Effect of excessive environmental heat on core temperature in critically ill patients. An observational study during the 2003 European heat wave

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Background. The primary goal of this study was to investigate the relation between the core temperature of critically ill patients and hot ambient temperatures during a heat wave. The second goal was to evaluate the impact of such a heat wave on the number of microbiological tests ordered.

Methods. During a heat wave, from August 3 to 22, 2003, we conducted an observational study in the surgical intensive care unit (ICU) of a French hospital that had no air-conditioning at the time. The core temperature of 18 critically ill patients and 36 health-care workers was measured with a non-contact, infrared tympanic membrane thermometer. The association between the core body temperature in infected and non-infected critically ill patients and the staff members, and the ambient temperature in the ICU was analysed using linear regression. The number of microbiological tests ordered was also recorded and compared with the same period in the previous year.

Results. The equation of the regression line for infected critically ill patients was: core temperature = 33.5 + 0.16 × ambient temperature ($R^2 = 0.53; P < 0.0001$). The regression line was steeper than that for the non-infected patients (0.077; $P < 0.0001$). The slopes of the regression lines for non-infected and control patients were similar ($P = 0.20$). More blood cultures were carried out during the heat wave than at the same period during the year 2002 (4.80 blood cultures per 1000 patient-days vs 2.47 per 1000 patient-days; $P = 0.0006$).

Conclusion. During a sustained high ambient temperature, hyperthermia can occur in critically ill infected patients and to a lesser extent in non-infected patients and health-care workers. The number of blood cultures requested rises substantially, leading to increased costs. Installation of air-conditioning is therefore recommended.

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Urban heat waves are among the deadliest of all weather emergencies.1 The European summer heat wave of 2003 was exceptional in its duration and intensity.2 In France, the number of heat-related deaths reached 14 800 by August 20.2 The human thermoregulatory system usually maintains a core body temperature near 37°C.3 Environmental temperatures between a warmth-response and cold-response threshold do not trigger autonomic thermoregulatory defences.3 Within this range, patients are poikilothermic, and body temperature changes passively depending on environmental conditions.

When the ambient temperature exceeds body temperature, heat is taken in from the environment.4 As soon as body temperature increases above the thermoregulatory set-point, heat dissipation depends on both autonomic and behavioural adjustments.4 In the normal subject, heat...
exposure to 46°C for 4 h increased the core temperature by only 0.75°C.7 Hyperthermia occurs when thermoregulatory mechanisms are overwhelmed by excessive environmental heat or impaired heat dissipation.4 Predisposing factors include cardiovascular diseases, neurological disorders, diabetes mellitus, obesity, the use of anticholinergic or diuretic medications, dehydration, and old age.46 Critically ill patients may present underlying diseases that impair thermoregulation in a hot environment.124 Moreover, many patients in the intensive care unit (ICU) are pyrexial,7 and this could also impair the efficiency of the thermoregulatory processes by up-regulation of the thermostatic set-point.8

The influence of high ambient temperatures on the core temperature of critically ill patients is of interest in the context of global warming and the predicted world-wide increase in the frequency and intensity of heat waves.910 The primary goal of this study was to investigate the relationship between the core temperature of critically ill patients and extreme high ambient temperatures. The second goal was to evaluate the impact of such a heat wave on the number of microbiological tests ordered.

Patients and methods
From August 3 to 22, 2003 we conducted an observational study in the surgical ICU of Henri Mondor hospital, a university teaching hospital located in suburban Paris. This 10-bed open unit is dedicated mainly to trauma and critically ill postoperative patients, and air-conditioning was not available at the time. Only patients with a clinical diagnosis of brain death were excluded. The ambient temperature in the ICU was measured early in the morning and during the afternoon in order to record a minimum and a maximum value on the same day when possible. The core body temperature was measured with a non-contact, infrared tympanic membrane thermometer (FirstTemp Genius 3000A; Sherwood Davis and Geck, Bondoufle, France) as recommended by the French authorities.11 Only the baseline temperature measured in the auditory canal was displayed. The tympanic membrane temperature is believed to reflect the temperature of the hypothalamus and thus the core body temperature.12 The outdoor temperature and relative humidity were provided by Météo France (Saint-Maur-des-Fossés, France) and the heat index was calculated. The heat index, or apparent temperature, is a function of the temperature and the relative humidity. It provides a measure of the evaporative and radiant transfer of heat between a typical human and the environment.2

