Levels of short (1-15 ms) electrical shocks from a 50 Hz supply inducing ventricular fibrillation in hyperbaric helium and oxygen

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ABSTRACT An investigation was undertaken to determine the ventricular fibrillation (VF) threshold of anaesthetised dogs subjected to external application of electric shocks between a foreleg and a hindleg. The shocks were 2-15 ms sections of 50 Hz sine waveform starting at peak current and were applied at a known time in the heart cycle. The object of the experiment was to determine if there was an increase in cardiac susceptibility to electrically induced VF at 31 atmospheres absolute (atm abs) in a helium and oxygen environment. The duration and position of that part of the cardiac cycle most vulnerable to induction of VF by electrocution was found (seven animals) using 4 ms shocks and then the minimum fibrillating current for shocks of 2-15 ms (min FC2-15) delivered at the most vulnerable point of the cycle (five animals). Body resistance was calculated from the data so gathered. Fibrillation thresholds were not changed by compression and there were no significant changes in the vulnerable period of the cardiac cycle. Min FC2 was significantly higher than for other durations under both control (3.21 A, SD 1.08) and test conditions (3.26 A, SD 0.39), $p = 0.001$. There was no difference in body resistance at 31 atm abs (395.5, SD 12.9) from control values at 1 atm abs (396.7, SD 10.9). From these data it was concluded that the heart is not more susceptible to the induction of VF at 31 atm abs in a helium oxygen environment and additional safety factors are unnecessary from this point of view.

Ventricular fibrillation is the most probable cause of death in immediately fatal electrical accidents and the minimum current flowing through the body required to produce this condition is taken as a guiding point for safety standards. The recent introduction of fast acting current breakers to the commercial market has made it necessary to define the maximum safe levels of short current pulses. It is possible to reduce shock duration to about 1 ms by using line insulation monitors that continuously measure insulation resistance. The cardiac cycle lasts 800 ms at a heart rate of 75 beats a minute and so in evaluating the effects of short shocks allowance must be made for the part of the cycle affected. It has been known for some time that the heart is most susceptible to electric shocks applied during the occurrence of the T wave of the electrocardiogram.¹ This vulnerable period has been mapped by using externally applied 4 ms electric shocks and occurs 110-200 ms after the upstroke of the R wave and is 35-40 ms in duration at currents of

2.5 A (figure).² The worst possible event therefore is best represented by application of the test shock during this vulnerable period. Using this approach we have identified the danger levels of 1-15 ms electrical shocks from a 50 Hz supply in normal atmospheric conditions.² Increasing use of electrical equipment in underwater technology and in proximity to divers requires that information should be available on the interaction of increased pressure and gaseous environment with susceptibility to electrical shock in order to quantify the requirement for additional safety factors.

Recommendations current at the time of this study specified a twofold increase in safety limits to cover the unknown effects of diving on an individual's susceptibility to electric shock.³ The aim of this study was to establish any alteration of the heart's susceptibility to short electrical shocks by conditions that simulated those of a diving habitat at 31 atmospheres absolute (atm abs) and therefore the necessity for safety factors above and beyond those presently in force for work at atmospheric pressure.

ECG vulnerable period. Top trace shows minimum 4 ms current required to induce fibrillation at various delay times. Data were taken from one animal and are typical of vulnerable period in other dogs. Lower trace shows part of ECG cycle in which vulnerable period occurred and trigger point for shocks on upstroke of R wave. Delay time refers to interval between R wave trigger and start of shock.

Methods

These tests were conducted using the Medical Research Council's hyperbaric primate facility, a 1.2 m³ vertically mounted cylindrical chamber rated to a working pressure of 201 atm abs. The vessel is equipped with an external, high pressure atmosphere conditioning unit designed to control temperature and the build up of humidity, carbon dioxide, and other organic contaminants by means of a heat exchanger and filter beds of alumina, activated charcoal, and soda lime. Chamber atmosphere is continuously analysed by mass spectrometry. During these experiments carbon dioxide concentrations in the chamber did not exceed 0.05 kPa (0.4 mm Hg) and relative humidity was kept to between 30% and 50%. Lighting is provided externally through portholes and the interior of the chamber is monitored on close circuit television.

In this series of experiments seven beagle dogs weighing between 12.5 and 18 kg were used as

subjects. The work was performed under general anaesthesia. Thirty minutes before induction of anaesthesia the dogs were premedicated by the intramuscular injection of 0.065 mg/kg fentanyl, 2 mg/kg fluanisone, and 0.02 mg/kg atropine. Anaesthesia was induced intravenously with pentobarbitone in a dose sufficient to enable endotracheal intubation (60–150 mg) and was maintained by the continuous intravenous infusion of pentobarbitone (1.5–3.0 mg/min) and ketamine (3.0–6.26 mg/min). After intubation the animals were allowed to breathe spontaneously. At atmospheric pressure the breathing gas composition was 30 kPa (225 mm Hg) oxygen in nitrogen and at pressure this was changed to 30 kPa (225 mm Hg) oxygen and 70 kPa (525 mm Hg) nitrogen in helium.

