Moderate Hypothermia Attenuates Pro-Arrhythmic Electromechanical Relations in the Left Ventricle – A Clinical Study

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Research

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Abstract

Background

Targeted temperature management is recommended after cardiac arrest, but the beneficial effects are controversial. The recently published TTM2 study reports that arrhythmias causing hemodynamic compromise are more common during moderate hypothermia. The causation is not explored. Experimentally, moderate hypothermia attenuates electromechanical relations with pro-arrhythmic impact. Mechanical systole outlasts the electrical systole to a greater extent giving increased electromechanical window positivity, and dispersion of electrical and mechanical activity are unaltered. In this prospective clinical study, we explored the effect of moderate hypothermia on electromechanical relations in un-insulted left ventricles. We hypothesized that during moderate hypothermia, prolongation of systolic duration would exceed electrical duration without dispersed electrical- or mechanical activity.

Methods

20 patients with normal left ventricular function, undergoing surgery on the ascending aorta and connected to cardiopulmonary bypass, were included. Measurements were obtained at 36 °C and 32 °C prior to aortic-repair, and at 36 °C after repair at spontaneous and paced heart rate 90 bpm. Comparable loading conditions were ensured and cardiopulmonary bypass was reduced to 20% of estimated maximum during the measurements. Global cardiac function was measured invasively and with echocardiography. Electromechanical window, dispersion of repolarization by ECG and mechanical dispersion by echocardiography, were calculated.

Results

At moderate hypothermia (32°C), mechanical systolic prolongation exceeded electrical prolongation so that electromechanical window increased (29 ± 30 to 86 ± 50 ms, $p < 0.001$). Dispersion of repolarization and mechanical dispersion remained unchanged. Myocardial function was preserved with maintained strain, fractional shortening and stroke volume. Similar electromechanical relations were present also at comparable increased heart rate during moderate hypothermia. After rewarming to 36°C, electromechanical alterations were reversed.

Conclusion

Moderate hypothermia increased electromechanical window positivity. Dispersion of repolarisation, mechanical dispersion, and myocardial function were unchanged. Moderate hypothermia did not induce adverse electromechanical changes in the left ventricle during standardized conditions, but rather an attenuation of pro-arrhythmic electromechanical relations.

Background

Targeted temperature management (TTM: 32-36°C) has been recommended for neuroprotection in comatose cardiac arrest survivors the last two decades (1), but the optimal targeted temperature and beneficial effects have been unclear. Moderate hypothermia affects electrical and mechanical systole, with prolonged QRS duration, QT interval, and systolic mechanical contraction at spontaneous heart rate (HR) (2–6). Electrical and mechanical events are closely linked and changes in the electromechanical relations may increase arrhythmic susceptibility (7–10). Previous clinical data have reported corresponding incidence of ventricular arrhythmic events during treatment with 32°C versus 36°C in cardiac arrest survivors (6, 11–14). However, the recently published TTM2 trial concludes that arrhythmias causing hemodynamic compromise are more common during moderate hypothermia compared to normothermia (15). Whether this is due to changes in electrical and mechanical relations is unexplored.

During normal conditions at normothermia, electrical systole, i.e. QT interval, ends immediately before the end of mechanical systole (16) giving a time difference represented by a positive electromechanical window (17). Mechanical peak strain in left ventricular segments occurs almost simultaneously with minimal physiological mechanical dispersion (18). Prolongation of the QT interval is a risk factor for ventricular arrhythmias (19), especially if QT outlasts the mechanical systole with change to a negative electromechanical window. A negative electromechanical window is suggested to be a more sensitive independent predictor for arrhythmic events than the QT prolongation alone (20, 21). QT prolongation is associated with increased dispersion of electrical repolarization and mechanical dispersion, which both further increase arrhythmic susceptibility (8, 9). In experimental *in vitro* models, dispersion of repolarization is profoundly increased <30°C, but there is an electromechanical shift ≥32°C with reduced dispersion of repolarization and an arrhythmogenicity similar to 36°C (22, 23). In an *in vivo* model we have demonstrated that moderate hypothermia induced electromechanical alterations, where electromechanical window negativity changed to a positive value, dispersion of repolarization was reduced, and mechanical dispersion remained unaffected despite HR reduction and prolonged electrical and mechanical systole (24). These changes consolidate and attenuate arrhythmic susceptibility. Whether this hypothermic impact on the electromechanical relations is accommodating in patients, is unknown. Considering the TTM2 findings, it is of clinical relevance to explore how moderate hypothermia affects myocardial electromechanical parameters.

