgren CEG, Ferrigno M (eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 291–294
Manley L (1990) Apnoeic heart rate responses in humans. A review. Sports Med 9:286–310
Masuda Y, Yoshida A, Hayashi F, Sasaki K, Honda Y (1981) The ventilatory responses to hypoxia and hypercapnia in the Ama. Jpn J Physiol 31:187–197
Masuda Y, Yoshida A, Hayashi F, Sasaki K, Honda Y (1982) Attenuated ventilatory responses to hypercapnia and hypoxia in assisted breath-hold divers (funado). Jpn J Physiol 32:327–336
McDonough JR, Barutt JP, Saffron JC (1987) Cardiac arrhythmias as a precursor to drowning accidents. In: Lundgren CEG, Ferrigno M (eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 212–229
Mithoefer JC (1959) Lung volume restriction as a ventilatory stimulus during breath-holding. J Appl Physiol 14:701–705
Molfino F, Zannini D (1964) L'uomo e il mondo straordinario. Minerva Medica, Turin
Neuman TS (1997) Near drowning. In: Bove AA (ed), Diving medicine. Saunders, Philadelphia, pp 217–226
Odaglia G (1985) Gli effetti delle alte pressioni sull'organismo. In: Cerretelli P, di Prampero PE (eds) Sport, ambiente e limite umano. Mondadori, Milan, pp 185–203
Olsen CR, Fanestil DD, Scholander PF (1962) Some effects of breath-holding and apneic underwater diving on cardiac rhythm in man. J Appl Physiol 17:461–466
Olszowka AJ, Rahn H (1987a) Gas store changes during repetitive breath-hold diving. In: Shiraki K, Yousef MK (eds) Man in stressful environments: diving, hyper- and hypobaric physiology. Thomas, Springfield, Ill., pp 41–56
Olszowka AJ, Rahn H (1987b) Breath-hold diving. In: Sutton JR, Houston CS, Coates G (eds) Hypoxia and cold. Praeger, New York, pp 417–428
Otis AB, Rahn H, Fenn WO (1948) Alveolar gas changes during breath-holding. Am J Physiol 152:674–686
Pan AW, He J, Kinouchi Y, Yamaguchi H, Miyamoto H (1997) Blood flow in the carotid artery during breath-holding in relation to diving bradycardia. Eur J Appl Physiol 75:388–395
Park YS, Rahn H, Lee IS, Lee SI, Kang DH, Hong SY, Hong SK (1983a) Patterns of wet suit diving in Korean women breath-hold divers. Undersea Biomed Res 10:203–215
Park YS, Rennie DW, Lee IS, Park YD, Paik KS, Kang DH, Suh DJ, Lee SH, Hong SY, Hong SK (1983b) Time course of deacclimatization to cold water immersion in Korean women divers. J Appl Physiol 54:1708–1716
Park YS, Shiraki K, Hong SK (1990) Energetics of breath-hold diving in Korean and Japanese professional divers. In: Lin YC, Shida KK (eds) Man in the sea. Best, San Pedro, Calif., pp 75–87
Paulev PE (1968) Cardiac rhythm during breath-holding and water immersion in man. Acta Physiol Scand 73:139–150

Paulev PE (1969) Respiratory and cardiovascular effects of breath-holding. Acta Physiol Scand [Suppl] 324:1–116
Paulev PE, Naeraa N (1968) Hypoxia and carbon dioxide retention following breath-hold diving. J Appl Physiol 22:436–440
Paulev PE, Wetterqvist H (1968) Cardiac output during breath-holding in man. Scand J Clin Lab Invest 22:115–123
Paulev PE, Honda Y, Sakakibara Y, Morikawa T, Tanaka Y, Nakamura W (1988) Brady- and tachycardia in light of the Valsalva and Mueller maneuver (apnea). Jpn J Physiol 38:507–517
Pendergast DR (1987) Energetics of breath-hold diving. In: Lundgren CEG, Ferrigno M (eds) The physiology of breath-hold diving. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 135–147
Plewes JL, Olszowka AJ, Farhi LE (1976) Amount and rate of CO_2 storage in lung tissue. Respir Physiol 28:359–370
Qvist J, Hill RD, Schneider RC, Falke KJ, Zapol WM (1986) Hemoglobin concentrations and blood gas tensions of free diving Weddell seals. J Appl Physiol 61:1560–1569
Qvist J, Hurford WE, Park YS, Radermacher P, Falke KJ, Ahn DW, Guyton GP, Stanek KS, Hong SK, Weber RE, Zapol WM (1993) Arterial blood gas tensions during breath-hold diving in the Korean ama. J Appl Physiol 75:285–293
Rahn H (1987) Breath-hold diving: alveolar O_2 and blackout. In: Bove AA, Bachrach AJ, Greenbaum LJ (eds) Underwater and hyperbaric physiology IX. Undersea and Hyperbaric Medicine Society, Bethesda, Md., pp 3–15
Rahn H, Fenn WO (1955) A Graphical analysis of the respiratory gas exchange. The O_2-CO_2 diagram. American Physiological Society. Washington, D.C.
