Hana Kai II: a 17-day dry saturation dive at 18.6 ATA. IV. Cardiopulmonary functions

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Smith, R. M., S. K. Hong, R. H. Dressendorfer, H. J. Dwyer, E. Hayashi, and C. Yelverton. 1977. Hana Kai II: a 17-day dry saturation dive at 18.6 ATA. IV. Cardiopulmonary functions. Undersea Biomed. Res. 4(3): 267–281.—Impedance plethysmography was used to measure resting cardiac stroke volume (SV) and thoracic conductive volume (TCV) in four divers at intervals during a prolonged dry saturation dive (17 days at 18.6 ATA and 7 days' decompression). Resting heart rate (HR), blood pressure (BP), and pulmonary minute ventilation ($\dot{V}_E$) were measured 4 times per day for the duration of the 30-day experiment. The vital capacity (VC) and its subdivisions IC and ERV were measured by spirometry every 3 days. In nonsmokers, VC fell significantly with time ($r = 0.64$), while VC in smokers increased nearly 400 ml during the first week at pressure before tending to fall with time. Compared to predive, the mean ERV was increased 629 ml at pressure, while $\dot{V}_E$ and respiratory rate were not changed. The increased ERV did not persist postdive and was probably the result of the increased work of breathing a dense gas (4.1 g/liters). Residual volume (RV) measured by nitrogen dilution before and after the dive increased 38% and remained significantly increased (22%) even after one year in 4 divers. It is suggested that hyperoxia (0.3 ATA P_{O_2}) combined with increased gas flow resistance caused the VC to fall and RV to increase. The major cardiovascular findings were a transient bradycardia associated with increased stroke volume leading to a significant increase in resting cardiac output associated with an increased rate of rapid ventricular filling, TCV, and BP at depth. Lowering the ambient temperature for 3 days did not re-establish the bradycardia, suggesting that hyperbaric bradycardia is not due to a subtle cold stress.

hyperbaric bradycardia	blood pressure
impedance plethysmography	cold
stroke volume	thoracic blood volume
cardiac output	lung volumes and ventilation

Human beings experience altered cardiorespiratory function with increased pressure. The two most consistently reported changes are bradycardia of apparently complex etiology and increased work of breathing due to increased gas density. Factors thought to play a role in hyperbaric bradycardia include: 1) increased oxygen tension (Shilling, Hawkins, and Hansen 1936; Fagraeus 1974); 2) cold (Hong, Moore, Lally, and Morlock 1973; Moore, Morlock, Lally

and Hong 1976); and 3) increased inert gas pressure or perhaps pressure per se (Smith 1975; Hong 1976; Hong, Smith, Webb, and Matsuda 1977a). The increased gas density at depth increases the work of breathing (Maio and Farhi 1967) which appears to cause a reflex alteration in the pattern of breathing such that either the tidal volume or end-expiratory lung volume is increased (McIlroy, Eldridge, Thomas, and Christie 1956).

To date, the mechanisms underlying the so-called hyperbaric bradycardia remain incompletely known. Similarly, the underlying neural mechanisms responsible for altered respiration at depth are largely unknown. The only cardiopulmonary change associated with long-term hyperbaric habitation which has been observed to persist postdive is an increased vital capacity (VC) attributed to a training effect on the inspiratory muscles (Wright, Fisher, Hendricks, Brody, and Lambertsen 1973).

The aims of the present study were threefold: 1) to measure the effects of increased ambient pressure on cardiac stroke volume (SV) and thoracic conductive volume (TCV) using noninvasive electrical impedance plethysmography (Kubicek, Kottke, Ramos, Patterson, Witsoe, Labree, Remole, Layman, Schoening, and Garamella 1974); 2) to document the resting heart rate and respiratory adaptations resulting from a prolonged dry dive to 18.6 ATA; and 3) to observe the effect of cold on the hyperbaric bradycardia.

METHODS

Subjects and dive protocol

Five divers were confined to the chamber *Aegir* at the sea surface for a 30-day experimental period. For convenience, the 30 days were divided into 3-day periods: sea level air control, *periods 1* (predive) and *9* (postdive); *periods 2–6* (17 days at 18.6 ATA $He-O_2$); and *periods 7*, and *8* (6 days' decompression). Three transition days (compression day, the last day at 18.6 ATA, and the last day of decompression) were not included in any period. Environmental parameters during each of these periods are described in a companion paper (Hong et al. 1977a). All data were obtained from 4 subjects, except residual volume (RV), which was measured in all 5 divers (see Hong et al. 1977a for a description of the subjects).

Respiratory measurements

Residual volume was measured before and after the dive by nitrogen dilution using the 3-breath method of Rahn, Fenn, and Otis (1949). Expired minute ventilation ($\dot{V}_E$) (via Parkinson-Cowan dry gas meter) was measured 4 times each day throughout the dive at 0630, 1030, 1530, and 1930 h. The VC and its components, inspiratory capacity (IC) and expiratory reserve volume (ERV), were measured in the standing position with a 13-liter Collins recording spirometer at 1200 h the second day of each period. Respiratory frequency (f) was measured daily at 1030 h during decompression via a mouthpiece thermocouple and a Beckman polygraph at the same time $\dot{V}_E$ was measured.

