Efficacy of Surface Electrodes for the Detection of Electrocardiogram Activity During Deep Accidental Hypothermia

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Abstract

Experiments on seven anaesthetised pigs were conducted where ECG was measured using surface, needle and epicardial electrodes during cooling of the pig to simulate deep accidental hypothermia. Data at four cooling conditions were examined: Control (skin at room temp, core 38c), Cool Skin (skin 2c, core 30c), Hypothermia and deep Hypothermia (skin 10c, core 15c). There was no significant change in the amplitude of ECG with cooling of the skin. However, there was a factor of 9 decrease in the ECG amplitude with decreasing core temperature. Core cooling slowly results in a decrease in the amplitude of the QRS complex and T wave, which is ultimately replaced by a 1Hz flutter, while the P wave remains relatively constant. This suggests that decreases in ECG amplitude is due to changes in the active bioelectrical behavior heart, rather than changes in the distribution of the ECG field in the body due to temperature dependant passive electrical impedance changes in the tissue.

1. Introduction

Every year in Denmark alone, more than 100 people die of accidental drowning in cold waters. Many of those who are rescued are found with surface skin temperatures around 0ºc, core temperatures around 23ºc, with no measurable ECG using surface electrodes. Often they are declared dead at the scene, however, upon further examination, are found to have a weak heart sound and are hastily treated for hypothermia [1,2] .

The present study attempts to recreate these conditions of deep accidental hypothermia, but under controlled experimental conditions in the anaesthetized animal model to determine the mechanism for the failure of the surface measurements to detect ECG. Since the skin temperature is around the freezing point of water, one possibility is that freezing of the electrode and/or skin and the associated changes in the various impedances [3] influence the measured ECG. Needle electrodes were tested along side of surface electrodes to test this hypothesis.

We aim to determine whether the source of the condition was due to instrumentation or electrode failures, and whether a solution to the problem could be found.

2. Methods

Acute experiments were performed on 7 adult female Danish Landrace pigs (~90kg). 1 hour prior to surgery, the pigs were premedicated with diazpham. Anaesthesia was initiated and maintained with intravenous injections of a ketamine, fentanyl, midazolam cocktail. The pigs were intubated with a cuffed tube in the trachea and a servo ventilator was used for ventilation. Airflow was adjusted according to the ventilation need to supplement the oxygenator. The blood pressure (BP) was monitored using a catheter in the left carotid artery. An extracorporial circuit was established between the right carotid artery through a 5.2mm arterial needle (Stökert Institute, Munich. Germany) and the right vena jugularis through a 29 French triple stage venous catheter (Edward Life Sciences). The circuit contained a centrifugal pump (Rotaflow, Jostra AB, Germany), oxygenator and a heat exchanger. The animals were treated with Heparin to an activated coagulation time (ACT) of 480 seconds. A complete description of this system appears elsewhere [4].

Surface and needle electrodes were placed on the right arm and left leg, roughly corresponding to the lead placement of Lead 2 of the Einthoven 3 lead configuration. The reference electrode was placed on the right upper leg. Pacemaker electrode leads were sewn on the epicardium at the right atrium and at the apex of the heart and used to measure the epicardial ECGs. ECGs were amplified (100x epicardial, 2500x surface/needle) and filtered (LP 1200Hz) using an Axon Cyberamp amplifier (Cyberamp 380 and 402 Smartprobes). Finally, signals were sampled at 2kHz (Mr.Kick, Aalborg Univ) with a 12-bit data acquisition card (National Instruments NI6040E). Thermocouples were placed just adjacent to

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Computers in Cardiology 2004;31:385−388.

the surface electrodes to measure skin temperature, and in the intraperitoneal cavity to measure core temperature.

The instrumented pig was placed in a small inflatable raft filled with crushed ice to cool the body of the pig from the exterior in addition to the cooling to the core provided by the extracorporeal circulation system. The external cooling the ice provided was necessary to simulate the cold skin conditions of the accidental hypothermia cases. During the course of the experiment, the blood temperature, oxygenation level, and blood flow rate of the pig were regulated through the extracorporeal circulation system. The blood temperature of the pig was slowly lowered to simulate deep hypothermia until either asystole was induced or the surface ECG showed no measurable activity. Once the bottom point was reached, the pig was slowly warmed and resuscitation from deep hypothermia was attempted. Simultaneous records of the various ECGs and temperatures were made for later off line analysis.

