Alice nel Paese delle Meraviglie

Trauma Stress Response

Risposta Sistemica

Ipotalamo	Attivaz. SNA Simpatico
Ipofisi ant.	↑ACTH ↑GH e Prolattina ↑↓TSH ↑↓FSH e LH ↑-ßendorfine
Ipofisi post.	↑Arginina Vasopressina
Surrene	↑Cortisolo ↑Aldosterone
Pancreas	↓Insulina ↑Glucagone
Tiroide	↓Tiroxina (T_4), T_3

★ la chirurgia non è una condizione naturale e lo stress response aumenta la morbidità

★ la risposta endocrina sistemica è attivata dagli impulsi nervosi periferici afferenti al midollo spinale e proiettati ai centri superiori

★ la diminuzione del “firing” delle vie afferenti sensitive determina una proporzionale diminuzione della risposta

Trauma Stress Response

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Trauma Stress Response

Risposta Locale

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- ruolo nella risposta infiammatoria ed immunitaria
- azione locale di mantenimento della risposta infiammatoria cellulare
- azione sistemica complessa ("***acute-phase response***" epatica),

★ la risposta locale è attivata dai mediatori cellulari dell'infiammazione: **citochine**

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Trauma Stress Response

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Veterinary
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Formerly the Journal of Veterinary Anaesthesia

Veterinary Anaesthesia and Analgesia, 2015 doi:10.1111/vaa.12275

RESEARCH PAPER

Stress-related biomarkers in dogs administered regional anaesthesia or fentanyl for analgesia during stifle surgery

Marta Romano*,†, Diego A Portela*,‡, Gloria Breghi* & Pablo E Otero§

- 4 gruppi
  - 2 ALR (PNB e Spinale)
  - Fentanil e Controllo
  - Valutazione Pain Scores, Glicemia, Cortisolo sierico

Table 3 Intraoperative heart rate (HR), mean arterial blood pressure (MAP) and end-tidal isoflurane (F_EIso) in dogs undergoing non-invasive diagnostic procedures [control (group CTR)] or orthopaedic surgery with different analgesic protocols: peripheral (femoral and sciatic) nerve blocks (group PNB); spinal (intrathecal) analgesia (group SPI), or variable rate intravenous infusion of fentanyl (group FEN) (n = 15 dogs in each group)

		Time point				
Variable	**Group**	**T_B**	**T_0**	**T_5**	**$T_{1/2}$**	**T_{END}**
HR (beats minute^{-1})						
	PNB	82 ± 23	81 ± 21	82 ± 22	81 ± 22	85 ± 22
	SPI	84 ± 17	85 ± 16	87 ± 18	86 ± 17	89 ± 17
	FEN	69 ± 14	68 ± 14	70 ± 15	66 ± 16*	65 ± 14†
	CTR	79 ± 18	79 ± 15	81 ± 15	79 ± 16	78 ± 16
MAP (mmHg)						
	PNB	80 ± 9	79 ± 9	78 ± 9	77 ± 8	79 ± 8
	SPI	74 ± 8‡	73 ± 9§	73 ± 8	75 ± 9‡	75 ± 10§
	FEN	77 ± 9	77 ± 11	76 ± 10	77 ± 10	79 ± 8
	CTR	85 ± 7	84 ± 7	85 ± 6**	87 ± 7	86 ± 7
F_EIso (%)						
	PNB	1.2 ± 0[a]	1.2 ± 0[a]	1.2 ± 0[a]	0.9 ± 0.2††[b]	0.9 ± 0.2††[b]
	SPI	1.2 ± 0[a]	1.2 ± 0[a]	1.2 ± 0[a]	1 ± 0.1††[b]	0.9 ± 0.2††[b]
	FEN	1.2 ± 0	1.2 ± 0	1.2 ± 0	1.2 ± 0.1	1.1 ± 0.1
	CTR	1.2 ± 0	1.2 ± 0	1.2 ± 0	1.2 ± 0.1	1.1 ± 0.1

T_B, 15 minutes before the beginning of the surgical or diagnostic procedure; T_0, beginning of the procedure; T_5, 5 minutes after the beginning of the procedure; $T_{1/2}$, half time of the duration of the procedure; T_{END}, end of the procedure. Different superscript letters indicate significant differences within the same group ($p < 0.0001$). *Significantly different from SPI at the same time point ($p = 0.02$). †Significantly different from PNB and SPI at the same time point ($p = 0.005$). ‡Significantly different from CTR at the same time point ($p = 0.004$). §Significantly different from CTR at the same time point ($p = 0.01$). **Significantly different from SPI and FEN at the same time point ($p = 0.002$). ††Significantly different from FEN and CTR at the same time point ($p < 0.0001$).