Impedance plethysmography

Kubicek et al. (1974) have developed and described the four-electrode Minnesota impedance cardiograph which noninvasively measures transthoracic electrical impedance (to the nearest 0.1 ohm) in response to a 100-kHz, 4mA excitation current. Changes in transthoracic impedance (Z_o) with each heart beat may be used to estimate cardiac SV as follows

$$SV = K \frac{L^2 T}{Z_o^2} \cdot dz/dt \quad (1)$$

where K = electrical resistivity of blood at 100 kHz (average assumed value = 135 ohm-cm)[1]; L = mean distance (cm) between the inner two (recording) electrodes (measured front and back); T = ventricular ejection time (s), determined from heart sounds and impedance waveform; Z_o = impedance (ohms, at 100 kHz) between the two electrodes (neck to xiphoid process); and dz/dt = first time derivative of change in Z_o with each heart beat (ohms/s).

For present use, the method was evaluated for accuracy with the single-breath nitrous oxide method (Lee and DuBois 1955) in four of the divers, who were seated erect in a body plethysmograph at 1 ATA prior to the dive (Fig. 1). Absolute SV determined simultaneously by the two methods correlated linearly ($r = 0.95$) with a slope not different from one; thus the impedance SV throughout this work was used without modification of Equation 1. In the present investigation it was assumed that this technique is as valid in the hyperbaric environment as in 1 ATA air. More recently, Lambertsen's group applied this technique at up to 16 ATA (Egawa 1976).

Schaefer, Allison, Dougherty, Carey, Walker, Yost, and Parker (1968) have defined thoracic conductive volume in terms of thoracic impedance as

$$TCV = \frac{K L^2}{Z_o} \quad (2)$$

where the symbols used have the same meaning as in Eq. 1. Luepker, Michael, and Warbasse (1973), using dogs, showed that the Minnesota impedance cardiograph quantitatively followed changes in both intravascular and extravascular thoracic fluid volumes including blood, and that the relationships were linear. However, since the entire chest is scanned, it is difficult to calibrate and impossible to locate the precise intrathoracic site of a fluid causing a change in TCV.

Finally, Lababidi, Ehmke, Durnin, Leaverton, and Lauer (1970) have correlated several cardiac events with changes in Z_o measured by the impedance cardiograph. Among these waveforms, one ("O" wave) was found to occur simultaneously with the rapid ventricular filling phase of the cardiac cycle. Here the assumption was that an increased rate of change of the impedance (dz/dt) reflects an increased rate of change of ventricular volume during rapid filling.

Cardiovascular measurements

Heart rate by palpation (30-s count) and blood pressure (BP) by aneroid sphygmomanometer were measured during spontaneous respiration while the subjects were seated erect, 4 times each day at 0630, 1030, 1580, and 1930 h.

Thoracic impedance (Z_o), dz/dt, heart sounds, and EKG were measured at preselected times with the Minnesota impedance plethysmograph (Instrumentation for Medicine, Inc., Model 304A). All data were recorded outside the habitat on a Beckman type R411 polygraph at a paper speed of 50 mm/s. Except for compression day, the surface electrodes (3M Co., No. M6001) were removed after each measurement. Seated erect and lying supine cardiac SV during apnea at FRC was calculated by Eq. 1. At least 3 consecutive heart beats were analyzed and the results averaged for each SV and HR determination.

[1]Baker, Judy, Geddes, Langley, and Hill (1971) have shown that adjustments in K to account for differences in hematocrit in normal male subjects did not substantially improve the accuracy of the stroke volume measurement compared to dye dilution. We have not attempted to correct for small changes in hematocrit observed on this dive.

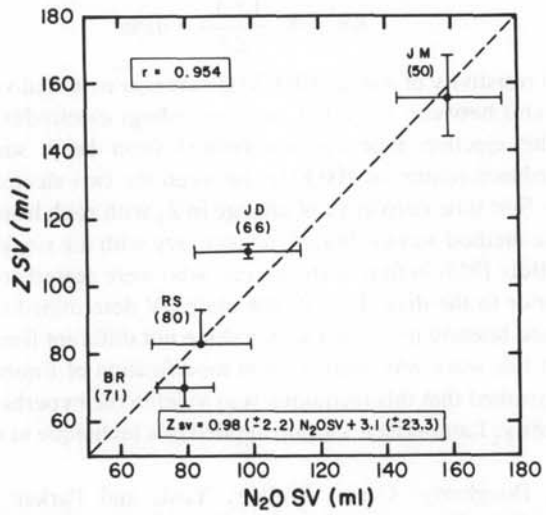

Fig. 1. Beat-by-beat calibration of impedance derived cardiac stroke volume (ZSV) vs. a nitrous oxide method (N_2OSV) performed simultaneously. Values are means ± SD for multiple heart beats during two 30-s determinations. Mean heart rate during measurement is shown in parentheses. Dashed line of identity and least squares linear regression equation (± SD) are also shown.

Thoracic impedance alone was also measured with subjects both lying supine and seated erect, at the end of a 3-min period in each posture and at 3 lung volumes. The order of Z_0 measurement was always: 1) end expiration (FRC); 2) full inspiration (TLC); and 3) full expiration (RV), with a 1-min interval between each lung volume. Each lung volume was held for about 5 s while measuring Z_0. Impedance at the 3 lung volumes was used to calculate changes in TCV from Eq. 2.

