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THE DIVING RESPONSE IN MAN: EFFECTS ON SYMPATHETIC ACTIVITY IN MUSCLE AND SKIN NERVE FASCICLES

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SUMMARY

1. Multi-unit recordings of muscle-nerve sympathetic activity (m.s.a) or skin-nerve sympathetic activity (s.s.a) were made in the left peroneal nerve of sixteen healthy volunteers during simulated diving by immersion of the face in a tub of water. The procedure was varied by the use of different water temperatures, by diving with snorkel breathing, by apnoea without diving, and by apnoea with a stream of air against the face instead of immersion in water.

2. Diving for 12 s elicited a pronounced activation of m.s.a., the mean increase from control periods being 360 %. The response was stronger with lower water temperatures. Immersion of the whole face evoked a stronger increase in m.s.a. than immersion of mouth and nose only. Diving without apnoea elicited a significant but weaker increase in m.s.a., whereas apnoea only for 12 s did not influence the sympathetic outflow. Cool air against the face during apnoea for 12 s was associated with a significant increase in m.s.a.

3. The increase in m.s.a. usually occurred before the bradycardia. On emersion, m.s.a. ceased abruptly, whereas the bradycardia persisted for a few seconds. Mental arithmetic during diving did not change the m.s.a. response but reduced the bradycardia. M.s.a. increased despite increasing blood pressure levels. On emersion, m.s.a. did not reappear until the pre-diving blood pressure level was attained.

4. S.s.a was inhibited on diving, with concomitant vasodilatation in the skin as recorded in the big toe.

5. It is concluded that the response of m.s.a. to diving is initiated by a central 'pattern recognition' of an input from facial receptors, that this input and the effects of apnoea, acting by mutual reinforcement, maintain the strong sympathetic outflow, and that the mechanism releasing m.s.a. on diving overrides the normal blood pressure regulatory function of m.s.a. Diving exerts differentiated influence on different parts of the sympathetic nervous system, as illustrated by the inhibition of s.s.a.

INTRODUCTION

Diving birds and mammals exhibit strong mechanisms for oxygen conservation during submersion in water, i.e. pronounced vasoconstriction in various organs and a profound degree of bradycardia (Zapol, Liggins, Schneider, Qvist, Snider, Creasy &

Hochachka, 1979; Blix & Folkow, 1983; De Burgh Daly, 1984). Contact of the 'face' with water is reported to be the crucial event eliciting the response (Andersen, 1963*a*; Dykes, 1974). The response is present in decerebrate animals, indicating a medullary integration of the cardiovascular adjustments to diving (Andersen, 1963*c*; Martner, Wadenvik & Lisander, 1977). A corresponding response is present in man, less dramatic but still reducing limb blood flow by 30–50 % (Heistad, Abboud & Eckstein, 1968; Campbell, Gooden & Horowitz, 1969). This effect implies an increased outflow of sympathetic vasoconstrictor impulses. These neural events have never been documented in man, however.

Sympathetic activity in human extremity nerves can be recorded directly by use of micro-neurography (for references see Wallin, 1984). Such nerve recordings have shown that sympathetic nerve activity appears as multi-unit volleys of impulses with interposed intervals of neural silence. Sympathetic outflow exhibits different characteristics in muscle and skin nerve fascicles. Muscle-nerve sympathetic activity (m.s.a.) appears as bursts of vasoconstrictor impulses governed by inhibitory baroreceptors which entrain the bursts in the cardiac rhythm (Delius, Hagbarth, Hongell & Wallin, 1972*a*; Fagius, Wallin, Sundlöf, Nerhed & Englesson, 1985). There is normally a close inverse relationship between blood pressure variations and m.s.a., with burst sequences occurring in response to transient blood pressure reductions (Delius, Hagbarth, Hongell & Wallin, 1972*b*; Sundlöf & Wallin, 1978). For unknown reasons there are great inter-individual variations with respect to the resting mean level of m.s.a. (Sundlöf & Wallin, 1977).

Skin-nerve sympathetic activity (s.s.a.) consists of vasoconstrictor and sudomotor impulses. It is primarily involved in thermoregulation, but is also activated by deep inspiration, arousal stimuli and emotional stress. Under resting conditions s.s.a. appears as wide, irregular bursts of impulse discharges occurring in an unpredictable fashion without any apparent linkage to cardiac rhythm (Hagbarth, Hallin, Hongell, Torebjörk & Wallin, 1972; Delius, Hagbarth, Hongell & Wallin, 1972*c*).

The present report comprises direct micro-electrode recordings of sympathetic impulses in human muscle and skin nerve fascicles during simulated diving by face immersion in water.

METHODS

Subjects

Nerve recordings were made in sixteen healthy volunteers, seven men and nine women aged 19–42 (mean 31) years. All subjects gave their informed consent. The recordings were approved by the Ethics Committee of the Medical Faculty, University of Uppsala.

