Febrilni pacient – kdy intervenovat a jak?

MUDr. Pavel Štětka
ARK FNUSA Brno

Pacient 1

- 69let muž, 3.den hosp. pro CAP- atb 3den
- PSV 5+10cmH20,Fio2 0.45 df 20, pao2 11.4
- Nor 5ml/hod , fisi 90, laktát 1,8
- Ramsay -1
- TT 38, 6 nasofarynx
- ???

Pacient 2

- 55let muž, 3.den hosp. po 20minKPR pro maligní arytmii při DKMP
- PSV 5+10cmH20,Fio2 0.45 df 20, pao2 13.4
- Nor 2ml/hod , SR 90, laktát 1,8
- Ramsay -4, analgsed.
- TT 38, 4 moč.měchýř
- Crp 90 leu 12 bronchorhea ,rtg bez infiltrace
- ????

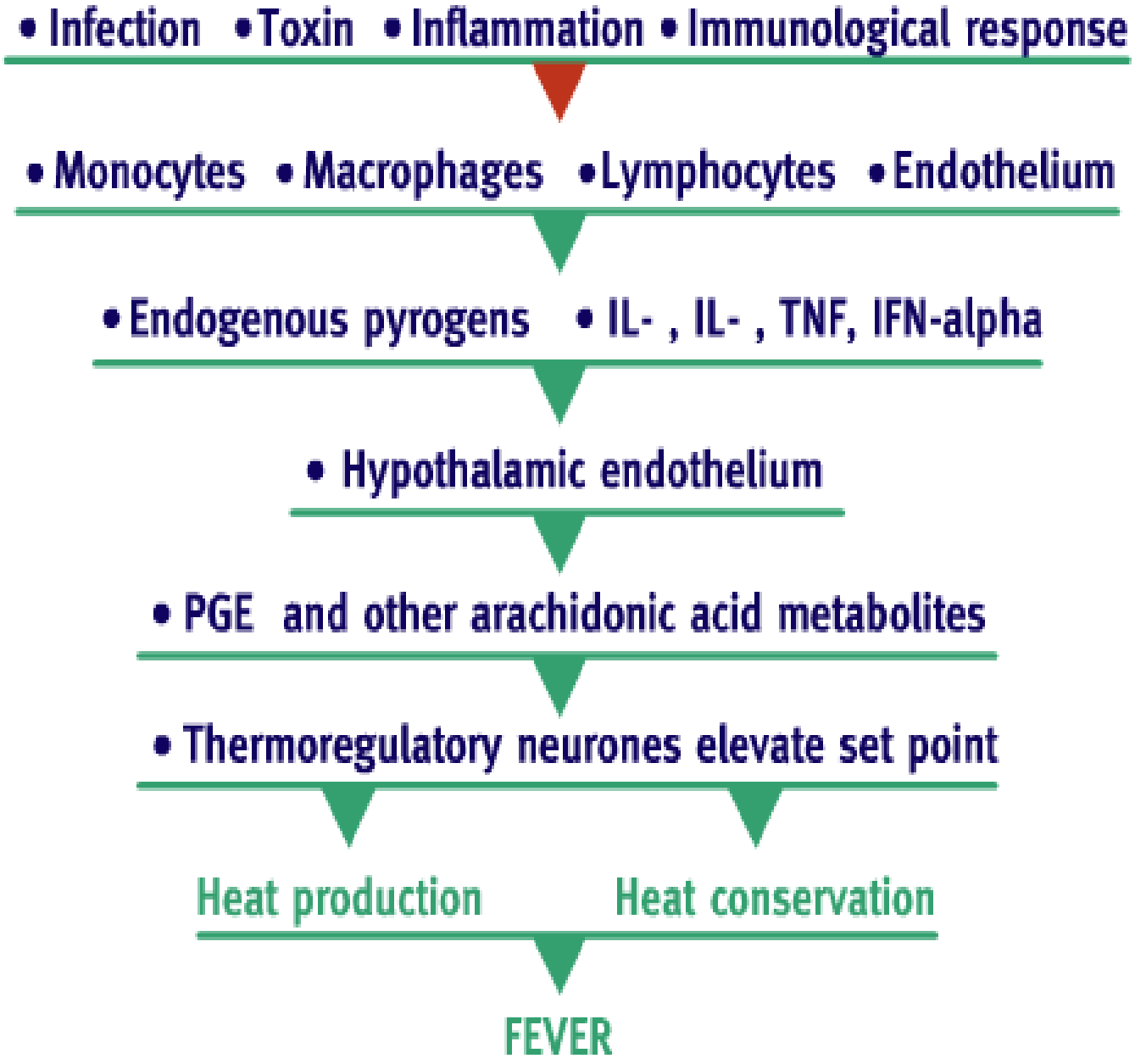
Otázky ?