The amplitude of the first time derivative of the impedance change associated with the rapid ventricular filling wave (Lababidi et al. 1970) was measured at end expiration in the seated position at the same time SV was measured. An internal calibration signal on the impedance cardiograph provided a 1-ohm per second reference.

Impedance data were obtained between 1000 and 1230 h (except during compression) according to the following schedule: *dive days (DD) 2–3* (predive 1.0 ATA control); *DD4* (compression, at 0930, 1130, 1430, and 1730 h); *DD5* (first day at 18.6 ATA); *DD14–15* (18.6 ATA, comfortable temperature); *DD19* (18.6 ATA, cold), *DD22* and *26* (decompression), and *DD29–30* (postdive 1.0 ATA control).

Statistics used were paired and unpaired *t* tests (two-tailed unless otherwise indicated) and linear regression analysis (Steel and Torri 1960).

RESULTS

Respiratory changes

Vital capacity and end-expiratory lung volume

Mean VC in the four subjects increased during *periods 2* and *3* and tended to fall thereafter (Fig. 2). Interestingly, nonsmokers *(JD, JM)* and smokers *(BR, RS)* responded differently: VC

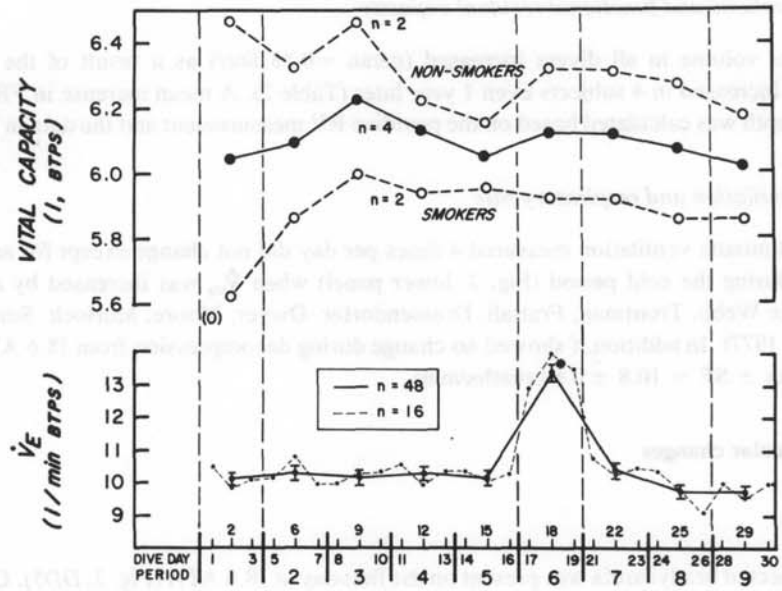

Fig. 2. Spirometric lung volumes during 9 experimental periods (top panel), and resting minute volume ($\dot{V}_E$) by period (solid line, ± SE) and by day (dashed line). Predive VC for smokers (5 separate daily measurements) is shown in parenthesis in *period 1*.

in nonsmokers fell significantly with time ($r = 0.64$, $P < 0.05$), while VC in smokers increased nearly 400 ml during *periods 2* and *3* before tending to fall.

Five predive determinations of IC and ERV were made on each subject prior to the dive, and including the *period 1* measurement, a mean predive value for each subject was calculated. Similarly, data for hyperbaric *periods 2* to *7* for each subject were pooled (Table 1). There is little doubt that ERV was increased at depth ($P < 0.01$). Furthermore, IC fell significantly at depth, but less than ERV increased, indicating a mean increase in the VC of some 250 ml at depth, due to the nearly 400 ml increase in VC during *periods 2* and *3* in smokers. The ERV returned to predive level during *periods 8* and *9*.

TABLE 1
Effect of habitation at 18.6 ATA on inspiratory capacity (IC) and expiratory reserve volume (ERV)

Subject	IC, ml		ERV, ml	
	1 ATA	18.6 ATA	1 ATA	18.6 ATA
JD	3978(±324)	3786(±204)	2167(±314)	2657(±221)
JM	3427(±306)	2779(±545)	2661(±299)	3403(±507)
BR	2768(±193)	2681(±120)	1720(±119)	2089(±108)
RS	3434(±245)	2862(±701)	3313(±352)	4229(±707)
$\bar{X}$ ±SE	3402(±248)	3027(±256)	2465(±342)	3095(±464)
$\bar{d}$	−375 ml		+629 ml	
Paired *t*	$P < 0.05$		$P < 0.01$	

Values are means ± SD, BTPS, of 6 determinations on different days.

Residual volume and functional residual capacity

Residual volume in all divers increased (mean = 0.53 liter) as a result of the dive and remained increased in 4 subjects even 1 year later (Table 2). A mean increase in FRC of 1.16 liters at depth was calculated based on the postdive RV measurement and the data in Table 1.

Minute ventilation and respiratory rate

Expired minute ventilation measured 4 times per day did not change except for an obvious increase during the cold period (Fig. 2, lower panel) when $\dot{V}_{O_2}$ was increased by about 100 ml/min (see Webb, Troutman, Frattali, Dressendorfer, Dwyer, Moore, Morlock, Smith, Ohta, and Hong 1977). In addition, f showed no change during decompression from 18.6 ATA to sea level (mean ± SE = 10.8 ± 0.6 breaths/min).

