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RESEARCH PAPER

Effects of peripheral cold application on core body temperature and haemodynamic parameters in febrile patients

Hossein Asgar Pour RN PhD

Lecturer, Doctor, Department of Surgical Nursing, Aydin Health School, Adnan Menderes University, Aydin, Turkey

Meryem Yavuz RN PhD

Professor, Lecturer, Department of Surgical Nursing, School of Nursing, Ege University, Izmir, Turkey

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This study designed to assess the effects of peripheral cold application (PCA) on core body temperature and haemodynamic parameters in febrile patients. This study was an experimental, repeated-measures performed in the neurosurgical intensive-care unit. The research sample included all patients with fever in postoperative period. PCA was performed for 20 min. During fever, systolic blood pressure, mean arterial blood pressure and arterial oxygen saturation (O_2Sat) decreased by 5.07 ± 7.89 mm Hg, 0.191 ± 6.00 mm Hg and 0.742% ± 0.97%, respectively, whereas the pulse rate and diastolic blood pressure increased by 8.528 ± 4.42 beats/ min and 1.842 ± 6.9 mmHg, respectively. Immediately after PCA, core body temperature and pulse rate decreased by 0.3°C, 3.3 beats/min, respectively, whereas systolic, diastolic, mean arterial blood pressure and O_2Sat increased by, 1.40 mm Hg, 1.87 mm Hg, 0.98 mmHg and 0.27%, respectively. Thirty minutes after the end of PCA, core body temperature, diastolic, mean arterial blood pressure and pulse rate decreased by 0.57°C, 0.34 mm Hg, 0.60 mm Hg and 4.5 beats/min, respectively, whereas systolic blood pressure and O_2Sat increased by 0.98 mm Hg and 0.04%, respectively. The present results showed that PCA increases systolic, diastolic, mean arterial blood pressure and O_2Sat, and decreases core body temperature and pulse rate.

Key words: fever, haemodynamic parameters, peripheral cold application.

INTRODUCTION

Monitoring and evaluating haemodynamic parameters (systolic/diastolic blood pressure, mean arterial blood pressure, pulse rate and arterial oxygen saturation) are important nursing initiatives, particularly for intensive-care unit (ICU) patients.[1–3] Additionally, core body temperature (CBT), arterial blood pressure, pulse, respiration and pain are basic vital signs and indicators of an individual's health status. Changes in physiological functions are reflected in the values of an individual's basic vital signs. Therefore, deviations from the normal values of vital signs indicate the disruption of homeostasis.[4] Measuring vital signs is an effective method of monitoring health status and assessing emerging issues. Accurately assessing vital signs plays an important role in patient

Correspondence: Hossein Asgar Pour, Adnan Menderes Universitesi, Aydin Saglik Yuksekokulu, Cerrahi Anabilim Dali, Aydin 09000, Turkey. Email: hasgarpour23@yahoo.com

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diagnosis and the delivery of accurate and appropriate interventions in nursing care.[5–7] The diagnostic value of standard monitoring parameters is high when these values are abnormal, as they are considered sensitive indices of the overall health of patients.[8–11] Measuring and monitoring vital signs are simple nursing skills, but nurses need to know how variations in vital signs affect haemodynamic parameters and the relationship between them. Furthermore, they need to know the nursing initiatives related to these changes.[10,12–14]

Fever is an adaptive response to a variety of infectious, inflammatory and foreign stimuli. The febrile response confers an immunological advantage to the host over invading microorganisms. Fever results from a cytokine-mediated reaction that results in the generation of acute phase reactants and controlled elevation of CBT.[15] Fever is defined as an increase in the CBT ≥ 38.3°C attributed to the upregulation of the thermostatic setpoint, which is controlled by the hypothalamus.[10,11,16] The incidence of fever ranges between 28% and 75% in critically ill patients, and fever has an infection and non-infectious causes.[11,17,18] Febrile episodes occur in roughly 50% of patients in the neurosurgical ICU. Fever in the neurocritical and neurosurgical patient population predominates with vascular injuries, such as intracerebral hemorrhage and subarachnoid hemorrhage. The highest rates of febrile episode occur in patients with subarachnoid hemorrhage (65%), followed by traumatic brain injury (40%) and intracranial hemorrhage (31%); no cause of fever was identified in 28% of patients, suggesting fever of central origin.[15]