The aim of the present clinical study was to assess the isolated effects of moderate hypothermia on electromechanical relations in normal functioning, un-insulted human hearts in a controlled standardized clinical setting. The study had a unique design approximately equal to the experimental model applied in our previous animal study (24). We hypothesized that during moderate hypothermia at 32°C, left ventricle systolic mechanical prolongation would exceed electrical prolongation, without increased mechanical and electrical dispersion.

Methods

The study was designed to isolate the pure temperature effects on left ventricular function and electromechanical relations. The study protocol for global left ventricular function data has previously been reported in detail (25). Patients without evident cardiac dysfunction, scheduled for elective ascending aortic surgery were included. To minimize the occurrence of myocardial function heterogeneity and change in left ventricular loading conditions, exclusion criteria were ejection fraction <55%, previous myocardial infarction, atrial fibrillation or planned aortic valve surgery. Written informed consents for collecting and publishing data, were obtained for all patients and the study was approved by the Regional Committee for Medical and Health research Ethics, South-East Norway (2013/565 B).

Anaesthesia, technical instrumentation, and surgical procedures

The patients were pre-medicated with diazepam (5-10 mg). Anaesthesia was induced by intravenous (iv.) fentanyl (3.5-7.5 mg/kg), midazolam (0.05-0.15 mg/kg), thiopental (2.5-7.0 mg/kg) and cisatracurium (0.15 mg/kg) and maintained with sevoflurane-inhalation (1.0-2.5%) and repeated doses of iv. fentanyl (1-2 mg/kg). The patients were monitored according to the department's standardized protocol during aortic surgery. Three-lead electrocardiogram (ECG) was obtained by surface leads. Blood pressure was registered from an arterial line in the radial artery. Central venous catheter (Arrow International Inc., Reading. PA, USA), and pulmonary artery catheter (Swan-Ganz CCO; Edwards Lifesciences Corporation, Irvine, CA, USA) were inserted. After induction of anaesthesia, the patients underwent the initial surgery with sternotomy and pericardiotomy, before atrial pacemaker leads (Medtronic streamline, Medtronic Inc., Mn, USA)) were sutured on the right atrium. The patients were cannulated and connected to cardiopulmonary bypass (CPB), (Stöckert S5, Sarin Group Deutschland GmbH, Munich, Germany). During CPB, ventilation was discontinued, and sedation was provided with propofol infusion (3.5 mg/kg/h). All patients received thiopental (1g) and methylprednisolone (2g) prior to graft procedure. When suturing distal anastomosis the patients were cooled to deep hypothermia (28 °C). 18/20 patients underwent brief circulatory arrest in this phase. Ice cold cardioplegia was infused in all patients before suturing of the proximal anastomosis. After completed aortic repair and cardiac reperfusion, the patients were rewarmed and weaned off CPB.

Study protocol

All measurements were made while the patients were on cardiopulmonary bypass (CPB) with open thorax, to achieve optimal comparable conditions. The measurements were made at three time points (Fig.1): T1 at 36 °C defined as baseline, T2 at 32 °C prior to graft surgery defined as moderate hypothermia, and T3 at rewarming to 36 °C after aortic repair and cardiac reperfusion. As the core temperature dropped <37 °C during surgical preparations, 36 °C was chosen as baseline temperature to avoid excessive time spent on rewarming to normothermia and also in accordance with the targeted temperature management recommendations in the current resuscitation guidelines. All recordings were made at spontaneous HR, and at atrial paced HR 90 beats per minute (bpm) to compensate for individual HR variability, and to adjust for the recognized hypothermia-induced bradycardia, both which could influence electrical and mechanical systolic duration. Two patients had spontaneous HR ≥90 bpm and

where not paced at T1. One patient got atrial fibrillation at 32 °C whereof measurements at T2 were not included.