Cardiovascular changes

Heart rate

The expected bradycardia was present on the first day at 18.6 ATA (Fig. 3, *DD5*). Compared to *DD3*, the HR was 8.4% lower on *DD5*. However, by *DD6* the HR had returned to the precompression rate. After 10 days at 18.6 ATA, the bradycardia suddenly reappeared during *period 5*. Compared to the predive HR the only statistically significant change in mean period HR was that which occurred postdive (*period 9*); HR for *period 9* was significantly increased compared to *period 8* as well. However, the postdive HR measured during the seated impedance experiments (which is based on several heart beats during apnea and fewer observations, and which followed several minutes of lying supine) was not significantly elevated (Fig. 4).

Blood pressure

Blood pressure, especially diastolic BP, tended to be elevated at depth, particularly in *periods 2* and *6* (Fig. 3). In addition, BP in *period 6* (cold) was significantly elevated over

TABLE 2
Effect of habitation at 18.6 ATA on pulmonary residual volume

Subject	Residual Volume, liter, BTPS		
	Predive *(DD0)*	Postdive *(DD31)*	1 Year Postdive
JD	1.49	1.80	1.83
EH	1.05	1.77	1.44
JM	1.64	2.05	——
BR	1.46	2.25	2.00
RS	1.43	1.85	1.59
$\bar{X}$	1.41	1.94	1.72
±SE	0.10	0.04	0.12
$\bar{d}$		0.53	0.36
Paired *t*		$P < 0.01$	$P < 0.02$

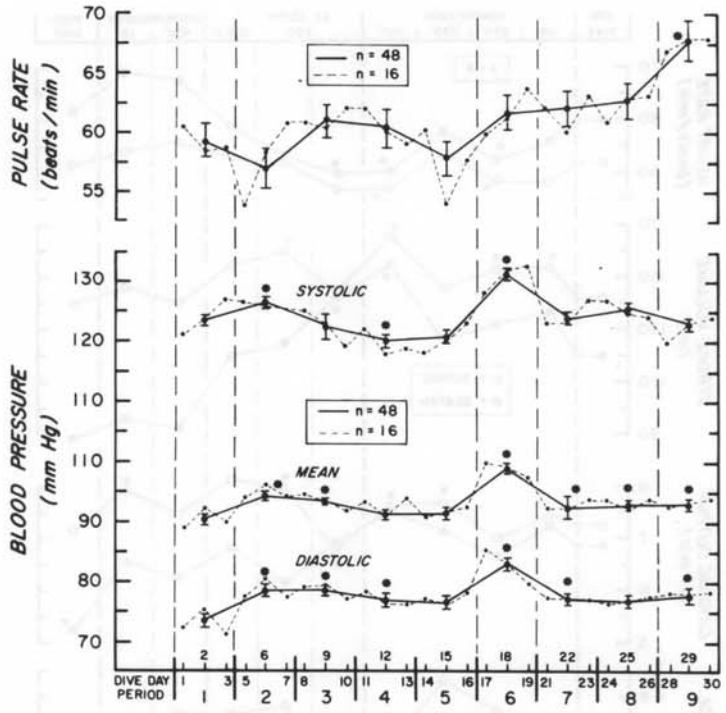

Fig. 3. Mean pulse rate and blood pressures plotted by period (± SE, heavy lines) and by day (dashed lines). Each value is obtained from 4 daily measurements on 4 seated, spontaneously breathing subjects. Starred values are significant at $P < 0.05$ (paired t test).

period 5 (warm). An exception to the general increase in BP at depth occurred in *period 4*, when systolic BP was significantly decreased compared to predive.

Thoracic conductive volume

Figure 4 summarizes the changes in calculated TCV measured at end expiration (FRC) and shows that this variable is somehow influenced by compression. The TCV tended to increase during compression to about 10.5 ATA (350 fsw) and then fell. When TCV was maximal (350 fsw), both urine flow (Hong, Claybaugh, Frattali, Johnson, Kurata, Matsuda, McDonough, Paganelli, Smith, and Webb, 1977b) and HR (Fig. 4) increased. On *DD5* the TCV was restored to near the predive value. Impedance was not measured again until *DD14–15*, when TCV was again increased relative to the predive value. The postdive TCV remained increased compared to predive. A similar temporal pattern of TCV (seated and supine) was seen at lung volumes TLC (Smith 1975) and RV (Table 3).

Stroke volume, cardiac output, and rate of rapid ventricular filling

In general, SV changed inversely to HR and tended to be increased early in the dive (Fig. 4). During compression, supine stroke volume increased more than the HR decreased, leading to a modest but significant increase in supine CO. On *DD5*, despite a bradycardia, supine SV fell and CO was significantly decreased. Throughout the rest of the dive supine SV tended to be