CBT increase from 37 to 39°C has been found to be followed by a 25% increase of oxygen consumption and energy expenditure in ICU patients.[11] These increases in the metabolic rate and serum levels of stress hormones are suggested to subsequently increase heart rate and arterial blood pressure in patients. Conversely, CBT increases can result in hypotension due to myocardial depression and vasodilation, especially in the veins of the kidneys, liver, skin, and upper and lower limbs.[5,11,19] Studies on increases in CBT and changes in haemodynamic parameters, that is arterial blood pressure, pulse rate and arterial oxygen saturation, are insufficient.[11] Although, different methods are used to reduce CBT in patients with fever, little research related to the effects of these methods on CBT and haemodynamic parameters have been performed.[20,21] Having a lack of sufficient resources and adding to new temperature measurement methods everyday makes the above diagnosis difficult. In this study, diagnosing the effects of fever and peripheral cold application (PCA) on haemodynamic parameters, which are the basic responsibilities of nurses, will be provided in the literature. PCA is a common, easy, non-invasive and inexpensive method that is usually provided as the first treatment option for febrile patients for 15–20 min. Shivering and other complications such as tissue ischemia can occur if PCA takes longer than 20 min, and it can have a direct effect on haemodynamic parameters.[2,3] The aim of this study was to investigate the effects of PCA on CBT and haemodynamic parameters in febrile patients.

MATERIALS AND METHODS

Design and participants

This repeated-measure, experimental study was conducted between 26 June 2009 and 16 May 2010 in the neurosurgical ICU at Ege University Hospital, Izmir, Turkey. The selection of samples was actualized in two periods with the convenience sampling method.

The inclusion criteria were as follows:

- Voluntary participation in the study
- No incidence of fever in the preoperative and operative periods
- At least two episodes of fever in the postoperative period
- Operation occurred at least 4 h prior to enrollment
- Do not receive any inotrope or cardiotonic agents during postoperative period.

A sample size of 20 achieves 99% power to detect a difference of −0.24143 between the null hypothesis mean of 0.99 and the alternative hypothesis mean of 0.24143 with an estimated standard deviation of 0.20018 and with a significance level (alpha) of 0.05000 using a two-sided Wilcoxon test assuming that the actual distribution is normal. According to the results of the power analysis, with power 0.99, a selection of 20 samples was sufficient (Fig. 1).

The research sample comprised of 35 patients who met the inclusion criteria, and the authors selected 35 samples in two periods. From all patients (n = 364) who were admitted at the neurosurgical ICU in postoperative period, 49 of the patients developed a fever for the first time and were eligible for enrollment in the first stage of sampling. PCA was performed for each patient, and CBT and haemodynamic parameters were measured during fever in the first stage of study. In the second stage of sampling, 14 of 49 samples did not enter the second stage

of the study. So, PCA was performed for 35 of 49 samples who developed a fever for second time and the same transactions of first stage repeated (Fig. 2).

Ethical approval

This study was approved by Ege University Institutional Review Board, and informed consent (written and verbal) was obtained from all participants or their relatives after explaining the aims and protocol of the study. Written approvals were obtained from the Institute of Health Sciences of Ege University (12 March 2009), Ethics Board of The Ege University School of Nursing (28 May 2009), the Ege University Hospital, and Neurosurgical Department Chief Physician (12 June 2009) and Directorate of Nursing Services of The Ege University Hospital (25 June 2009).