Nerve recordings

The nerve signals were recorded with an insulated tungsten micro-electrode (tip diameter about 5 μm), which was inserted manually through the intact skin into the underlying left peroneal nerve at the fibular head. A low-impedance reference electrode was placed subcutaneously 1–2 cm away. The nerve was localized with the aid of electrical stimuli delivered through the recording electrode. When the nerve was encountered, an electrode position within a muscle nerve fascicle was identified by muscle twitches evoked by the electrical stimuli and by the appearance of afferent, mechanoreceptive activity elicited by stretching or tapping the appropriate muscle. Similarly, a skin nerve fascicle was identified by paraesthesiae without muscle twitches following electrical stimulation, and afferent impulses evoked by gentle skin touch but not by muscle tapping. Thereafter minor adjustments of the electrode position were made until the characteristic pattern

of multi-unit m.s.a. or s.s.a. was recorded. The evidence that the signals are of sympathetic origin are in summary: (1) the activity is efferent, as shown by the application of local anaesthesia proximal and distal to the recording site; (2) the impulses are conducted with a velocity of about 1 m/s; (3) the activity is reversibly abolished by the sympathetic ganglion-blocking agent trimetaphan: (4) changes in nerve activity are succeeded by changes of sympathetic effector organ activities (Delius *et al.* 1972*a*, *b*, *c*; Hagbarth *et al.* 1972; Fagius & Wallin, 1980).

The nerve signal was amplified in two steps (total gain 50000) and fed through a 700–2000 Hz bandpass filter and an amplitude discriminator for improving signal-to-noise ratio. An R–C integrating network (time constant 0·1 s) provided a mean voltage display of the multi-unit neural activity (the relationship between discriminated original and mean voltage neurogram is shown in Fig. 1).

An electrocardiogram (e.c.g.) was recorded by chest electrodes and respiratory movements by a strain gauge strapped around the chest with a rubber band. In two experiments blood pressure was recorded invasively through a catheter in the left brachial artery connected to a pressure transducer (EMT 35) and an electromanometer (EMT 31: Siemens–Elema, Stockholm, Sweden). A photo-electric plethysmograph (van Gogh, Amsterdam, Netherlands) was used in four experiments for monitoring skin blood flow changes in the big toe of the left foot.

All recorded signals were stored on tape (FM tape recorder, Sangamo Sabre VI, Sangamo Weston-Schlumberger, Sarasota, FL, U.S.A.) for subsequent analysis. During the experiments the signals were displayed on a storage oscilloscope.

General procedure

The experiments were carried out in a laboratory with an ambient temperature of 22–24 °C. The subjects were lying horizontally prone on a comfortable table. The head and shoulders were supported by pillows which were temporarily removed and replaced by a tub of water with its surface approximately at the level of the table. The subject then held his head immediately above the water surface for about 30 s before immersing the face (forehead, eyes, nose, mouth and chin) at the end of inspiration; the term 'diving' is hereafter used for this procedure. The time taken to get into the immersed position was 0·5–1·0 s. The duration of the immersion was usually 12 s; in some experiments immersions of 30 s were added. No hyperventilation preceded the dives, but the subjects were instructed to make the last inspiration before immersion slightly deeper than normal. Before a 30 s dive a deeper last inspiration was allowed. Diving for 12 s did not evoke a desire to breathe, but following emersion (or after apnoea without diving for 12 s) the first one to three breaths were slightly deeper than before; during the 30 s dive a need for air was felt by some subjects. Detailed instructions were given to avoid a Valsalva-like manoeuvre and the diving procedure was practised before the experiment started.

Experimental procedure during recordings of m.s.a.

Repeated diving for 12 s was performed with a water temperature of 20 °C in fourteen experiments (in three subjects two recordings were made on different days). In seven of these experiments a number of variations were added: diving in water at 9 °C (seven experiments) and 34 °C (four experiments); diving in water at 20 °C without concomitant apnoea by use of a snorkel and a nasal clip (seven experiments); apnoea without diving with the head held in the same position as during immersion (seven experiments); apnoea in the same position with simultaneous application of a stream of cool or warm air against the face by use of an electric fan and a domestic hair dryer (seven and four experiments respectively). All these manoeuvres were performed for 12 s and repeated 2–4 times in each experimental session.

In four experiments diving for 30 s in water at 20 °C was performed twice, first as a plain prolongation of the immersion and then with superimposed mental arithmetic: the subjects were instructed to add numbers given orally by the experimentor every third second and to give the sum after emersion.

In a few subjects prolonged (25–30 s) or maximal apnoea without concomitant diving was added.

Experimental procedure during recordings of s.s.a.

Diving for 12 s in 20 °C water with apnoea was performed by five subjects, repeated 2–4 times. In one subject prolonged dives of 30 s with and without mental arithmetic were included and in another diving in 10 °C water was added.

Analysis procedure

The main analysis was made from a 2·5 mm/s paper display of the recorded signals, provided by an ink-jet recorder (Siemens–Elema, Stockholm, Sweden). The mean voltage neurogram was amplified to a degree giving a similar amplitude of bursts in all recordings; i.e. amplification differed between subjects (cf. below). For detailed comparison of instantaneous heart rate and occurrence of bursts of m.s.a., 25 mm/s paper displays of the diving manoeuvres were used.

M.s.a. The total outflow of m.s.a. per unit time can be defined as the product of the number of bursts and the strength of the bursts (corresponding to the area under the bursts in the mean voltage neurogram, which is closely related to the burst amplitude, cf. Fig. 1). Since the amplitude of the recorded bursts is critically dependent on the intraneural position of the electrode, it cannot be used for comparison of the strength of the sympathetic outflow on different recording occasions, but within a given recording, relative burst amplitude is representative of the strength of the activity when the response to a manoeuvre is compared with a control period (provided the electrode position is unchanged during the manoeuvre). The total m.s.a. during the dives or alternative procedures was therefore calculated as the product of the number of bursts and the mean burst amplitude (measured in millimetres with a ruler in the mean voltage neurogram) during the manoeuvre and expressed in arbitrary units. With repeated manoeuvres a mean value was calculated. Thus a mean level of total sympathetic activity for each manoeuvre performed was obtained for each experimental session. The sympathetic outflow during the 12 s manoeuvres was compared with two different control periods: (a) the mean total m.s.a. during the 12 s period immediately prior to immersion or corresponding procedure, i.e. with the head unsupported; (b) the mean total m.s.a. of ten randomly chosen 12 s periods during rest with the head resting on pillows.