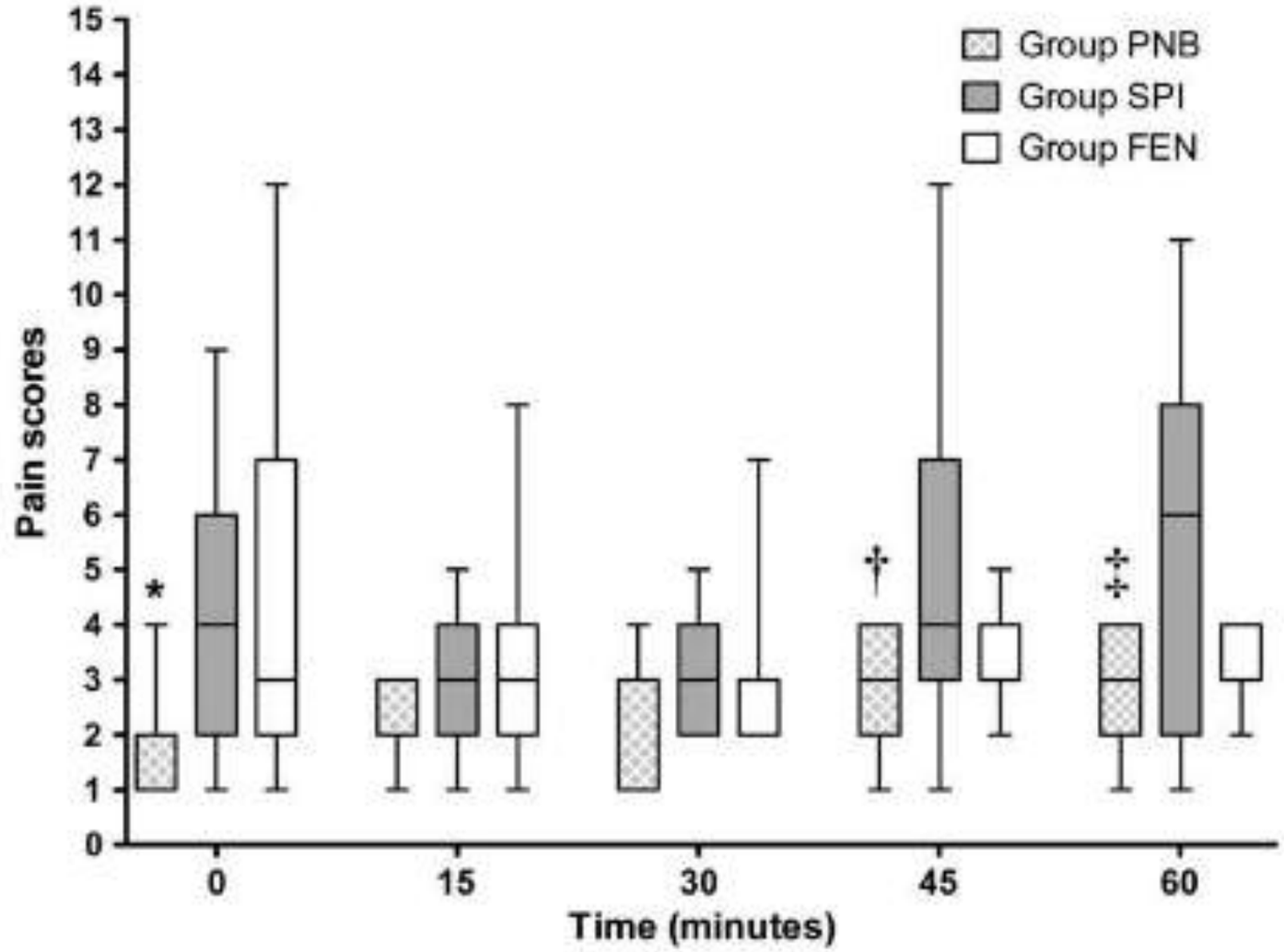


Figure 1 Postoperative pain scores evaluated for 60 minutes starting from the regaining of consciousness (T_0) using the short form of the Glasgow Composite Measure Pain Scale (maximum score possible: 20) in dogs submitted to femoro-tibial joint surgery (tibial tuberosity advancement or tibial plateau levelling osteotomy) and administered femoral and sciatic nerve blocks (group PNB), spinal (intrathecal) anaesthesia (group SPI) or a variable rate intravenous infusion of fentanyl (group FEN) as analgesic protocols (n = 15 per group). Boxes represent interquartile ranges; the horizontal line within the box represents the median value, and the upper and lower whiskers represent maximum and minimum values. *Significantly lower compared with SPI and FEN (p = 0.0064). †Significantly lower compared with SPI (p = 0.021). ‡Significantly lower compared with SPI (p = 0.024).