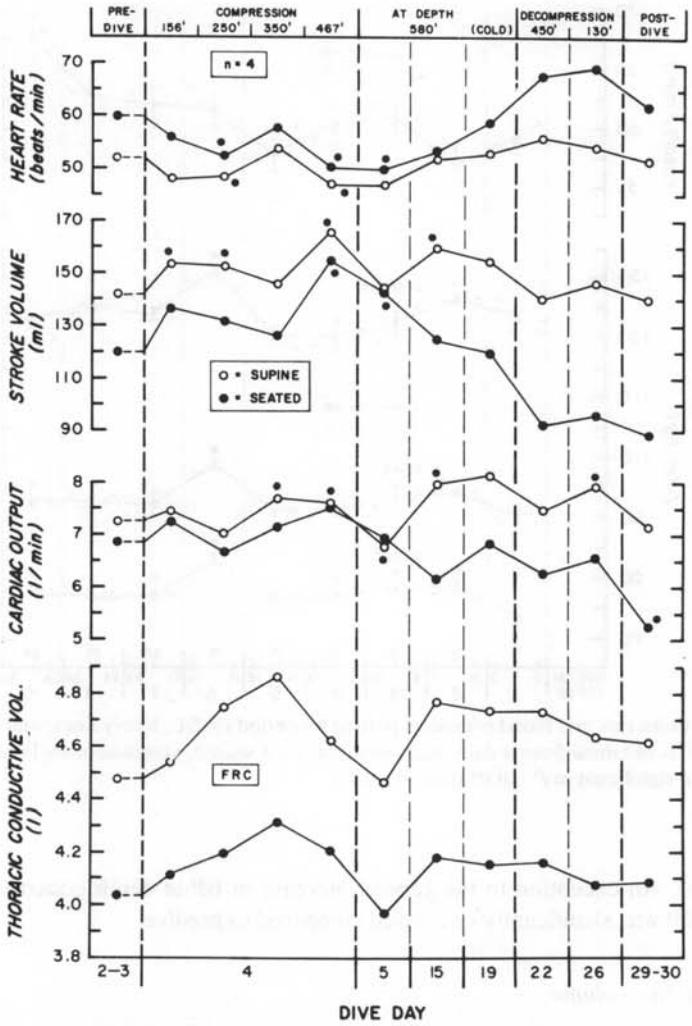

Fig. 4. Heart rate and impedance-derived stroke volume, cardiac output, and thoracic conductive volume all measured at end-expiratory lung volume (FRC) in 2 postures. HR and SV determined from at least 3 consecutive heart beats.

maintained above control levels, in striking contrast to the seated SV which tended to fall after the initial increase. The progressive fall in seated SV led to a significant reduction in seated CO postdive compared to predive (Fig. 4). However, the (seated) rate of rapid ventricular filling was significantly increased during and after the dive (Table 4).

DISCUSSION

Respiration

While the mechanisms are far from clear, Campbell (1968) has suggested " . . . that all subjects react to an increased airways resistance with increased residual volume, functional

TABLE 3
Mean calculated thoracic conductive volume for each subject at residual lung volume lying supine

Dive day *DD*	Depth, fsw	Thoracic Conductive Volume, ml *JM*	*JD*	*RS*	*BR*	$\bar{X}\pm SE$	$\bar{d}$*
1–2	0	4619	5115	4105	4068	4477±247	
	150	4911	5693	3909	3823	4584±445	107
4	250	4657	6549	3970	3903	4770±617	293
	350	5161	6598	3859	3945	4888±643	412
	467	4911	5885	3970	3924	4673±464	196
5	580	4994	4624	4501	3860	4495±236	18
14–15	580	5397	5567	4110	4158	4808±391	331
18–19	580 (cold)	4676	5427	4312	4632	4762±236	285**
22	450	4734	5981	4314	4345	4844±391	367
26	130	5122	5372	4308	4130	4733±303	256**
29–30	0	5256	5398	4067	4049	4693±367	216

*Mean difference from *DD 1–2* (1 ATA); **One-tailed paired *t* test; $P < 0.05$.

residual capacity, and total lung capacity." Since the relationship between increased gas density and increased airways resistance is firmly established (Maio and Farhi 1967), these same lung volume changes might be expected to occur in hyperbaric environments. In the present experiment, a gas density of 4.1 g/liter should have tripled the work of breathing and reduced the maximal voluntary ventilation (MVV) by about 40% (Maio and Farhi 1967). Indeed, Dressendorfer, Hong, Morlock, Pegg, Respicio, Smith and Yelverton (1977) found a 40% reduction in MVV at 18.6 ATA during the present dive. We found no change in $\dot{V}_E$ (Fig. 2) or in the respiratory rate (f), while most authors have reported an increased V_T with reduced f at depth and none have reported an increased ERV of the magnitude seen here (0.63 liter).

TABLE 4
First derivative of impedance change during rapid ventricular filling measured at FRC, seated erect

Dive Day, *DD*	Depth, fsw	Ventricular Rapid Filling Wave, ohm/s × 10 *JM*	*JD*	*RS*	*BR*	$\bar{X}\pm SE$	$\bar{d}$*
1–2	0	0.3	0.2	2.5	0.4	0.9±0.6	
	150	2.0	1.5	4.0	1.0	2.1±0.7	1.28**
	250	3.0	0.0	2.5	1.5	1.8±0.7	0.90
4	350	3.0	1.0	3.0	2.5	2.4±0.5	1.53**
	467	2.0	1.5	4.0	0.0	1.9±0.8	1.03
5	580	3.0	1.5	4.0	2.5	2.8±0.5	1.90**
14–15	580	3.0	3.0	3.0	2.0	2.8±0.3	1.90**
18–19	580	2.5	0.0	3.5	3.0	2.3±0.8	1.40
22	450	1.0	1.5	3.5	3.5	2.4±0.7	1.53**
26	130	3.0	2.0	3.0	2.0	2.5±0.3	1.65**
29–30	0	1.5	1.0	2.5	1.5	1.6±0.3	0.78**

*Mean difference and paired *t* compared to *DD 1–2;* **significant at $P < 0.05$.

