ASA ABSTRACTS

A358

Title: Lidocaine Sensitive Electrode

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INTRODUCTION: The measurement of plasma drug concentration is a therapeutic tool widely applied in drug dosage adjustment. Plasma concentration of lidocaine is used to be assayed by flurorescence polarization immunoassay, RIA, HPLC. These methods, however, are complicated and expensive for routine use. We developed a coated wire lidocaine sensor. This electrode is not only small size but also enables to measure continuously. In the following, we report some properties of this electrode.

METHODS: The coated wire electrode sensitive to lidocaine cation was prepared as follows: the copper wire (0.8 mm^{ϕ}) was coated with the PVC-membrane including 5 mg of ion-pairs of tetraphenylborate ion with protonated lidocaine cations, 100 mg of dioctylphtalate, 110mg of PVC and 1.5 ml of tetrahydrofuran. The electromotive force was measured with ion-meter (PHL-40, DKK, Tokyo) in constantly stirred solution. The temperature was controlled by circulating water from a thermostatic bath through a double-wall beaker. The electrode was examined on the dose-electorical responses, effects of pH and ionic selectivities. Interaction of lidocaine with albumin was also investigated.

RESULTS: The Nernstian response of this lidocaine electrode was 55.9 mV / decade and the standard relative deviation of slope was 0.9 %. The response time of the electrode was within 30 seconds in the lidocaine concentration range of 10^{-2} to 10^{-5} mol / l. Reproducibility of electrode potential was confirmed by increasing or decreasing lidocaine concentrations. Fluctuations of the electrode potentials were within 1 % with the concentration changes from 10^{-4} to 10^{-2} mol / l or from 10^{-2} to 10^{-4} mol / l. The pH in the solution did not affect the electrode potentials within the pH ranges 2.1 - 7.7. In order to exclude the influences of other cations and various substances contained in the body fluid, selectivity coefficients (SC) for the lidocaine electrode were determined. None of the investigated species were found to interfere the electrode potentials, as shown by the very small values of SC (from -2.36 of $CaCl_2$ to -4.28 of urea). The result of the interaction of lidocaine with albumin revealed number of binding site of lidocaine per one albumin molecule and the dissociation constant. They were 0.6 and 849.6 respectively.

DISCUSSION: Coated wire electrode sensitive to lidocaine were prepared. The electrode exhibits a linear response with Nernstian slope within the lidocaine concentration range 10^{-4} to 10^{-2} mol / l. This electrode has a good reproducibility, stability, short responce time and small selectivity coefficients. The concerned electrode is expected to give useful information concerning molecular mechanism and pharmacokinetics of local anesthetics on applying as a sensor in direct potentiometry and potentiometric titration of lidocaine. Furthermore, this type of electrode can be miniaturized easily, so it will be applied for *in vivo* sensor to monitor blood lidocaine concentration continuously.

A359

TITLE: CHANGES IN PROXIMAL AND DISTAL SODIUM REABSORPTIONS DURING STANDARDIZED ANESTHESIA AND SURGERY IN MAN.

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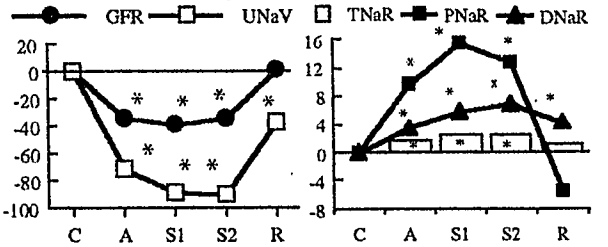
Anesthesia and surgery are known to induce sodium retention. However the exact renal location of this Na retention have never been studied in human. The aim of this study was to differentiate segmental Na tubular transport by measuring separately proximal (PNaR) and distal (DNaR) Na fractional reabsorptions during standardized anesthesia and surgery.

The protocol carried institutional approval and informed consent from each patients. 8 patients (3 males, 5 females) of ASA grade 1 and aged 48.9 ± 3 years were scheduled for cholecystectomy (cholelithiasis). Patients had normal renal function before surgery. Standardized anesthetic procedure was: premedication by 100 mg of hydroxizine, then induction by bolus dose of Propofol (Pf) (2 $mg.kg^{-1}$) associated with fentanyl (0.2 $mg.kg^{-1}$) and vecuronium bromide (0.08 $mg.kg^{-1}$) followed by an infusion of Pf (3-6 $mg.kg^{-1}.h^{-1}$) with nitrous oxide (50% inspired). Fentanyl (0.05 mg) and vecuronium bromide were injected if necessary. Patients were under mechanical ventilation and were extubated immediately after the end of surgery. Intravenous fluid replacement (0.9% sodium chloride solution) was approximatively 500 $ml.h^{-1}$. The duration and the procedure of surgery were similar in all cases. Urine and blood samples were collected before anesthesia (C), after anesthetic induction and just prior to the begining of surgery (A), at the time of cholecystectomy (S1), before the end of surgical procedure (S2), and thereafter 2 hours after the end of surgery (R). The following parameters were measured or calculated: Heart rate (HR), mean blood pressure (MBP), inulin clearance (GFR), urinary Na excretion (UNaV), total (TNaR) and PNaR (lithium clearance) fractional Na reabsorptions, plasma renin activity (PRA), aldosterone (PA) and atrial natriuretic factor (ANF). Clearances and fractional reabsorptions were calculated from the standart formula. DNaR was calculated as distal reabsorption of end proximal Na delivery. Results were exprimed as mean ± SEM. Wilcoxon test was used to compare data (* $p < 0.05$ vs C).

	C	A2	S1	S2	R
HR	73±4	60±2*	66±2	61±2*	64±3*
MBP (mmHg)	93±4	82±4*	96±5	100±4	95±6

Baseline values were: UNaV=366 µmol/min; GFR=102 ml/min/1.73m^2; TNaR=97.2%; PNaR=68.0%, DRNa=91.3%, PRA=88 ng/l/min, PA=168 pmol/l and ANF=54 pg/ml. The following figures show the percent changes in GFR and Na tubular transports during anesthesia, surgery and recovery.



The percent changes in PRA, PA and ANF were respectively 191*, 107* and -18% at A; 175*, 235* and -11% at S1; 134*, 293* and 5% at S2 and -8, 164* and 50% at R.

During anesthesia and surgery, the severe decrease in UNaV is due to both a decreased GFR and an increased TNaR. Both PNaR and DNaR participated to the increase in TNaR. At R, GFR and PNaR returned to control levels but UNaV remained lower because of persistant high DNaR. Hormonal changes rather than the slight observed changes in MBP could account for this intense Na reabsorption in the whole nephron segments.

1-Renal Physiol, Basel 10:261-271, 1987.

2-Anesthesiology 71:260-277, 1989