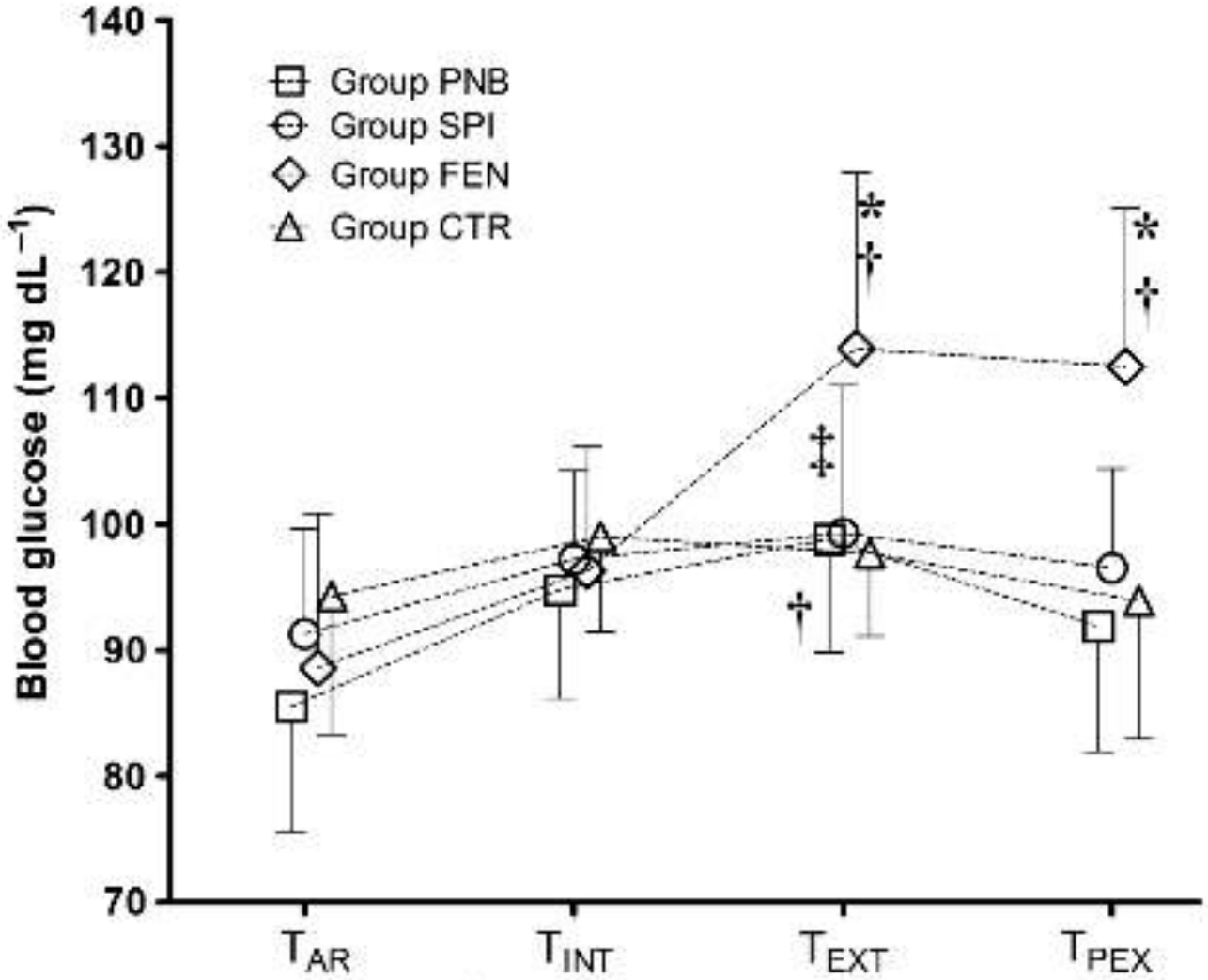


Figure 2 Peri-anaesthetic blood glucose (mg dL^{-1}) concentrations in dogs undergoing anaesthesia for non-invasive diagnostic procedures (group CTR) or orthopaedic surgery (tibial plateau levelling osteotomy or tibial tuberosity advancement) with different analgesic protocols (n = 15 dogs per group). Group PNB: peripheral femoral and sciatic nerve blocks; group SPI: spinal analgesia; group FEN: variable rate intravenous infusion of fentanyl. Time points: T_{AR}, before anaesthesia; T_{INT}, after endotracheal intubation; T_{EXT}, after extubation; T_{PEX}, 1 hour after extubation. *Significant differences compared with PNB, SPI and CTR ($p < 0.0001$). †Significant differences compared with T_{AR} ($p < 0.0001$). ‡Significant differences compared with T_{AR} ($p = 0.0016$).