Increased expiratory reserve volume

McIlroy et al. (1956) were the first to show that normal subjects react reflexly (within one breath) to the sudden imposition of nonelastic respiratory loads by one of two typical responses: 1) by immediately increasing V_T and decreasing f; or 2) by increasing the ERV with little or no change in V_T and f. Apparently, the latter mechanism was dominant in Hana Kai II divers. Although it is not certain if an increase of 0.63 liter in the ERV is big enough to shift the lung to a less compliant portion of the pressure-volume curve (Otis, Fenn, and Rahn 1950), such an increase in the ERV is expected to produce more negative inspiratory pressures (see upper panel of Fig. 5, and Flynn, Berghage, and Coil 1972), which might increase the thoracic blood volume (TBV) (Cudkowicz and O'Neill, 1965). This must also increase the passive elastic recoil pressure of the lung (Otis et al. 1950), allowing expiration to remain largely passive. This seems adaptive in the sense that active expiration would raise intrapleural pressure toward or above ambient, tending to collapse airways upon expiration and causing a further increase in airway resistance. In addition, airway resistance is less at larger lung volumes (Tsung, Godfrey, and Shepard 1959). Thus, the increase in ERV appears to be part of an adaptive response to an increased airways flow resistance. As the gas density decreased

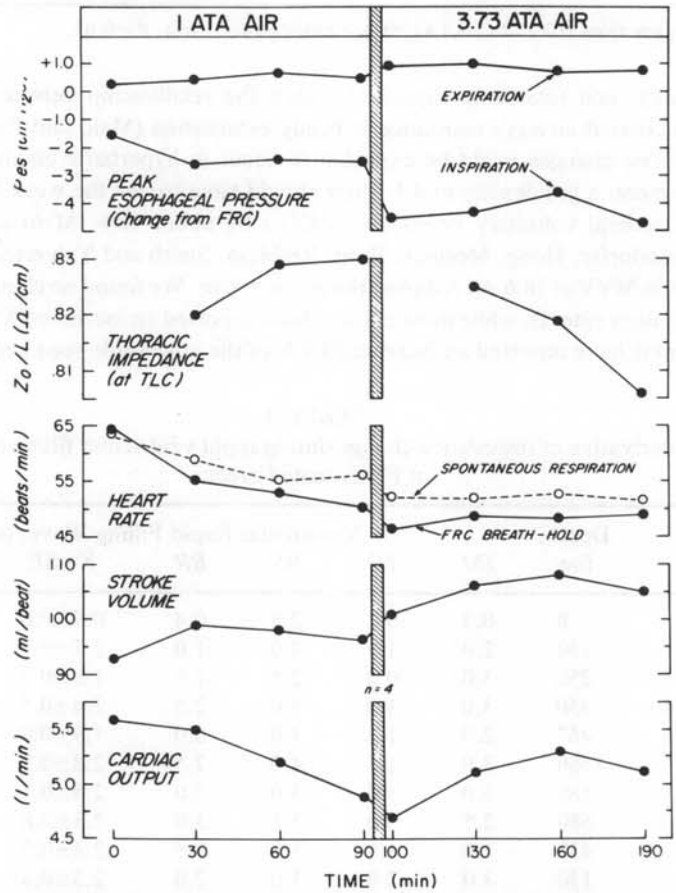

Fig. 5. Responses of 4 Hana Kai II divers upon compression to 3.73 ATA breathing air. Plotted transthoracic impedance (Z_o/L) was measured at full inspiration (TLC). Note that increase in Z_o/L represents a decrease in TCV.

during decompression, the ERV returned to the predive value, suggesting that lung compliance in the mid-volume range was not measurably changed according to this rough estimate.

Increased residual volume

In contrast to the reversible adaptive increase in ERV, the VC tended to fall after the first week at depth, while the RV showed persistent increase (Table 2). Since the VC increased during the first week of the dive in two smokers (Fig. 2), it is possible to speculate that part of the increased RV postdive is due to restoration of ventilation to alveolae distal to formerly occluded airways. Indeed, the three smokers resumed smoking after *DD30,* and one year postdive the RV had decreased 280 ml (due to renewed airway occlusion?), while one nonsmoker's RV remained increased (Table 2). Thus, restoration of airway patency in clean hyperbaric atmospheres cannot explain the entire increase in RV measured postdive, particularly in nonsmoking subjects.