S.s.a. The nerve activity was analysed during diving and equally long control periods (immediately prior to immersion) and by measuring the area under the mean voltage neurogram to an added base line and expressed in arbitrary units. Only the period immediately before diving was chosen as the control period, since s.s.a. increased considerably in most subjects prior to diving, presumably due to psychic tension before the procedure (under true testing conditions s.s.a. may be entirely absent). The measurement was made by use of a digitizing board (Talos, Phoenix, AZ, U.S.A.) connected to a computer (Apple Computer, Cupertino, CA, U.S.A.).

Statistical methods. Two-tailed Student's *t* test for paired samples, linear regression and *t* test of the regression line were applied.

RESULTS

M.s.a.

The outflow of m.s.a. was the same in the two different 12 s control periods. In all subjects, diving was associated with an intense increase in m.s.a. and a bradycardia, as exemplified in Fig. 1. Mean reduction in heart rate was 14 % (range 5–31 %). Mean total outflow of m.s.a. (in arbitrary units) in fourteen experimental sessions was 39 ± 6 ($\pm$S.E. of mean) in the control period and 153 ± 22 during diving ($P < 0{\cdot}001$; Fig. 3*A*). The mean percentage increase in m.s.a. from control period to diving in 20 °C water was 360 % (range 54–1050 %). In a given subject the increase was fairly constant and without any sign of adaptation (maximum number of dives in one experiment was eleven). Upon emersion some subjects exhibited one or two further strong bursts, then the activity ceased abruptly (Fig. 1) and remained decreased for up to 1 min (the delay before reappearance of normal m.s.a. correlated inversely with the amount of activity at rest).

Fig. 2 illustrates, and Fig. 3*B* summarizes the effect of different water temperatures and alternative manoeuvres on m.s.a. A water temperature of 9 °C (perceived by the subjects as cool or cold; one subject labelled this water unpleasant) was associated with a significantly stronger increase in m.s.a. than a temperature of 20 °C (perceived as slightly cool, not unpleasant). Diving in water at 34 °C, which was perceived as lukewarm, still elicited a clear increase in m.s.a. (Fig. 2).

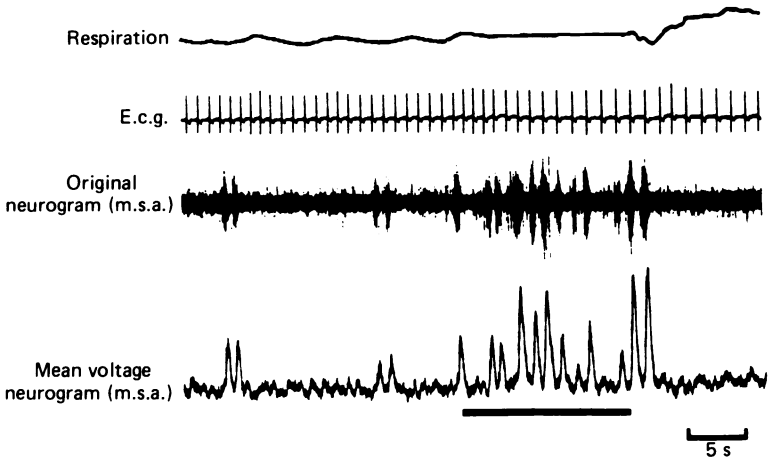

Fig. 1. Typical strong increase in m.s.a. on diving in water at 20 °C. Diving indicated by horizontal bar. Traces from above: respiration (inspiration upwards), e.c.g., original (discriminated) neurogram, mean voltage neurogram.