The surgical setting with CPB enabled standardization with accurate control of body temperature by a heat-exchanger connected to the CPB, and loading conditions. To obtain comparable and near-to-normal cardiac working conditions, CPB flow was carefully reduced to 20% of the estimated maximum flow, and loading conditions were made comparable by clamping venous drainage adjusted to mean arterial pressure (MAP) >50 mmHg and central venous pressure (CVP) ± 10% of baseline value. All measurements were performed during stable phase, with no change in anaesthesia or hemodynamic support. Surgical manipulation was paused during the measurements and between T1 and T2. Low dose norepinephrine (0.01 mg/kg/min) was used in two patients during T1 and T2, and low dose nitroprusside infusion (0.25-0.5 mg/kg/min) was continued in five patients at T3. These exceptions were controlled for and had no influence on statistical significance, thus data from all the 20 patients included are presented.

Transoesophageal echocardiographic recordings

A Vivid E95 scanner (GE Vingmed Ultrasound, Horten, Norway) was used for echocardiographic 2D and Pulsed Wave- (PW) Doppler recordings with a 5 MHz transesophageal echocardiographic probe (6VT-D, GE Vingmed Ultrasound, Horten, Norway). The recordings were obtained from mid-esophageal two- and four-chamber and long axis views, and transgastric short axis view at mid-papillary level. All recordings were analysed offline by designated software (EchoPac version 203, GE Healthcare, Horten, Norway). Measurements were made from three consecutive heart cycles and averaged. Four isolated measurements from segment 2 were for-shortened at T2 in three different patients at spontaneous heart rate; hence these measurements were excluded from the calculation of mechanical dispersion. Data were de-identified, and the investigator was blinded to patient ID and situation.

Calculations of the electrical events

The standardized electrocardiogram (ECG) lead II, was used for electrical measurements and was synchronized with the echocardiographic scanner (Fig.2). Electrical systole was represented by QT interval and measured from ECG onset-QRS to end of T-wave (Te), and this was also HR corrected (QTc) (26). The manual tangent method was used to determine Te, defined by the intersection of the isoelectric line with the tangent to the steepest downslope of the T-wave (27). Dispersion of repolarization was measured from the ECG T-wave as inter-individual variation in duration of the T-peak to T-end interval (TpTe). T-peak (Tp) was defined as the first maximum positive or negative deflection of the T-wave from the isoelectric line (28).

Calculation of mechanical events

Aortic- and mitral valvar opening and closing were registered from echocardiographic recordings (Fig.2). Ejection time (ET) was measured from aortic valve opening (AVO) to aortic valve closing

(AVC). Isovolumic contraction time (IVCT) and isovolumic relaxation time (IVRT) were measured from mitral valve closing (MVC) to AVO and AVC to mitral valve opening (MVO) respectively. Diastolic filling time was measured from MVO to MVC. Mechanical systole was defined by the interval from onset of QRS to AVC (QAVC). Electromechanical window was calculated as the difference between mechanical and electrical systole measured in the same beat (QAVC – QT).

Regional strain and mechanical dispersion

Speckle tracking echocardiography was used to obtain longitudinal strain in four-chamber, two-chamber and long axis views (29, 30) from 2D recordings with frame rate 52 ± 11 ms. Endocardial border was traced manually, and segments were adjusted to the myocardial thickness. Global longitudinal strain was derived from 18 segments and the peak systolic strain values were measured. Time to peak strain was defined from QRS-onset in ECG to peak longitudinal strain in each segment, (Fig.2). Mechanical dispersion was calculated as standard deviation of time to peak strain in the 18 segments model (9).

Global cardiac function

HR, MAP and CVP were obtained from the patients monitor (Siemens SC8000 patient monitor, Siemens Healthcare Erlangen, Germany). Cardiac output was measured from the pulmonary artery catheter by thermodilution with infusion of 10 mL 4 °C NaCl 9 mg/ml (Vigilance II, Edwards Lifesciences Corporation, Irvine, CA, USA), and cardiac index and stroke volume index were calculated. Fractional shortening was calculated from echocardiographic measured end-diastolic and end-systolic dimensions from short axis view, and systolic mitral ring peak velocity (s') from mitral ring tissue PW-Doppler registrations.

