need to silence the alarm before searching for the cause. If the cause has not been remedied in this period, then the alarm is repeated, and urgency is indicated by an alarm that repeats its signal at faster speed and higher pitch, rather than louder. The total number of such sounds would be limited, since studies in civil aviation have shown diminished ability to recall the significance of more than seven sounds,[7] and pilots believe that all noncritical alarms should be silenced during high workload periods.[8]

Since heart rate, arterial blood pressure, and oxygenation are such fundamental aspects of patient care that anesthesiologists should be continually aware of these variables, it is questionable that these monitors actually need auditory alarms that are so often spurious. If these monitors are fitted with alarms, then the results of this study would suggest that the situation could be improved by using the newer type of alarm sounds outlined in the previous paragraph. This is in contrast to alarm devices such as disconnection alarms or inspired oxygen monitors, which rarely sound, but, when they do so, are likely to denote extreme patient hazard. With an integrated approach to monitoring and alarm systems as a whole, some alarms may be able to be omitted, with a reduction in the number of auditory alarms and an increase in their significance. Gaba *et al.*[9] stated that this approach needs to be validated, and alarms improved so that their benefits clearly outweigh their annoyance and potential confusion. The anesthesiologist's vigilance and ability to integrate information remains the most important source of patient monitoring. We conclude that there is an unacceptably high incidence of spurious alarms during routine anesthesia monitoring.

References

1. O'Carroll TM: Survey of alarms in an intensive therapy unit. Anaesthesia 41:742–744, 1986
2. Sury MR, Hinds CJ, Boustred M: Accidental disconnection following inactivation of Serventilator alarm. Anaesthesia 41:91, 1986
3. McIntyre JWR: Ergonomics: Anaesthetists' use of auditory alarms in the operating room. Int J Clin Monit Comput 2:47–55, 1985
4. Cooper JB, Couvillon LA: Accidental breathing system disconnections, Interim Report to the Food and Drug Administration. Cambridge, Arthur D. Little Inc, 1983
5. Emergency Care Research Institute Health Devices. 16:39–44, 50–51, 1987
6. Hyman WA, Drinker PA: Design of medical device alarm systems. Med Instrum 17:103–106, 1983
7. Patterson R: Guidelines for auditory warning systems on civil aircraft, Civil Aviation Authority Report 82017. London, Department of Transport, 1982, 67
8. Veitergruber JE, Doucek GP, Smith WD: Aircraft alerting systems criteria study, Federal Aviation Authority Report FAA Rd-76-222. Washington, Federal Aviation Authority, 1977, 57
9. Gaba GM, Maxwell M, DeAnda A: Anesthetic mishaps: Breaking the chain of accident evolution. ANESTHESIOLOGY 66:670–676, 1987

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Prevention of Hypokalemia during Axillary Nerve Block with 1% Lidocaine and Epinephrine 1:100,000

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We observed a patient in whom a decrease in amplitude of the T wave on the ECG occurred after axillary block with 1% lidocaine and 1:100,000 epinephrine with associated hypokalemia.

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Key words: Anesthetics, local: lidocaine. Anesthetic techniques: axillary; regional. Ions: hypokalemia. Sympathetic nervous system: catecholamines; epinephrine.

Following the initial observations of Bachromejew[1] and D'Silva,[2] epinephrine produced an initial, transient increase in plasma potassium (K^+) concentration, followed by a more prolonged decline in animals.[3,4] To determine when this finding applies to humans, we investigated the influence of propranolol on changes in plasma K^+ and ECG induced by axillary nerve blockade with lidocaine and epinephrine.

Materials and Methods

Thirty adult patients of both sexes undergoing elective brachial operations under axillary blockade were studied. The subjects were free of metabolic and cardiovascular disease and electrolyte disorders, and each gave informed consent to the study. Secobarbital (75–100 mg) was given im 1 h prior to the anesthetic blockade. On arrival in the operating room, a 5% fructose solution with balanced electrolytes was started iv, and infused at a rate of 100 ml/h. Control arterial blood pressure, heart rate, and lead II of the ECG were recorded. ECG was continuously monitored and arterial blood pressure and heart rate were recorded every 5 min during operation and arterial blood sampling accomplished before blockade in all patients. Patients were randomly allocated to two groups (each n = 15). In group 1, axillary block using 1% lidocaine with epinephrine 1:100,000 was performed (control group). In group 2, 2 mg of propranolol were given iv 5 min before an identical axillary blockade. All nerve blocks in this study were performed by one of the authors (YT). At 15, 30, 60, 90, and 120 min after block in both groups, ECG was recorded and femoral arterial blood sampling was done. Arterial blood gases were analyzed and blood glucose, plasma K^+, Cl^-, and Na^+ were also measured.

