ABSTRACTS

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A COMPUTER-BASED EXPERT SYSTEM FOR CONSULTATION ON THE USE AND INTERPRETATION OF HIGH-RESOLUTION ELECTROCARDIOGRAPHY

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Although High-Resolution Electrocardiography (HR-ECG) is commonly used by electrophysiologists to evaluate patients for the presence of substrates for arrhythmia, its clinical role and proper interpretation remain confusing for the non-specialist. We therefore created a computer-based expert system which assists in interpreting the findings of HR-ECG studies, provides consultation on the significance of the findings for a given patient, and allows interactive access to the related medical literature. Information was cross-referenced from 209 current articles on HR-ECG, and the prospective studies pertaining to risk of significant ventricular arrhythmia after MI and as a cause for syncope were reviewed. A risk table was developed from the pooled data, utilizing ejection fraction, and findings on HR-ECG and Holter monitor to predict a patient's risk of arrhythmia.

The expert system was developed using a commercially-available expert system building tool running under the MS-DOS operating system. The risk of significant ventricular arrhythmia (either after MI or as a cause of syncope) is taken as the goal of the analyais. Supporting data is gathered as needed by the process of backward chaining. The user interface is menu driven, and requires a minimum of typing. An extensive body of knowledge is available during a consultation session in the form of hypertext, providing explanations of terms, and descriptions of the reasoning used. All conclusions are supported by references to the relevant medical literature, and can be printed out along with the associated abstracts, if desired. Intermediate results and forecasting can be displayed as tables which highlight the data from the prospective studies which are relevant to the specific patient being evaluated. This consultation system can provide a useful tool to make the expertise on the proper use and interpretation of HR-ECG widely available to all clinicians.

COMPUTER MODELS TO PREDICT DEATH, COMPLICATIONS, LATE DEATH, AND OTHER LATE EVENTS IN PATIENTS UNDERGOING CARDIAC INTERVENTIONS

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We have developed a generalized PC based computer program to display a graphical representation of survival and other end points for patients undergoing therapeutic cardiovascular procedures. The program will allow several patient profiles to be entered, and the resulting survival curves to be overlayed.

Data for this demonstration of the program was retrieved from the Emory University Cardiac Data Bank software system - CCARD (Cardiac Clinical And Research Data base). The Cardiac Data Bank uses a Hewlett Packard 9000/835 to store clinical information for reporting and clinical research. The data base now contains 88,138 catheterization forms, 15,792 PTCA forms, and 21,488 coronary surgery forms. Systematic data collection at Emory began in 1973.

Outcome data has been developed for PTCA, coronary surgery, and valvular surgery. For late events and late death analysis, a Cox proportional hazards regression model was developed. The events considered here include myocardial infarction, reoperative surgery, and coronary angioplasty. A number of prognostic factors were selected that were determined to be predictors of patient survival. The program applies the covariate values to the hazard function vector and then proceeds to create a survival curve for that patient profile. Multiple survival eurose can be created and overlayed to examine the effect of variation in values of individual covariates. For in-hospital death analysis, stepwise logistic regression analysis was performed. All data analysis was performed using the BMDP statistical analysis program.

A PC based computer program was developed to integrate these sets of statistical information with an easy to use interface to examine the relationships of the various prognostic factors. A menu allows the user to select the analysis, and then prompts for values for each of the prognostic factors.

The program is written in Borland Turbo C 2.0 and Gnuplot 2.0 is used to display the curves and other data.

PREDICTION OF ARRHYTHMOGENIC AREAS IN INFARCT-RELATED VENTRICULAR TACHYCARDIA—A COMPUTERIZED SHORT-CUT

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Successful surgical or catheter ablation of ventricular tachycardia (VT) requires obtaining detailed mapping information, which can take large amounts of time. If mapping could be targeted to sample likely arhythmogenic regions, mapping time could be decreased and accuracy enhanced. A method has previously been devised to predict the site of origin (SO) of VT in a large proportion of patients with infarction-related VT, using four features present on the 12-lead ECG in VT: infarct location; bundle branch block pattern; QRS axis; precordial R wave progression (1 of 8 patterns). The algorithm developed was accurate, but complex and difficult to apply; a computerized (Macintosh) version has now been developed (based on a database of over 300 endocardially-mapped [catheter and/or intraoperative] VT's correlated with their corresponding 12-lead ECGs) in which the operator checks off features of the ECG in VT and the program graphically displays (on a schematic diagram of the left ventricular endocardial region, and can thus facilitate mapping by directing catheter placement toward areas likely to be VT-SO's. The program is designed to serve not only as a clinical tool to optimize endocardial mapping in infarct-related VT, but to aid in familiarizing the user with left ventricular endocardial fluoroscopic anatomy. Other features include a mini-tutorial for identifying catheter positions on fluoroscopy and a graphical summary of the mapping findings for all 300+VT's in the database.

COMPUTER MODEL OF INTRA-CARDIAC CONDUCTION AND HEART-

PACEMAKER INTERACTION: AN INTERACTIVE EDUCATIONAL VERSION

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Based on the previous experience with an investigational version of a computer model of cardiac rhythm disturbances and heart-pacemaker interaction, a new interactive educational version of this model has been developed and implemented on IBM PC AT compatible computers. This version of the model, written in Turbo Pascal, consists of the main simulation core and of the library of educational segments.

In the core model, the heart tissues are simulated by a network of excitable elements linked together by special connectors. The speed of excitation transmission, duration of repolarisation phase, and their cycle rate dependences are individually programmable for each element. Selected elements may represent the tissue in contact with sensing and pacing electrodes. The interaction between natural intracardiac processes and the actions of an implantable pacemaker are modelled in this way. The core part of the model also generates its output which has two parts: a schematic electrocardiogram and colour coded tracing of the excitation and repolarisation changes of individual elements of the modelled heart.

The educational library consists of segments which correspond to the physiological situation and to selected pathologies of intra-cardiac conduction and heart-pacemaker interactions, such as the pre-excitation, atrioventricular (AV) and AV nodal resentant tachycardias, and the dual chamber pacemaker driven tachycardia. Each segment contains a special network of cardiac structures and initial setting of parameters of individual elements and connectors of the model. The 'anatomical' network of each segment enhances the relevant pathology, such as a conduction block, a pre-excitation fibre, or dual AV nodal pathways.

The initial setting parameters of each segment enables the relevant pathology to be reproduced. In an interactive mode, the user of the package can modify both the network of the modelled tissue and the parameters of individual elements. In this way, different clinical features can be demonstrated, such as the termination of a pacemaker driven tachycardia due to an extension of the ventricular refractory period of the device, initiation of an AV nodal reentrant tachycardia by an atrial extrastimulus, or the lack of AV reentrant tachycardia initiation due to a modification of AV nodal decremental conduction.