Rahn H, Yokoyama T (1965) Physiology of breath-hold diving and the Ama of Japan. NAS-NRC Publ. 1341, Washington, D.C.
Rebuck AS, Slutsky AS, Mahutte CK (1977) A mathematical expression to describe the ventilatory response to hypoxia and hypercapnia. Respir Physiol 31:107–116
Rennie DW, Covino BG, Howell BJ, Song SH, Kang BS, Hong SK (1962) Physical insulation of Korean diving women. J Appl Physiol 17:961–966
Sackner MA, Feisal KA, DuBois AB (1964) Determination of tissue volume and CO_2 dissociation slope of the lungs in man. J Appl Physiol 19:374–380
Sasamoto H (1965) The electrocardiographic pattern of the diving Ama. In: Rahn H, Yokoyama T (eds) Physiology of breath-hold diving and the Ama of Japan. Publication 1341. NAS-NRC, Washington, D.C., pp 271–280
Schaefer KE (1965) Adaptation to breath-hold diving. In: Rahn H, Yokoyama T (eds) Physiology of breath-hold diving and the Ama of Japan. Publication 1341. NAS-NRC, Washington, D.C., pp. 237–252
Schaefer KE, Carey CR (1962) Alveolar pathway during 90-foot breath-hold dives. Science 137:1051–1052
Schaefer KE, Allison RD, Dougherty JH Jr, Carey CR, Walker R, Yost F, Parker D (1968) Pulmonary and circulatory adjustments determining the limits of depth in breath-hold diving. Science 162:1020–1023
Schagatay E, van Kampen M, Emanuelsson S, Holm B (2000) Effects of physical and apnea training on apneic time and the diving response in humans. Eur J Appl Physiol 82:161–169
Scholander PF (1940) Experimental investigations on the respiratory function in diving mammals and birds. Hvalradets Skrifter, Norske Videnskaps-Akademie, Oslo 22, pp 1–131
Scholander PF, Hammel HT, LeMessurier H, Hemmingsen E, Garey W (1962) Circulatory adjustments in pearl divers. J Appl Physiol 17:184–190
Sebert P, Sanchez J (1981) Sexual and postural differences in cardioventilatory responses during and after breath holding at rest. Eur J Appl Physiol 47:209–222
Song SH, Kang DH, Kang BS, Hong SH (1963) Lung volumes and ventilatory responses to high CO_2 and low O_2 in the Ama. J Appl Physiol 18:466–470
Song SH, Lee WK, Chung YA, Hong SK (1969) Mechanisms of apneic bradycardia in man. J Appl Physiol 27:323–327
Sterba JA, Lundgren CEG (1985) Diving bradycardia and breath-holding time in man. Undersea Biomed Res 12:139–150
Sterba JA, Lundgren CEG (1988) Breath-hold duration in man and the diving response induced by face immersion. Undersea Biomed Res 15:361–375
Strømme SB, Kerem D, Elsner R (1970) Diving bradycardia during rest and exercise and its relation to physical fitness. J Appl Physiol 28:614–621
Sundblad P, Linnarsson D (1996) Influence of apnea on cardiovascular responses to neck suction during exercise. Am J Physiol 271:H1370–H1374
Teruoka G (1932) Die Ama und ihre Arbeit. Arbeitsphysiologie 5:239–251
Tibes U, Stegemann J (1969) End tidal partial pressures, O_2 uptake and CO_2 output following apneic diving and breath-hold in water and in air. Pflügers Arch 311:300–311
Tipton MJ, Kelleher PC, Golden F (1994) Supraventricular arrhythmias following breath-hold submersions in cold water. Undersea Hyperbaric Med 21:305–313
Warkander DE, Ferrigno M, Lundgren CEG, McCoy K (1996) Some physiological parameters in a breathhold dive to 107 msw (351 fsw) in the Ocean (abstract). Undersea Hyperbaric Med 23:S75
West JB, Tsukimoto K, Mathieu-Costello O, Prediletto R (1991) Stress failure in pulmonary capillaries. J Appl Physiol 70:1731–1742
Zapol WM (1996) Diving physiology of the Weddel seal. In: Fregly MJ, Blatteis CM (eds) Handbook of physiology. Environmental physiology. Oxford University Press, New York, pp 1049–1056
Zapol WM, Liggins GC, Schneider RC, Qvist J, Snider MT, Creasy RK, Hochachka PW (1979) Regional blood flow during simulated diving in the conscious Weddel seal. J Appl Physiol 47:968–973