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TITLE: CAUDAL STEROIDS FOR THE TREATMENT OF PELVIC PAIN DUE TO CANCER

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Systemic steroids have been used as co-analgesics[1] and have been advocated in the treatment of bone pain, pain related to pressure on local structures by edema surrounding large tumor masses, and pain due to nerve compression.[2,3] Although there are anecdotal reports on the use of caudal or epidural steroids with local anesthetic in patients with pain due to cancer, it is not clear whether the steroid (S) or the local anesthetic (LA) is the essential drug for providing pain relief. This study was designed to evaluate the pain relieving effects of caudal administration of S by comparing the following groups: LA, S & LA, and S.

Methods: After approval by the hospital's Human Research Committee thirty-one patients with a diagnosis of cervical, rectal, or vulvar carcinoma who were experiencing severe pain in spite of narcotic administration were enrolled in this study. All patients received, in a double blind randomized manner, one of the following administered caudally in a 10ml volume:

1) 1% Lidocaine
2) 80mg Methylprednisolone in normal saline
3) 30mg Methylprednisolone in 1% Lidocaine

Pain was monitored using the Visual Analog Scale (VAS) prior to injection, 30 minutes post injection, and daily until pain returned. Significant pain relief was defined as 50% or more reduction in VAS. Pain medications were continued at the same dose from pre-injection throughout the study when possible. The degree and duration of pain relief and the number of patients obtaining significant pain relief were subjected to statistical analysis.

Results: All three groups experienced significant pain relief at 30 mins. post-block. Duration of pain relief using the Generalized Wilcoxon and Savage test for comparing survival curves showed that the three groups differ $p<0.00005$ (see Table 1). Using the same statistical tests the two extended survival groups do not differ significantly ($p \geq .38$).

Discussion: It appears that the benefits of caudally administered LA & S are due primarily to the anti-inflammatory action of the S. Although addition of a LA is useful to confirm needle placement, it is not necessary for long term pain relief and has the disadvantage of possible intravascular or spinal injection.

References:

1) Post Grad Med J, 59:702-6, 1983.
2) N Engl J Med, 313:84-95, 1985.
3) J Pain Sympt Mgmt, 3:39-43, 1988.

TABLE 1
MEAN DURATION PAIN RELIEF IN DAYS

Steroid n=10	164.71 ±	S.E. = 17.35
Steroid & Local n=11	182.36 ±	S.E. = 37.82
Local n=10	0.08 ±	S.E. = 0.121

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Title: EFFECT OF FLUMAZENIL ON LIDOCAINE INDUCED CONVULSION IN RATS

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Flumazenil, the specific antagonist of benzodiazepines, has received a clinical attention as an effective antagonist of benzodiazepine induced sedation (1)(2). However, it is not certainly known whether flumazenil antagonizes the anticonvulsant property of benzodiazepines. The purpose of this study was to investigate the effect of flumazenil on lidocaine induced convulsion with and without diazepam pretreatment in rats.

After institutional approval, twenty-eight awake Wistar rats were divided into 4 groups of 7 each, and pretreated with I.V. diazepam and/or flumazenil through the femoral vein cannula at 6 min and 3 min before lidocaine infusion. Control group received normal saline(n.s.), diazepam group, 0.2mg/kg diazepam and n.s., diazepam+flumazenil group, 0.2mg/kg diazepam and 0.2mg/kg flumazenil, and flumazenil group, n.s. and 0.2mg/kg flumazenil. All groups received a continuous I.V. infusion of lidocaine (15mg/ml) at a rate of 4mg/kg/min until tonic/clonic convulsion occurred. Blood pressure and heart rate were continuously recorded and the convulsant doses of infused lidocaine were calculated. Blood samples were drawn from the femoral artery cannula for determination of blood gases and plasma concentrations of lidocaine at the onset of convulsion.

The pretreatment with diazepam increased both convulsant lidocaine doses (Table1, Fig1) ($p<0.01$) and plasma lidocaine concentrations at the onset of convulsion (Table1, Fig2) ($p<0.01$). Flumazenil reversed these diazepam values. (Fig1 and 2). The pretreatment with flumazenil alone neither changed convulsant lidocaine doses (Table1, Fig1) nor plasma lidocaine concentrations at the onset of convulsion (Table1, Fig2). Blood gases were maintained within normal ranges and blood pressure did not change until convulsion in all groups.

Moreau et al.(3) reported that 10mg/kg I.P. flumazenil reversed the anticonvulsant action of 3mg/kg I.P. diazepam against convulsion induced by intracerebroventricular injection of N-methyl - D-aspartic acid in mice. Our study also demonstrated that flumazenil antagonized the anticonvulsant effect of diazepam during lidocaine infusion. It may be, therefore, suggested that one should use flumazenil with great care for patients who received both benzodiazepines and large doses of local anesthetics during anesthesia and surgery, and for patients who are on benzodiazepines as anticonvulsant therapy.

In conclusion, our data showed that I.V. flumazenil reversed the anticonvulsant property of I.V. diazepam against lidocaine induced convulsion, and flumazenil itself had no effect on lidocaine induced convulsion in rats.

Statistical analysis: ANOVA followed by Duncan's method.

References: (1)Br. J. Anaesth. 58: 1005-1011, 1986.
(2)Anesthesiology 70: 899-904, 1989.
(3)Br. J. Pharmacol. 98: 1050-1054, 1989.

Table 1.

Group	Lidocaine Dose (mg/kg)	Lidocaine Concentration (μg/ml)
control	40.0 ± 1.9	10.8 ± 0.5
diazepam	54.9 ± 1.3 #,¶,§	15.3 ± 0.7 #,¶,§
diazepam+flumazenil	41.8 ± 1.6	11.5 ± 0.4
flumazenil	42.8 ± 1.6	10.8 ± 0.4

Values are mean±SE; n=7
#: p<0.01 vs control, ¶: p<0.01 vs diazepam+flumazenil, §: p<0.01 vs flumazenil

Fig 1.

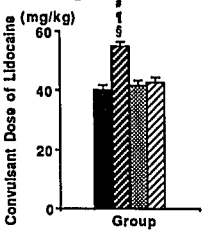

Fig 2.