Mechanical factors such as an increased FRC (Campbell 1968), airway obstruction (Finucane and Colebatch 1969), or an increased thoracic blood volume (Hauge, Bø, and Waaler 1975; Goldberg, Mitzner, Adams, Menkes, Lichtenstein, and Permutt 1975) have been implicated as the possible causes for an increase in the RV. During Hana Kai II both the airway resistance and thoracic conductive volume were increased. Interestingly, the increase in RV in airway obstruction is not readily reversible and may continue indefinitely (persistence at least 6 weeks after relief of airway obstruction due to asthma, Finucane and Colebatch 1969; persistence unchanged for 4 weeks following removal of airway obstruction in place for only two weeks in dogs, Buhain, Brody, and Fisher 1972). Similar changes may have occurred on Hana Kai II, but it is doubtful that mechanical forces, acting alone, caused the chronic increase in RV, because Wright et al. (1973) found that 14 days at similar gas density (but normoxic) did increase the ERV by about 400 ml in human subjects, but the RV decreased by nearly the same volume and was not increased postdive.

The result of Wright et al. suggests that the difference in RV may be caused by: 1) pressure (4.0 ATA vs. 18.6 ATA); 2) inert gas (nitrogen vs. helium); or 3) P_{O_2} (0.2 ATA vs. 0.3 ATA). We know of no evidence to suggest an effect of pressure per se on pulmonary mechanics. Furthermore, the evidence for a specific effect of helium on lung tissue is not conclusive (Harrison and Solomon 1975). On the other hand, a chronic mild hyperoxia (or hyperoxic-gas density interaction) has been shown to induce certain morphometric changes. For instance, Weibel, Lewerenz, and Kaplan (1968) found changes suggestive of interstitial fibrosis and edema in the lungs of rats, dogs, and monkeys after 8 months' exposure to an oxygen pressure of only 0.23 ATA. The minimal toxic concentration of oxygen for a 3-wk continuous exposure is unknown for humans (Nash, Blennerhassett, and Pontoppidan 1967; Clark and Lambertsen 1971). However, the elaborate enzymatic defense system of the body against metabolic by-products of oxygen suggests that any long-term increase in P_{O_2} may be harmful (Fridovich 1975). It is possible that during Hana Kai II, a controlled P_{O_2} of about 225 mmHg combined with P_{O_2}'s exceeding 300 mmHg for the first two days at pressure and with oxygen therapy for decompression sickness in four of the divers (see Hong et al. 1977a) may be somehow related to the increased RV.[2] Obviously, more critical studies are needed in the future to understand this interesting phenomenon.

[2]*Subject EH* had an increase in RV in the absence of oxygen therapy during decompression (Table 2).

Circulation

Impedance–compression effects

During compression, TCV increased and then fell as urine flow increased (Fig. 2, Hong et al. 1977b). Sjöstrand (1953) has shown that the thorax is an important blood reservoir and that an increase in thoracic blood volume (TBV) is shared by the lungs and heart in the ratio 3:1, respectively. Changes in TBV are known to affect cardiovascular and body fluid regulation via high or low pressure intravascular stretch receptors. Thus, the increase in TCV during compression may be the cause of the early increase in urine flow, which seems to have produced a systemic dehydration early in this dive (see Hong et al. 1977b). Impedence data on *DD5* (Fig. 4, Table 3) support this view, strongly suggesting that the systemic mobilizable blood volume was indeed reduced the first day after compression. A normal seated CO (Fig. 4) associated with increased BP on *DD5* implies peripheral vasoconstriction probably related to loss of systemic blood volume (Hong et al. 1977b) or hyperoxia rather than to cold.

In view of the importance of thoracic impedance changes during compression, a postdive experiment to confirm essential findings was performed. Figure 5 shows the results obtained in four Hana Kai II divers when they were compressed with air to 3.73 ATA in a deck decompression chamber (gas density similar to 18.6 ATA heliox). Peak inspiratory esophageal pressure (P_{es}) nearly doubled with only a slight increase in peak expiratory P_{es}. Thoracic impedance, which had been tending to increase with time predive (due to insensible water loss and perhaps to accumulation of blood in the legs of these resting subjects) tended to fall progressively at pressure, indicating an increase in TCV (no fluid intake was allowed). Stroke volume exhibited a slow rise at depth, while HR fell rapidly. These data essentially confirm the findings at 18.6 ATA.

Hong et al. (1977b) argue that suppression of insensible water loss is not the explanation for the marked increase in urine flow observed when TCV was maximal during compression, and it is doubtful that pulmonary edema due to a high P_{O_2} could develop this rapidly upon compression (Clark and Lambertsen 1971). The remaining probable causes of the increased thoracic fluid volume upon compression are: 1) bradycardia due to a pressure effect; 2) altered intrathoracic mechanics due to increased gas density; or 3) increased circulating blood volume due to a gas osmotic effect (Kylstra, Longmuis, and Grace 1968).

Further experiments (Smith, Haanio, Kim, and Watt 1976) have shown that compression to 3.73 ATA in the same deck decompression chamber but breathing He:O_2 (80:20) did not cause Z_o to fall; instead, it continued to increase with the same slope it had during 90 min of air breathing precompression. That this occurred in the face of a bradycardia and total gas tensions of similar magnitude on heliox or air suggested that neither bradycardia nor inert gas osmosis are the cause of the increased TCV upon compression. Thus, increased gas density at pressure and consequently more negative intrathoracic pressure may be the cause of a redistribution of blood into the thorax capable of altering body fluid balance in the hyperbaric environment.