3. Results

3.1. ECG and temperature

Decreasing the body temperature and inducing hypothermia has several effects on the ECG, which are summarized in Figure 1 from a typical experiment. The left panel of the figure shows the core and skin temperatures measured during the experiment. The right panel shows the corresponding average ECG cycle as measured by the surface electrode. In addition to the standard protocol of cooling and warming the animal, the effect of freezing the skin with compressed CO_2 immediately around the surface electrode was tested in this experiment between minutes 40 - 60. It shows that during freezing of the skin and electrode, there is very little change in the core temperature and very little change in the ECG. The ECG changes are mostly related to changes in the core temperature, as measured with the thermocouple in the intraperitoneal cavity. During

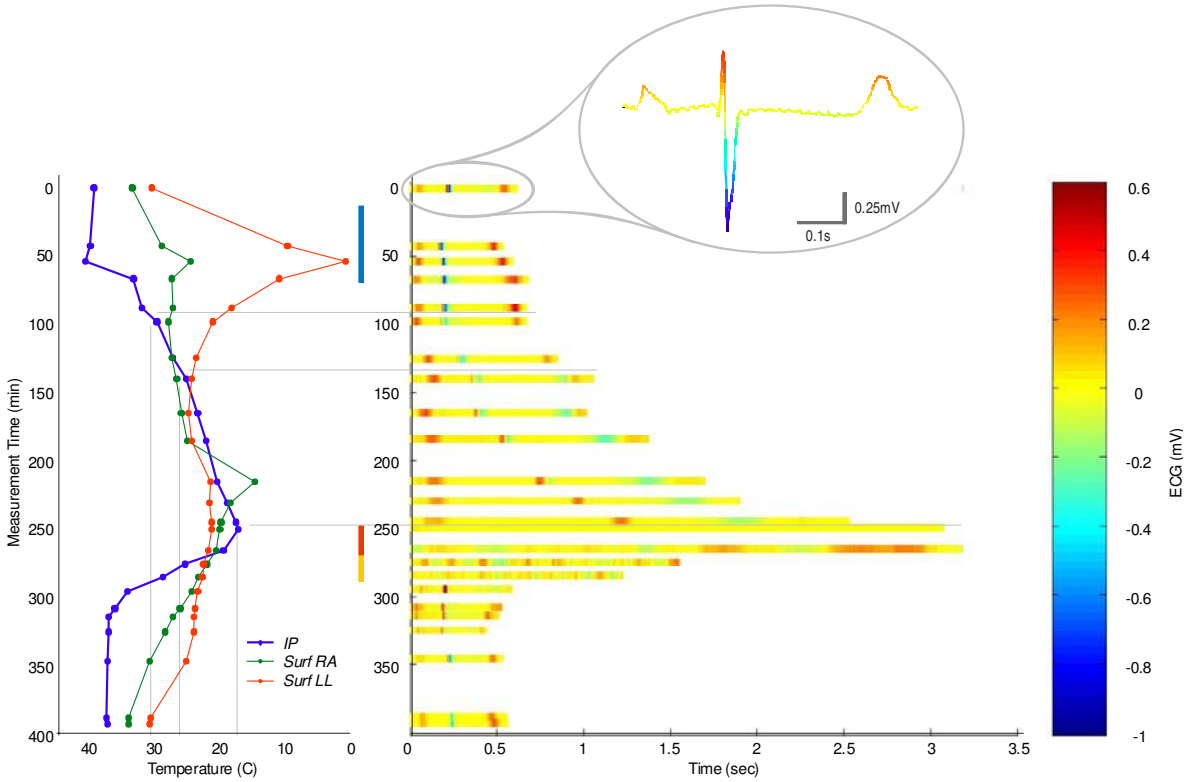

Figure 1. The temperature and average ECG time course of a typical experiment. The left panel shows the core temperature and the temperatures of the skin in the vicinity of surface electrodes place on the Right Arm (RA) and Left Leg (LL). The right panel shows the average ECG waveform recorded by the surface electrode (Einthoven Lead 2) corresponding to the times and temperatures in the left panel. The ECG lengthens, and its amplitude decreases with decreasing core temperature. Freezing the surface electrode and skin near the surface electrode (blue bar) shows little to no effect on either the period or amplitude of the ECG.

cooling, the length of the ECG increases, while the amplitude of the QRS complex notably decreases. The P-wave generally lengthens in time, though its amplitude is less affected by the decrease in temperature. Finally, 250 minutes after the start of the experiment, at the bottom temperature, only the P-wave remains, though it is difficult to distinguish from baseline noise. Moments later, the heart activity ceases followed by ventricular fibrillation. The BP at this point indicates no cardiac function. At this point, we started warming the animal and the core temperature slowly increases. At approximately minute 290 at a core temperature of ~30ºc, ventricular fibrillation spontaneously converts to a sinus rhythm. We continue to warm the animal until minute 310 when the core temperature reaches normal body temperature.

3.2. Surface vs. needle electrodes

The lead 2 ECG from gel type surface electrodes and needle electrodes were simultaneously measured during cooling. Figure 2 shows a typical records taken during four points of the experiment corresponding to the initial measurement (control), maximum skin cooling (cold skin), during cooling when the skin temperature and core temperatures were at 28ºc (cooling), and at the minimum temperature (cold core). There is very little difference between the ECG measured using needle electrodes and surface electrodes.