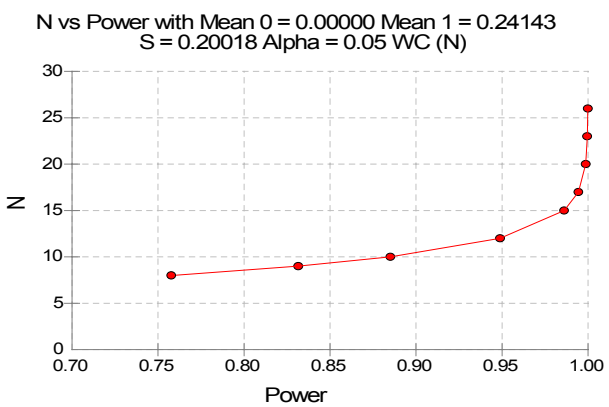

Figure 1. Numeric results for Wilcoxon test.

Pilot study

According to the results of a study by Klein and *et al.*, tympanic membrane temperature has been shown to be strongly correlated with pulmonary artery temperature ($r = 0.91$), whereas their mean difference is small ($0.07 \pm 0.41°C$), thus it was considered to properly represent core temperature.[22] In the present study, tympanic membrane temperature was considered as CBT. In order to determine the relationship between tympanic membrane and axillary temperature, a pilot study was conducted by Kiekkas *et al.* which the mean difference between tympanic membrane and axillary temperatures was not significantly differ when tympanic membrane temperature value was above or below 38.3°C. Simple linear regression was then used to estimate tympanic membrane temperature values on the basis of axillary temperature values. Pearson's correlation coefficient was 0.98 ($P < 0.001$), whereas the coefficient of determination was 0.96 ($P < 0.001$). The regression equation derived was used for the adjustment of axillary temperature values to core temperature ones:

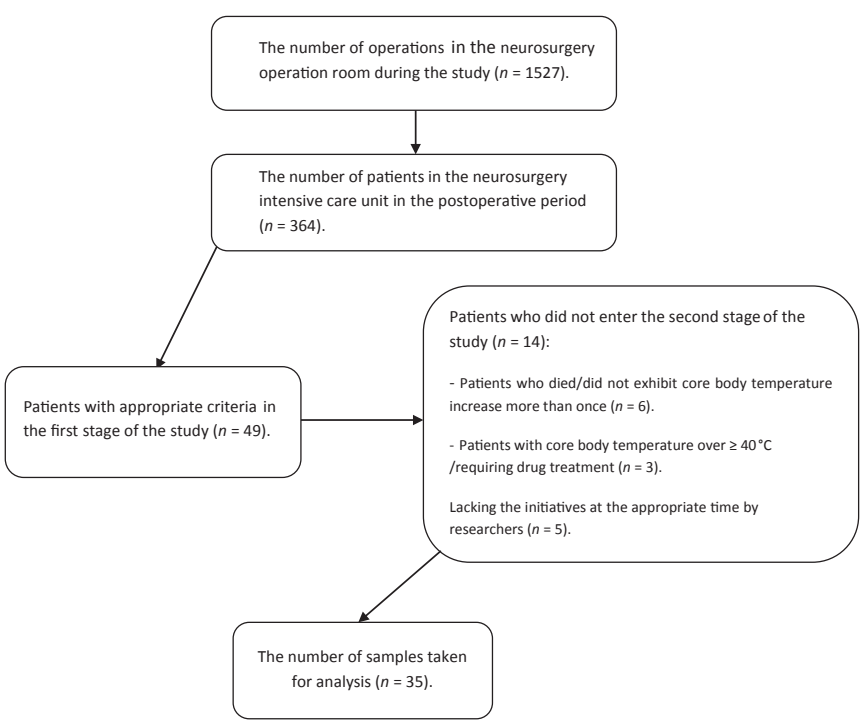

Figure 2. The stages of sample selection.

tympanic membrane temperature (°C) = 0.889 + 0.982 × axillary temperature (°C).[11]