The ECG was monitored using non-polarising electrodes (Medicotest). Arterial pressure was measured from a 20 cm cannula of 0.8 mm internal diameter inserted through the femoral artery and lying in the descending aorta. The pressure transducer used was an Elcomatic type EM 750 and was connected to a Roche Kontron type 224 signal conditioner. An impedance pneumograph signal was also available from the ECG electrodes via a Roche Kontron 107 monitor. Estimations of arterial pH, PaCO₂, and PaO₂ were made 30 minutes after the induction of anaesthesia before the application of electric shocks to the animal and again just before compression in six animals (Instrumentation Laboratory blood gas analyser 613). Dog 3 was not so monitored due to analyser failure. Oesophageal temperature was measured in each dog (Yellow Springs 401 thermistor probe) and maintained at 36–38°C using electric heating lamps at atmospheric pressure and the pressure chamber life support system at pressure. Electric shocks were applied between the right foreleg and the left hindleg through metal plates (5 × 3 cm). The plates were closely applied and electrical contact assured by the use of conducting gel. Defibrillation was achieved using another two plates (7 × 6 cm) similarly applied to each side of the chest by 60 or 100 J from a standard dc defibrillator (Cambridge Medical).

The test shock was derived from a specially constructed machine (ERA Technology Ltd) that was triggered by the ECG R wave.⁴ The shock consisted of a short (up to 15 ms) section of 50 Hz sine waveform that could be started at any required phase of the 50 Hz signal and at an accurately controlled time from the R wave trigger (delay time—figure). The generator provided shock impluses (up to 3 kV peak) entirely independent of the power mains and, once the required delay, phase, and duration were set on its controls (to 1 ms accuracy), it would deliver the shock at the set delay time after the first R wave after release

of the "fire" button. The actual current flowing through the animal was monitored via an isolating transformer in series. All shock sequences contained one peak of the sine waveform and the current level quoted refers to this peak.

The ECG, arterial pressure, and current stimulus received by the animal were recorded on a chart recorder (Mingograph) and also on a four channel FM tape recorder (Racal Store 4) for subsequent analysis.

Initially, in order to locate the sensitive time in the cardiac cycle, the shock generator was set to deliver 1 kV (equivalent to about 3 A) for a pulse duration of 4 ms beginning at the peak of a sine wave. The peak start was chosen to avoid the zero crossing, where it was suspected that there might be anomalous effects, and to maximise the delivered energy. The delay time was set to 160 ms and the first shock delivered. If the heart fibrillated then defibrillation was promptly carried out and a second shock at a lower voltage given. The level of shock was then gradually increased to find the minimum level required to induce ventricular fibrillation. If no fibrillation occurred at 160 ms then 140 ms was tried and if necessary 180 ms. Having found one point in the vulnerable period, the delay times just before and after were explored, starting at low voltage and increasing in 100 V stages till fibrillation occurred. In this way the vulnerable period of the cardiac cycle was mapped in relation to the current required to induce fibrillation and time (figure). The results may be read on the basis that fibrillation did not occur at 0.3 A less than the indicated value. In five animals the most sensitive delay time was selected from the data acquired and the fibrillation threshold for shocks of 2–15 ms, starting at this time in the cardiac cycle, determined.

Each electrical stimulus induced a small transient fall in arterial pressure and an associated increase in respiratory and pulse rates. A similar but greater effect was seen after defibrillation subsequent to a shock and ventricular fibrillation. After each stimulus sequence therefore the animal was allowed to recover and shocks were not repeated until respiratory rate, pulse rate, and arterial pressure had returned to pre-shock levels. This took one to 10 minutes.

After mapping of the vulnerable period of the cardiac cycle and determination of fibrillation in response to shocks of different durations the animals were compressed to 31 atm abs at a rate of 1 atm/min by addition of 100% helium to the pressure chamber. Fifteen minutes were allowed for saturation of the heart muscle with helium at pressure and the vulnerable period again mapped as described above. The response to shocks of different durations was determined in the five animals that had been investigated in this manner at atmospheric pressure.

Results

The data gathered were assessed with reference to factors that might increase the likelihood of ventricular fibrillation. These were the resistance of the animal, the duration of the vulnerable period, and the response to shocks of different duration at the most sensitive part of the vulnerable period.

