

Effect of immersion, submersion, and scuba diving on heart rate variability

J D Schipke, M Pelzer

Abstract

Background—Heart rate variability (HRV) describes the cyclic variations in heart rate and offers a non-invasive tool for investigating the modulatory effects of neural mechanisms elicited by the autonomic nervous system on intrinsic heart rate.

Objective—To introduce the HRV concept to healthy volunteers under control conditions and during scuba diving. In contrast with more established manoeuvres, diving probably activates both the sympathetic and parasympathetic nervous system through various stimuli—for example, through cardiac stretch receptors, respiration pattern, psychological stress, and diving reflex. A further aim of the study was to introduce a measure for determining a candidate's ability to scuba dive by providing (a) standard values for HRV measures (three from the time domain and three from the frequency domain) and (b) physiological responses to a strenuous manoeuvre such as scuba diving.

Methods—Twenty five trained scuba divers were investigated while diving under pool conditions (27°C) after the effects of head out immersion and submersion on HRV had been studied.

Results and conclusions—(a) Immersion under pool conditions is a powerful stimulus for both the sympathetic and parasympathetic nervous system. (b) As neither the heart rate nor the HRV changed on going from immersion to submersion, the parasympathetic activation was probably due to haemodynamic alterations. (c) All HRV measures showed an increase in the parasympathetic activity. (d) If a physiological HRV is a mechanism for providing adaptability and flexibility, diving should not provoke circulatory problems in healthy subjects. (e) Either a lower than normal HRV under control conditions or a reduction in HRV induced by diving would be unphysiological, and a scuba diving candidate showing such characteristics should be further investigated.

(Br J Sports Med 2001;35:174–180)

Keywords: immersion; submersion; scuba diving; autonomic nervous system; heart rate variability

Heart rate is not predictably regular, and the healthy heart is characterised by an apparently non-homoeometric variability. This heart rate variability (HRV) is the cyclic variations in

heart rate that are manifestations of neural controlling systems elicited by the autonomic nervous system.^{1–3} Such variability is an important mechanism of adaptability and flexibility.

A number of cardiac and non-cardiac diseases^{4–7} are characterised by a loss of complex variability and increasing periodic behaviour. For example, physiological HRV is lost in patients minutes to months before sudden cardiac death,^{8–10} and a reduced HRV has become accepted as a risk factor for postinfarction patients,^{11–13} which has proved to be a strong and independent predictor of mortality after acute myocardial infarction.^{14 15}

If HRV is the result of both different physiological perturbances in cardiovascular balance and counteracting responses, and if the autonomic nervous system has a significant effect on HRV,^{5 16} measures of HRV should either show the physiological interplay among the neural regulatory outflows from the heart¹⁷ or provide clinical information about the status of the autonomic nervous system.¹⁸

As submersion and scuba diving cause psychological stress, the sympathetic nervous system must be activated. The parasympathetic nervous system, on the other hand, will be activated through the diving reflex.^{19 20} Therefore we hypothesised that, during scuba diving, the HRV would be increased simultaneously through sympathetic and parasympathetic activation.

It was one purpose of this study to establish further HRV measures under control conditions and to assess the effect of scuba diving on HRV measures in normal healthy people. In doing so, we also hoped to provide a tool to determine the qualification of candidates for diving, assuming that unphysiologically low HRV measures under control conditions or unphysiological responses to diving would indicate that the autonomic nervous system was in some way disturbed. Because of the complex effect of diving on the autonomic nervous system, the effects of immersion and submersion on HRV were first investigated.

The investigations were performed on 25 healthy trained scuba divers. From the many measures used to assess HRV, we chose three established ones from the time domain and three from the frequency domain which were well established and suitable for assessing short term variation.²¹

Methods

VOLUNTEERS

The measurements were performed on 25 volunteers who were experienced scuba divers (20 men, five women). All were members of the

Department of
Surgery, Research
Group Experimental
Surgery, Heinrich-
Heine-Universität
Düsseldorf,
Düsseldorf, Germany
J D Schipke
M Pelzer

Correspondence to:
Dr Schipke, Zentrum für
Operative Medizin I,
Forschungsgruppe
Experimentelle Chirurgie,
Heinrich-Heine-University
Düsseldorf,
Universitätsstraße 1,
D-40225 Düsseldorf,
Germany
schipkej@uni-duesseldorf.de

Accepted 29 January 2001

German Underwater Club in Düsseldorf. Informed consent was obtained from each. The mean (SD) age was 33 (10) years, and the mean (SD) body mass was 73 (10) kg. Established scuba divers have to have regular medical examinations (every two years in those aged 40 or under, and every year in those older than 40). No disorders of the respiratory and cardiovascular systems were found on review of the medical histories; a more detailed investigation of health status seemed unnecessary.

