Selected Abstracts from the Third Annual Soviet/American Conference on Emergency Medical Care

Introduction

The Third Soviet/American Conference on Emergency Medical Care was held 17–19 May 1991 in the cities of Leningrad [now St. Petersburg], Odessa, and Moscow. Formal presentations of papers were held at the Djanelidze Institute of Emergency Medical Research in Leningrad. While in Moscow, the group of delegates from the United States paid informal visits to the Skifosovosky Center for Emergency Medical Research, the Disaster Medicine Institute, and the Institute for Reanimatology. In all cases, the U.S. delegation was well-received and cordially hosted. The delegation learned much about the Soviet system of medicine, about the current directions of emergency medical research in the Soviet Union, and we were impressed by what our Soviet colleagues were able to accomplish under difficult circumstances.

The Fourth Annual Soviet/American Conference on Emergency Medical Care (Slavic) will be held 29 May through 12 June 1992. Again, the main host will be the Djanelidze Institute for for Emergency Medical Research in St. Petersburg, Russia. The program will be expanded to include three days of formal presentations: one day on surgical/trauma-related emergencies; one day on medical emergencies; and one day on management and administration of emergency services. In particular, the Soviets asked that we include one day on this last topic, in contrast to earlier years (when, because of the great dissimilarities in health-care funding mechanisms, the subject had been avoided) because of their need for rapid reorientation to a market/insurance-based system.

The intermediate stop will be the city of Kiev. Kiev has an important medical research center with which we have arranged an exchange and a visit, and the program remains flexible. In Moscow, our hosts will be the Institute for Reanimatology (involved in basic science, resuscitation, and critical care research) and the Sklifosovosky Institute for Emergency Medicine Research (the main research/teaching institution for emergency medicine in Moscow).

The medical system, is undergoing rapid and massive change. It is our intention to keep open the lines of communication between our medical colleagues in the former USSR and American physicians during this stressful period in the evolution of their country. Anyone who wishes to join us for the Fourth SACEMS may call the office of the Society for International Advancement of Emergency Care (SIAEMC) at (410) 987-5616, or write to SIAEMC, P.O. Box 179, Millersville, MD 21108, USA.

Theodore E. Harrison, MD, FACEP
Society for International Advancement of Emergency Care

Non-Competitive Antagonists in Management of Acute Clonidine Poisoning

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Poisoning by clonidine (C) causes hemodynamic disturbances (hypotension, bradycardia) which are followed by central adverse effects (somnolence, visual reduction), and metabolic displacement (decrease of sympathetic outflow, hyperglycemia, hypoprolactinemia, decreased oxygen consumption).

According to the data of the Regional Poisoning Center (Djanelidze Emergency Institute, St. Petersburg, Russia), the frequency of acute poisoning by C was 5% of all poisoned patients. The ingested dose was about 50 times more than the therapeutic one. Rapid absorption causes rapid onset of drowsiness and myasthenia; a long prehospital period made the admitted patients soporific (Glasgow coma score 14).

Of a total of 80 patients, 20 severe cases were observed (mean blood pressure 60/40 mmHg, heart rate 40±2.4) with reduction of cardiac output to 30%, and peripheral resistance to 18% of normal. The primary cardiac toxicity was sinoatrial (S-A) blockade.

The initial clinical treatment included charcoal administration and supportive therapy (dextrose, vasopressor, forced diuresis). The competitive antagonists—direct (dopamine 15 mcg/kg/min) and indirect (atropine sulfate 15 mcg/kg six times a day), both in loading and maintenance doses removed myocardial depression, but did not improve mental activities (Glasgow coma score 15). Moreover, episodes of subepicardial ischemia (Y1–Y2) following administration did not allow an increase in the dose of antagonists.

Methoproclamide (0.5mg - loading dose + 0.25 mcg/kg/h —maintenance dose) as a selective D2 receptor blocker was considered to be non-competitive antagonist of alpha 2 receptor agonist C. Being infused, it increased cardiac output 8%, moderated some metabolic changes, and activated the consciousness of patients (Glasgow coma score-19).