Statistical analyses

Sample size calculation was performed based on previous experimental results, to provide 80 % statistical power to identify ≥20 % change in left ventricular function with a two-sided alpha level of 0.05. Statistical analyses were calculated in SPSSv.26 software (SPSS, Inc., Chicago, IL, USA). Data are presented as mean ± standard deviation. One-way analysis of variance (ANOVA) for repeated measures was used to determine whether there were any statistical significant differences between the three sets of scores and comparisonsbetween time points T2 and T1, and T3 and T1, were performed by paired Student's *t* test. *P* value <0.05 was considered statistically significant, and post-hoc Bonferroni correction was done with alpha level 0.025.

Results

Twenty patients were included (Table 1). Moderate hypothermia (32°C) significantly reduced HR from 70 ± 14 to 53 ± 11 bpm, p <0.001. Rewarming reversed these changes (65 ± 11 bpm, p = 0.079).

Electromechanical relations at moderate hypothermia

At baseline (T1; 36 °C), electrical systole ended just before the mechanical systole, giving a positive electromechanical window (Fig.3). During moderate hypothermia (T2; 32 °C) duration of the electrical and mechanical systole both increased (Table 2). The mechanical prolongation exceeded the electrical prolongation, and electromechanical window became more positive in all patients (Fig.3). Dispersion of repolarization was unchanged during moderate hypothermia (Fig.3). Mechanical contraction pattern was altered, as time to peak systolic strain was prolonged and systolic mitral ring peak velocity was slowed (Table 3). However, mechanical dispersion was not significantly increased (Fig.3) and myocardial function was preserved (Table 3). All changes were evident also at comparable increased HR except deformation parameters. Fractional shortening, strain and indexed stroke volume, were reduced at increased HR during moderate hypothermia.

Electromechanical relations after rewarming

After rewarming (T3; 36 °C), both electrical and mechanical systole were shortened, but still prolonged compared to baseline. However, electromechanical window was reversed. (Fig.3). Dispersion of repolarization was slightly increased (Fig. 3). Time to peak systolic strain was partially reversed, but still prolonged compared to baseline (Table 3). Mechanical dispersion remained unchanged (Fig.3) and myocardial function was preserved (Table 3). The findings also corresponded after rewarming at comparable HR 90 bpm.

Discussion

This prospective clinical study explored the isolated effects from moderate hypothermia on electromechanical mechanisms in patients with normal cardiac function. Electromechanical window positivity increased at 32 °C, while dispersion of repolarization, mechanical dispersion and myocardial function remained unchanged. The electromechanical changes were reversed after rewarming. These results indicate that moderate hypothermia does not induce adverse electromechanical relations in the myocardium, but rather causes a consolidation and an attenuation of electromechanical arrhythmic susceptibility.

Electromechanical interactions represent a complex interplay. QT interval with negative electromechanical window is strongly associated with arrhythmic events (7). Nevertheless, QT prolongation during moderate hypothermia is not associated with increased ventricular arrhythmias or mortality (31, 32). In the present study we found prolonged QT interval during moderate hypothermia. However, mechanical systolic prolongation exceeded the electrical prolongation and the electromechanical window increased to a moderate positive value at 32 °C independent of HR. Our findings are consistent with experimental results describing increased electromechanical window at temperatures <36 °C (24, 33). Although the electromechanical window effect seems to have a U-shaped curve with increased arrhythmic risk at both negative and excessive positive values (34), clinical data strongly suggest that corresponding positivity reduces arrhythmic susceptibility (7). This moderate

temperature related increase in electromechanical window can be beneficial if persistent negative values after cardiac arrest.

Arrhythmias are primarily explained as electrical interference with mechanical impact. However a pathological electromechanical relationship may not solely be driven by electrical abnormalities. Both QT prolongation and structural changes in the myocardium may increase dispersion of repolarization and mechanical dispersion (22, 35). Changes in physiological or structural conditions may induce electromechanical heterogeneity and impair functional parameters. In patients with coronary artery disease, and after myocardial infarction, electrical and mechanical dispersion is often enhanced due to heterogeneity between healthy and dyskinetic myocardial tissue, which increases arrhythmogenicity (36, 37).