DATA ACQUISITION

The measurements were performed in an indoor swimming pool between 1900 and 2100 hours. The air and water temperature was about 27°C. The volunteers wore swimsuits. To reduce artefacts on the electrocardiographic (ECG) signal, the electrodes were attached after the skin had been rubbed with an ECG gel. Small electrodes (N-00-S, blue sensor; Medicotest) that could easily be covered with waterproof tape were used. Two pairs of electrodes were attached in a modified Einthoven position: right clavicle and 5th rib left front axillary line (channel 1), and 6th rib right midclavicular line and 6th rib left midclavicular line (channel 2). All electrodes were attached over bones to avoid extensive motion artefacts.

The ECG was recorded using a two channel Holter monitor (Tracker; Reynolds Medical) protected in a waterproof pocket (TMT; ewa-marine). The signal was stored on a cassette (D90; IEC I/ type I Normal; TDK).

The Holter monitor was powered with batteries that operate safely for 24 hours. Because our measurements were completed within less than one hour, and because we tested the batteries before use, we can exclude any significant decreases in voltage. In addition, the tape speed control was phase locked to exact tolerances so that oscillations of the tape speed were avoided.

PROTOCOL

The volunteers were familiarised with the protocol while the electrodes were attached and the recorder put in place. The protocol consisted of four 10 minute steps:

- (1) control: sitting at pool side (without speaking);
- (2) vertical immersion (head out);
- (3) vertical submersion—that is, additional immersion of the face using mask and snorkel;
- (4) scuba diving: kneeling on the pool floor (4 m).

DATA ANALYSIS

The recordings were analysed with a long term ECG analyser (Pathfinder 4; Reynolds Medical) and appropriate software (RR Tools). The ECG signal was synchronised with the tact signal and digitised at a sampling rate of 128 Hz. The channel that proved most suitable for evaluation was chosen. A typical QRS signal was stored at the beginning of the ECG analysis and later used by the program to separate regular from aberrant beats, which were removed. In addition, the signal was visually

inspected on a monitor during the entire protocol.

The relation between two consecutive RR intervals was calculated to eliminate supraventricular extra beats and pauses. Subsequently, all beats with a ratio below 0.63 or over 1.75 were removed. From the remaining data, a five minute interval was selected from each of the four 10 minute protocol steps during which the heart rate was as stable as possible.

The following three statistical time domain measures were chosen: standard deviation of the normal to normal intervals (SDNN)—that is, the square root of variance. This measure reflects all the cyclic components responsible for variability during recording.²¹ The root mean square of successive differences of NN intervals (RMSSD) and the percentage of the differences of successive NN intervals greater than 50 milliseconds normalised to all differences within the interval (pNN50) determine high frequency (HF) variations in heart rate.²¹

To interpret the HRV in the frequency domain, tachograms were made by sampling at 2 Hz. For each of the five minute intervals, a frequency spectrum was obtained through fast Fourier transformation. To account for the relatively short interval—that is, to minimise spectral leakage—a Hamming window²² was used.

Three measures from the frequency domain were assessed: spectral density within the low frequency (LF) range and spectral density within the HF range. In accordance with studies in the literature, the threshold between the LF and the HF range was set at 0.15 Hz.²³⁻²⁶ The lower limit of the LF range was chosen to be 0.05 Hz, and the upper limit of the HF range was chosen to be 0.45 Hz.²⁴⁻²⁷ We did not determine the very low frequency component, because, if assessed from short term recordings (less than five minutes, for example), it is regarded as a dubious measurement.²¹

The LF component is associated with sympathetic activity^{17 28 29} or, according to others, with both sympathetic and parasympathetic activity^{5 7} modulated substantially by baroreflex activity.³⁰ The HF component is generally associated with parasympathetic activity.^{21 30 31} Thus, the ratio (R) between LF and HF can be used as an index of the sympathetic-parasympathetic balance. Obviously, an increase in sympathetic tone generally causes an increase in the LF/HF ratio, and an increase in parasympathetic tone decreases this ratio.¹⁸

STATISTICAL ANALYSIS

The described measures of HRV were analysed with a statistics program (SYSTAT) on an IBM-compatible computer. A one way analysis of variance for repeated measurements was used for the four protocol steps. The differences between the steps were tested with a subsequent post hoc test (Fisher's least significant differences). Data are presented as mean (SD). A p value of less than 0.05 was considered significant.