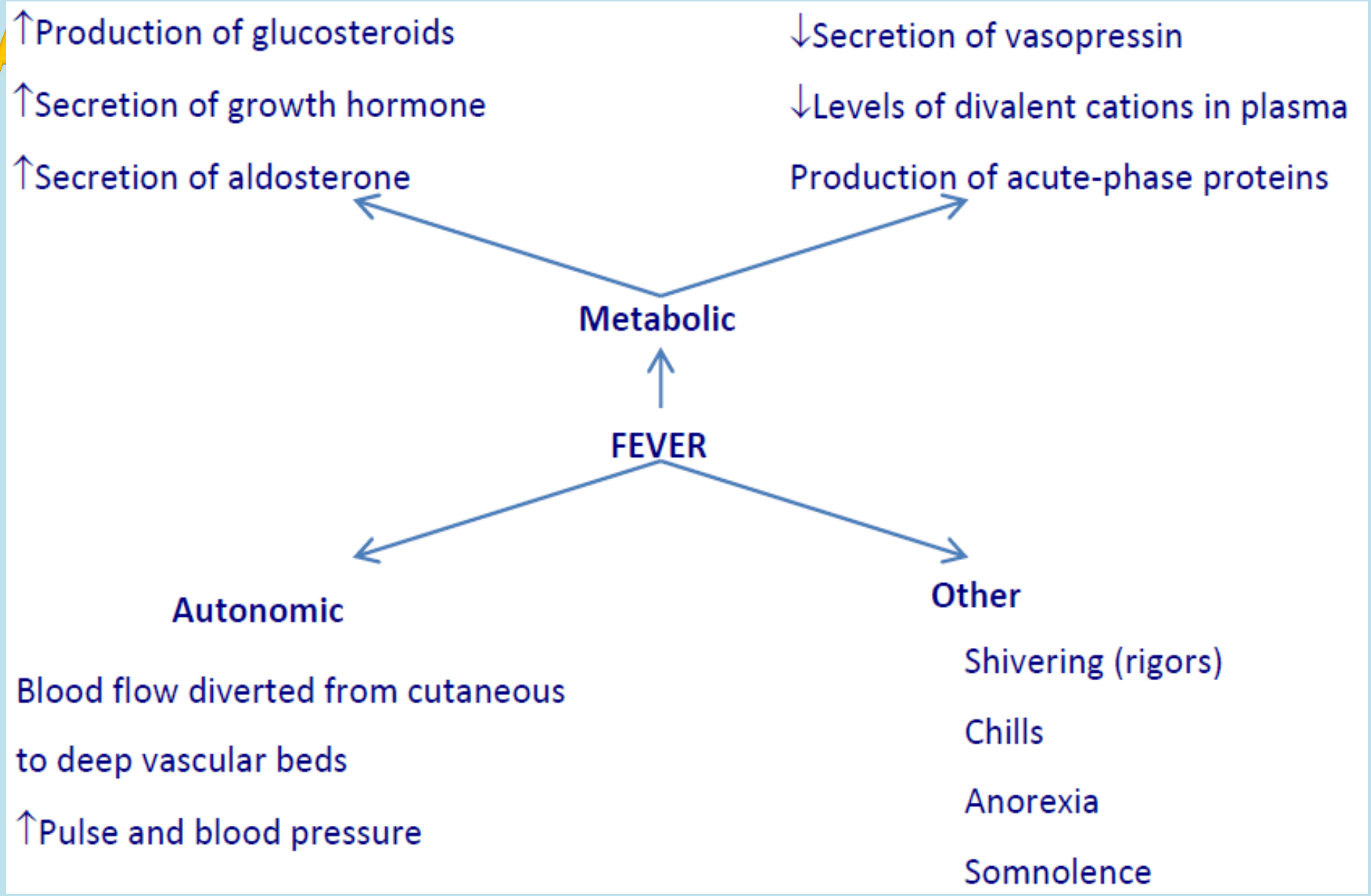
- Je horečka škodlivá pro ICU pacienty?
- Je racionální horečku léky nebo fyzikálními prostředky snižovat?