The simultaneous administration of differentiating antagonists decreased ischemia periods.

Several studies have investigated the results of early activation of mental activities to moderate the manifestation of "post-toxic asthenia" after acute poisoning.

These data indicate that non-competitive antagonists can minimize the toxic consequences of C, limit the hospitalization period by 1.6 times, and may be useful in the prehospital period.

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An Overview of the 5th National Conference on CPR and Emergency Cardiac Care

EMERGENCY CARDIAC CARE UPDATE

April 9-12, 1992

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Pediatric CPR Update - *M. Hazinski, RN*
ECC Training Programs - *J. Billi, MD*
Citizen CPR: What's Left of the Dream Revisited - *A. Braslow, PhD*
The Psychological Impact of Resuscitation on the Rescuer - *R. Swanson, MD*
We are all Elijah's Children - *M. Eisenberg, MD, PhD*
CPR in the Year 2000 - *L. Cobb, MD*
CPR: Lifesaving Therapy or Burdensome Futility? - *T. Crimmins, MD*
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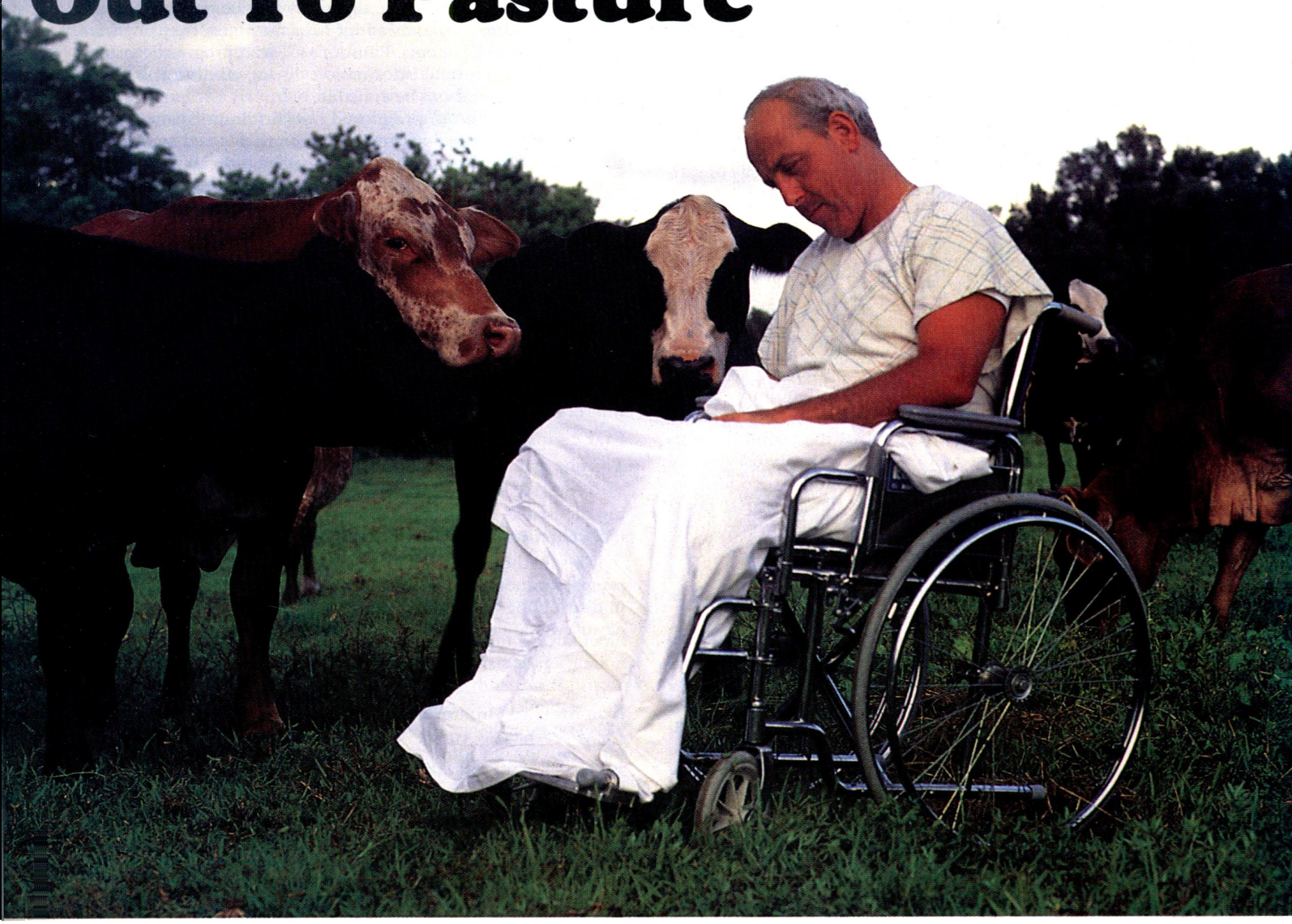