Data collection instrumental

Axillary and tympanic membrane temperatures were used to measure the CBT. To measure CBT via the axilla, mercury-glass thermometer and tympanic membrane, a commercial infrared tympanic thermometer (Felix, Thermo-Scan Thermometer 952; Hubdic Co. Ltd., Gyeonggi-do, South Korea), haemodynamic parameter non-invasive monitoring system (Bruker TMSN – 910CD/Ni12v1.9 Ah cable; EMCO 4950 MPM; Emco Meditek, Gujarat, India), and peripheral dry cold application materials (ice packs placed at a total of 7 points (24 × 12 cm) and covers and soft cloths) were used. Calibration of the commercial infrared tympanic thermometer and non-invasive monitoring system before the study and each 6 months were performed by the relevant company. Measurements of CBT via an axillary method and systolic/diastolic blood pressure, mean arterial blood pressure, pulse rate and arterial oxygen saturation by a monitor were performed at admission (preoperative period).

Data collection

In this study fever was defined as an increase of CBT ≥ 38.3°C. CBT and haemodynamic parameters were measured at 1 h intervals by recording on the ICU daily chart in admission and postoperative period. When CBT was >37.8°C, haemodynamic parameters values were measured and recorded every 15 min to the study questionnaire. Patients were placed in semi-Fowler's position when fever was manifested during postoperative period. The axillary, groin, abdomen and below-knee regions were scrubbed with a clean-dry cloth and checked for open sores and erythema before PCA. Ice packs were placed on the cleared regions for 20 min. Immediately after PCA and 30 min after the end of PCA, CBT of the tympanic membrane and haemodynamic parameters were measured and recorded. The regions that were subjected to PCA were examined in terms of redness. In the second stage of this study, PCA was also provided to patients during the second incidence of fever and the same transactions repeated. Therefore, PCA was performed at two different times for each patient. Then, data measured at both times were analyzed for each patient, and the means were calculated. During the process, the temperature of the ICU was measured and recorded.

Data analysis

The data analysis was performed by the University Biostatistics and Medical Informatics Main Field of Study by using SPSS version 16.0 for Windows statistics program (IBM, Chicago, IL, USA). Data analyses were performed for all participants who completed the study. Friedman and Wilcoxon signed-rank tests were used to check the normality of distribution of continuous variables. Data are presented as mean (standard deviation). To compare standard monitoring parameters at admission, 1 h before the onset of fever, and before, immediately after, and 30 min after the end of PCA paired samples *t*-test was used. Statistical significance was set at $P < 0.05$.

RESULTS

Thirty-five patients with at least two episodes of increased CBT were included in this study. The mean age of patients was 49.71 ± 17.65 years, and 62.9% patients were men. Regarding chronic disease, 31.4% patients had hypertension, and 5.7% had diabetes mellitus. Regarding disease diagnosis, 68.6% patients had brain hemorrhage, and the mean operation time was 3.85 ± 0.68 h. Fever appeared in 51.4% patients after the third day of operation. During fever, 62.9% patients were semi-conscious, and 54.2% exhibited no infections. Furthermore, the average temperature of the ICU was 24.3°C ± 1.32°C.

The average CBT at admission was 37.30°C ± 0.47°C, compared with 37.37°C ± 0.34°C 1 h before the onset of fever ($P = 0.515$). The average CBT before PCA was 38.51°C ± 0.27°C. Compared with the temperature 1 h before the onset of fever, a significant increase of 1.144°C ± 0.38°C was observed before PCA ($P = 0.001$). The average CBT immediately after PCA was 38.19°C ± 0.33°C. Compared with the CBT before PCA, a significant decrease of 0.328°C ± 0.24°C was observed immediately after PCA ($P = 0.001$). The average CBT 30 min after the end of PCA was 37.94 ± 0.41°C. Compared with the CBT before PCA, a significant decrease of 0.570°C ± 0.29°C was observed 30 min after the end of PCA ($P = 0.001$) (Fig. 3).