- SCCM --horečka na ICU= teplota jádra nad 38.3 °C
- Normální TT do 37st.C, cirkadiánně 0.5-1stC
- horečka je způsobena dysbalancí mezi produkcí a ztrátou tepla,
- Doba trvání
- Kde měřit? i.v., moč.m., nasofarynx,rectum ??
- Přesně a opakovaně

Febrilie

- Endothermie =homeostatický mechanismus savců i ptáků-význam vs. pomocník v boji s infekcí
- Zlepšuje produkci Ig, aktivaci T lymfocytů, zvyšuje funkci neutrofilů a makrofágů
- Weinstein-spontánní bakteriální peritonitis , TT nad 38 , snížení mortality
- 10% zvýšení VO2 na 1stC
- CNS léze, nepoměr DO2/VO2


• Infection • Toxin • Inflammation • Immunological response
• Monocytes • Macrophages • Lymphocytes • Endothelium
• Endogenous pyrogens • IL- , IL- , TNF, IFN-alpha
• Hypothalamic endothelium
• PGE and other arachidonic acid metabolites
• Thermoregulatory neurones elevate set point
Heat production
Heat conservation
FEVER


↑Production of glucosteroids
↑Secretion of growth hormone
↑Secretion of aldosterone
↓Secretion of vasopressin
↓Levels of divalent cations in plasma
Production of acute-phase proteins
Metabolic
FEVER
Autonomic
Blood flow diverted from cutaneous
to deep vascular beds
↑Pulse and blood pressure
Other
Shivering (rigors)
Chills
Anorexia
Somnolence

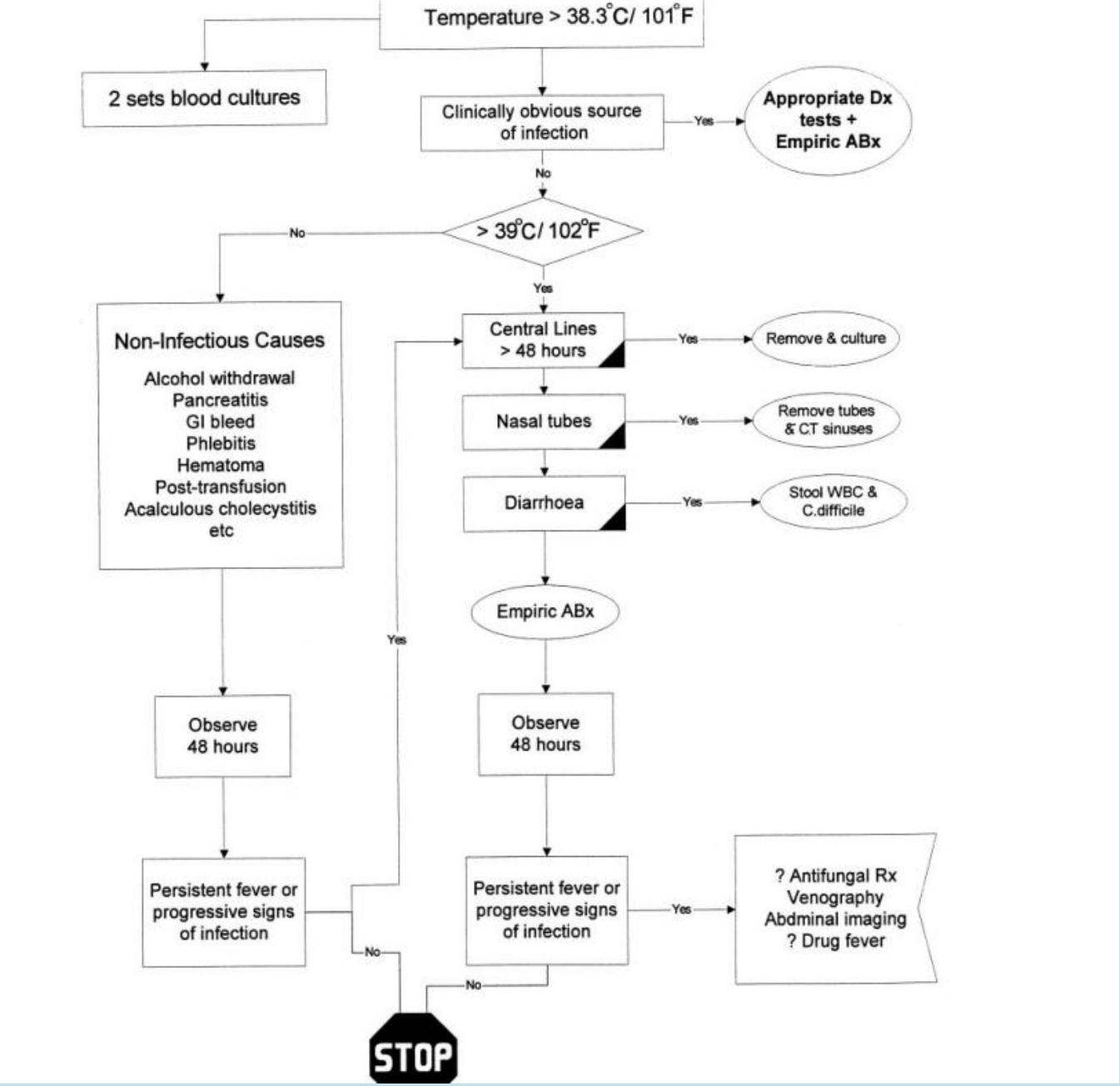
- 30% pacientů ICU - horečka
- Až 90% ICU pacientů se sepsí je febrilních ,ikdyž na začátku je až 35% normotermních
- 10% hypotermních-zvýšená mortalita**!!!**
- příčina horečky infekční , neinfekční n. smíšená , **39stC** hranice
- identifikace příčiny je často obtížná , což komplikuje i rozhodnutí - léčit n. neléčit