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A Toxicology Screening Algorithm for Prehospital Setting and Emergency Department

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Drug toxicity can be evaluated during the prehospital period by an algorithm that classifies simple mechanisms of toxicity of various drugs with the cholinergic and adrenergic systems.

The study involved three hundred patients who had been poisoned by neurotransmitter acting drugs. Data were gathered on clinical condition, hemodynamics (cardiac output [CO], blood pressure [BP], peripheral resistance[TPR]), and ECG picture. Patients were categorized as either one of two groups. The first group had tachycardia, constricted pupils, and dry skin (*chronopositive* group). The second group had bradycardia, constricted pupils, and moist skin (*chrononegative* group).

The first group presented with: 1) an anticholinergic syndrome (caused by atropine sulfate, H1 blockers, N-central blockers), that included delirium, increased cardiac sounds, sinus tachycardia, slightly increased CO and BP, and almost normal TPR; 2) an adrenergic syndrome (amphetamines, theophylline, ephedrine, L-dopa, etc) with psychosis, flapping first heart sound, systolic murmur, high CO, high BP, and low peripheral resistance (and increased frequency of arrhythmias); or 3) an alpha blockade syndrome (phenothiazines, TCA, etc.) with a delirium-soporific condition, split heart sounds, frequent His bundle blockade, low CO and BP, and low TPR.

The second group was represented by: 1) cholinergic syndrome (opiates, cardiac glycosides, reserpine, etc.) with weakness of the first cardiac tone, bradycardia, low CO, BP, PTR; 2) alpha-2-adrenergic syndrome (clonidine, verapamil, alpha-methadopa), with diminished heart sounds, low CO, BP and PTR, more expressive than the previous group, or 3) beta-blockade syndrome (propranolol, quinidine, etc.) with faint first heart sound, split second heart sound, QRS prolongation, low CO, BP, and high PTR.

The recognition of the two groups can be performed in the prehospital setting and the definition of the specific syndrome done in the Emergency Department. The whole algorithm can be represented as a scale. Sixty special clinical features have been marked and have been placed following the codes for the closer definition (1-convulsions, 5-vomiting, 27-hyperthermia, etc.)

The scale and its algorithm show that poisoning can be described with the help of a computer simulation. This could be helpful both in laboratory investigation and in the clinical assessment and administration of appropriate antagonists.

Evaluation of Peritoneal Exudation by Means of Protein Dilution in Patients with Acute Pancreatitis

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Experimental studies in 50 dogs with different models of acute pancreatitis revealed strict dependency between the rapidity of peritoneal exudation, average time of hemorrhagic necrotic foci formation, and mortality (Spierman correlation ranks + 0.855 and 0.924). The study concluded that the severity of acute peritonitis (AP) influences the incidence of necrotic foci formation and can be quantitatively evaluated by means of coefficient of peritoneal exudation (CPE).