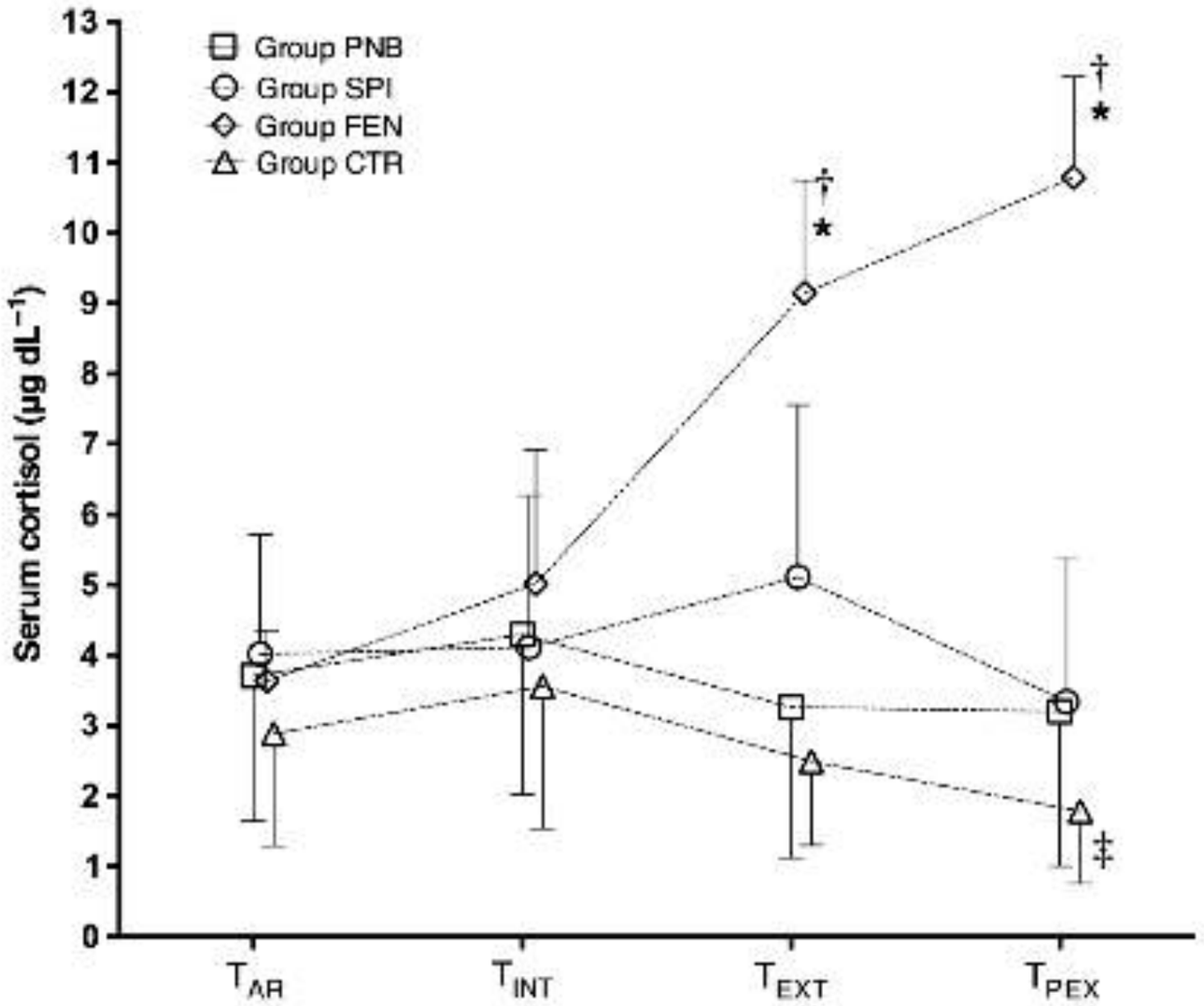


Figure 3 Peri-anaesthetic serum cortisol concentrations (µg dL^{-1}) in dogs undergoing anaesthesia for non-invasive diagnostic procedures (group CTR) or orthopaedic surgery (tibial plateau levelling osteotomy or tibial tuberosity advancement) with different analgesic protocols (n = 15 dogs per group). Group PNB: peripheral femoral and sciatic nerve blocks; group SPI: spinal analgesia; group FEN: variable rate intravenous infusion of fentanyl. Time points: T_{AR}, before anaesthesia; T_{INT}, after endotracheal intubation; T_{EXT}, after extubation; T_{PEX}, 1 hour after extubation. *Significant differences compared with T_{AR} ($p < 0.0001$). ‡Significantly lower compared with T_{INT} ($p = 0.008$). †Significantly higher compared with CTR, PNB and SPI ($p < 0.0001$).

Catabolismo

Can J Anesth/J Can Anesth
DOI 10.1007/s12630-014-0274-y

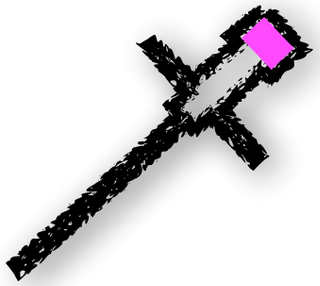
REVIEW ARTICLE/BRIEF REVIEW

Perioperative catabolism
Catabolisme périopératoire

Thomas Schricker, MD, PhD ·
Ralph Lattermann, MD, PhD

- 2015
- Stress e Catabolismo
- Neuroassiale vs. Oppioidi

Patients undergoing major surgery are exposed to metabolic and endocrine alterations in carbohydrate, protein, and insulin metabolism, often summarized as the catabolic response. Preventing stress in an effort to minimize this catabolic response to surgery represents



Neuraxial anesthesia

Segmental blockade of nociceptive signals at the spinal cord level provides the most effective pain relief after intraperitoneal procedures. Apart from optimal pain control, neuraxial blockade has anticatabolic effects that may contribute to better outcome.[117]

Epidural and intrathecal administration of local anesthetics prevents or blunts the neuroendocrine stress response, which results in improved insulin sensitivity with a positive influence on glucose and protein catabolism.[118] By attenuating insulin resistance and facilitating exogenous glucose utilization, neuraxial techniques reduce the amount of energy that is required to maintain protein balance. If the energy load of parenteral feeding can be decreased, use can be made of peripheral veins and hyperglycemia can be avoided. Epidural analgesia together with the perioperative

Opioids

High-dose opioid anesthesia attenuates most of the endocrine and metabolic responses to surgery, but it is used rarely for procedures of short and intermediate duration.[122] Newer short-acting narcotics, such as sufentanil, alfentanil, and remifentanil, prevent intraoperative catabolism, also when used at a smaller dose. Postoperative catabolic changes, however, are either unaffected or even more pronounced.[28]

Catabolismo

Anesth Analg 2005;101:1202–8

The Anticatabolic Effect of Neuraxial Blockade After Hip Surgery

Ralph Lattermann, MD, MSc*, Geesche Belohlavek, MD*, Sigrid Wittmann, MD*, Bernd Füchtmeier, MD†, and Michael Gruber, PhD*

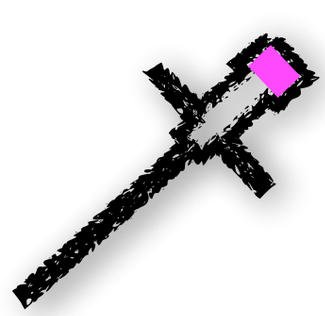
Departments of *Anesthesia and †Trauma Surgery, University of Regensburg, Germany

technique. Neuraxial block of afferent and efferent signals with epidural local anesthetics has often been shown to modulate protein economy after abdominal surgery, most likely mediated through its suppressive effect upon the hypothalamopituitary-adrenal stress response (6). Although the impact of epidural blockade upon protein economy after abdominal surgery has been extensively studied, its potential role in modifying protein catabolism after lower extremity surgery remains unclear. The findings of one study re-