The average pulse rate at admission was 89.8 ± 17.8 beats/min, compared with 93.4 ± 17.6 beats/min 1 h before the onset of fever ($P = 0.383$). The average pulse rate before PCA was 102.0 ± 20.8 beats/min. Compared with the pulse rate 1 h before the onset of fever, a significant increase of 8.528 ± 4.42 beats/min was observed before PCA ($P = 0.001$). The average pulse

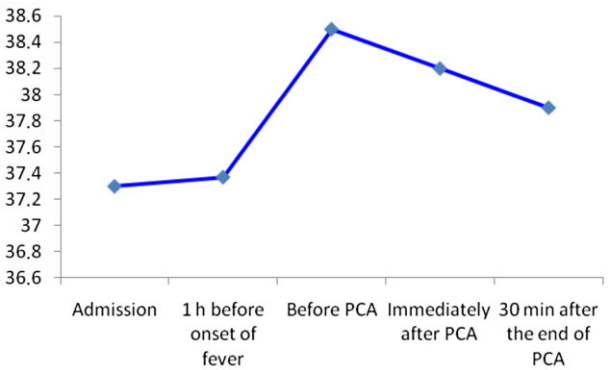

Figure 3. Distributions of CBT at admission, 1 h before the onset of fever, before PCA, immediately after PCA and 30 min after the end of PCA.

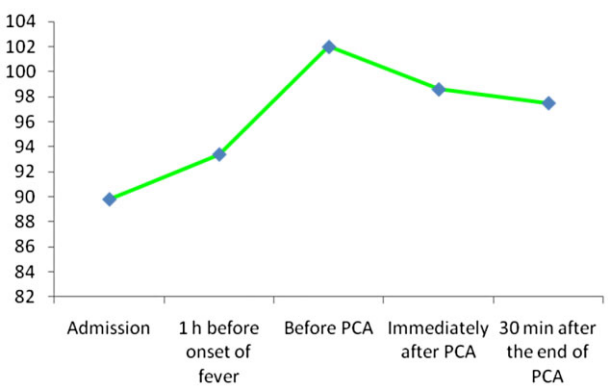

Figure 4. Distributions of pulse rate at admission, 1 h before the onset of fever, before PCA, immediately after PCA and 30 min after the end of PCA.

rate immediately after PCA was 98.6 ± 21.1 beats/min. Compared with the pulse rate before PCA, a significant decrease of 3.385 ± 5.84 beats/min was observed immediately after PCA ($P = 0.002$). The average pulse rate 30 min after the end of PCA was 97.5 ± 20.2 beats/min. Compared with the pulse rate before PCA, a significant decrease of 4.50 ± 8.82 beats/min was observed 30 min after the end of PCA ($P = 0.005$) (Fig. 4).

The average arterial oxygen saturation at admission was 96.4% ± 2.3%, compared with 96.01% ± 2.11% 1 h before the onset of fever ($P = 0.433$). The average arterial oxygen saturation before PCA was 95.2% ± 2.7%. Compared with the arterial oxygen saturation 1 h before the onset of fever, a significant decrease of 0.742% ± 0.97% was observed before PCA ($P = 0.001$). The average arterial oxygen saturation immediately after PCA was 95.5% ± 2.4%. Compared with the arterial

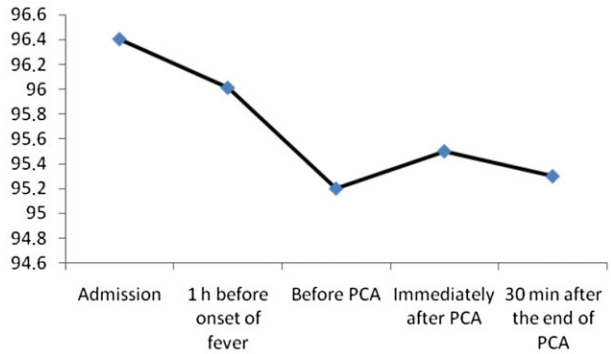

Figure 5. Distributions of arterial oxygen saturation at admission, 1 h before the onset of fever, before PCA, immediately after PCA and 30 min after the end of PCA.

oxygen saturation before PCA, an insignificant increase of 0.271% ± 1.13% was observed immediately after PCA ($P = 0.166$). The average arterial oxygen saturation 30 min after the end of PCA was 95.3% ± 3.1%. Compared with the arterial oxygen saturation PCA, an insignificant increase of 0.042% ± 1.99% was observed 30 min after the end of PCA ($P = 0.900$) (Fig. 5).