Ransom's early prognostic signs only may be evaluated within 48-hours of admission and cannot help the emergency physician in stratification of patients. Ransom's CT-scan prognostic criteria give much more useful information during admission, but CT-scanning may not always be available.

In such conditions, prognostic classification of patients with AP in the emergency department may be performed by means of CPE evaluation. This method is important especially in the first hours following onset of severe/fulminant AP.

After laparocentesis, 1–2 ml of peritoneal exudate should be aspirated and total protein in this fluid should be estimated (P1). Then, 1-liter of any saline solution used for intravenous (IV) fluid replacement should be introduced into the peritoneal cavity and lavage catheter should be closed for 30 minutes. Afterward, it should be opened and the mixture of exudate and saline should be obtained for the second total protein analysis (P2). The CPE is calculated according to the following formula.

CPE = \1000 x [(P2),(P1 - P2) T])

where: CPE = coefficient of peritoneal exudation (ml/h),

P1, P2 = total protein in the first and second samples (g/l),

T = time from the onset of the disease (hours).

Severe AP was recognized if CPE was more than 10 ml/h during the first 24 hours of the disease. In cases of shock or myocardial infarction, CPE is not informative.

This method was tried in 120 patients and helped to improve prognostication at the time of admission. Frequency of mistakes in distributing patients into mild or severe forms during the first day of AP was decreased from 25% to 13%.

Non-Invasive Monitoring of Ventricular Fibrillation

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Introduction: Several preclinical studies have demonstrated that after five minutes of ventricular fibrillation, initial defibrillation attempts, as recommended by the American Heart Association, do not lead to successful defibrillation with return of spontaneous circulation. In addition, there is increasing biochemical, histological, and clinical evidence to suggest that the cumulative energy imparted to the fibrillating myocardium through defibrillation can cause myocardial injury and ventricular dysfunction. These studies also demonstrate that following 7.5 minutes of ventricular fibrillation, if reperfusion is augmented with epinephrine or a mechanical assist device prior to fibrillation, there is an increase in the percentage of subjects that can be defibrillated successfully, compared to those who are defibrillated prior to reperfusion. Therefore, estimates of the duration of ischemia could help direct therapy during ventricular fibrillation. This study sought to determine whether signal analysis of the electrocardiagram (ECG) during ventricular fibrillation could be used to estimate this time duration during ventricular fibrillation.

Methods: Eleven swine were fibrillated electrically and the ventricular fibrillation (VF) ECG signal was recorded continuously. Following ten minutes of VF, the ECG signal was digitized. The digitized ECG signal then was analyzed using a fast Fourier transform (FFT) analysis. The FFT analysis allowed calculation of the amplitude and median frequency of the VF and ECG.

Results: Although there was a large intersubject variability in the amplitude of the VF-ECG signal, the mean coefficient of variation for the median frequency during the 10 minutes of VF was

small, and permitted modeling of the data. The model for the median frequency estimated VF ischemia times to within +1.3 minutes.

To demonstrate the feasibility of this technology in humans the VF signal was recorded in humans who had a cardiac arrest during Holter monitoring. The FFT analysis of these signals demonstrated that although there are quantitative differences in the ECG signal during VF compared with swine, there are qualitative similarities. Specific frequency parameters demonstrate a dynamic and repeatable pattern with respect to time during VF in humans.

Conclusion: These studies suggest that it is feasible to estimate the duration of ischemia during VF cardiac arrest. This technology will permit allocation of treatment based on ischemia times. Additional studies now are being conducted to determine whether this technology can be used to determine the optimum time to defibrillate during VF cardiac arrest and to non-invasively monitor the response to therapy during CPR. [Variation of this paper published as: Brown CG: Median frequency: A new parameter for predicting defibrillation success rate. *Ann Emerg Med*: 1990;20:787–791.]