- 2005
- Effetto anti-catabolico CSE

The effect of CSE on the catabolic stress response during and after hip surgery, however, has not been studied. Using stable isotope tracers, we demonstrated that CSE inhibits the increase in amino acid oxidation on the first postoperative day, and thus, it is a useful anticatabolic strategy in patients undergoing hip surgery. Because protein catabolism has been identified as an important factor contributing to postoperative fatigue, prolonged convalescence, and increased morbidity, preservation of whole body protein may be a possible mechanism by which CSE exerts it beneficial clinical effects after hip surgery.

Another characteristic feature of the metabolic response to surgery is the increase in plasma glucose concentration, a consequence of both stimulated glucose production and impaired glucose use. Considering the adverse effects of hyperglycemia, such as compromised immune function(19), increased wound infection (20), enhanced protein catabolism (21), poor clinical outcome after myocardial infarction (22), and cerebral ischemia (23), preservation of perioperative glucose homeostasis represents a clinically relevant goal. In agreement with previous investigations (6), CSE in the present protocol inhibited the increase in glucose plasma concentration during hip surgery. However, observations based on



In conclusion, we have found that CSE inhibits the increase in plasma glucose concentration during hip surgery but does not modify glucose metabolism on the first postoperative day. In addition, CSE reduces protein loss one day after hip arthroplasty and thus represents a useful anticatabolic strategy for patients undergoing hip surgery.

Immunocompetenza

"I would have everie man write what he knows and no more."—MONTAIGNE

BRITISH JOURNAL OF ANAESTHESIA

VOLUME 81, NO. 6 DECEMBER 1998

EDITORIAL

Opioids and the immune system

- 1998
- Ruolo oppiodi endogeni
- Rilascio da parte di granulociti

Corticotropin releasing hormone (CRH) is an important hormone released under stress conditions from both the hypothalamus and cells of the immune system. Injection of CRH has been shown to produce analgesia when injected into sites of local inflammation, an effect which is blocked by co-injection of antiserum to β-endorphin.[8] This again suggests a possible mechanism whereby local activation of the endogenous opioid system from immunocompetent cells provides both an analgesic action and anti-inflammatory effect.

Immunocompetenza

British Journal of Anaesthesia **101 (1):** 40–4 (2008)
doi:10.1093/bja/aen078 Advance Access publication April 8, 2008

BJA

Pain and the immune system

H. L. Rittner*, A. Brack and C. Stein

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**Corresponding author. E-mail: heike.rittner@charite.de*

- 2008
- Meccanismo d'azione e rilascio oppioidi endogeni
- Azione anti tolleranza oppioidi endogeni

Opioid peptides in leucocytes

In peripheral inflamed tissue, opioid peptides such as β-endorphin, met-enkephalin, dynorphin, and endomorphins are produced by leucocytes and released upon certain types of stimulation.[17 39] Opioid peptides can bind to opioid receptors on sensory neurons. These receptors are synthesized in dorsal root ganglia and are transported intra-axonally to peripheral nerve endings. Three types of opioid receptors such as μ-(MOP), δ-(DOP), and κ-(KOP) are expressed in sensory neurons.[24 26 27] Agonist binding elicits potent exogenous or endogenous analgesia in inflamed tissue.[39]