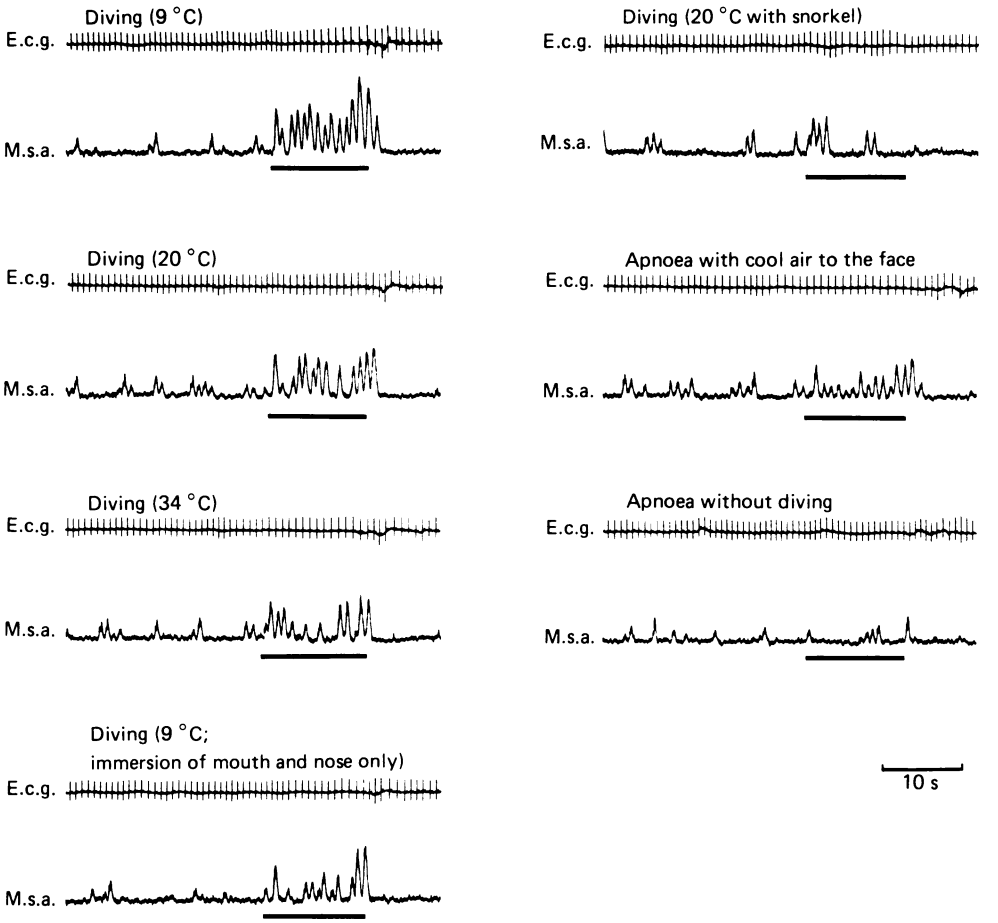

Fig. 2. Responses of m.s.a. to diving and alternative manoeuvres in one subject. Manoeuvres indicated by horizontal bars. Upper traces, e.c.g.; lower traces, mean voltage neurogram. Same time scale in all panels.

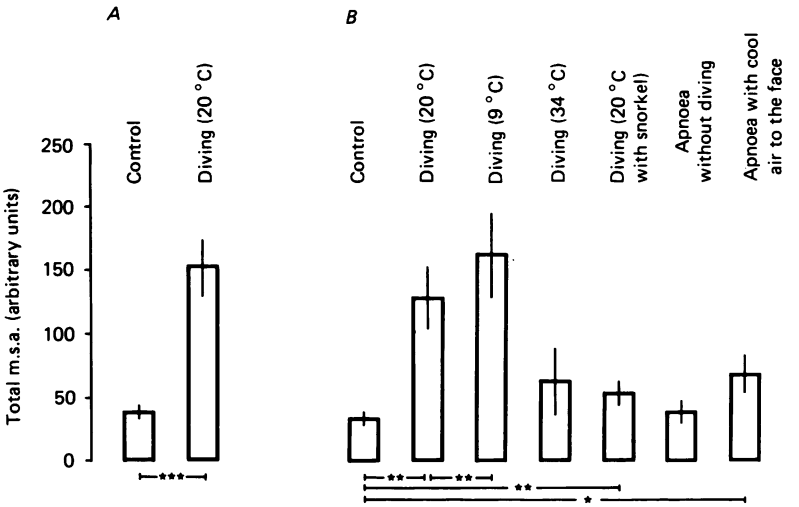

Fig. 3. Response of m.s.a. to diving, or alternative procedures, for 12 s periods. Arbitrary units; mean, S.E. of mean. *A*, outflow at control and on diving in water at 20 °C; $n = 14$. *B*, outflow at control and on diving in water at different temperatures or with alternative manoeuvres; $n = 7$ (except diving at 34 °C where $n = 4$). Significant differences indicated at bottom: *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

Diving (20 °C) while breathing through a snorkel caused a moderate but significant increase in m.s.a.: often an initial activation followed by a relative neural silence, as seen in Fig. 2. Apnoea for 12 s without diving did not cause any change in m.s.a. (Figs. 2 and 3*B*). A prolonged apnoea without diving regularly brought about a strong activation of m.s.a. after about half a minute, as shown in Fig. 4; this increase coincided with an increasing desire to breathe. Apnoea for 12 s with cool air against the face from a fan also elicited a significant increase in m.s.a., although much less pronounced than that seen with diving (Figs. 2 and 3*B*). Apnoea (12 s) combined with warm air was not associated with any detectable change in m.s.a.

Immersion of the lower part of the face only (nose, mouth and chin) with simultaneous apnoea evoked a clear increase in m.s.a. at both 9 °C and 20 °C (not tested at 34 °C), but the activation was stronger when the whole face was immersed (Fig. 2). Manual occlusion of the mouth and nose with apnoea for 12 s did not elicit any response. One subject with a strong increase in m.s.a. during diving was instructed to 'pretend to dive'; no change was seen in the neurogram.

Relationship between m.s.a. and heart rate. An analysis was made to see whether changes in sympathetic outflow and heart rate were dependent on one another. There was a tendency towards a lower heart rate with a stronger increase in m.s.a. during diving, but no close inverse relationship was found. Bradycardia was more pronounced with lower water temperatures and was qualitatively correlated with the stronger increase in m.s.a.