The average systolic blood pressure at admission was 135.2 ± 27.5 mm Hg, compared with 132.9 ± 14.5 mm Hg 1 h before the onset of fever ($P = 0.678$). The average systolic blood pressure before PCA was 127.8 ± 15.7 mm Hg. Compared with the systolic blood pressure 1 h before the onset of fever, a significant increase of 5.07 ± 7.89 mm Hg was observed before PCA ($P = 0.002$). The average systolic arterial blood pressure immediately after PCA was 129.2 ± 17.8 mm Hg. Compared with the systolic arterial blood pressure before PCA, an insignificant increase of 1.40 ± 8.72 mm Hg was observed immediately after PCA ($P = 0.349$).The average systolic blood pressure 30 min after the end of PCA was 128.8 ± 19.5 mm Hg. Compared with the systolic blood pressure before PCA, an insignificant increase of 0.985 ± 11.89 mm Hg was observed 30 min after the end of PCA ($P = 0.627$) (Fig. 6).

The average diastolic blood pressure at admission was 79.1 ± 19.3 mm Hg, compared with 77.2 ± 11.6 mm Hg 1 h before the onset of fever ($P = 0.610$). The average diastolic blood pressure before PCA was 79.1 ± 12.5 mm Hg. Compared with the diastolic blood pressure 1 h before the onset of fever, an insignificant increase of 1.842 ± 6.9 mm Hg was observed before PCA ($P = 0.074$). The average diastolic blood pressure immediately after PCA was 81.0 ± 11.4 mm Hg. Compared with the

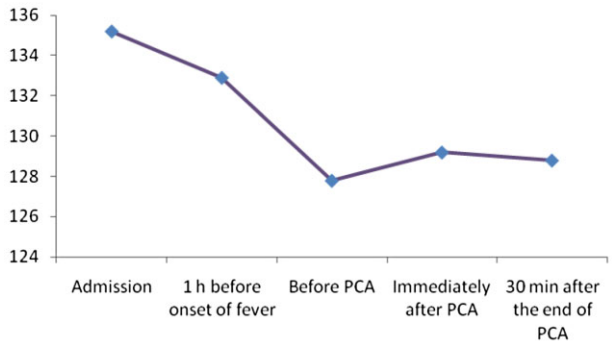

Figure 6. Distributions of systolic blood pressure at admission, 1 h before the onset of fever, before PCA, immediately after PCA and 30 min after the end PCA.

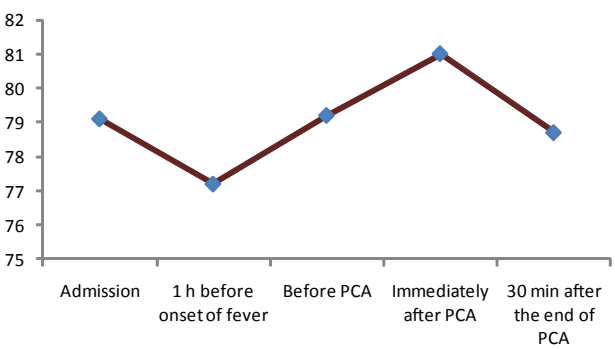

Figure 7. Distributions of diastolic blood pressure at admission, 1 h before the onset of fever, before PCA, immediately after PCA and 30 min after the end of PCA.

diastolic blood pressure before PCA, an insignificant increase of 1.871 ± 8.00 mm Hg was observed immediately after PCA ($P = 0.176$). The average diastolic blood pressure 30 min after the end of PCA was 78.7 ± 12.3 mm Hg. Compared with the diastolic blood pressure before PCA, an insignificant decrease of 0.342 ± 9.52 mm Hg was observed 30 min after the end of PCA ($P = 0.833$) (Fig. 7).