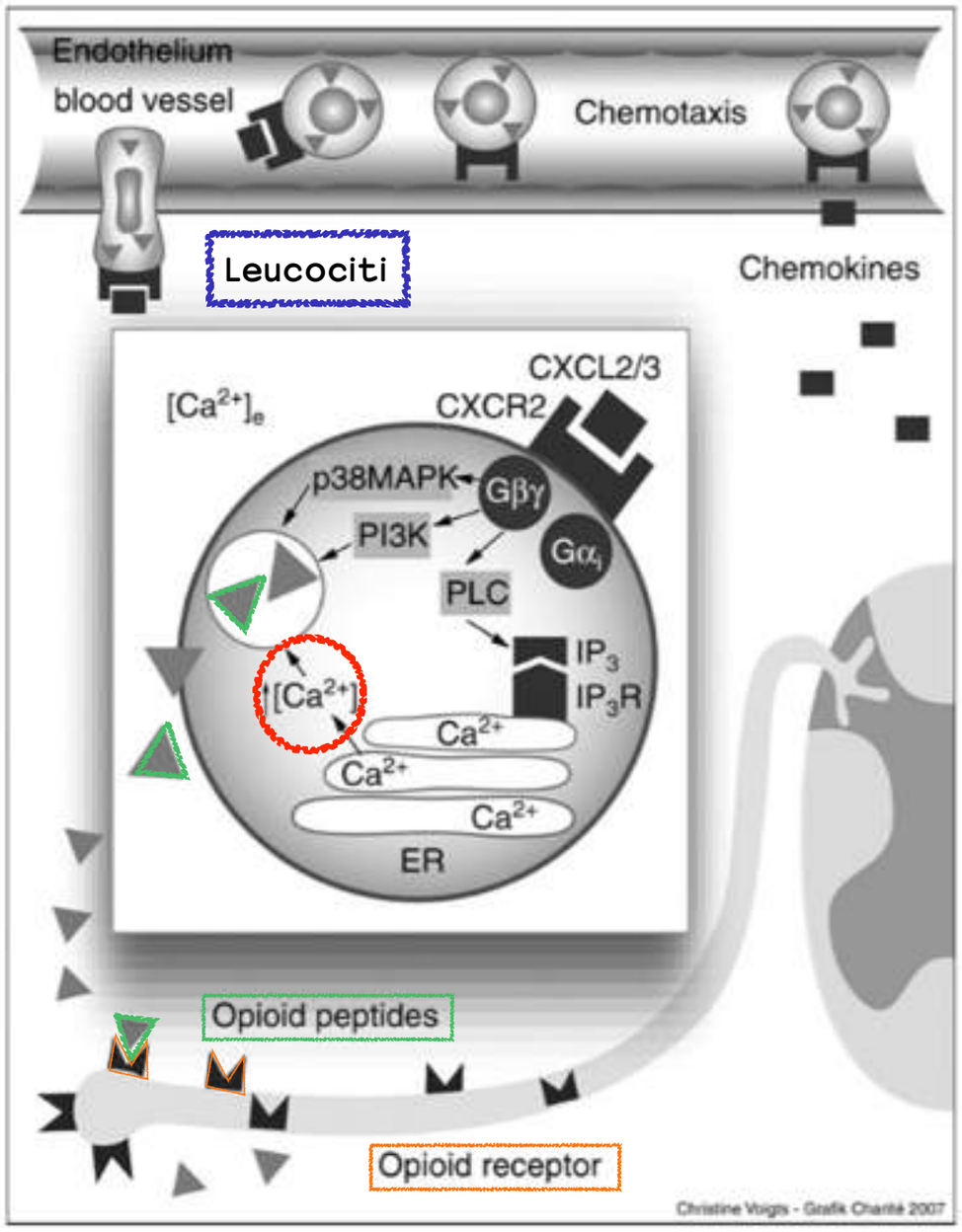

Fig 1 Intracellular mechanisms of opioid peptide release from granulocytes. Opioid peptide-containing granulocytes migrate into inflamed tissue. Opioid peptides are released and bind to opioid receptors on peripheral sensory neurons leading to analgesia. CXCL2/3 produces antinociception by triggering opioid peptide release. CXCL2/3-induced opioid release requires elevation of intracellular Ca^{2+} via flux from the endoplasmatic reticulum (ER). In addition, activation of phosphoinositol-3-kinase (PI3K) and p38 mitogen-activated kinase (MAPK) are involved. PLC, phospholipase C; IP_3, inositol 1,4,5-triphosphate.

Chemokines regulating migration of opioid peptide-containing leucocytes

Chemokines are chemotactic mediators cotrolling cell trafficking under physiological and pathological conditions. Chemokines are not only important under various inflammatory conditions but also play a role in pain and analgesia. Although many studies examined the hyperalgesic action of chemokines, recent evidence also points towards their antinociceptive effects.

Chemokines released from leucocytes and endothelial cells upregulate and increase the avidity of adhesion molecules and thereby migration into inflamed tissue.

In early inflammation, CXCL1 and CXCL2/3, binding to their receptor CXCR2 on granulocytes, are expressed in CFA inflammation.[4 5]

$CXCR2^+$ granulocytes also contain opioid peptides. Pre-treatment of rats with antibodies against CXCL1 or CXCL2/3 substantially decreases the number of opioid-containing granulocytes but not of monocytes/macrophages accumulating in the inflamed tissue[4] and in consequence abolishes antinociception mediated by endogenous opioid peptides. An intact chemokine cascade is, therefore, a prerequisite for peripherally mediated endogenous opioid antinociception and inhibition of only one of the steps is sufficient to impair this mechanism.

Immunocompetenza

Effects of anaesthesia on the human immune system

John D Hunter

Hospital Medicine, September 1999, Vol 60, No 9

While the magnitude of surgical trauma is thought to have a more pronounced effect on the immune response than anaesthesia itself, in-vivo and in-vitro work suggests that anaesthetic agents have a significant immunomodulatory effect.

The potential for anaesthesia to influence the immune system has long been recognized and immunomodulation induced by anaesthesia may be implicated in the dissemination of tumour metastasis and the incidence of postoperative infection. Of the many studies of the effects of anaesthesia on the immune system, some give

- 1999
- Short term depression
- Durata infusioni
- Pazienti Critici
- Vantaggi ALR

TABLE 1.
Effects of anaesthesia and surgery on the human immune system

Immunomodulatory effect				Anaesthetic drugs and techniques	Reference
T lymphocyte function	Lymphocyte proliferation	In-vivo studies suggest a decrease in T cell responsiveness		Combination of general anaesthesia + surgery	Hole (1984), Stevenson et al (1990)
		In-vitro studies yield conflicting results	No demonstrable effect	Intravenous induction agents	Devlin et al (1994)
			Decreased T cell response	Nitrous oxide, halothane	Bruce (1976), Stevenson et al (1990)
	NK cell cytotoxicity	In-vivo studies suggest a decrease in NK cytotoxicity		Exposure to nitrous oxide, enflurane and halothane	Woods and Griffiths (1986)
		In-vitro studies also suggest a decrease in cytotoxicity		Combination of general anaesthesia + surgery	Tonnesen and Wahlgreen (1988)
				Opioid analgesics	Beilin et al (1996), Yeager et al (1995)
Antigen processing	Downregulation of MHC class II expression on lymphocytes and monocytes			High dose fentanyl anaesthesia	McBride et al (1994)
Cytokines	Decreased IL-6 release			Alfentanil	Crozier et al (1994)
	Increased TNF-α from monocytes			Thiopentone, propofol and ketamine	Rossano et al (1992)
Neutrophil function	Neutrophil polarization inhibited			Propofol, thiopentone	O'Donnell et al (1992)
	Respiratory burst inhibited			Propofol, midazolam, ketamine and methohexitone	Heine et al (1996)
	Neutrophil chemotaxis inhibited			Halothane, enflurane, and propofol	Moudgil and Forrest (1984), Jensen et al (1993)

