microelectrode voltage-clamp. Activation of PKC was induced by phorbol ester PMA (1μM).
Results: an inhibitory effect could be observed in all homomeric channels. In homomeric Kir2.1 channels inhibition was only weak (-10%). However, in homomeric Kir2.2 channels (-81%) and in homomeric Kir2.3 channels (-58%) the inhibitory effect was very pronounced. Heteromeric channels exhibited differing reactions. In Kir2.1/Kir2.2 heteromers (-24%) and Kir2.1/Kir2.3 heteromers (-10%) the inhibitory effect was markedly reduced. However, in Kir2.2/Kir2.3 heteromers the inhibitory effect was very pronounced (-88%). With Kir2.2 mutant channels lacking PKC phosphorylation sites we investigated the relevance of the different subunits of the multimeric channels. We found that co-expression of the mutant channels with wild-type channels resulted in a marked reduction of the inhibitory effect. We propose that phosphorylation of several subunits of one multimer is necessary to accomplish the inhibitory effect.
Conclusion: Kir2.x heteromeric channels are inhibited by a proteinkinase c dependent phosphorylation. Kir2.2 channel subunits play a major role in this regulation, whereas Kir2.3 channel subunits contribute to a lesser extent. The inhibitory effect probably occurs only after phosphorylation of several subunits. These results elucidate further details of the molecular mechanisms of adrenergic regulation of cardiac IK1 current.

351
Effect of cilnidipine and amlodipine on QT dispersion in patients with hypertension

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Purpose: QT dispersion has been used as a marker for life threatening ventricular arrhythmia. This type of arrhythmia associated with an excessive QT dispersion has been reported with anti-arrhythmic drugs, but it has not yet been reported with Ca antagonist. We therefore evaluated the changes in QT dispersion during the administration of cilnidipine and amlodipine.
Methods: forty-one untreated patients with essential hypertension had QTd measured from a surface 12-lead electrocardiogram, and two-dimensional echocardiography performed to measure LVM. We investigated the effects of 6 months of treatment with the cirnidipine (a novel L+N type calcium channel blocker, n=20) and the amlodipine (n=21) on left ventricular mass (LVM) and QT dispersion (QTd) in a single-blind study.
Results: there are no significant differences in the lowering of BP between cilnidipine and amlodipine at 6 months of treatment. The LVM were reduced significantly with both drug. The QT dispersion were reduced significantly with both drug, but there was a consistent trend toward greater reduction in the cilnidipine group. This effect was a correlation between the change in QTd and change in SBP in amlodipine group that was not seen in the cilnidipine group. In cilnidipine group, we found correlation between the change in QTd and change in norepinephrine level. Also, our data demonstrated significant relationships between QTd and LVM with both drugs.
Conclusion: this favorable effect of cilnidipim and amlodipine may reduce sudden cardiac death in high-risk hypertension patients.

352
Is the delta wave consequence of the annular or non-endocardial accessory pathway insertion?

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The slow onset of the delta wave has been suggested to be related to an AV annular insertion of the accessory pathway, leading to a late engagement of the His-Purkinje system by the activation wavefront. To test this hypothesis, we compared QRS complexes recorded during maximal preexcitation and during ventricular pacing at the successful endocardial ablation site in the mitral annulus.
Methods: A 12 lead maximally preexcited ECG was recorded while pacing (400-600 ms) the atrium from the coronary sinus in 41 patients. All patients had a single overt left free wall AV accessory pathway, which was successfully ablated from the endocardial ventricular side of the mitral annulus. A second ECG was recorded while pacing at the successful ablation site at the same cycle length. Maximally preexcited (QRSpx) and paced (QRSpc) QRS complex morphologies were compared to determine the number of leads with major, and the number of leads with minor changes (Ch). QRS complex duration (QRSd) and intrinsic deflection time (IDt) were analysed at 50-200 mm/sec and measured in ms.
Results: see table

	Major Ch	Minor Ch	QRSd	IDt V2
QRSpx			167.1±16.1	92.5±12.2
QRSpc			150.2±12.4	64.8±15.1
	0.2±0.5	1.7±0.9	p<0.001	p<0.001

Conclusion: The slow onset of the delta wave in the Wolff-Parkinson-White syndrome should be explained by mechanisms other than an AV annular insertion of the accessory pathway.

353
Immediate effects of atrial contribution loss in sequentially paced patients demonstrated by non-invasive monitoring using the Finometer device

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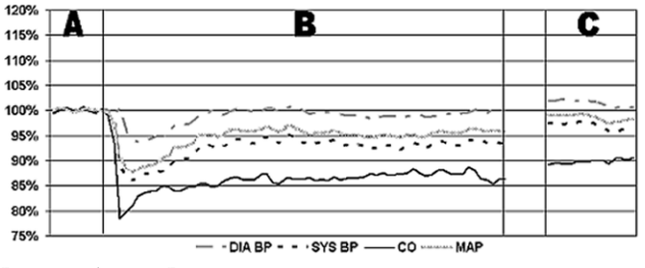
Introduction: the aim of the study was to find out which of the output parameters of a non-invasive finger arterial pressure evaluation device (Finometer®) is most sensitive to an abrupt change in systolic output of the left ventricle, induced by loss of atrial contribution.
Patients and Methods: a total of 15 hemodynamically stable patients aged 67±15 years were enrolled. All patients had dual chamber pacemakers enabling alternation between an atrioventricular delay (AVD) 170ms (period A) to an AVD 50ms (period B), while maintaining a constant heart rate and constant RR intervals. Beat-to-beat changes in systolic (sBP), diastolic (dBP) and mean (MP) arterial pressure and cardiac output (CO - calculated by the device from the shape of the individual pressure waves) were analyzed in a period of seven minutes in each setting.
Results: after the switchover, an average drop in CO (22%), sBP (15%), dBP (7%) and MP were recorded (period B). At the end of the seven-minute period (period C) all parameters returned to initial values apart from CO (lower by 9.4%, P < 0.001).



Percentage change vs Beats

Conclusions: the abrupt fall in atrial contribution causes a drop in arterial pressure and CO. While the CO remains decreased, arterial