Strong bursts of m.s.a. were often seen immediately upon immersion of the face (Figs. 1, 2 and 4), whereas bradycardia usually arose after a short delay. Detailed analysis of the temporal course revealed that two subjects displayed an immediate marked activation of m.s.a. with concomitant bradycardia on diving. In ten of the

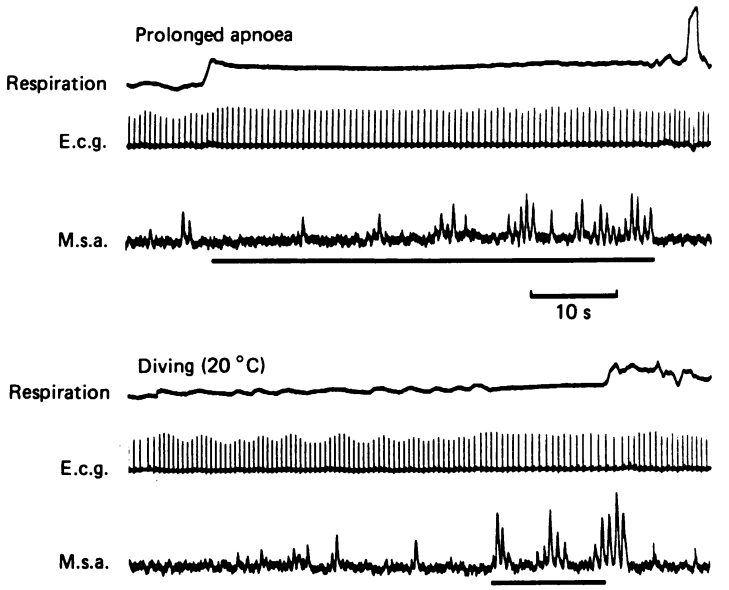

Fig. 4. Comparison of responses of m.s.a. to prolonged apnoea without diving and to diving in water at 20 °C for 12 s in one subject. Manoeuvres indicated by horizontal bars. Traces from above: respiration (inspiration upwards), e.c.g., mean voltage neurogram. Late increase in m.s.a. with apnoea only and immediate increase on diving.

fourteen experimental sessions strong bursts of m.s.a. appeared upon diving before the bradycardia (defined as the occurrence of an R–R interval longer than the longest one during the preceding control period). Bradycardia appeared before m.s.a. in one subject (this subject displayed an unusually low amount of m.s.a. at rest), whereas the time relationship was variable in one.

A reflex latency can be defined from a heart beat to the succeeding burst of m.s.a. (Fagius & Wallin, 1980): in peroneal nerve recordings the latency is about 1·3 s. Thus a given burst of m.s.a. can be identified as corresponding to a certain R–R interval. Beat-to-beat analysis showed no correlation between the strength of individual sympathetic bursts and the duration of corresponding R–R interval during diving; the longest R–R interval was not even necessarily associated with a burst of m.s.a. For the strong bursts appearing immediately on diving in some subjects, it could be estimated that the corresponding R–R interval could occur before immersion (even when it was taken into consideration that the immersing manoeuvre was not instantaneous).

In contrast to the abrupt cessation of m.s.a. following emersion, the bradycardia persisted for 2–10 s (most clearly seen in Figs. 1 and 6*B*).

Fig. 5 illustrates the effect of mental arithmetic during diving for 30 s, perceived as considerable mental stress by the subjects. The bradycardia was substantially less than in a 30 s dive without arithmetic; mean numbers of heart beats during the dive (four experiments) were thirty-seven and twenty-eight with and without arithmetic respectively. The activation of m.s.a. was unaffected by the mental stress, mean total sympathetic outflow (in arbitrary units) being 266 and 251 respectively.

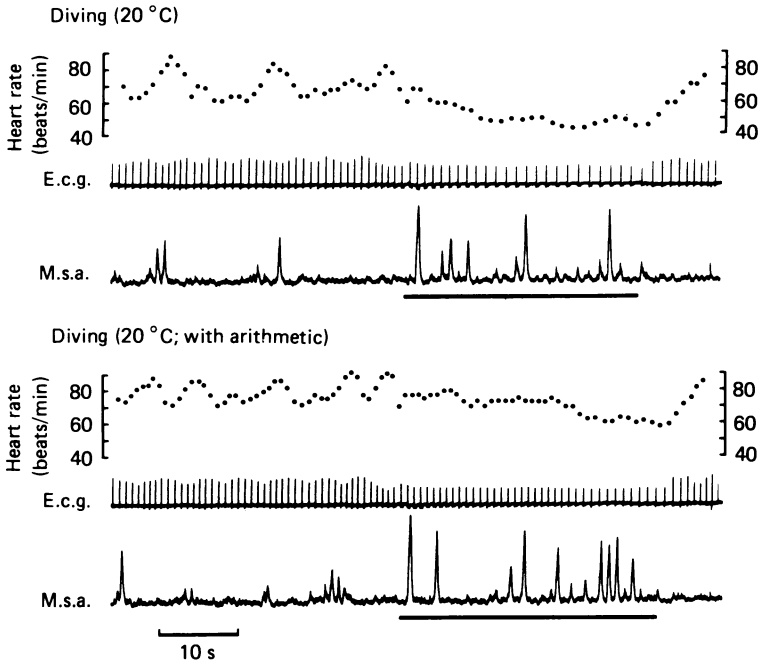

Fig. 5. Responses of m.s.a. and heart rate to diving in water at 20 °C in one subject, with mental arithmetic added. Diving indicated by horizontal bars. Traces from above: instantaneous heart rate, e.c.g., mean voltage neurogram. Bradycardia less pronounced but increase in m.s.a. unchanged by addition of mental stress.

Relationship between m.s.a. and blood pressure. Blood pressure increased substantially in all fifteen dives performed with blood-pressure recording (Fig. 6). The delay from immersion to onset of blood pressure rise varied, since immersion could occur during different phases of spontaneous blood pressure fluctuations; on all occasions blood pressure went up within 5 s and when the immersion coincided with a spontaneous rise in blood pressure, the blood pressure level immediately became higher than during the preceding control period.

Blood pressure fluctuations were more pronounced during diving. M.s.a. occurred predominantly during falling phases of blood pressure (as in the resting condition), although at a successively higher blood pressure level, as illustrated in Fig. 6*A*. In one subject, exhibiting a 'maximal' sympathetic outflow during diving, m.s.a. appeared despite an ongoing increase in blood pressure (Fig. 6*B*).