The data were subjected to statistical analysis using the programme SPP, a statistics package for personal computers,⁵ run on an Apricot personal computer.

VULNERABLE PERIOD

The vulnerable period was plotted for each animal as in the figure. The resultant trace was roughly U shaped with the down strokes of the U lying between 4 and 7 A. The possible differences in vulnerable period were assessed by comparing the duration of the period at 5 A (VP 5). The data from 31 atm abs were not normally distributed and difference between the two pressures was assessed using a Wilcoxon matched pairs test. Mean VP 5 at atmospheric pressure was 31.1 ms (SD 8.8) and 34.9 ms (SD 13.7) at 31 atm abs (table 1). There was no suggestion of difference due to pressure ($p = 0.67$).

RESISTANCE

Resistance was calculated by the application of Ohm's law to the peak voltage of the shock delivered to the animal and the peak observed current. Values of peak current and voltage were taken from the data recorded during six shocks at atmospheric pressure and six at 31 atm abs. From these data resistance was calculated and the means of the six values so obtained are presented in table 1 and were used for comparison of the two different atmospheric conditions. The standard deviation round these mean values was never

Table 1 Body resistance and duration of the vulnerable period at 1 and 31 atm abs. (Data quoted for resistance were calculated from Ohm's law from the values of voltage and current during six randomly selected shocks of more than 1 kV. Duration of the vulnerable period was taken at a current level of 5 A)

Pressure (atm abs)	Resistance (ohms)		Duration of vulnerable period (ms)	
	1	31	1	31
Dog No:				
1	392.3	400.0	30	60
2	416.0	408.0	50	30
3	399.4	405.5	30	20
4	400.3	391.7	30	40
5	394.2	396.2	25	35
6	395.8	395.8	30	35
7	379.4	371.6	22.5	20
Mean	396.7	395.5	31.1	34.3
SD	10.9	12.0	8.8	13.7

Table 2 Minimum 50 Hz current (A) required to induce ventricular fibrillation. (All shocks contained one peak of the sine waveform and the current level quoted refers to this peak. The vulnerable period was mapped using 4 ms shocks and all data in this table refer to shocks starting at the most sensitive delay time so identified)

Shock duration (ms)	2		4		6		8		10		13		15	
Pressure (atm abs)	1	31	1	31	1	31	1	31	1	31	1	31	1	31
Dog No:														
1				2.37		2.50								
2				2.03		2.13								
3				1.97		2.52								
4	3.03	3.23	1.86	1.74	2.0	2.5	1.73	1.97	1.75	1.48	1.86	2.09		1.77
5	2.50	2.77	1.73	2.07			1.97	1.4	1.72	1.45	1.77	1.20	2.01	1.82
6	3.75	3.0	1.73	2.07			3.01	2.02	2.53	2.3	2.6	2.12	2.33	2.30
7	2.02	3.7	1.97	2.75	2.06	2.04	1.77	2.0	1.48	2.36	1.63	2.08	1.82	2.13
	4.77	3.6	3.2	3.2	3.16	3.77	2.7	2.60	2.7	2.85				
Mean	3.21	3.26	2.16	2.42	2.41	2.77	2.24	2.0	2.04	2.09	1.97	1.87	2.05	2.0
SD	1.08	0.39	0.49	0.48	0.65	0.90	0.58	0.42	0.54	0.61	0.43	0.45	0.26	0.25

more than 17. The shocks analysed were chosen at random from the experimental data. With shocks of less than 1 kV non-linear current versus time and current versus voltage relations have been observed that were not seen during shocks of higher voltages.⁶ Accordingly only shocks of more than 1 kV have been considered. At atmospheric pressure mean resistance was 396.5 (SD 12.0). The data at increased and atmospheric pressure were compared using a paired *t* test. There was no difference (*p* = 0.66).

FIBRILLATION THRESHOLDS IN RESPONSE TO SHOCKS OF DIFFERING DURATION

In five animals the minimum peak current required to induce fibrillation was determined for shocks of 2–15 ms and the data obtained are presented in table 2. There were no gross differences between the two pressure conditions. The fibrillation current for all five animals had been determined for shock durations of 2, 4, 8, and 10 ms and these data were subjected to analysis of variance together with data from a further two animals at 4 ms. This provided for testing the effects of pressure (on two levels—one and 31 atm abs) and duration of shock (on four levels—two, four, eight and 10 ms). Pressure had no effect on fibrillation current. There was also no interaction between the two factors. There was, however, a

significant effect due to the duration of the shocks (*p* = 0.1×10^{-3}). This effect was further assessed by a contrast of means. Mean fibrillation current for shocks of 2 ms were significantly greater than the mean of the other three durations (*p* < 0.001). Fibrillation current for shocks of four, eight, and 10 ms did not differ significantly from each other at the 5% level under both environmental conditions.