The group data were compared using unpaired Student's *t* test and analysis of variance (ANOVA). If ANOVA showed significant differences between groups, Tukey test was employed. *P* values less than 0.05 were considered significant.

Results

Results are expressed as the means ± SD. There were no significant differences between the groups regarding patients' age, gender, or body weight. Mean ages were 32 ± 16 and 30 ± 15 yr and mean body weight 63 ± 12 and 59 ± 9 kg in groups 1 and 2, respectively. The male/female ratio was 13/2 in both groups. The mean doses of lidocaine and epinephrine given were 6.9 ± 1.9 mg/kg and 6.9 ± 1.9 μg/kg in group 1, and 6.6 ± 1.5 mg/kg and 6.6 ± 1.5 μg/kg in group 2, respectively. There was no significant difference in the doses of lidocaine and epinephrine between groups.

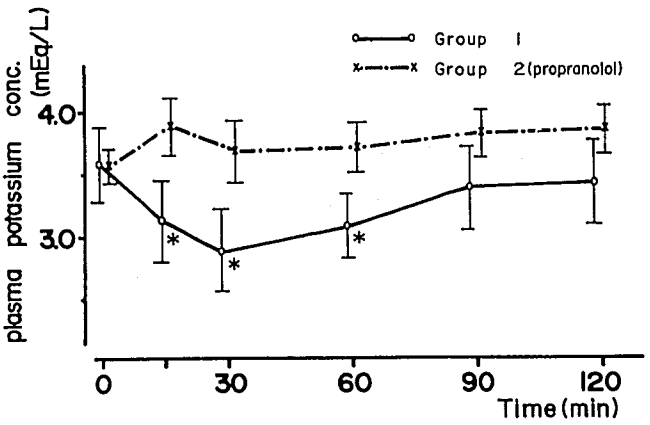

FIG. 1. Changes in plasma potassium concentrations after axillary blockade. Group 1: no pretreatment with propranolol. Group 2: pretreatment with propranolol. Mean plasma K^+ were decreased significantly in group 1 at 15, 30, and 60 min after blockade (*$P < 0.05$).

There was no significant difference in plasma (K^+) before blockade between groups. Mean plasma K^+ was 3.6 ± 0.3 mEq/l before blockade and significantly de-

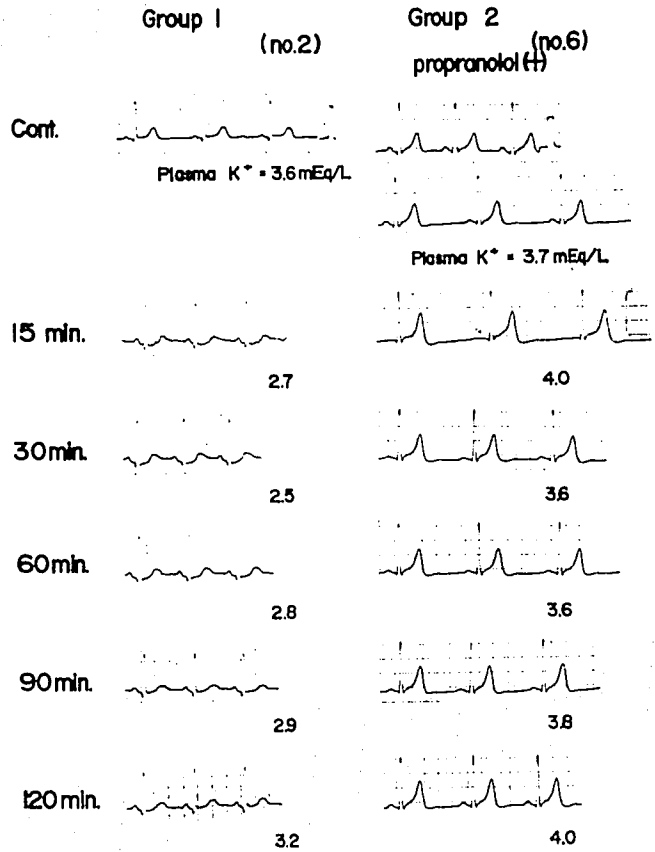

FIG. 2. Examples of ECG changes after axillary blockade. Group 1: no pretreatment with propranolol. Group 2: pretreatment with propranolol. Decreases in the ampulitude of the T wave in group 1 were accompanied by the plasma potassium changes.