The average mean arterial blood pressure at admission was 97.8 ± 21.2 mm Hg, compared with 95.7 ± 12.02 mm Hg before the onset of fever ($P = 0.624$). The average mean arterial blood pressure before PCA was 95.1 ± 12.7 mm Hg. Compared with the arterial blood pressure 1 h before the onset of fever, an insignificant decrease of 0.191 ± 6.00 mm Hg was observed before PCA ($P = 0.973$). The average mean arterial blood pressure immediately after PCA was 96.5 ± 13.6 mm Hg. Compared with the arterial blood pressure before PCA,

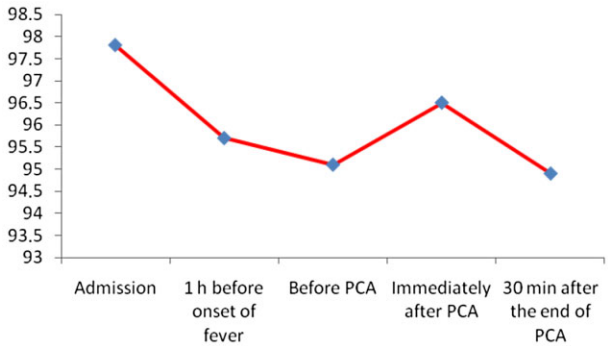

Figure 8. Distributions of mean arterial blood pressure at admission, 1 h before the onset of fever, before PCA, immediately after PCA and 30 min after the end of PCA.

an insignificant increase of 0.984 ± 7.13 mm Hg was observed immediately after PCA ($P = 0.420$). The average mean arterial blood pressure 30 min after the end of PCA was 94.9 ± 14.5 mm Hg. Compared with the mean arterial blood pressure before PCA, an insignificant decrease of 0.607 ± 8.90 mm Hg was observed 30 min after the end of PCA ($P = 0.689$) (Fig. 8).

DISCUSSION

Standard monitoring of ICU patients includes the observation of electrocardiogram, heart rate, arterial blood pressure and arterial oxygen saturation.[8,11] The diagnostic value of monitoring standard parameters is very important, as these are considered sensitive indicators of the overall health of patients.[9–11] During fever, increases in the metabolic rate and serum levels of stress hormones (cortisol and norepinephrine) are thought to subsequently increase the pulse rate and arterial blood pressure. In contrast, body temperature increases can result in hypotension due to myocardial depression and vasodilation.[11,14,23] Subsequent increases in oxygen consumption, respiratory quotient and cardiac output add a considerable burden to these patients, who might be unable to compensate for the increased metabolic demand.[13,18,24] In a study of the adverse effects of fever on cardiac performance, evidence of decreased left ventricular performance and increased heart rate were observed during the febrile episode. Because all patients maintained a normal or high cardiac index in association with increases in heart rate during the febrile state, the tachycardic response of the febrile patient can serve to maintain cardiac output when myocardial performance is impaired.[25] In a study of fever and haemodynamic parameters in ICU patients,

body temperature was increased due to decreases in systolic blood pressure and arterial oxygen saturation and an increase in the pulse rate. As a result, a 1°C increase in CBT, a 4.7 beats/ min increase in heart rate, a 2.7 mm Hg decrease in systolic blood pressure and a 0.4% decrease in arterial oxygen saturation were observed.[11] In the present study, the increase in CBT was caused by decreases in systolic blood pressure, mean arterial pressure and arterial oxygen saturation, and increases in diastolic blood pressure and pulse rate. According to these results, changes in haemodynamic parameters occur during elevations in CBT. In this study, a degree celsius increase in CBT, decreased by 4.43 mm Hg in systolic blood pressure, 0.166 mm Hg mean arterial blood pressure and 0.64% arterial oxygen saturation, and increase of 1.61 mm Hg in diastolic blood pressure and 7.46 beats/ min pulse rate.