Data were collected prospectively for each patient. The severity of illness was evaluated with the first-day new Simplified Acute Physiology Score (SAPS II).13 Various other variables were noted: age, sex, reason for admission to the ICU, prescription of norepinephrine, need and duration of mechanical ventilation, need for sedation, and mortality in the ICU. Changes in the plasma concentrations of sodium and creatinine and the white blood cell count were recorded. Analgesia and sedation were adjusted to obtain clinical comfort for the patient, as judged by nurses and physicians, by continuous infusions of fentanyl 100–400 µg h⁻¹ and midazolam 2–10 mg h⁻¹ or propofol 150–250 mg h⁻¹.

Moderate hypothermia was provided for all patients with severe neurotrauma14 and for patients with hypoxaemia who were at risk from the increased oxygen consumption accompanying fever.15 Antipyretic therapy was provided for other patients with a sustained tympanic temperature above 38.5–39°C: paracetamol was administered i.v. External cooling with ice slush and the use of an ice-tunnel (constructed using sheets plunged into iced water and a bedside fan) was also used.

Control group
The tympanic membrane temperature was also recorded in 39 controls, composed of physicians, nurses and nurse assistants working in the ICU. This control group consisted of 22 men and 17 women with a mean (range) age of 34.5 (19–59) years.

Infection criteria and microbiological tests
ICU-acquired infection was defined as an infection that began at least 48 h after ICU admission. In patients receiving mechanical ventilation of the lungs, the diagnosis of nosocomial pneumonia was considered when they developed a new and persistent lung infiltrate and had purulent tracheal secretions, confirmed by bacterial culture of a blind protected telescoping catheter ≥10³ c.f.u. ml⁻¹ of at least one pathogen.16 Other nosocomial infections were prospectively surveyed and detected according to standard surveillance and definitions.17

The numbers of blood cultures, protected telescopic catheter cultures and urine cultures ordered during the study period were reported. The same data were also provided for the year 2002 for comparison. For this analysis, all the patients were included.

According to French law (Law No. 88–1138, December 20, 1988), this study did not need Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale approval or informed consent because, as an observational study, it did not modify current diagnostic or therapeutic strategies. All data were anonymized.

Statistical analysis
Data were computerized and analysed using the Statview 5.0 statistical package (SAS Institute, Cary, NC, USA) or Microsoft® Excel 2000. We expressed continuous variables as the mean (SD) or as the median (25th–75th percentiles) when appropriate. The χ²-test or Fisher’s exact test were used to compare proportions and rates, and continuous variables were compared using the Student’s t-test or the
Mann–Whitney U-test when appropriate. Linear regression of core temperature on ambient temperature was performed for infected and non-infected critically ill patients, and for the control group. Differences between the slopes of the three regression lines were tested by analysis of variance (ANOVA). The study period was split into two periods: the first period was from August 3 to 15 and the second from August 16 to 22, according to the duration of the heat wave. Variations of biological variables during the two periods were compared using ANOVA. Statistical significance was defined as a P-value of \( < 0.05 \).

**Results**

**Heat wave and ICU temperature (Fig. 1)**

During 9 consecutive days, the highest and lowest recorded ICU temperatures were above 35°C and 30°C, respectively. The heat index in the ICU was continuously above 32.1°C during the same period, at which level it remained until August 13, when it started decreasing. Interestingly, despite a fall in the outside temperature during the night, the ICU temperature remained at above 30°C until August 13.

**Effect of the heat wave on core temperature in critically ill patients and controls**

Thirteen men and five women (11 trauma, four complicated surgery, three medical) were studied prospectively for a mean of 10 days (range, 2–20 days). Their mean (range) age was 49 (16–84) yr and their mean (SD) SAPS II score was 30 (16) [associated probability of death: 32% (24)%]. Ten patients received mechanical ventilation of the lungs, eight medication for sedation, and six norepinephrine. Nosocomial infection was diagnosed in 10 patients (four pneumonia, three sepsicaemia, and three miscellaneous infections). Only two patients died (11.1%).