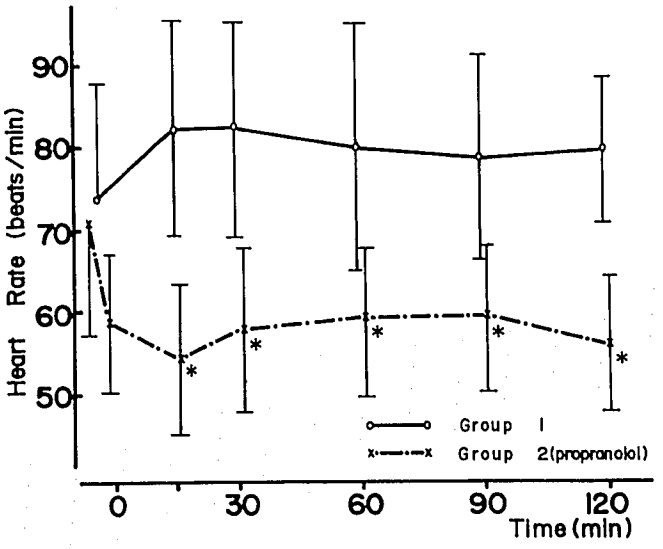

FIG. 3. Changes in heart rate after axillary blockade. There was a significant difference in heart rate between groups (*$P < 0.05$).

creased to a maximum level of 2.9 ± 0.3 mEq/l at 30 min after blockade and returned to control values at 120 min in group 1. There was no significant change in K^+ in group 2 (fig. 1).

The amplitude of the T wave was decreased in all cases of group 1 at 30 min after blockade, but slightly increased at the same time in group 2. A U wave was observed in ten cases (67%) of group 1 at 30 min after blockade, but not observed in group 2 (fig. 2). Dysrythmias were not seen, and no patients complained of chest pain in either group during the study.

There was a significant difference in heart rate between groups 1 and 2 as shown in figure 3. There were no significant differences in the degree of change in arterial blood pressure, arterial blood gases, blood glucose, plasma sodium, and chloride between groups.

DISCUSSION

To slow the absorption of local anesthetics from the site of injection, vasoconstrictors have been added to local anesthetic solutions. For this purpose, epinephrine is the most commonly used drug. While the optimal concentration has been controversial, most authorities now agree that 1:200,000 is the most acceptable.[5] Although the concentration of epinephrine (1:100,000) used in the present study was higher than commonly advocated, this amount has been used for more than 25 yr in Japan, and is thus prepared by a pharmaceutical company (ASTRA, Japan).

The principal side effects of epinephrine are arterial hypertension, bradycardia or tachycardia, and cardiac dysrythmias. These reactions are more likely to occur if the local anesthetic solution with epinephrine is accidentally injected iv.[5] With respect to the ECG in normal persons, epinephrine decreases the amplitude of the T wave in all leads. In animals given relatively larger doses of epinephrine, additional effects are seen on the T wave and S-T segment. After an initial decrease in amplitude, the T wave may become biphasic and the S-T segment deviates either above or below the isoelectric line before abnormal ventricular complexes appear. Such S-T segment changes are similar to the downward displacement found in patients with angina pectoris during spontaneous or epinephrine-induced attacks of pain. Accordingly, these electrical changes have been attributed to myocardial ischemia.[6] However, Struthers *et al.*,[7] in their study on infusion of epinephrine intravenously in man, showed that circulating epinephrine may induce dysrhythmias both directly, *via* changes in ventricular repolarization, and indirectly, *via* epinephrine-induced hypokalemia.

In laboratory animals, intravenous administration of epinephrine produces an initial, transient increase in plasma K^+ concentration followed by a more prolonged decline to levels below controls; the duration of initial K^+ increase is about 5 min.[1-4,8] The initial hyperkalemic phase of the response, however, has not been well documented.[4] Only one study[9] shows that a consistent, minimal early increase in plasma K^+ occurs, while, in two others,[10,11] small increases in K^+ were observed in only a minority of subjects studied. No increase was recorded by several other workers.[12-16] Examination of the methodology in these studies revealed that the epinephrine had not always been administered intravenously (intramuscular or subcutaneous routes having been used); the times at which the first sample was taken varied from 0.5–15 min; and analyses had usually been performed on peripheral venous samples.[4]

In our study, epinephrine was not given iv, and sampling was done from the femoral artery; the first sample was taken 15 min after injection. We did not measure plasma K^+ during the 5 min after blockade, and ECG changes did not develop during that time. Vick *et al.*[8] concluded that epinephrine acts directly to increase uptake of K^+ by both liver and skeletal muscle, and that the effects are mediated through adrenergic beta-receptors.