To prevent complications regarding these changes, interventions to reduce the CBT are important during fever. Although different methods are used to reduce CBT in patients with fever, little research regarding the effects of these methods on CBT and haemodynamic parameters have been performed.[20,26] In the only study in which patient body temperature returned to normal after a fever episode, a significant decrease in the heart rate was combined with a significant increase in mean arterial blood pressure. Of importance was that when the fever was infectious, the decrease in heart rate after the temperature returned to normal was greater than that when no infection was present. On the contrary, the increase in mean arterial blood pressure was greater when the fever was non-infectious. Therefore, changes in CBT and haemodynamic parameters occur when the metabolic rate is depressed.[25] PCA is a common method that is usually provided as the first treatment option for febrile patients for 15–20 min. Shivering might occur if PCA takes longer than 20 min, and it can have a direct effect on haemodynamic parameters. In PCA, ice packs or ice bags are placed on the axillae, groins and below the knees.[2,3] In this study, the authors used PCA for 20 min, and no shivering was observed during and after PCA.

In the present study, immediately after PCA, CBT decreased by 0.3°C, and pulse rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and arterial oxygen saturation increased by 3.3 beats/ min, 1.40 mm Hg, 1.87 mm Hg, 0.98 mmHg and 0.27%, respectively. Thirty minutes after PCA, CBT, diastolic blood pressure, mean arterial blood pressure and pulse rate decreased by 0.57°C, 0.34 mm Hg, 0.60 mm Hg and 4.5 beats/min, respectively, whereas systolic blood pressure and arterial oxygen saturation increased by 0.98 mm Hg and 0.04%, respectively.

Consequently, knowledge about the effects of fever on haemodynamic parameters will be of a benefit to nurses in terms of quality and efficiency of care. Thus, accurate and careful measurements of haemodynamic parameters play an important role in preventing complications before, during and after interventions related to the fever.[10,13,16,27]

STUDY LIMITATION

One of the limitations of this study was that it investigated neurosurgery patients in the postoperative period. For practical reasons, the present study assessed only the small amount patient. The result cannot generalize. Another limitation of the present study was that the results were based on only 35 patients' assessment. We expect that some results will change if more patients have participated in further assessments. Furthermore, standard monitoring parameters are also affected by numerous physiological reflexes. These, in turn, are affected by some imponderable factors, which cannot be completely excluded from such a study. These factors mainly include patient clinical status, such as the presence of pain or pre-existing illnesses and types of drugs administered.

CONCLUSION AND IMPLICATIONS FOR NURSING PRACTICE

In this study, increases in CBT resulted in decreases in systolic blood pressure, mean arterial blood pressure and arterial oxygen saturation, and increases in diastolic arterial pressure and pulse rate. PCA increases systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and arterial oxygen saturation, and decreases CBT and pulse rate. Therefore, PCA should be applied for 20 min to the axillae, groin, abdomen and below-knee regions in neurosurgical febrile patients. Furthermore, accurate and careful monitoring of haemodynamic parameters before and during fever can be helpful to determine the process of fever treatment. Information about the effects of PCA on CBT and haemodynamic parameters and mechanisms related to these changes in neurosurgical febrile patients will be of benefit to the quality and efficacy of care. Neurosurgical ICU staff, particularly during postoperative care, should be proactive in ensuring that they have a formal, evidence-based management plan for fever

that conforms to the relevant clinical guidelines and integrates multidisciplinary care. Neurosurgical ICU staff should ensure that febrile patients receive individual assessment, care and treatment managements, including multidisciplinary goals, to obtain optimal patient outcomes.

AUTHORS' CONTRIBUTIONS

HA and MY were responsible for the study conception and design and drafting of the manuscript.

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CONFLICT OF INTEREST

No conflict of interest has been declared by the authors.

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