Table 2—*Common Infectious Causes of Fever in the ICU*

Infectious Causes
VAP
Sinusitis
Catheter-related sepsis
Primary Gram-negative septicemia
C difficile diarrhea
Abdominal sepsis
Complicated wound infections

Table 1—*Noninfectious Causes of Fever in the ICU*

Noninfectious Causes
Alcohol/drug withdrawal
Postoperative fever (48 h postoperative)
Posttransfusion fever
Drug fever
Cerebral infarction/hemorrhage
Adrenal insufficiency
Myocardial infarction
Pancreatitis
Acalculous cholecystitis
Ischemic bowel
Aspiration pneumonitis
ARDS (both acute and late fibroproliferative phase)
Subarachnoid hemorrhage
Fat emboli
Transplant rejection
Deep venous thrombosis
Pulmonary emboli
Gout/pseudogout
Hematoma
Cirrhosis (without primary peritonitis)
GI bleed
Phlebitis/thrombophlebitis
Adrenal insufficiency
IV contrast reaction
Neoplastic fevers
Decubitus ulcers



Young and Saxena *Critical Care* 2014, **18**:206
http://ccforum.com/content/18/2/206

CRITICAL CARE

REVIEW

Fever management in intensive care patients with infections

Paul J Young[1]*, Manoj Saxena[2]

	Design, setting, and participants	Key findings
Laupland et al. 2008 [30]	Retrospective cohort study of patients admitted to four ICUs in Calgary between 2000 and 2006; *n* = 24,204 ICU admissions in 20,466 patients	• Fever of ≥ 38.3 °C developed during 44 % of ICU admissions and high fever ≥ 39.3 °C during 8 % of admissions • Fever was not associated with increased ICU mortality but high fever was associated with a significantly increased risk of death
Young et al. 2011 [31]	Inception cohort study in three tertiary ICUs in Australia and New Zealand over six weeks in 2010 identifying patients with fever ≥ 38 °C and known or suspected infection; *n* = 565	• 9 % of patients admitted to ICU had or developed a fever and known or suspected infection • Paracetamol was administered to about $^{2}/_{3}$ of patients with fever and known or suspected infection on any given day
Selladurai et al. 2011 [32]	Retrospective cohort study of patients admitted to a single tertiary ICU in Australia with sepsis between December 2009 and August 2010; *n* = 106	• 69 % of septic patients received paracetamol at least once during their first seven days in ICU • 88 % of septic patients with a fever > 38 °C received paracetamol during their first seven days in ICU • Septic patients with a fever > 38 °C were 6.8 times (95 % CI 1.9-24.7) more likely to receive paracetamol than septic patients who were not febrile
Lee et al. 2012 [33]	Inception cohort study of consecutive patients admitted to 25 ICUs in Japan and Korea for more than 48 hours over three months in 2009; *n* = 1,425	• NSAID use independently associated with increased 28-day mortality in patients with sepsis (adjusted OR 2.61; 95 % CI 1.11-6.11; p = 0.03) but with a trend towards a decreased 28-day mortality in patients without sepsis (adjusted OR 0.22; 95 % 0.03-1.74; p = 0.15) • Paracetamol use independently associated with increased 28-day mortality in patients with sepsis (adjusted OR 2.05; 95 % CI 1.19-3.55; p = 0.01) but with a trend towards a decreased 28-day mortality in patients without sepsis (adjusted OR 0.58; 95 % 0.06-5.26; p = 0.63)