The core body temperature was recorded 272 times in critically ill patients and 194 times in controls. The changes in core temperature over time in critically ill patients with or without infection are reported in Figure 2. During the heat wave, the mean core temperature of infected patients was always above 38.5°C and was above 39°C for 5 consecutive days despite the use of external...
cooling and prescription of paracetamol. We were unable to achieve mild hypothermia in patients with neurotrauma or hypoxaemia using active cooling. At the same time, non-infected patients and controls had a moderate increase in their core temperature. When the ICU temperature fell below 30°C, the mean core temperature of the patients never exceeded 38°C (Fig. 2A) and that of controls ranged between 36.5 and 37.5°C (Fig. 2B).

**Fig 2** (a) Time course of core temperature in infected and non-infected critically ill patients during the heat wave. (b) Time course of core temperature in controls during the heat wave. In both cases, the x axis represents the date and time of temperature measurements (for example 3; 21 means August 3 at 9 p.m.).
The results of linear regression analysis of core temperature on ICU temperature for infected and non-infected critically ill patients are shown in Figure 3. The equation of the regression line for control patients was: core temperature = 35.0 + 0.08 · ICU temperature \((R^2=0.48; P<0.0001)\) and core temperature = 33.50 + 0.16 (ICU temperature) for infected patients \((R^2=0.53; P<0.0001)\). Patients with current infection showed the steepest slope, which was significantly different from those of non-infected patients \((P<0.0001)\).

Table 1  Comparison of the number of microbiological tests ordered during August in the years 2003 and 2002. First period vs 2nd period: \(^a P=0.0001; \ ^b P=0.51; \ ^c P=0.88; \ ^d P=0.78; \ ^e P=0.46; \ ^f P=0.21\)

<table>
<thead>
<tr>
<th>Microbiological tests ordered</th>
<th>Year 2003, August</th>
<th>Year 2002, August</th>
<th>2003 vs 2002 (1st period)</th>
<th>2003 vs 2002 (2nd period)</th>
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<td>3 to 15 (1st period)</td>
<td>16 to 22 (2nd period)</td>
<td>3 to 15 (1st period)</td>
<td>16 to 22 (2nd period)</td>
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<td>Blood cultures</td>
<td></td>
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<tr>
<td>n</td>
<td>63</td>
<td>16</td>
<td>21</td>
<td>12</td>
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<td>Per 1000 patient-days</td>
<td>4.80</td>
<td>2.10(^a)</td>
<td>2.47</td>
<td>2.26(^d)</td>
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<td>4</td>
<td>4</td>
<td>5</td>
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<tr>
<td>Per 1000 patient-days</td>
<td>0.76</td>
<td>0.52(^b)</td>
<td>0.47</td>
<td>0.94(^e)</td>
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<tr>
<td>Urine cultures</td>
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<tr>
<td>n</td>
<td>13</td>
<td>8</td>
<td>8</td>
<td>2</td>
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<tr>
<td>Per 1000 patient-days</td>
<td>0.99</td>
<td>1.05(^c)</td>
<td>0.94</td>
<td>0.37(^f)</td>
</tr>
</tbody>
</table>

The results of linear regression analysis of core temperature on ICU temperature for infected and non-infected critically ill patients are shown in Figure 3. The equation of the regression line for control patients was: core temperature = 35.0 + 0.08 · ICU temperature \((R^2=0.48; P<0.0001)\) (data not shown). Differences between the three slopes were significant after ANOVA \((P<0.0001)\). Patients with current infection provided the steepest slope, which was significantly different from that of non-infected patients \((P<0.0001)\). The slopes of non-infected and control patients were similar \((P=0.20)\).

Impact of the heat wave on selected biological variables and on prescription of microbiological tests

The mean values of serum sodium concentration and white blood cell count were significantly higher during the heat wave than after the heat wave [141 (5) vs 139 (3) mmol litre\(^{-1}\) \((P=0.036)\) and 14.7 (8.5) vs 11.0 (4.5) \times 10^6 \text{ litre}^{-1} \((P<0.001)\) respectively]. The serum creatinine concentration remained stable during the study period [83 (35) µmol litre\(^{-1}\)].

More blood cultures were carried out during the heat wave than at the same period during the previous year (Table 1).
There was no difference in the numbers of protected telescopic catheter specimens and urine cultures ordered. The two periods (2002 vs 2003) were comparable in terms of admission numbers (22 patients each), case mix (trauma patients 64 vs 55%), mean age [52 (16–79) vs 47 (16–84) yr], sex ratio (men, 77 vs 72%), and severity of illness [mean SAPS II score 34 (18) vs 36 (14)].