In the present clinical study, we found that despite hypothermia induced QT prolongation and changed contraction pattern, dispersion of repolarization was not increased, either at spontaneous or comparable increased HR. This is consistent with experimental and clinical studies on moderate hypothermia (22, 23, 38). In ischemic experimental models a direct temperature-related beneficial attenuation of arrhythmic susceptibility has been observed due to a reduction in transmural dispersion of repolarization and conduction-slowing with stabilized ionic-channel function and prevention of conduction block (5, 39). In our study moderate hypothermia prolonged and slowed myocardial contraction. This has previously been reported in experimental studies (4, 40). However, mechanical dispersion was only slightly and not significantly increased at spontaneous HR, and remained completely stable at comparable HRs. Despite the electrical and structural influences of the myocardium during moderate hypothermia, there was a consolidation of dispersion of repolarization and mechanical dispersion. This may indicate a preserved physiological contraction pattern and stable arrythmogenicity during moderate hypothermia.

After rewarming electrical and mechanical systole were still prolonged, but the interrelation was restored and the electromechanical window returned to baseline value. This indicates a rapid reversibility of the isolated temperature effect on electromechanical relations during moderate hypothermia.

Although the mechanisms are uncertain, we carefully suggest that the increased electromechanical window positivity during moderate hypothermia may provide a myocardial attenuation of arrhythmic risk rather than increased susceptibility. In addition, moderate hypothermia itself did neither increase dispersion of repolarization or mechanical dispersion, despite the QT prolongation and contraction slowing. In the TTM2 study an increased incidence of arrhythmias causing hemodynamic compromise was reported during moderate hypothermia (15), but the nature of arrhythmias was not further described. According to our findings electromechanical alterations in the myocardium do not explain the phenomenon. We found that moderate hypothermia induced electromechanical alterations which rather seem to be favourable concerning the risk for ventricular arrhythmias. This hypothermic effect could have clinical relevance in selected groups of patients after cardiac arrest. Electromechanical window registration is easily accessible bedside during echocardiographic assessment, and implementation of

the parameter in clinical practice may provide valuable information when choosing the optimal targeted temperature in cardiac arrest survivors.

Limitations

Some aspects of this study warrants caution. The open thorax may induce electrical changes and influence mechanical performance (41, 42). In addition, electromechanical relations may be affected of the surgical setting including cardioplegia, changes in autonomic tone (43), catecholamine levels (44), preoperative beta-blockade (7) and age (45). The sample size was restricted and represented a selected group of patients undergoing surgery on the ascending aorta. However, these aspects were compensated by the advantageous study model closely approximating an experimental model. All measurements were performed during comparable conditions including HR pacing, at all time-points. Thus each patient acted as its own control. The aim of the study was to elucidate the isolated effects of moderate hypothermia whereof measurements were performed in a population without overt cardiac disease. However, we cannot exclude that the changes in electromechanical relations may act differently in hypothermic patients post cardiac arrest.

Conclusions

In patients with normal left ventricular function, moderate hypothermia increased electromechanical window positivity without induced change in dispersion of repolarization, mechanical dispersion, or myocardial function. These results indicate that moderate hypothermia itself does not induce pro-arrhythmic adverse electromechanical changes in the left ventricle despite prolonged electrical and mechanical systole. The increased electromechanical window positivity is rather suggested to attenuate pro-arrhythmic electromechanical relations which may be beneficial in selected groups of patients after cardiac arrest.

Abbreviations

AVO=Aortic valve opening

AVC=Aortic valve closing

Bpm=beats per minute

CPB=Coronarypulmonary bypass

CVP=Central Venous Pressure

ECG= Electrocardiogram

HR= Heart rate

LV=left ventricle

MAP=Mean arterial pressure

MVO=Mitral valve opening

MVC=Mitral valve closing

TTM=Targeted Temperature Management

IVCT=Isovolumic contraction time

IVRT=Isovolumic relaxation time

Declarations

- Ethics approval and consent to participate: Yes
- Consent for publication: Yes
- The datasets used and analysed during the current study are available from the corresponding author on reasonable request.
- The authors declare that they have no competing interests
- There was no external funding for the research reported
- All authors have contributed substantially in conducting the underlying research and drafting this manuscript. All authors read and approved the final manuscript.

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Tables

Table 1. Patient Characteristics and General Methodology

Variable	Value
Male /Female (n)	12 / 8
Age (year)	63 ± 14
Body mass index (kg/m^2)	26 ± 3.3
Beta-blockade (n)	8
Time per measurement (min)	7 ± 2
Time, cooling from T1; 36° to T2; 32° (min)	10 ± 3
Duration of hypothermia < 36 °C (min)	81 ± 24
Total cardiopulmonary bypass time (min)	127 ± 26
Cross-clamp time (min)	48 ± 23
Surgery time (min)	181 ± 28

Data expressed as Mean ± SD and numerical (n).