BLOOD GAS RESULTS

Blood gas results are presented in table 3. Anaesthesia depresses respiration and this is reflected in a raised P_aCO_2 (normal range 4.7–6.0 kPa, 35–45 mm Hg) and lower than normal pH (normal range 7.35–7.45) both before and after determination of fibrillation thresholds. The before and after values of pH, P_aCO_2 , and P_aO_2 were compared using paired Student's *t* tests. There were no significant differences in blood gases from control after determination of fibrillation levels at 1 atm abs. Standard bicarbonate levels were not normally distributed and were assessed by a Wilcoxon matched pairs test that showed a small but suggestive decrease over control levels (*p* = 0.059).

DEFIBRILLATION

Although we made no attempt to assess any change in defibrillation energy required under the two pressure conditions we did not see any overt increase in the energy required to defibrillate at 31 atm abs.

Discussion

The animals in this study exhibited a degree of respiratory acidosis at 1 atm abs that may have been slightly potentiated by the development of an accompanying metabolic acidosis induced by the methods used for the determination of fibrillation thresholds. The gas mixture used at pressure during this study is about five times more dense than air at 1 atm abs and such respiratory gas densities have been associated

Table 3 Blood gas data before and after determination of fibrillation thresholds at atmospheric pressure. (Values quoted are the mean and SD from six animals. Standard bicarbonate (S Bic) levels are also quoted)

	Before shocks	After shocks
Time after start of anaesthesia (min)		
pH	41 (7.4)	132 (25.9)
P_aCO_2 (kPa)	7.284 (0.025)	7.262 (0.046)
P_aO_2 (kPa)	6.89 (0.51)	6.703 (0.79)
S Bic (mmol/l)	15.4 (3.8)	14.2 (3.2)
	22.5 (1.9)	20.9 (2.1)

with hypoventilation⁷ which would accentuate anaesthetic induced respiratory depression and antagonise respiratory compensation for a developing metabolic acidosis. It is quite possible, therefore, that the animals in this trial were more acidotic at pressure than at the surface. Both metabolic and respiratory acidosis have been reported to reduce the current required to induce ventricular fibrillation,^{8,9} although the pH changes associated with such changes were much larger than those seen in this study and the work differed from that presented here in that fibrillation thresholds were determined after thoracotomy and exposure of the heart. Later work on electric shock in intact animals with normal acid base balance indicated that the maintenance of normal body chemistry was not associated with a change in fibrillation thresholds but that deviation about the mean was reduced.¹⁰ The present study shows that at 31 atm abs there was no difference in fibrillation threshold neither did there seem to be an increase in the SD around the mean values obtained. If acidosis had reduced fibrillation thresholds then this was counteracted by a compensating protective effect due to the changed environment. This would seem to be unlikely in view of the known action of pressure in slowing conduction velocity in cardiac tissue,¹¹ an action that might be expected to render the heart more vulnerable.¹⁰ We conclude that although the experimental animals were probably acidotic at pressure the degree of acidosis was insufficient to affect fibrillation thresholds.

It was considered that susceptibility to ventricular fibrillation could be increased at pressure by a reduction in body resistance, an increased susceptibility of the heart to electric current, or a widening of the vulnerable period. We detected no change in any of the variables measured. A further risk factor might lie in change of shape of the vulnerable period from, for example, a V shape at atmospheric pressure to a U shape at pressure. On mapping the vulnerable periods for each animal at 1 and 31 atm abs we detected no gross changes in the shape of the vulnerable window and therefore considered that statement of the lowest fibrillation current during the period and duration of the period was an adequate method of assessment.

Before this study recommendations for using electricity during diving operations incorporated a two-fold increase in safety limits to cover the unknown effects of diving on an individual's susceptibility to electric shock.³ Safety regulations for shocks of more than 10 seconds duration from a 50 Hz source at atmospheric pressure are based on the current level that causes involuntary muscle spasm and inability to release a conductor. For shorter shocks they rely on fibrillation thresholds determined in dogs or pigs.¹² In an earlier study we detected no increased sus-

ceptibility of dogs to ventricular fibrillation in response to shocks of five seconds duration at 6 atm abs in air.¹³ Data from this trial indicate no significant effect of compression to 31 atm abs on the fibrillation threshold in dogs with 50 Hz shocks of 2–10 ms duration. These data do not support the incorporation of extra safety factors, based on fibrillation threshold, into the use of electricity in diving.

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