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ANALYSIS OF HOLTER MONITORING FOR THE ASSESSMENT OF VENTRICULAR PREMATURE CONTRACTIONS AND THE EVALUATION OF DRUG EFFICACY -ESTIMATION OF AN EFFICIENT METHOD OF ANALYSIS-

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To establish the plain and correct method for assessing ventricular premature contractions (VPCs) using Holter monitoring and its clinical application for evaluating drug efficacy, consecutive two or three days Holter monitorings before and after administration of medicine were undertaken in 40 patients and the recordings were analysed using both manual method (M) and the automatic analyser (Aa).

The calculated VPC frequencies obtained from one minute and one hour monitorings, observed value multiplied by 1440 (VPC 1 m $\times$ 1440) and observed value multiplied by 24 (VPC 1 h $\times$ 24), respectively, were significantly different from the observed value by M (VPC-M) ($p < 0.001$). VPC counts of Aa had a significant correlation with VPC-M ($r = 0.820$, $p < 0.001$), but they were also significantly different from each other ($p < 0.01$). Sum total of the number of VPCs for one minute in every one hour multiplied by 60 (Σ VPC 1 m $\times$ 60) correlated significantly with VPC-M ($r = 0.933$, $p < 0.001$) and no difference was remarked between them. The 95% confidence intervals of variability between first and second day regarding VPC-M was 88% and the characteristics of VPCs also varied day by day. The minimal interval of VPC (VPC-MI) had a correlation with VPC-M ($r = 0.574$, $p < 0.001$) and were mainly observed when the drug efficacy was considered to be lost in the medicated stage.

Conclusion 1) The most efficient method to evaluate VPCs is the combination of the physician's pattern analysis of the compression recordings of Holter monitorings at a glance and the use of Σ VPC 1 m $\times$ 60 for VPC frequency. 2) Spontaneous day by day variations of VPCs are remarkable and a 24 hours monitoring is insufficient to evaluate the severity of VPCs and the efficacy of drugs. 3) VPC-MI might be an index for assessing drug efficacy.

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A STUDY OF THE ACCELERATING EFFECT OF VERAPAMIL ON JUNCTIONAL AUTOMATICITY IN PATIENTS WITH SICK SINUS SYNDROME

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In order to evaluate the accelerating effect of Verapamil (Ver) on junctional automaticity (JA), atrial overdrive suppression tests were performed before and after intravenous administration of 5mg of Ver in patients (pts) with SSS. Number of junctional beats and their

recovery time (JRT) during 10 recovery cycles after pacing were measured for each pacing rate. In 15 pts junctional recovery beats were noticed both before and after administration of Ver, enabling comparison of Ver effect on JA. Acceleration of JA with Ver was defined as more than 10% shortening in JRT after administration of Ver. JA was accelerated in 10 pts. Among these, max JRT was shortened from 4256 ± 1474 to 2236 ± 996 mSec, ratio of junctional beats among recovery beats increased from 50.2 ± 22.2 to $80.9 \pm 17.7\%$ ($p < 0.01$), the pacing rate at which Wenkebach block appeared (W-rate) decreased from 152 ± 24 to 133 ± 20 BPM, max JRT during pacing rates less than W-rate also shortened from 3542 ± 1382 to 2208 ± 1018 mSec ($p < 0.01$) and systolic arterial pressure during 1:1 AV conduction pacing changed from 134 ± 34 to 122 ± 17 mmHg ($n=6$, N.S.). The 10 pts were divided into two groups according to the manner of acceleration of JA. After administration of Ver 7 pts showed lesser fluctuations in JRT (< 400 mSec) over different pacing rates [Group I] while the other 3 showed larger fluctuations (> 1500 mSec) [Group II]. Max JRT of group I and II were 1667 ± 461 and 3540 ± 468 mSec ($p < 0.01$), respectively. Lack of warming up of JA was noted in 3 of group I and none of group II. Organic heart disease was noted in 6 of group I and none of group II.

Thus Ver accelerated JA in 10/15 pts with SSS. Although the mechanism of acceleration is unknown, suppression of AV node conduction and fall in arterial pressure with Ver did not seem to play a major role. And the two distinctive manner of JA acceleration noted in the 10 pts suggests involvement of at least two mechanisms.

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EFFECT OF ADENOSINE-5'-TRIPHOSPHATE ON ATRIOVENTRICULAR REENTRANT TACHYCARDIA IN INFANCY AND CHILDHOOD

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The effects of adenosine-5'-triphosphate (ATP) on atrioventricular reentrant tachycardia (AVRT) were studied in 6 patients aged 2 months to 6 years. There were 3 patients with WPWdynftomr, 2 of them being complicated by Ebstein's anomaly. Nineteen of 22 paroxysms were terminated by a rapid intravenous injection of ATP of 2 to 15 mg with a mean dose of 0.78 ± 0.48 mg/kg (SD). Termination of the tachycardia occurred at 7 to 17 sec after the injection, with a mean value of 10.3 ± 3.0 sec and was caused by a blockade of the antegrade A-V conduction. Immediately after the termination of AVRT, transient sinus bradycardia was observed in all cases and A-V block developed in one. Two patients complained brief chest discomfort. All of these lasted very short and did not require any treatment. These results suggest that ATP is a safe and effective drug for quick termination of AVRT in infants and children. Emphasis should be placed on the rapid bolus injection of the drug. Also, pretreatment with sedatives appeared to give higher success rate.

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ELECTROPHYSIOLOGIC EFFECTS AND THERAPEUTIC USE OF ADENOSINE AND ATP FOR TERMINATING PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

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