Discussion
Our results suggest that thermoregulation failed in critically ill infected patients during the heat wave when the ambient temperature was above 30°C. The rise in core temperature occurred despite the use of external cooling and antipyretics. The number of blood cultures ordered was substantially increased.

The human thermoregulatory system usually maintains a core body temperature near 37°C. A variety of disorders can elevate body temperature; those resulting from thermoregulatory failure are properly called hyperthermia, whereas those resulting from intact homeostatic responses with up-regulation of the thermostatic set-point for body temperature are categorized as fever or pyrexia. Hyperthermia may be caused by excessive heat production, diminished heat dissipation, or malfunction of the hypothalamic thermostat. Disorders of excessive heat production could be excluded in our patients and there was no clinical or radiological evidence of central thermoregulatory failure. In this case, hyperthermia resulted from regulatory failure in the warm environment.

The temperature recorded in the ICU followed the outside temperature, but the minimum temperature remained at a higher level. Such elevation of the minimum temperature did not allow recovery from the severe heat stress experienced during the day. The heat index in the ICU ranged between 32.1°C and 40.6°C during this period, a range in which heat exhaustion is possible. Vasodilatation and increased skin blood flow (in concert with sweating) are essential for heat dissipation during heat exposure. Concurrent with cutaneous vasodilatation, the evaporation of sweat decreases the skin temperature, thereby cooling the blood in the dilated skin vessels before it returns to the core.

There are several possible explanations for the occurrence of hyperthermia in critically ill patients during the heat wave. First, volume depletion leads to cutaneous vasoconstriction and decreased sweating, which impair heat dissipation. Critically ill patients are clearly at risk as about 50% of them suffer from volume depletion, and some also receive diuretic medications. The higher serum sodium concentrations recorded during the heat wave provided some indirect evidence for volume depletion. Moreover, an inability to increase cardiac output because of salt and water depletion, cardiovascular disease, or medications that interfere with cardiac function can impair heat tolerance and result in increased susceptibility to heat illness. Secondly, norepinephrine, prescribed in some patients, causes cutaneous vasoconstriction and therefore limits heat dissipation. Third, sedatives can impair thermoregulation. However, propofol and opioids increase the sweating threshold only slightly, whereas midazolam decreases it slightly. Finally, the great majority of ICU patients are unable to utilize the normal behavioural responses to hyperthermia.

Fever is a common clinical finding in ICU patients and is a cardinal manifestation of nosocomial infection. The fact that infected patients had a more impressive increase in body temperature during the heat wave suggests that the two mechanisms are probably additive. Thus, critically ill non-infected patients only had a moderate increase in core temperature. Because fever and hyperthermia may occur simultaneously, it is always important to look for infectious causes of fever in patients with elevated body temperatures, even if there is an explanation for hyperthermia. As a consequence, the number of blood culture rose substantially, leading to increased costs of ICU care.

Health-care workers during physical work also presented a moderate increase in core temperature. They perceived discomfort and physical strain (heat stress), but none of them needed to stop working.

The consequences of hyperthermia are well described. For some critically ill patients in situations of limited oxygen delivery or hypoxaemic respiratory failure, tissue hypoxia can develop. High body temperature also has the potential to exacerbate the lung damage incurred by adverse patterns of mechanical ventilation. Patients with healthy cardiovascular systems may tolerate the stress of hyperthermia, but patients with underlying heart disease may suffer ischaemia, arrhythmias, hypotension or congestive heart failure. Furthermore, local and systemic insults associated with heat stress, such as splanchnic hypoperfusion, can alter the immunological and barrier functions of the intestines and favour the development of multiple organ failure. We did not observe an increased rate of death, which was at variance with the results of another French study on heat stroke. However, with 18 patients it was very unlikely that any relationship between excessive core temperature and outcome would be found. Hospitalization in an ICU and the level of care provided have probably contributed to reduction in the consequences of hyperthermia.

In conclusion, when the ambient temperature in the ICU rises above 30°C, hyperthermia can occur in infected critically ill patients and to a lesser extent in non-infected critically ill patients and health-care workers. In such circumstances, physical cooling should be set up and paracetamol prescribed to lower the hypothalamic set-point in infected patients. Since the only known protective measure against heat is to withdraw from it, the long-term solution is the widespread installation of air-conditioning.

Acknowledgements
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