On emersion there was neural silence despite a pronounced fall in blood pressure; as shown in Fig. 6*B*, m.s.a. did not reappear until the blood pressure had returned to its pre-diving level.

S.s.a.

S.s.a. was studied during diving in water at 20 °C. One subject also dived in water at 9 °C. Spontaneous s.s.a. was at a high level immediately before immersion in most subjects. In no subject did an increase in nerve activity occur: instead, the neural outflow was more or less inhibited during diving (Fig. 7). On some occasions the

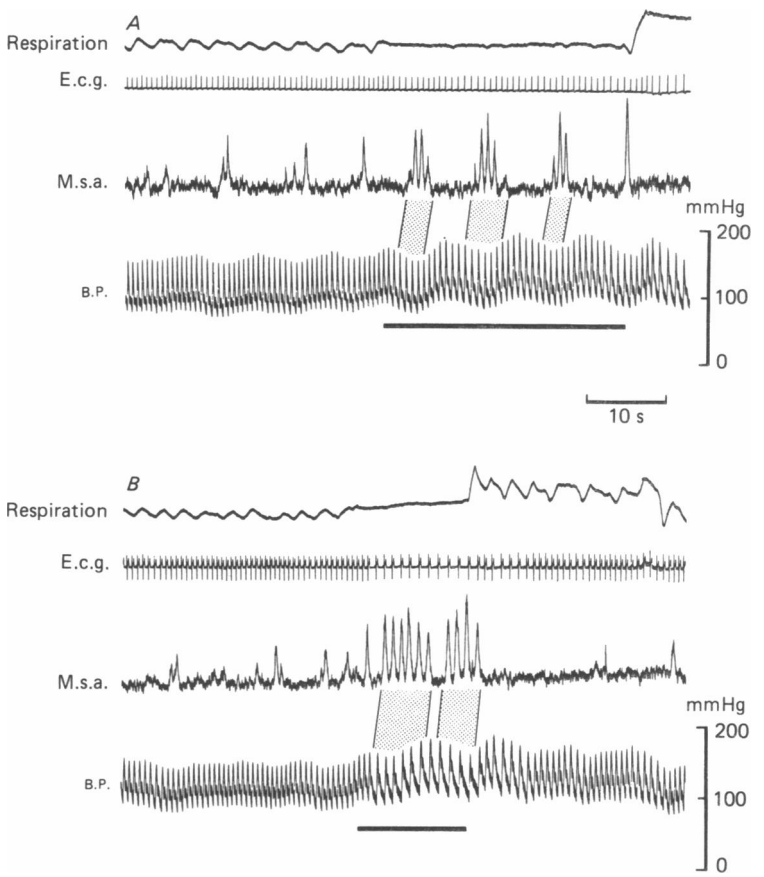

Fig. 6. Relationship between m.s.a. and blood pressure on diving in water at 20 °C in two subjects. Diving indicated by horizontal bars. Traces from above: respiration (inspiration upwards), e.c.g., mean voltage neurogram, intra-arterial blood pressure recording. Shaded areas indicate time relationship (reflex delay) between blood pressure and m.s.a. *A*, 30 s dive. M.s.a. appearing during falling phases of blood pressure fluctuations, at a successively higher level of blood pressure. *B*, 12 s dive. M.s.a. occurring with simultaneous increase in blood pressure. Note delay of reappearance of m.s.a despite falling blood pressure after emersion until pre-diving level of blood pressure is regained.

inhibition was pronounced; on others less so, some activity still being seen. Mean outflow of s.s.a (in arbitrary units) was 148 ± 12 ($\pm$S.E. of mean) and 109 ± 10 during the control period and diving for 12 s respectively ($P < 0{\cdot}001$; sixteen observations in five experimental sessions). The inhibition was seen also on diving in 9 °C water (perceived by this particular subject as unpleasant).

Plethysmographic recordings from the big toe displayed an increasing pulse amplitude towards the end of the dive. In one subject prolonged dives for 30 s were performed (Fig. 7): the inhibition of s.s.a., concomitant with an increase in pulse amplitude indicating toe vasodilatation, was seen during diving without mental stress, whereas the addition of mental arithmetic was associated with ongoing outflow of s.s.a. and a constant degree of vasoconstriction during the procedure.