Table 2. Electrical and Mechanical Parameters

	Spontaneous Heart Rate			Heart Rate 90 Beats per Minute		
Variable	**36 °C**	**32 °C**	**36 °C Post**	**36 °C**	**32 °C**	**36 °C Post**
R-R interval (ms)	862 ± 170	1156 ± 254	937 ± 176	662 ± 29	669 ± 18	666 ± 5
p		<0.001	0.025*		0.164	0.347
QRS-complex (ms)	63 ± 4	68 ± 5	67 ± 9	62 ± 5	66 ± 5	65 ± 8
p		<0.001	0.006		<0.001	0.005
QT-interval (ms)	397 ± 49	497 ± 79	429 ± 68	353 ± 31	391 ± 42	381 ± 36
p		<0.001	<0.001		<0.001	<0.001
QTc-interval (ms)	431 ± 46	463 ± 45	449 ± 63	434 ± 39	478 ± 52	467 ± 44
p		<0.001	0.021		<0.001	<0.001
QAVO (ms)	130 ± 24	141 ± 19	138 ± 23	129 ± 20	145 ± 20	143 ± 26
p		0.224	0.114		0.098	0.065
QAVC (ms)	425 ± 43	588 ± 67	465 ± 36	391 ± 33	470 ± 47	412 ± 41
p		<0.001	<0.001		<0.001	0.038*
Isovolumic contraction time (ms)	46 ± 22	45 ± 16	43 ± 25	42 ± 21	44 ± 17	53 ± 26
p		0.603	0.862		0.560	0.363
Ejection time (ms)	299 ± 38	450 ± 71	326 ± 29	261 ± 35	320 ± 43	267 ± 42
p		<0.001	0.004		<0.001	0.304

Data expressed as Mean ± SD, $p < 0.05$ is considered significant. * represents $p \geq 0.025$ after Bonferroni correction. *P*-value represents comparison between groups: at baseline 36 °C versus moderate

hypothermia 32 °C, and after rewarming post-surgery at 36 °C versus baseline 36 °C, at spontaneous and increased heart rate 90 beats per minute.

Table 3. Echocardiographic Functional Parameters

	Spontaneous Heart Rate			Heart Rate 90 Beats per Minute		
Variable	**36 °C**	**32 °C**	**36 °C Post**	**36 °C**	**32 °C**	**36 °C Post**
Global longitudinal strain (%)	16 ± 9	14 ± 10	16 ± 9	15 ± 9	10 ± 7	16 ± 22
p		0.078	0.546		<0.001	0.214
Time to peak systolic strain (ms)	395 ± 58	521 ± 90	421 ± 55	352 ± 52	390 ± 78	378 ± 51
p		<0.001	<0.002		<0.001	<0.001
Systolic mitral ring peak velocity (s') (cm/s)	4.8 ± 1.3	3.3 ± 0.9	3.9 ± 1.1	4.9 ± 1.3	4.2 ± 1.3	3.9 ± 1.1
p		<0.001	0.012		0.141	0.152
Fractional Shortening (%)	38 ± 8	42 ± 13	41 ± 11	34 ± 9	36 ± 9	40 ± 9
p		0.033*	0.251		0.402	0.292
Stroke volume index ($mL/beat/cm^5$)	33 ± 9	34 ± 14	35 ± 7	27 ± 7	22 ± 10	26 ± 6
p		0.798	0.609		<0.004	0.273
Cardiac index ($L/min/m^{2)}$	2.3 ± 0.6	1.8 ± 0.7	2.3 ± 0.5	2.4 ± 0.5	2.0 ± 0.8	2.3 ± 0.5
p		0.004	0.686		0.005	0.183

Data expressed as Mean ± SD, $p \leq 0.05$ is considered significant. * represents $p \geq 0.025$ after Bonferroni correction. *P*-value represents comparison between groups: at baseline 36 °C versus moderate hypothermia 32 °C, and after rewarming post-surgery at 36 °C versus baseline 36 °C, at spontaneous and increased heart rate 90 beats per minute.

