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POSTER SESSION II

A717

Title: EFFECT OF ACUTE HYPERTENSION ON LIDOCAINE INDUCED CONVULSION IN RATS

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The addition of epinephrine to lidocaine is harmful in the event of accidental intravascular injection (1). However, the mechanism by which epinephrine lowers the convulsant doses of lidocaine is not clear. The preliminary study showed that acute hypertension itself might play a principle role. The purpose of this study was to determine if acute hypertension induced by epinephrine(E), norepinephrine(N) or phenylephrine(P) have the same effects on lidocaine induced convulsion in rats.

After institutional approval, fifty-three awake Wistar rats were divided into 7 groups. All groups received a continuous I.V. infusion of lidocaine (15mg/ml) through a femoral vein cannula at a rate of 4 mg/kg/min until tonic/clonic convulsion occurred. Control group received plain lidocaine. Doses of additive E, N and P had been predetermined to increase systolic arterial pressure(SAP) to 185±10mmHg (10μg/ml, 7.5μg/ml, 50μg/ml, respectively). Doses of sodium nitroprusside(SNP) to prevent the increase in SAP by E, N and P were also predetermined in the preliminary study(7.5μg/kg/min, 12.5μg/kg/min,15μg/kg/min,respectively). 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 addition of E, N, and P produced acute hypertension, and there were no significant differences in SAP at 1min of infusion and at the onset of convulsion among 3 groups. SAP did not change in the control and SNP groups(Table 1). Blood gases were maintained within normal ranges until convulsion in all groups. Acute hypertension groups(E,N,P) showed significantly smaller convulsant doses of lidocaine compared with control (p<0.01)(Fig 1), and also significantly lower plasma concentrations of lidocaine (p<0.01)(Fig 2). In the SNP groups, the addition of E and N, but not P still decreased convulsant doses of lidocaine significantly (p<0.01)(Fig 1).

Our data showed that the same degree of acute hypertension induced by three different inotropic agents decreased the convulsant doses and the plasma concentrations of lidocaine to the same extent. Acute hypertension can cause the breakdown of the blood-brain barrier in animals (2). Therefore, acute hypertension observed in this study may have caused the breakdown of the blood-brain barrier, thereby increasing permeability to lidocaine. Although SNP decreased SAP to the control level, E and N, but not P(no ß-adrenergic effect) still decreased the convulsant doses of lidocaine. Therefore, our results indicate that not only acute hypertension but also ß-adrenergic effect can contribute in lowering the threshold of lidocaine induced convulsion.

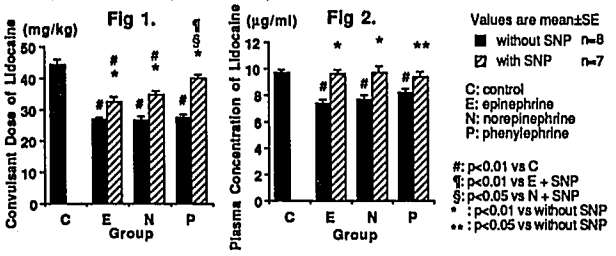
Statistical analysis: ANOVA followed by Duncan's method or Student's t-test.

References: (1) Anesthesia Progress 32: 57-61, 1985.
(2) Circ. Res. 64: 658-664, 1989.

Table 1.

	Group	0 min	Time 1 min	Convulsion
SAP (mmHg)	C (n=8)	128±3	126±3	126±3
	E (n=8)	124±2	183±2 Δ #	189±3 Δ #
	E+SNP (n=7)	128±4	129±4 *	134±4 *
	N (n=8)	128±2	188±2 Δ #	190±2 Δ #
	N+SNP (n=7)	127±3	127±2 *	128±3 *
	P (n=8)	131±3	187±1 Δ #	193±2 Δ #
	P+SNP (n=7)	129±3	127±3 *	132±4 *

Values are mean±SE. C: control, E: epinephrine, N: norepinephrine, P: phenylephrine
Δ: p<0.01 vs 0 min, #: p<0.01 vs C, *: p<0.01 vs without SNP



A718

TITLE: A NEW APPROACH TO NEUROLYTIC BLOCK OF THE CELIAC PLEXUS:TRANS-INTERVERTEBRAL DISC TECHNIQUE

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Introduction: Technical difficulties are frequently encountered and complications such as kidney puncture cannot be completely avoided with the conventional approach to celiac plexus block. The purpose of this study was to examine a new technique for celiac plexus block which has been developed in our institution: trans-intervertebral disc approach.

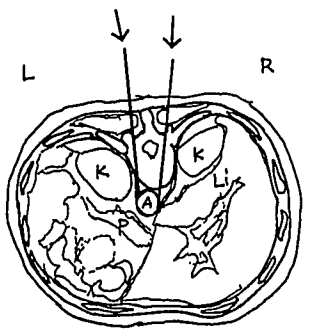
Method: After institutional approval and informed consent were obtained, trans-intervertebral disc celiac plexus block was performed on 42 patients (age 35-80). All patients were suffering intractable upper abdominal pain due to malignancy. Our approach for celiac plexus block was as follows: with the patient in the prone position, the skin was prepped and 1% lidocaine was injected at the needle insertion sites, 3.5 to 5 cm bilateral to the midline and at the level of the tip of the L1 spinous process. Under fluoroscopy, a 23 gauge 15cm long needle was inserted through both right and left insertion points towards the midline (Fig). When the tip of the needle encountered the intervertebral disc, the needle was further advanced until the needle tip just penetrated the disc. This was determined by the loss of resistance technique. The location of the needle was verified by X-ray before and after instillation of 5ml of 1% lidocaine with 4ml of contrast medium. After pain relief was obtained, 99.5% ethyl alcohol 10-20ml was injected through the needle.

Results: All 42 patients obtained complete pain relief immediately after the block. Incidence of aortic puncture was 50% without sequelae and 17 patients developed mild hypotension. Puncture of the intervertebral disc did not result in any complications.

Discussion: Celiac plexus block using a conventional technique such as the transaortic approach (1) and trans-crural approach (2) sometimes cannot be performed on patients who have organomegaly or anatomical anomalies such as deformity of the vertebrae. The success rate of our technique using the transintervertebral disc approach was 100% with no severe complications. In conclusion, the technique of neurolytic celiac plexus block using the trans-intervertebral disc approach was found to be reliable and safe with a low incidence of complications.

References: (1)Pain 16:333-341, 1983
(2)Int Anesth Clin 16:157-162,1978

Arrows indicate needle direction.



K: kidney
A: aorta
P: pancreas