Results

Under control conditions, the respiration rate was 13 (3) breaths/min and remained almost unchanged (12 (2) breaths/min) during head out immersion. During submersion (11 (2) breaths/min) and while diving (7 (2) breaths/min), the respiration rate was significantly lower than under control conditions. In addition, the respiration rate during diving was significantly lower than during immersion and submersion.

Heart rate did not differ significantly between the first three steps: control 82 (10) beats/min; immersion 75 (12) beats/min; submersion 80 (12) beats/min. During diving, heart rate was significantly reduced (71 (11) beats/min).

The three time domain measures all showed an increase in HRV compared with control conditions (fig 1). SDNN was 45 (19) milliseconds under control conditions and significantly higher in the three other steps (immersion: 64 (19) milliseconds; submersion: 63 (24) milliseconds; diving: 78 (31) milliseconds). RMSSD was similar although there was a

larger scatter. This measure was also significantly increased: control 23 (9) milliseconds; immersion 47 (26) milliseconds; submersion 43 (33) milliseconds; diving 53 (26) milliseconds. The pNN50 was relatively low under control conditions (5 (7)%) but increased significantly during immersion (19 (18)%), submersion (16 (13)%), and diving (22 (18)%). For none of these measures were the differences between immersion, submersion, and diving significant.

In the LF range, the spectral density was 484 (257) arbitrary units (au) under control conditions (fig 1). This measure was slightly increased during immersion (613 (240) au) and submersion (564 (300) au); the increase was significant during diving (774 (330) au).

The spectral density in the HF range under control conditions was 325 (150) au (fig 1). This value was significantly increased during immersion (646 (370) au), submersion (600 (360) au), and diving (646 (360) au). There were no significant differences between these last three values. It should be mentioned that, because of the low respiration rate during diving (7 breaths/