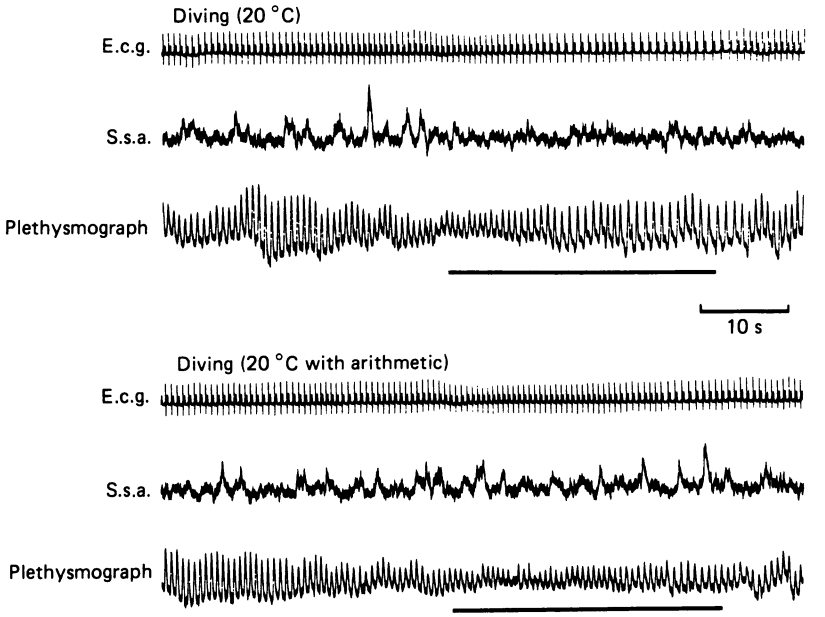

Fig. 7. Typical inhibition of s.s.a. on diving and influence of superimposed mental stress in one subject. Diving indicated by horizontal bars. Traces from above: e.c.g., mean voltage neurogram, plethysmographic recording of blood flow in big toe. Inhibition of s.s.a. on diving with successive increase in pulse amplitude is abolished by simultaneous mental arithmetic.

DISCUSSION

Only the peroneal nerve was explored in the present study, but the observed effects on m.s.a. probably apply to other extremity muscle nerves as well, since m.s.a. has been shown to be equally pronounced in different upper and lower extremity nerves at rest (Sundlöf & Wallin, 1977). Similarly, s.s.a. appears in parallel in the nerves supplying the glabrous skin of the hand and foot (Bini, Hagbarth, Hynninen & Wallin, 1980*b*).

The present study shows that a pronounced increase in sympathetic discharge to skeletal muscle is induced during short-lasting diving in man; from previous studies there is strong evidence that the activity derives from vasoconstrictor fibres (Wallin, 1984). The same manoeuvre causes a reduction in sympathetic outflow to the skin. Such a differentiation of sympathetic activity to different vascular beds during diving has been reported also in ducks (Folkow, Nilsson & Yonce, 1967; Blix & Folkow, 1983). The increase in m.s.a. is in accordance with the reduction in limb blood flow that occurs during diving in man (Heistad *et al.* 1968; Campbell *et al.* 1969; Song, Lee, Chung & Hong, 1969). The whole response, including bradycardia, is similar, although less pronounced, to that seen in diving animals (Blix & Folkow, 1983). The pattern of cardiovascular changes seems to be specific for the diving procedure: a Valsalva manoeuvre was avoided and the change in pulse frequency and blood pressure is clearly different from that seen in the Valsalva manoeuvre (Delius *et al.* 1972*b*).

Stimuli eliciting the increase in m.s.a.

Water contact with the face (or corresponding anatomical structure) has been found to be the major determinant for the vasoconstrictor and heart rate response to diving in both animals (Andersen, 1963*a*, *c*; Dykes, 1974) and man (Kawakami, Natelson & DuBois, 1967; Campbell *et al.* 1969; Moore, Lin, Lally & Hong, 1972). In the present study, on diving with apnoea the increase in m.s.a. was stronger with lower water temperatures (Figs. 2 and 3), as reported previously for diving bradycardia (Kawakami *et al.* 1967; Song *et al.* 1969; Moore *et al.* 1972). However, a clear increase was seen also in lukewarm water (Fig. 2), corresponding to the vasoconstriction reported by Campbell *et al.* (1969), using a water temperature of 34 °C. Immersion of only the lower part of the face induced a weaker increase than when the whole face was immersed at the same temperature (Fig. 2). During diving with snorkel breathing, a significant increase in m.s.a. was seen, primarily during the first few seconds of immersion. Cool air against the face instead of water, with simultaneous apnoea, also evoked a 'diving response'; previous studies of diving bradycardia (Kawakami *et al.* 1967; Moore *et al.* 1972) have shown that wetting the face is not necessary for evoking the response. All these findings suggest that cold receptors and probably mechanoreceptors of the face are involved in initiating the response of m.s.a. It is reasonable to assume that a composite input with spatial summation from many facial receptors is identified by 'pattern recognition', presumably in central sympathetic vasomotor neurones, thereby evoking the response. Whether other afferent pathways, e.g. from the larynx (Tchobroutsky, Merlet & Rey, 1969; Drummond & Jones, 1979), may play a role in eliciting the response cannot be assessed from the present data.

Apnoea only for 12 s was not associated with any change in m.s.a. (Figs. 2 and 3*B*). During a prolonged apnoea the increase in m.s.a. (Fig. 4), coinciding with a need to breathe, is assumed to be induced by peripheral chemoreceptors (Folkow & Neil, 1971; De Burgh Daly, 1984). An asphyxia of any importance is not likely to develop during apnoea of 12 s duration, yet apnoea on diving still elicited a prompt increase in m.s.a., considerably stronger than what would be expected from simple summation of the responses seen on diving without apnoea and apnoea without diving (Figs. 2, 3 and 4). Thus, impulses from facial receptors and effects of apnoea seem to act by mutual reinforcement in evoking the diving response. Similar mechanisms have been reported to underlie diving bradycardia in the duck (Andersen, 1963*b*) and the diving muskrat (Drummond & Jones, 1979). Whether chemoreceptors or any other effects of apnoea cause the reinforcement cannot be concluded from the present results.