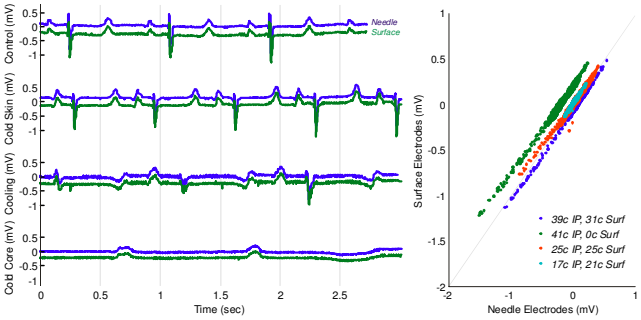

Figure 2. Lead 2 records from surface and needle electrodes. The left panel shows records taken at control and 3 stages of cooling. The right panel shows ECGs taken from the surface electrode plotted against that from the needle electrodes. The needle and surface electrodes record the same values.

3.3. Surface vs. epicardial ECG

The progression of the ECG as recorded by the surface electrode and epicardial electrodes is summarized in figure 3. The peak-to-peak amplitudes of the P, QRS, and T waves measured from the surface and epicardial electrodes are shown as a function of core temperature in figure 4. The ECG recorded from the epicardial leads are considerably larger than that recorded from surface electrodes, but still show a similar progression in the amplitudes of the ECG waves with decreasing

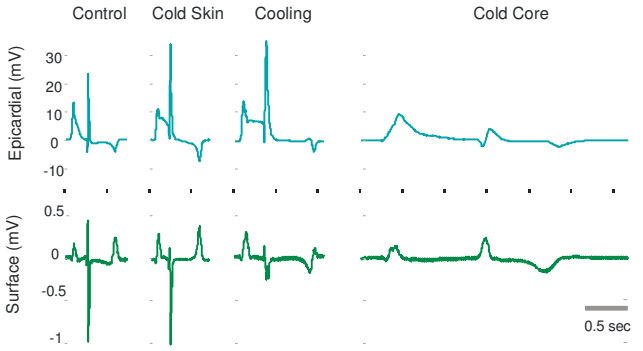

Figure 3. ECGs measured using epicardial (top) and surface (bottom) electrodes at control and 3 states of cooling. Note the lengthening of the ECG and significant decrease in the amplitude of the QRS complex.

temperature. It can be generally observed that the P-wave attenuates more slowly and persists at a lower core temperature than the other components. The QRS complex attenuates with decreasing core temperature and

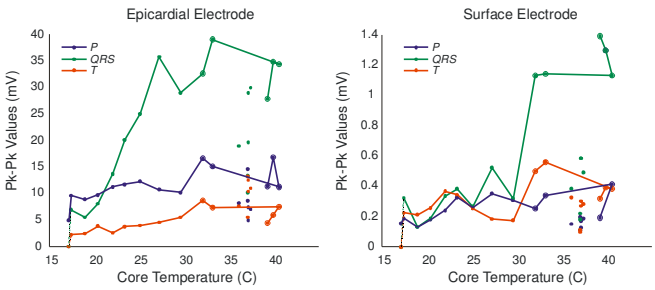

Figure 4. Peak to peak measures of the amplitude of the P, QRS and T waves for epicardial electrodes (left) and surface electrodes (right) as a function of core temperature.

is the ECG component that is most sensitive to temperature, possibly suggesting cold conduction block of ventricular activity. The amplitude of the QRS complex, however, appears to attenuate more rapidly in the surface electrode records starting at around 30ºc as compared to that of the epicardial electrode.

4. Discussion and conclusions

We were able to recreate most of the conditions of deep hypothermia in the acute animal preparation that allowed us to observe changes in the ECG reported in cases of deep hypothermia. Typically, depending upon the size of the animal, preparation spent approx 2 hours in

deep hypothermia < 30c. About half of the pigs could be resuscitated and eventually weaned from the ECC system.

The experiments attempted to model accidental hypothermia and to measure the changes in surface ECG during the course of inducing deep hypothermia and resuscitating the pig back. The animal was packed in ice during the procedure since we found that the ECC system cools the pig from the inside out. It is the opposite of the direction of heat loss in accidental hypothermia, where heat is lost to the environment and the body is cooled from the outside in. Further local cooling of the skin in the vicinity of the surface and needle electrodes to attempt to freeze the electrode and skin did not show an appreciable change in the ECG from the control measurement, nor was there a significant difference between the ECG measured with the surface electrodes or the needle electrode. These observations give weight to the suggestion that changes in electrode, skin or tissue impedance do not play a large role in the decreased ECG amplitude seen during accidental hypothermia.

Major changes in the ECG were only observed in relation to changes in the core temperature of the animal. These two factors combine to suggest that the decrease in the recorded ECG during deep hypothermia is related to changes in the electrical activity of the heart rather than changes in the extracellular current distribution within the body. These changes included the lengthening of the ECG, which could be reflecting slowing of the membrane channel dynamics and contractile properties of the cardiac muscle. We observed that the QRS complex is most sensitive to decreasing core temperature, though the RT interval remains generally unchanged. It suggests that during cooling, there is progressive conduction block taking place to progressively block ventricular contraction, perhaps at the AV node. However, we found with our setup that even though the amplitude of the ECG decreased, it was still observable down to the point where the heart slowed to cardiac arrest even with surface electrodes.

Acknowledgements

The authors would like to thank support for this work from the Hertha Christensen Foundation.

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