Laupland et al. 2012 [34]	Inception cohort study of patients admitted to French ICUs contributing to the Outcomerea database between April 2000 and November 2010; $n = 10{,}962$	• 25.7 % of patients had a fever of ≥ 38.3 °C at ICU presentation • Fever was not associated with increased mortality but hypothermia was an independent predictor of death in medical patients
Young et al. 2012 [35]	Retrospective cohort study of 636,051 patients in Australia, New Zealand and the UK admitted to the ICU between 2005 until 2009	• Elevated body temperature in the first 24 hours in ICU was associated with an increased risk of mortality in patients without infections and a decreased risk of mortality in patients with infections
Niven et al. 2012 [36]	Interrupted time series analysis of cumulative fever incidence in ICUs in Calgary from 2004–2009	• The cumulative incidence of fever ≥ 38.3 during ICU admission decreased from 50.1 % to 25.5 % over the 5.5 years of the study

Bernard et al. 1997 [43]	Double blind placebo-controlled trial of ibuprofen in patients with severe sepsis in seven centers in North America; $n = 455$	• Ibuprofen significantly reduced temperature, heart rate, oxygen consumption, and lactic acidosis in patients with severe sepsis • Ibuprofen did not alter the incidence or duration of shock or ARDS and had no significant effect on 30-day mortality (37 % ibuprofen-treated group vs. 40 % placebo-treated group)
Memis et al. 2004 [44]	Double blind placebo-controlled trial of lornoxicam in patients with severe sepsis in one center in Turkey; $n = 40$	• No significant difference between lornoxicam and placebo was demonstrated in terms of hemodynamic parameters, biochemical parameters, cytokine levels, or ICU mortality (35 % lornoxicam-treated group vs. 40 % placebo-treated group)
Morris et al. 2011 [45]	Multicenter, randomized trial comparing the antipyretic efficacy of a single dose of placebo, 100 mg, 200 mg, or 400 mg of i. v. ibuprofen in hospitalized patients of whom > 90 % had infections; $n = 120$ (53 critically ill)	• All doses of ibuprofen tested were effective in lowering temperature • There were no significant difference between treatment groups with respect to ventilation requirements, length of stay or in-hospital mortality (4 % placebo, 3 % 100 mg ibuprofen, 7 % 200 mg ibuprofen, 6 % 400 mg ibuprofen)
Haupt et al. 1991 [46]	Multicenter, placebo-controlled randomized trial of ibuprofen in patients with severe sepsis; $n = 29$	• Ibuprofen significantly reduced body temperature • There was no significant difference between the treatment groups in terms of in-hospital mortality (30.8 % in the placebo group vs. 56.3 % in the ibuprofen group)
Schulman et al. 2006 [47]	Single center, unblinded, randomized trial of aggressive vs. permissive temperature management in febrile patients in a trauma ICU; $n = 82$	• There was no significant difference between the treatment arms in terms of the number of new infections • The in-hospital mortality was 15.9 % in the aggressive treatment group and 2.6 % in the permissive treatment group ($p = 0.06$)
Niven et al. 2012 [48]	Multicenter, unblinded randomized trial of aggressive vs. permissive temperature management in febrile ICU patients; $n = 26$	• The mean daily temperature was lower in the patients assigned to aggressive fever management • The in-hospital mortality was 21 % in the aggressive treatment group and 17 % in the permissive treatment group ($p = 1.0$)
Schortgen et al. 2012 [49]	Multicenter, randomized controlled trial of external cooling in patients with fever and septic shock receiving mechanical ventilation in seven centers in France; $n = 200$	• External cooling significantly reduced body temperature • External cooling did not alter the proportion of patients who had a 50 % reduction in vasopressor dose after 48 hours • Day-14 mortality was significantly lower in the patients assigned to external cooling but there was no significant difference between the groups in terms of ICU or in-hospital mortality

závěr

- Horečka je signál
- Onem. CNS, stavy spojené s nepoměrem mezi DO2/VO2 -cíl normotermie
- Horečka infekčního původu – spíše neléčit
- Horečka neinfekčního původu –spíše léčit
- paracetamol , !na fyz.chlazení

závěr

- Horečka je signál -**příčina??**
- Onem. CNS, stavy spojené s nepoměrem mezi DO2/VO2 -cíl normotermie
- Horečka infekčního původu – spíše neléčit
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- **Správné atb?**

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- Crp 90 leu 12 bronchorea ,rtg bez infiltrace
- **Léčit horečku(paracetamol i fyz.metody a zároveň pátrat po infekčním zdroji – pneumonie??**