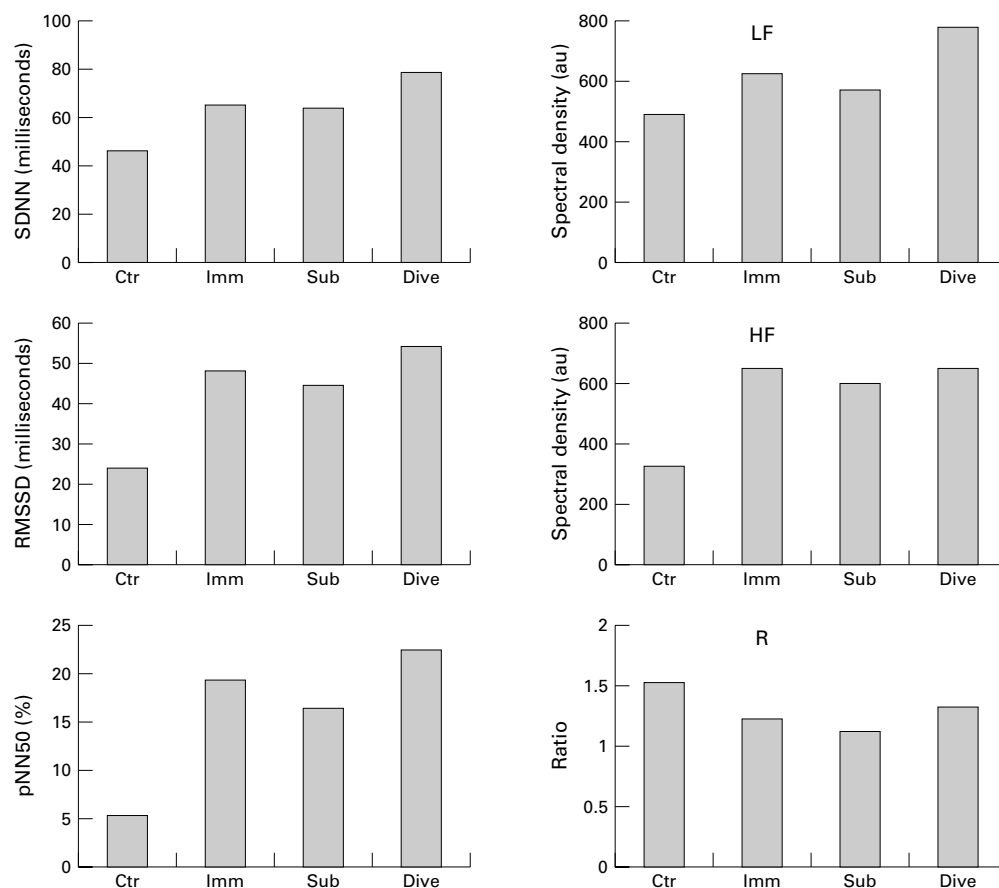


Figure 1 Changes in heart rate variability (HRV). The three time domain measures (standard deviation of the normal to normal intervals (SDNN), root mean square of NN intervals (RMSSD), and percentage of the differences of successive NN intervals greater than 50 milliseconds normalised to all differences within the interval (pNN50)) all exhibited a significant increase in HRV compared with control conditions. For none of these measures were the differences between immersion, submersion, and diving significant. In the low frequency (LF) range, the spectral density was slightly increased during immersion and submersion; the increase was significant during diving. The spectral density in the high frequency (HF) range under control conditions was significantly increased during immersion, submersion, and diving. There were no significant differences between these last three values. Because of the low respiration rate during diving, one portion of the respiratory sinus arrhythmia had shifted from the HF range into the LF range. The ratio (R) between the spectral density in the LF range and the HF range showed reciprocal behaviour to the spectral density in the HF range. Ctr, Control; Imm, immersion; Sub, submersion; Dive, diving.

min), one portion of the respiratory sinus arrhythmia (RSA) had shifted from the HF range into the LF range.

The ratio between the spectral density in the LF range and the HF range showed reciprocal behaviour to the spectral density in the HF range (fig 1). The ratio decreased significantly from control (1.5 (0.7)) to immersion (1.2 (0.6)) and submersion (1.1 (0.5)). The decrease from control conditions to diving (1.3 (0.4)) was not significant.

Discussion

The main finding of this study is that HRV in 25 healthy experienced scuba divers was significantly increased during immersion and submersion and even more so during scuba diving. Although it is recognised that HRV increases with decreasing heart rate, this study shows that this also holds for bradycardia induced by scuba diving. In contrast with our hypothesis, diving predominantly increased only the parasympathetic activity in the experienced divers.

CRITIQUE OF METHODS

Some of the factors that may have influenced our results are addressed in the following. The protocol was chosen to investigate the effects of diving by increasing the number of stimuli in a stepwise fashion. This was also done because, so far, no data on the duration of the stimuli (immersion, submersion, diving) are available. Thus a persisting response of one intervention could have offset the baseline values of the following intervention within a random order protocol.

On the other hand, our results could have been affected by the relatively low water temperature (27°C). This temperature is regarded as thermoneutral for swimming but not for resting conditions (35–36°C).³² Thus some increase in sympathetic activity could have been temperature induced,³³ but because of the limited duration (<10 minutes) and because both heart rate and respiration rate were lowest during diving, sympathetic activity was probably not increased during diving—that is, at the end of the protocol. Scuba diving in water at temperatures lower than in the pool—for example, as found in many open European waters—would probably produce an increase in sympathetic activity.³³

In addition, the reproducibility of HRV is controversial. Poor reproducibility in the time domain (variation 20–33%) and also in the frequency domain (variation 37–75%) have been reported for short term analyses.³⁴ However, good reproducibility for short term analysis has also been reported.¹⁷

In this study, traditional HRV measures were chosen, although all physiological variables—for example, heart rate—may be controlled by chaotic processes,⁹ in which case the correlation between heart rate and respiration would not be linear. Thus it cannot be excluded that part of the activity in the LF range is generated from the RSA.³⁵ Diving could, on the other hand, increase the complexity of the heart rate, making it similar to noise and therefore no

longer able to be described by discrete frequencies. The increased HRV would then reflect a faulty positive effect of diving on the heart and circulatory system.⁹

The HRV measures vary not only with the status of the autonomic nervous system but also with several other factors, such as filtering, algorithm, age, sex, and time of day. We feel that these factors are unlikely to have affected the trends in our data, which were acquired under almost identical circumstances, processed in the same manner, and compared only within individuals.