Figures

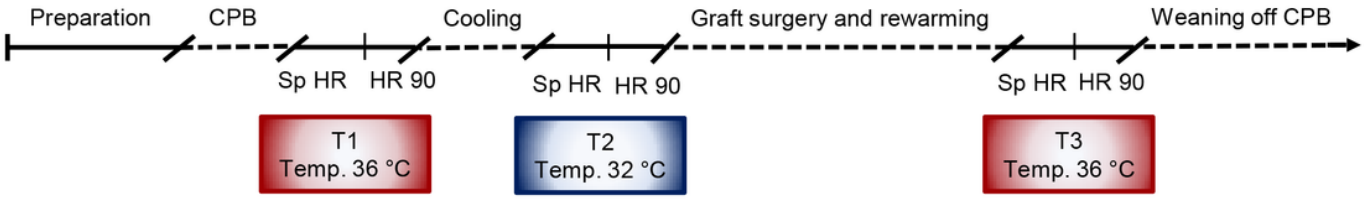

Figure 1

Timeline showing procedural sequence in relation to temperature and measurements at T1, T2 and T3. CPB; cardiopulmonary bypass, Sp HR; spontaneous heart rate, HR 90; atrial paced heart rate 90 beats per minute.

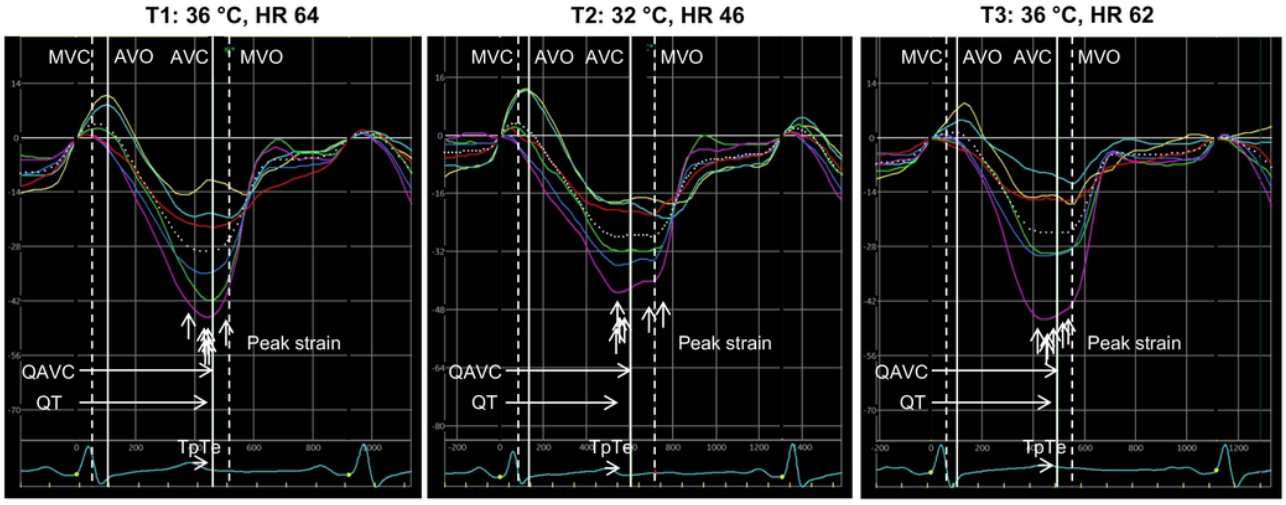

Figure 2

Longitudinal strain by speckle tracking echocardiography in apical long axis view at baseline: T1 (36 °C), at moderate hypothermia: T2 (32 °C) and after rewarming: T3 (36 °C), at spontaneous heart rate (HR).

White lines indicate aortic valve opening (AVO) and closing (AVC), and white dotted lines mitral valve opening (MVO) and closing (MVC). QAVC, QT-interval and TpTe are marked. Electromechanical window is represented by the difference QAVC-QT, dispersion of repolarization by the inter-individual variance in TpTe, and mechanical dispersion as variation in time to peak strain, marked with white vertical arrows. Electromechanical window positivity is increased, while dispersion of repolarization and mechanical dispersion are unchanged at moderate hypothermia. Electromechanical window is returned to baseline value after rewarming.