There is general agreement that epinephrine causes a decrease in plasma K^+ in humans[10-13,15,17] that can be abolished by non-selective beta-adrenoceptor blockade.[8,16] Recently, Struthers *et al.*[18] and Brown *et al.*[19] demonstrated that hypokalemia induced by epinephrine was prevented by $beta_2$-blockade. In our study, we prevented the development of hypokalemia and ECG changes in group 2 by pretreatment with propranolol. Thus, we observed no cardiac dysrhythmias, although a decrease in amplitude of the T wave occurred and a U

wave developed, even though hypokalemia was present. These benign responses might have been a result of an antiarrhythmic action of lidocaine.

The results of the present study indicate that the decrease in amplitude of the T wave, development of U waves, and hypokalemia after axillary blockade with lidocaine and epinephrine were induced by epinephrine, and that these were prevented by pretreatment with propranolol. Hypokalemia can be associated with a variety of cardiac dysrhythmias. On the basis of our findings, we recommend that the ECG should be monitored during blockade, and that pretreatment with beta-adrenergic blockade may be beneficial when local anesthetics with epinephrine are given. Also, epinephrine should be avoided in patients with K^+ deficiency.

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REFERENCES

1. Bachromejew IwR: Über den Komplex: Innensekretion-Nerven-Ionen. Die Beteiligung des Schilddrusenapparates an der Regelung der Ca- und K-Ionen. Pflugers Arch 231:426–441, 1932
2. D'Silva JL: The action of adrenaline on serum potassium. J Physiol (Lond) 82:393–398, 1934
3. Ellis S: The metabolic effects of epinephrine and related amines. Pharmacol Rev 8:486–562, 1956
4. Lim M, Linton RAF, Band DM: Continuous intravascular monitoring of epinephrine-induced changes in plasma potassium. ANESTHESIOLOGY 57:272–278, 1982
5. Cousins MJ, Bridenbaugh PO: Neural Blockade in Clinical Anesthesia and Management of Pain. Philadelphia, JB Lippincott, 1980, p 103
6. Winer N: Norepinephrine, epinephrine and the sympathomimetic amines, The Pharmacological Basis of Therapeutics. Edited by Goodman LS, Gilman A, Rall TW, Murad F. New York, Macmillan, 1985, pp 145–180
7. Struthers AD, Reid JL, Whitesmith R, Rodger JC: Effect of intravenous adrenaline on electrocardiogram, blood pressure, and serum potassium. Br Heart J 49:90–93, 1983
8. Vick RL, Todd EP, Luedke DW: Epinephrine-induced hypokalemia: Relation to liver and skeletal muscle. J Pharmacol Exp Ther 181:139–146, 1972
9. Brewer G, Larson PS, Schroeder AR: On the effect of epinephrine on blood potassium. Am J Physiol 126:708–712, 1939
10. Allot EN, McArdle B: Further observations on familial periodic paralysis. Clin Sci 3:229–239, 1937
11. Pekkarnien A, Hortling H: The effect of continuous intravenous infusion of adrenaline on the circulation and blood chemistry. Acta Endocrinol (Copenh) 6:193–214, 1951
12. Castleden LIM: The effect of adrenaline on the serum potassium level in man. Clin Sci 3:241–245, 1937
13. Keys A: The response of the plasma potassium level in man to the administration of epinephrine. Am J Physiol 121:325–330, 1938
14. Jacobson WE, Hammarsten JF, Heller BI: The effects of adrenaline upon renal function and electrolyte excretion. J Clin Invest 30:1503–1506, 1951
15. Paget M, Routier G: Sur l'hypokaliemie post-adrenalinique. J Sci Med Lille 81:539–549, 1963
16. Massara F, Tripodina A, Rotunno M: Propranolol block of epinephrine-induced hypokalaemia in man. Eur J Pharmacol 10:404–407, 1970
17. Dury A, Holler JW, Smith C: The changes in plasma potassium level after epinephrine in normal human beings and in persons with epilepsy. J Clin Invest 31:440–444, 1952
18. Struthers AD, Reid JL, Whitesmith R, Rodger JC: The effects of cardioselective and non-selective β-adrenoceptor blockade on the hypokalaemic and cardiovascular responses to adrenomedullary hormones in man. Clin Sci 65:143–147, 1983
19. Brown MJ, Brown DC, Murphy MB: Hypokalemia from beta$_2$-receptor stimulation by circulating epinephrine. N Engl J Med 309:1414–1419, 1983

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69:112–116, 1988

End-tidal, Transcutaneous, and Arterial p_{CO_2} Measurements in Critically Ill Neonates: A Comparative Study

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Non-invasive estimates of arterial CO_2 (Pa_{CO_2}), such as end-tidal ($PetCO_2$) and transcutaneous CO_2

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Key words: Anesthesia: pediatric. Carbon dioxide: alveolar; arterial; end-tidal; tension; transcutaneous.