IL = interleukin; MHC = major histocompatability complex; NK = natural killer; TNF = tumour necrosis factor

Devlin et al (1995) examined the effects of propofol and thiopentone on delayed type hypersensitivity (DTH) reactions and T lymphocyte proliferation in healthy volunteers. This is of interest since DTH responses test not only the ability of the immune system to recognize foreign antigens, but also its effectiveness in dealing with them. They demonstrated that both drugs caused no depression of PHA-induced T cell proliferation, but caused significant depression of DTH reactions to skin multi-test antigens. This may be of clinical importance since a reduced DTH response is associated with increased mortality in surgical patients (Christou et al, 1995).

Intra- and postoperatively administered opiates may be implicated in the suppression of immune function. Beilin et al (1996) compared NK cytotoxicity in patients receiving high doses (75–100 μg/kg) and low doses (up to 5μg/kg) of fentanyl as part of their anaesthetic regimen. In both groups there was a similar suppression of NK cell activity. However, patients receiving the high doses of fentanyl still had significantly impaired NK cell cytotoxic activity at 48 hours, whereas in the low dose fentanyl group the NK cell cytotoxic activity had returned to normal.

Yeager et al (1995) examined the effects of morphine on NK cell natural cytotoxicity in healthy volunteers. They demonstrated significant suppression of NK cell natural cytotoxicity 2 and 24 hours after the onset of intravenous morphine exposure, which persisted for 24 hours after termination of the morphine in those receiving higher doses. The mechanisms by which opioids suppress

REGIONAL ANAESTHESIA AND THE IMMUNE RESPONSE

There is evidence that regional anaesthesia may lessen the immunosuppressive changes seen postoperatively, by attenuating the endocrine stress response to surgery. Hashimoto et al (1995) showed that extradural anaesthesia prevents changes in lymphocyte subpopulations in gastrectomy patients. There was a decrease in CD4+ and an increase in CD8+ lymphocytes measured after skin incision in those receiving general anaesthetic, which was not observed in patients receiving epidural anaesthesia. The authors suggested that extradural anaesthesia may be immunologically beneficial during the postoperative period.

NK cell cytotoxicity may also be altered by extradural anaesthesia (Tonnesen and Wahlgreen, 1988). Postoperative NK activity was measured in two groups of patients undergoing abdominal hysterectomy: one group received extradural anaesthesia, and the other neuroleptanaesthesia. In the latter group NK activity was impaired for 3 days after the operation while no significant suppression of NK function was seen in the extradural group. The reason for this is unknown but may be related to an alteration in the stress response to surgery induced by extradural anaesthesia.

Immunocompetenza

REVIEW

Acute pain the immune system and opioimmunosuppression

Keith Budd[a,*], E.A. Shipton[b]

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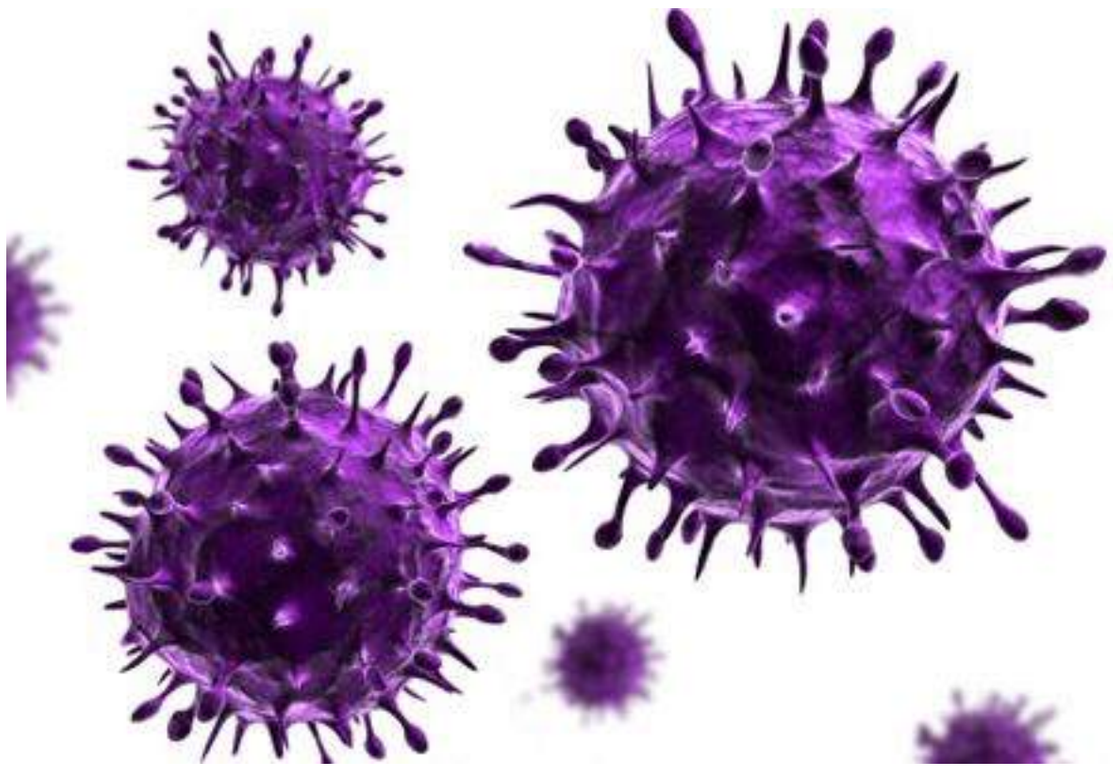
[b] *University of Otago, Christchurch, New Zealand*