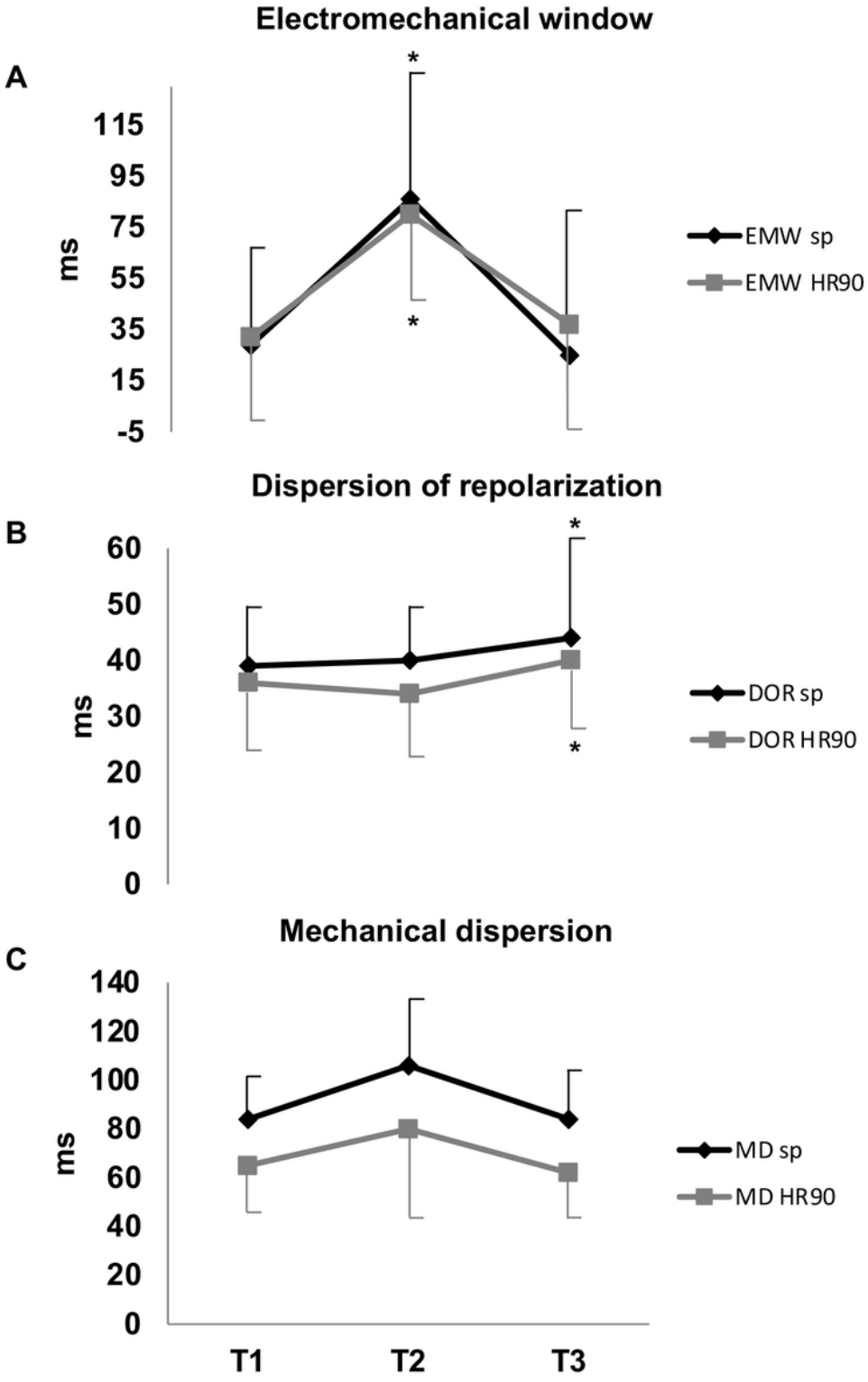

Figure 3

Electromechanical relations at baseline: T1 (36 °C), at moderate hypothermia: T2 (32 °C) and after rewarming: T3 (36 °C), at spontaneous heart rate and heart rate 90 beats per minute respectively. A, Electromechanical window (EMW); B, Dispersion of repolarization (DOR) and C, Mechanical dispersion (MD). Mean values are presented with standard deviation. □ represents estimates and * denotes significant interaction between groups: T2 versus T1, and T3 versus T1.

Supplementary Files

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