Impedance-steady state effects

Impedance measurements during and after *period 5* are consistent with the interpretation that after the initial compression effect, the thoracic fluid volume remained increased at depth (Fig. 4, Table 3). These results, taken together with the sustained increases in the rate of rapid ventricular filling (Table 4), and lying CO (Fig. 4), also suggest a systemic rehydration after 10

days at 18.6 ATA. Additional evidence (see Hong et al. 1977b) also points to a systemic rehydration in *period 5*.

The causes of these complex changes in body fluid distribution with time at depth remain unknown, but several likely possibilities will be mentioned. First, the sustained increase in TCV may reflect an increase in total body water related to the suppression of insensible water loss (see Hong et al. 1977b). Total body water postdive was increased 800 ml and may account for the increased TCV postdive (Table 3). Second, hyperoxia may cause pulmonary interstitial edema over the course of several weeks (see Clark and Lambertsen 1971).[3] Subtle oxygen poisoning also may have caused an increased cardiac afterload and reduced cardiac contractile power (Jacobs, Williams, and Schenk 1970) leading to the progressive fall in seated SV (Fig. 4) which might increase TCV. Third, an effect of gas density which alters respiratory mechanics and cardiac pre-load has been previously discussed (Smith 1975). Recently, Goldberg et al. (1975) have suggested that more negative intrathoracic pressure also constitutes an increased afterload on the heart. This, in combination with increased transmural pressure at the aortic baroreceptor and increased BP due to peripheral vasoconstriction may also participate in the forces tending to increase TCV.

It is difficult to see how bradycardia or gas osmosis could play a role in this second (sustained) increase in TCV.

Hyperbaric bradycardia

Most human studies in the hyperbaric field have confirmed the existence of a hyperbaric bradycardia (see Hong 1976 and Hong et al. 1977a for references), but the underlying mechanism(s) and significance of this response remain obscure. Lowering the ambient temperature during *period 6* did not cause any bradycardia while it significantly increased blood pressure (Fig. 3) and $\dot{V}_{O_2}$ (see Webb et al. 1977). Thus, it seems doubtful that a subtle hyperbaric cold stress (Moore et al. 1976) is responsible for the hyperbaric bradycardia, which confirms the conclusion of Matsuda, Nakayama, Itoh, Kirigaya, Kurata, Strauss, and Hong (1975). Furthermore, on the present dive, Dressendorfer et al. (1977) observed a similar decrease in HR during maximal exercise at 18.6 ATA breathing both hyperoxic and normoxic gas mixtures, which suggests a cause other than hyperoxia for the hyperbaric bradycardia. A direct effect of pressure or inert gas is suspected (Örnhagen and Hogan 1976).

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[3]If pulmonary interstitial edema was a reality on this dive, the TCV changes may be about 35% less than those reported here (Fig. 4, Table 3) since we have assumed that the fluid was whole blood rather than serum (see Luepker et al. 1973). Our unpublished observations suggest that the circumferential electrode arrangement used here is quite insensitive to lung gas volume compared to lung fluid volume changes, and no correction for changing lung gas volume has been attempted.

Smith, R. M., S. K. Hong, R. H. Dressendorfer, H. J. Dwyer, E. Hayashi, and C. Yelverton. 1977. Hana Kai II: Plongée fictive à saturation, 17 jours à 18,6 ATA. IV. Fonctions cardiopulmonaires. Undersea Biomed. Res. 4(3): 267–281.—Le débit systolique du sujet en repos et le volume thoracique conductif ont été mesurés par la pléthysmographie à impédance chez 4 plongeurs au cours d'une plongée fictive à saturation (17 jours à 18,6 ATA, 7 jours de décompression). La fréquence cardiaque, la pression artérielle, et la ventilation minute (V_E) ont été mesurées 4 fois par jour pendant l'expérience entière (30 jours). La capacité vitale (CV) et ses composants, la capacité inspiratoire (CI) et le volume de réserve expiratoire (VRE) ont été mesurés par spirométrie tous les 3 jours. La CV chez les non-fumeurs a baissé significativement par rapport au temps ($r = ,64$), tandis qu'une augmentation de la CV de 400 ml a été observée chez les fumeurs pendant la première semaine; elle a baissé ensuite. Le VRE moyen a augmenté de 629 ml en pression, par rapport à la valeur pré-plongée; le V_E et la fréquence respiratoire n'ont pas change. L'augmentation de la VRE a disparu après la plongée; elle resulte sans doute du travail accru de la respiration d'un gaz dense (4,1 gm/l). Le volume residuel (VR) mesuré par dilution à N_2 avant et après la plongée, a augmenté par 38 o/o, et est resté élevé (22 %) même après une année chez 4 plongeurs. Il paraît que l'hyperoxie (0,3 ATA P_{O_2}) avec la résistance accrue au débit du gaz, ait provoqué une baisse de la CV et une augmentation du VR. Une bradycardie transitoire et un débit systolique accru ont produit des augmentations importantes du débit cardiaque, du volume thoracique conductif, de la pression artérielle, et de la vitesse de remplissage ventriculaire. Une baisse de la température ambiante pendant 3 jours n'a pas ramené la bradycardie, ce qui laisse supposer que la bradycardie en hyperbarie ne soit pas due au stress thermique.

bradycardie en hyperbarie
pléthysmographie à impédance
pression artérielle
volume sanguin thoracique
plongée à saturation en hélium–oxygène
volumes pulmonaires et ventilation
débit systolique
débit cardiaque
froid

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