The following example elucidates the effect of different durations of ECG monitoring. The 24 hour SDNN varies between 150 and 200 milliseconds in healthy subjects^{36–37} and is regarded as pathological if it decreases to ≤50 milliseconds.^{3–38} The short term SDNN has been reported to be shorter.²¹ In one study on seated controls of similar age (23 *v* 33 years), this measure was similar to ours (51 *v* 45 milliseconds). Likewise, the RMSSD was similar to ours (26 *v* 23 milliseconds).

IMMERSION

Head out immersion in water (vertical body position) produces distinct haemodynamic responses. Because of buoyancy, the intrathoracic volume and central venous pressure increase.³⁹ The increase in reflex cardiac filling induces bradycardia and an increase in both stroke volume and cardiac output.^{39–41} The moderate systemic vasodilatation, however, does not support reduction of the central venous pressure, because the hydrostatic pressure counteracts the blood flow into the peripheral circulation.³⁹ Thus the impact of bathing—that is, immersion in water—on the systemic and pulmonary circuits is equivalent to a radical blood transfusion.⁴⁰

Centralisation of the circulation, activation of cardiac stretch receptors, cold, and psychological stress suggest that immersion in cold water (27°C) stimulates the sympathetic and parasympathetic nervous system. In fact, all six measures showed an increase in HRV, which may leave the heart rate unchanged. Because the heart rate had the tendency to decrease, the parasympathetic tone may have affected the HRV more than the sympathetic tone.

Although it is appreciated that data from healthy subjects cannot readily be extrapolated to patients, one example is presented. Bathing is apparently not harmful for patients with heart disease. However, if they show signs of cardiac insufficiency, bathing can induce pulmonary oedema or even acute heart failure.³⁹ This could be important for postinfarction patients during therapeutic procedures such as swimming and water gymnastics. Presumably, the increased HRV in water actually has a protective effect because parasympathetic activation reduces the danger of sudden cardiac death during or after myocardial infarction.⁴² In addition, moderate physical exercise, such as swimming, improves the long term prognosis of postinfarction patients⁴³ and induces bradycardia by increasing parasympathetic activity.

SUBMERSION

The additional immersion of the head in water may influence heart rate or HRV through the diving reflex, which should induce parasympathetic activation. The significance of this reflex in humans is, however, controversial.^{19, 20} Because none of the HRV measures in the time domain changed significantly, our results suggest that the diving reflex is not solely induced by face immersion in humans. The slightly increased heart rate may be explained by the increased effort of breathing against the increased hydrostatic pressure on the submerged chest.

The simultaneous moderate increases in both the LF density and the HF density and the unchanged ratio suggest that the sympathetic-parasympathetic balance was not affected by the transition from immersion to submersion. As found for immersion, the respiration rate during submersion (11 breaths/min = 0.18 Hz) probably does not affect HRV by shifting from the HF range to the LF range.

SCUBA DIVING

Man is not made to live under water, so scuba diving provokes psychological stress. Bradycardia, mediated through parasympathetic dominance, has been reported to be a consequence of increased pressure;⁴⁴ furthermore there is no effect on heart rate with ambient pressures up to 2 bar.⁴⁵ Therefore the pressure increase of 0.4 bar in this study should not have much affected any of the variables investigated in this study.

As heart rate in this study was decreased during scuba diving compared with control conditions, diving in a pool obviously does not cause substantial stress to the experienced diver. In contrast, diving in deeper and colder water with currents and limited visibility perhaps after a strenuous boat ride would probably activate the sympathetic system thereby increasing the heart rate.

We observed a distinct reduction in the respiration rate in all volunteers. Slow deep respiration at thermoneutral temperature at the surface (28°C during rest) has no effect on heart rate.⁴⁶ However, controlled respiration close to the spontaneous rate stimulates the parasympathetic system probably through more effective activation of pulmonary receptors.¹⁷ Respiration was not controlled in this study, but the significant noise that develops during expiration through a regulator could have resulted in feedback, which may have reduced the heart rate through parasympathetic activation. Whether slow deep respiration during scuba diving in water at a temperature slightly below thermoneutral (35–36°C during rest) has an effect on heart rate—similar to the diving reflex—is not known.

The clear increase in HRV in the LF range is probably due to a shift in the RSA from the HF range into the LF range. Dominance of the parasympathetic system, on the other hand, is unlikely because the heart rate did not decrease further. Possibly, HRV is more sensitive than heart rate to a shift in the tone of the autonomous nervous system.