Relationship between m.s.a., heart rate and blood pressure

The degree of change of m.s.a. and heart rate on diving differed considerably between subjects. Some inverse correlation between changes in m.s.a. and heart rate during diving was present, indicating that the two effects are to some extent related to each other. However, the lack of any relationship between the strength of individual bursts of m.s.a. and the length of the corresponding R–R intervals shows that the increase in m.s.a. is not only a compensatory phenomenon due to bradycardia. This assumption is reinforced by the finding that longer R–R intervals were not

associated with a fall in blood pressure. Instead, a number of observations suggest that the vasoconstrictor effect may be the primary response to diving: (a) in most subjects increased m.s.a. appeared before the bradycardia on immersion, and on emersion m.s.a. disappeared before the bradycardia in all subjects; (b) reflex latency determination for the strong bursts appearing immediately on immersion showed that corresponding R–R intervals occurred before submersion, indicating that a reflex mechanism, overriding and working faster than the baroreflexes, was activated; (c) the attenuation of diving bradycardia by mental arithmetic, described previously by Ross & Steptoe (1980), with persisting increase in m.s.a. (Fig. 5), indicates that heart rate, but not vasoconstriction, may be modulated by supramedullary cerebral influence. Blix & Folkow (1983) give strong arguments for the view that peripheral vasoconstriction is the primary event, and a prerequisite for, the subsequent bradycardia in diving animals. The above findings indicate that the situation is similar in man.

The relationship between blood pressure and m.s.a. on diving exhibited two distinct features. First, m.s.a. was still time-locked in the cardiac rhythm, indicating that carotid baroreceptors inhibited sympathetic outflow with each systolic blood pressure wave in their ordinary manner (Fagius *et al.* 1985). Secondly, m.s.a. appeared at a higher blood pressure level but still predominantly during falling phases of blood pressure (Fig. 6*A*), in accordance with earlier observations that m.s.a. counteracts fall of blood pressure in a dynamic fashion (Sundlöf & Wallin, 1978; Wallin & Eckberg, 1982). Moreover, m.s.a. could appear despite an ongoing sharp rise in blood pressure (Fig. 6*B*), which is not seen ordinarily (Sundlöf & Wallin, 1978; Wallin & Sundlöf, 1979). It seems reasonable that effects of m.s.a. contributed to the rise in blood pressure as seen in Fig. 6. These observations imply a resetting of the baroreceptor control of blood pressure to a higher level of pressure. The two different patterns seen in Fig. 6 would then be an example of different magnitude of this resetting between individuals. The cause of the resetting is unknown, but seems to be a complex reflex mechanism (e.g. subserving oxygen conservation demand), which is activated on diving and which is given priority over ordinary blood pressure regulation. After diving, this specific response mechanism is probably immediately switched off and the baroreflexes returned to their normal working range; after emersion m.s.a. did not reappear until the blood pressure had fallen to its pre-diving level (Fig. 6*B*).

Controversy has existed as to whether the diving response in man (mainly studied as diving bradycardia) is due to primary neural reflex events or to adaptation via baroreflexes to pressure changes in the thorax (Paulev, 1968; Song *et al.* 1969). Intrathoracic pressure mechanics were not assessed in the present study, but the prompt initiation of the effects during short-lasting dives, and the relationships between m.s.a., heart rate, blood pressure and baroreceptor function discussed above, suggest that mechanical adaptation was not a major determinant of the effects observed.

Skin-nerve sympathetic outflow

Vasoconstrictor impulses of s.s.a. govern skin blood flow of thermoregulatory importance (Hagbarth *et al.* 1972; Bini, Hagbarth, Hynninen & Wallin, 1980*a*),

presumably by acting on arterial–venous (a.–v.) shunts (Roddie, 1983). The present inhibition of s.s.a. during diving with concomitant digital vasodilatation (Fig. 7) is in agreement with findings in ducks (Folkow *et al.* 1967). Blix & Folkow (1983) suggest that a.–v. shunts are kept open in animals during diving since they are 'ideally suited for moving oxygen-containing venous blood towards the heart during submersion'. Thus the reduction in s.s.a. probably represents another integrative part of the cardiovascular adjustment to diving, even in man.

Exposure of the body to cool air or exposure of one hand to ice water normally induces increases in skin sympathetic vasoconstrictor activity (Bini *et al.* 1980*a*; Fagius & Blumberg, 1985). In the present study diving induced reduction of s.s.a. and digital vasodilatation despite a cool or even cold water temperature, suggesting that s.s.a., like m.s.a., is during diving governed by regulators that override those operating under normal circumstances. The influence on s.s.a. of psycho-emotional stimuli (Hagbarth *et al.* 1972; Delius *et al.* 1972*c*) was still present, as shown by the lack of inhibition of s.s.a. on diving with mental arithmetic (Fig. 7).

Thus diving exerts opposite effects on m.s.a. and s.s.a. in man, thereby emphasizing the differentiation shown previously between these two subdivisions of the sympathetic nervous system (Wallin, 1984). The associated bradycardia is due to both vagal activation (Heistad *et al.* 1968) and inhibition of sympathetic outflow to the heart (Folkow *et al.* 1967; Gandevia, McCloskey & Potter, 1978). At rest there is evidence for a coupling between m.s.a. and sympathetic impulses to the heart (Wallin & Nerhed, 1982) and following baroreceptor deafferentation a very high level of m.s.a. coincided with intense tachycardia (Fagius *et al.* 1985). Consequently, the diving response also includes a specific differentiation between sympathetic outflow to muscle vessels and the heart.

Most diving animals exhale reflexly on submersion (Andersen, 1963*a*), whereas man inspires. Due to this and other differences between species the response to immersion in diving animals and in man has been suggested to be due to different mechanisms (Paulev, 1968). In contrast to this view, the present results indicate that the human response to diving basically exhibits the same characteristics as those seen in expert animal divers, although the effects evoked are much more pronounced in the latter. The strongly differentiated response of different parts of the autonomic nervous system seems well suited for maximal economical adjustment to a milieu lacking in oxygen.

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