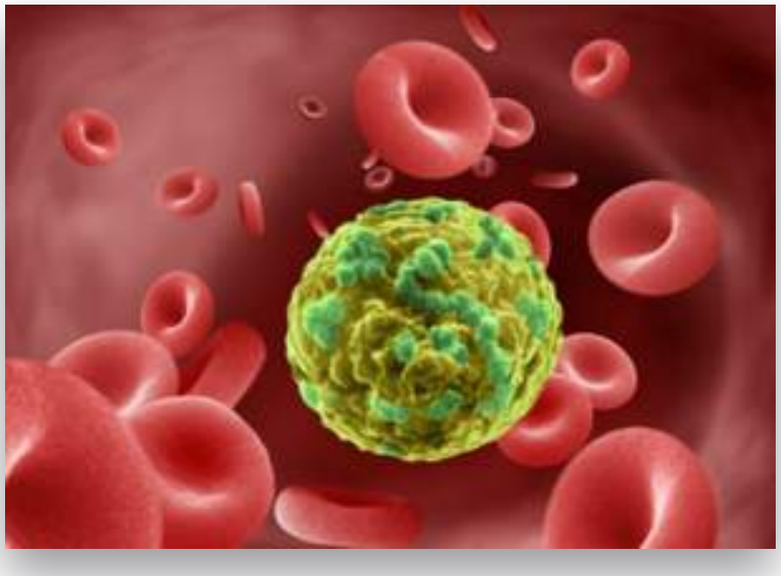
- 2004
- Ruolo immunosoppressivo oppioidi esogeni
- Differenze tra tipologia di oppioidi

9. Immunosuppression by exogenous opioids

During the past half century, a large body of evidence has been gathered showing significant effects by a majority of opioid analgesics in common clinical utilisation on the human immune system [56,71–74].

Some of the more profound depressant effects of the opioid agents are seen to lead to an increased susceptibility to bacterial and viral infection, especially following surgery and trauma [75], reduced defence mechanisms in cancer patients indicated by their reduced survival time and increased incidence of secondary growths [76,77] and a greater susceptibility to HIV infection in drug abusers [78,79].





In vivo administration of morphine-like agents to rodents induces a decrease of multiple immune parameters affecting almost all of the cell types of the immune system, NK cells being shown to be particularly sensitive to morphine modulation [88–90]. Evidence is also available showing significant opioid-induced decrease of T-cell proliferation following both acute and chronic administration [88,91–95]. In contrast, effects on B-lymphocytes are less pronounced [96–97].

12. Exogenous opioid differentiation in immune suppression

It has already been seen that the degree to which and manner in which each individual opioid affects the immune system can be significantly different. It is, therefore, clinically important to define between those opioids that depress immune function significantly and those that are more appropriate to use. The two most important differentiating factors are opioid receptor affinity and molecular structure. Opioids with high affinity for the μ-opioid receptor have been found to induce significant immunosuppression, whilst those with κ-receptor or δ-receptor selectivity do not [59,89]. There is no information currently available concerning the performance of agents with affinity at the nociceptin receptor. Antagonists at the μ-opioid receptor tend to enhance the function of the immune system [119].

The immunosuppressive effects of opioids are independent of their analgesic effects and appear to be a function of their molecular structure.

The basic opioid structure may be represented by that of morphine (Fig. 1), which is known to produce a high level of immunosuppression.

13. Conclusions

The majority of opioid analgesics in current clinical practice have been shown to have or to have the propensity for producing suppression of the immune system in humans. There is also an increasing

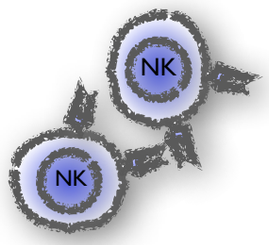
Selecting opioid analgesics which do not depress the immune system is not difficult. They are those which structurally have a carbonyl substitution at C6, a single bond between C7—C8 and, preferably, an hydroxyl group at C14. Agents complying with these constraints have pharmacological properties which make them suitable to replace those opioid analgesics that have immunodepressant properties. They include buprenorphine, hydromorphone, oxymorphone, oxycodone and tramadol which, in certain clinical circumstances, provide additional advantages as well as immunostability.

Ruolo immuno-soppressivo

La IASP (International Association for the Study of Pain - 1986) definisce il dolore come "*un'esperienza sensoriale ed emozionale spiacevole associata a danno tissutale, in atto o potenziale. E' un esperienza individuale e soggettiva, a cui convergono componenti puramente sensoriali (nocicezione) relative al trasferimento dello stimolo doloroso dalla periferia alle strutture centrali, e componenti esperenziali e affettive, che modulano in maniera importante quanto percepito*".

Perché ALR deve piacere

Antibiotico



Perché ALR deve piacere

Antibiotico