Parasympathetic activity is reflected primarily through the RSA.⁵ On the other hand, the RSA is predominantly determined by vagal efferents.⁵ In this study, part of the RSA was already located in the LF range and thus increased the density in this range. During scuba diving, the RSA fell almost exclusively in the LF range (0.12 Hz), imitating increased sympathetic activity.

RMSSD and pNN50 are both measures that predominantly represent HF variations. Thus they normally reflect parasympathetic activity.^{16, 38} The fact that both increased during immersion, submersion, and scuba diving compared with control conditions suggests increased parasympathetic activity. From the preceding conclusions, however, one could infer that RMSSD, pNN50, and the HF range do not display the whole parasympathetic activity, because one portion is contained in the LF range. The spectral density in the LF range on the other hand, conceivably even “incorrectly” increases after the RSA shifts into the LF range during reduced rates of respiration, thereby reducing the density in the HF range, which is predominantly determined by the RSA. It is noticeable that the HF range during scuba diving did not decrease, and RMSSD and pNN50 even increased. Thus, an unknown increase in the parasympathetic tone must have compensated for the RSA related portion that was shifted into the LF range at the low respiration rate during diving. Thus, correct interpretation of HRV measures has to account for possible changes in the respiration rate during the time course of data collection.

Bearing in mind that only 25 healthy scuba divers were investigated in an almost ideal environment, the following summary is presented.

- Heart rate and respiration rate did not vary much over the whole protocol, except for respiration rate during scuba diving, which was significantly decreased.
- The time domain is better suited than the frequency domain for short term investigations because of the smaller scatter, if fast Fourier transformation derived measures are used.
- Immersion under swimming pool conditions (27°C) is a powerful stimulus for both the sympathetic and parasympathetic nervous system.
- Because neither the heart rate nor the HRV changed on going from immersion to submersion, the parasympathetic activation was probably due to haemodynamic alterations and not the diving reflex.
- There were no major differences between the three measures from the time domain (SDNN, RMSSD, and pNN50), all showing an increase in HRV during diving compared with control conditions.
- The densities in the LF range and the HF range were both significantly increased during diving compared with control conditions, indicating a simultaneous increase in sympathetic and parasympathetic activity. Consequently, the balance between these two increased activities remained almost

unchanged. Major changes in the respiration rate between experimental steps should be avoided because they could shift the RSA related density from one frequency domain into the other.

- The results support our hypothesis that diving should activate both the sympathetic and parasympathetic nervous system.
- The HRV is not helpful in determining whether or not a person is suited for scuba diving. This is because, although reliable data on HRV are available for patients—for example, those with heart disease—no such data are available for healthy people. The significance of a reduced HRV in a healthy person is not known—for example, whether it represents an increased risk of sudden cardiac death.
- However, if a candidate for scuba diving has decreased HRV under control conditions compared with normal or if the HRV decreases in response to diving, then he/she should be examined further.
- If variations in heart rate are an important mechanism of adaptability and flexibility, and HRV is increased during diving, then normal dives should not provoke circulatory problems in experienced divers.

The study was generously supported by Mr Hartgenbusch from Reynolds Medical, Germany. The excellent secretarial help of Mrs Wieland is greatly appreciated.

- 1 Chess GF, Tam RMK, Calaresu FR. Influence of cardiac neural inputs on rhythmic variations of heart period in the cat. *Am J Physiol* 1975;228:775–80.
- 2 Hirsch JA, Bishop B. Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate. *Am J Physiol* 1981;241:H620–9.
- 3 Bigger JT, Fleiss JL, Rolnitzky LM, et al. Stability over time of heart period variability in patients with previous myocardial infarction and ventricular arrhythmias. *Am J Cardiol* 1992;69:718–23.
- 4 Appel ML, Berger RD, Saul JP, et al. Beat to beat variability in cardiovascular variables: noise or music? *J Am Coll Cardiol* 1989;14:1139–48.
- 5 Malliani A, Pagani M, Lombardi F, et al. Cardiovascular neural regulation explored in the frequency domain. *Circulation* 1991;84:482–92.
- 6 Malik M, Camm AJ. Heart rate variability and clinical cardiology. *Br Heart J* 1994;71:3–6.
- 7 Kamath MV, Fallen EL. Power spectral analysis of heart rate variability: a noninvasive signature of cardiac autonomic function. *Crit Rev Biomed Eng* 1993;21:245–311.
- 8 Schwartz PJ, De Ferrari GM. The influence of the autonomic nervous system on sudden cardiac death. *Cardiology* 1987;74:297–309.
- 9 Goldberger AL. Is the normal heartbeat chaotic or homeostatic? *News in Physiological Sciences* 1991;6:87–91.
- 10 Lown B. Neural activity and ventricular fibrillation. *N Engl J Med* 1976;294:1165–70.
- 11 Cripps TR, Malik M, Farrell TG, et al. Prognostic value of reduced heart rate variability after myocardial infarction: clinical evaluation of a new analysis method. *Br Heart J* 1992;65:14–19.
- 12 Algra A, Tijssen JGP, Roelandt JRTC, et al. Heart rate variability from 24-hour electrocardiography and the 2-year risk for sudden death. *Circulation* 1993;88:180–5.
- 13 Brachmann J, Schweizer M, Hilbel T, et al. Stellenwert neuer Verfahren zur Diagnoseverbesserung bei ventrikulären tachykarden Arrhythmien. *Internist* 1993;34:417–22.
- 14 Kleiger RE, Miller JP, Bigger JT, et al. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *Am J Cardiol* 1987;59:256–62.
- 15 Malik M, Farrell T, Cripps T, et al. Heart rate variability in relation to prognosis after myocardial infarction: selection of optimal processing techniques. *Eur Heart J* 1989;10:1060–74.
- 16 Esperer HD. Die Herzfrequenzvariabilität, ein neuer Parameter für die nichtinvasive Risikostratifizierung nach Myokardinfarkt und arrhythmogener Synkope. *Herzschr Elektrophys* 1992;3:1–16.
- 17 Pagani M, Lombardi F, Guzzetti S, et al. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. *Circ Res* 1986;59:178–93.
- 18 Bianchi A, Bontempi B, Cerutti S, et al. Spectral analysis of heart rate variability signal and respiration in diabetic subjects. *Med Biol Eng Comput* 1990;28:205–11.
- 19 Hong SK, Moore TO, Seto G, et al. Lung volumes and apneic bradycardia in divers. *J Appl Physiol* 1970;29:172–6.
- 20 Moore TO, Lin YC, Lally DA, et al. Effects of temperature, immersion, and ambient pressure on human apneic bradycardia. *J Appl Physiol* 1972;33:36–41.
- 21 Task Force of The European Society of Cardiology, The North American Society of Pacing and Electrophysiology. Heart rate variability. *Eur Heart J* 1996;17:354–81.
- 22 Hamming RW. *Coding and information theory*. 5th ed. Englewood Cliffs: Prentice Hall, 1980.
- 23 Saul JP, Arai Y, Berger RD, et al. Assessment of autonomic regulation in chronic congestive heart failure by heart rate spectral analysis. *Am J Cardiol* 1988;61:1292–9.
- 24 Breuer H-WM, Skyschally A, Schulz R, et al. Heart rate variability and circulating catecholamine concentrations during steady state exercise in healthy volunteers. *Br Heart J* 1993;70:144–9.
- 25 Bigger JT, Fleiss JL, Steinman RC, et al. Correlations among time and frequency domain measures of heart period variability two weeks after acute myocardial infarction. *Am J Cardiol* 1992;69:891–8.
- 26 Hayano J, Sakakibara Y, Yamada A, et al. Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. *Am J Cardiol* 1991;67:199–204.
- 27 Stein KM, Borer JS, Hochreiter C, et al. Prognostic value and physiological correlates of heart rate variability in chronic severe mitral regurgitation. *Circulation* 1993;88:127–35.
- 28 Guzzetti S, Piccaluga E, Casati R, et al. Sympathetic predominance in essential hypertension: a study employing spectral analysis of heart rate variability. *J Hypertens* 1988;6:711–17.
- 29 Lombardi F, Sandrone G, Pernpruner S, et al. Heart rate variability as an index of sympathovagal interaction after acute myocardial infarction. *Am J Cardiol* 1987;60:1239–45.
- 30 Bigger JT, Jr, Fleiss JL, Rolnitzky LM, et al. Time course of recovery of heart period variability after myocardial infarction. *J Am Coll Cardiol* 1991;18:1643–9.
- 31 Katona PG, Jih F. Respiratory sinus arrhythmia: noninvasive measure of parasympathetic cardiac control. *J Appl Physiol* 1975;39:801–5.
- 32 Ulmer HV. In: Schmidt RF, Thews G, eds. *Physiology des Menschen*. 26th ed. Berlin, Heidelberg, New York: Springer, 1995:697–707.
- 33 Bonde-Petersen F, Schultz-Pedersen L, Dragsted N. Peripheral and central blood flow in man during cold, thermoneutral, and hot water immersion. *Aviat Space Environ Med* 1992;63:346–50.
- 34 Breuer H-WM, Skyschally A, Wehr M, et al. Schlechte Reproduzierbarkeit von Parametern der Herzfrequenzvariabilität. *Z Kardiol* 1992;81:475–81.
- 35 Kitney RI, Fulton T, McDonald AH, Linkens DA. Transient interactions between blood pressure, respiration and heart rate in man. *J Biomed Eng* 1985;7:217–24.
- 36 Brüggemann T, Andresen D, Voller H, et al. Heart rate variability from Holter monitoring in a normal population. *IEEE Computer Society Press* 1991;10:337–40.
- 37 Cook JR, Bigger Jr JT, Kleiger RE, et al. Effects of atenolol and diltiazem on heart period variability in normal persons. *J Am Coll Cardiol* 1991;17:480–4.
- 38 Casolo GC, Stroder P, Signorini C, et al. Heart rate variability during the acute phase of myocardial infarction. *Circulation* 1992;85:2073–9.
- 39 Gauer OH. Die hydrostatische Wirkung von Bädern auf den Kreislauf. *Dtsch Med J* 1955;6:462–6.
- 40 Hood WB Jr, Murray RH, Urschel CW, et al. Circulatory effects of water immersion upon human subjects. *Aerospace Medicine* 1968;39:579–84.
- 41 von Nieding G, Löllgen H, Koppenhagen K, et al. In: Gerstenbrand, Lorenzoni, Seemann, eds. *Tauchmedizin 2*. Schlütersche: Verlagsanstalt und Druckerei, 1982:244–9.
- 42 Vanoli E, Schwartz PJ. Sympathetic-parasympathetic interaction and sudden death. *Basic Res Cardiol* 1990;85(suppl 1):305–21.
- 43 Claus J, Eisenriegler E, Grodzinski E, et al. Besondere Gesichtspunkte des körperlichen Trainings während der Rehabilitationsphase von Koronarpatienten. *Herz* 1991;16:199–209.
- 44 Sagawa S, Miki K, Tajima F, et al. Cardiovascular responses to upright tilt in man during acute exposure to 3 atm abs air. *Undersea Biomed Res* 1992;19:97–106.
- 45 Kessel R, Reyes M, Praml G. In: Gerstenbrand, Lorenzoni, Seemann, eds. *Tauchmedizin 3*. Schlütersche: Verlagsanstalt und Druckerei, 1984:108–15.
- 46 Brown TE, Beightol LA, Koh J, et al. Important influence of respiration on human R-R interval power spectra is largely ignored. *J Appl Physiol* 1993;75:2310–17.

Take home message

Heart rate variability (HRV) describes cyclic variations of the heart rate and presents a non-invasive tool to investigate the modulatory effects of neural mechanisms on the intrinsic heart rate. The investigations on 25 trained scuba divers show that (a) immersion under pool conditions is a powerful stimulus for both the sympathetic and parasympathetic nervous systems, (b) the parasympathetic activation is probably due to haemodynamic alterations, and (c) all six HRV measures exhibit an increase in the parasympathetic activity during scuba diving. If a physiological HRV is a mechanism for providing adaptability and flexibility, diving should not provoke circulatory problems in healthy subjects.

Commentary

Scuba diving involves a large number of physiological and psychological stresses, but the ways in which these affect the autonomic nervous system are poorly understood. This is at least partly because physiological measurement in the diver's environment can be difficult. This study provides new information about the parasympathetic and sympathetic control of heart rate during immersion, submersion, and scuba diving. However, we need to bear in mind that measurements made during a brief shallow dive in a heated pool may not mimic proper diving. Most diving is deeper, when the effects of raised partial pressures of nitrogen and oxygen are present. Much diving, say around Britain, is in water that is much colder, dark, and murky, with strong currents. It is often strenuous and exhilarating and sometimes scary.

PETER WILMSHURST