Adrenergic Drug Therapy in Cardiopulmonary Resuscitation

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Several recent studies have defined the minimal myocardial and cerebral blood flow requirements during ventricular fibrillation cardiac arrest. These studies demonstrate that the minimal myocardial blood flow required to meet the metabolic demands of the fibrillating myocardium is approximately 50 ml/min/100g (one-third of the coronary flow required during normal sinus rhythm flow). The cerebral blood flow requirements approximate 10 ml/min/100g (20–25% of normal sinus rhythm flow) during cardiac arrest. In animal models of out-of-hospital cardiac arrest in which the ischemia time prior to cardiopulmonary resuscitation (CPR) is 10 minutes, myocardial and cerebral blood flow, as measured with radio-labelled microspheres, is less than 5 ml/min/100g to the myocardium, and less that 3 ml/min/100g to the cerebral cortex. These studies demonstrate that standard external CPR following this duration of ischemia does not generate sufficient blood flow to meet the metabolic demands of the heart and brain.

In order to improve myocardial and cerebral blood flow during CPR, alpha-adrenergic agonists have been employed. These drugs increase peripheral vascular resistance and, thus, aortic diastolic pressure (the major, driving force for myocardial blood flow). In addition, alpha-adrenergic agonists have been shown to shunt blood flow from the extra-cerebral to the intra-cerebral vasculature, and to prevent carotid artery collapse during CPR, thus improving cerebral blood flow.

Although the American Heart Association recommendation for alpha-adrenergic drug therapy during CPR is approximately 0.01–0.02 mg/Kg of epinephrine, until 1985, no studies had examined the dose-response effects of alpha-adrenergic drugs on myocardial and cerebral blood flow during CPR. In a swine model of ventricular fibrillation cardiac arrest, the dose response effects were examined for the following alpha-adrenergic agonist drugs: epinephrine, norepinephrine, methoxamine, phenylephrine, and dopamine. These studies demonstrate that the 0.2 mg/Kg of epinephrine, and norepinephrine 0.12–0.16 mg/Kg, significantly improve myocardial blood flow, cerebral blood flow, and resuscitation rates compared to other alpha-adrenergic agonists. These investigations have led to the hypothesis that to increase peripheral vascular resistance effectively during CPR and thus, myocardial and cerebral hemodynamics, drugs with post-synaptic, alpha-2 agonist properties are required (epinephrine and norepinephrine). The rationale of the need for larger doses of these drugs than currently recommended may be related to the development of a tolerance at the alpha-receptor during ischemia. Recent clinical studies have demonstrated hemodynamic improvement following 0.02 mg/Kg of epinephrine in humans following prolonged cardiac arrest and CPR.

In an effort to determine the efficacy of high-dose (0.2 mg/Kg) versus standard-dose (0.02 mg/Kg) of epinephrine in out-of-hospital cardiac arrest, a multi-center, clinical trial was conducted. This study will examine the effectiveness of these epinephrine regimens on hospital discharge rates and neurologic outcome in refractory ventricular fibrillation, asystole, and electrical-mechanical dissociation.

Mother and Child—Trauma in Pregnancy: A Review

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Trauma is the most common cause of death in women under 35 years of age. Six to seven percent of all pregnancies are complicated by trauma. Management of the gravid trauma victim differs from the non-pregnant patient because of the anatomic and physiologic changes of pregnancy and the need to consider fetal well-being. Emergency physicians may be faced with pregnant victims of catastrophic or life-threatening trauma as well as those with apparently trivial injuries. Less common mechanisms of injury including thermal and electrical burns occur in pregnant patients seeking emergency care. This review discussed these topics in the context of the medical literature. Pathophysiology, clinical presentation, and emergent management were stressed. A review of the available data on automobile restraint systems in pregnancy was included with recommendations on use.

Understanding the pregnant trauma patient begins with knowledge of the maternal physiologic response to pregnancy. Important changes in these systems occur in the cardiovascular, respiratory, and hematologic systems. The article reviews the changes in these systems with respect to effect on emergency physicians and maternal, fetal responses to trauma. Likewise, anatomic changes predispose the pregnant patient to trauma. Individual organ systems become more or less vulnerable to blunt and penetrating injuries as pregnancy progresses. Specific aspects of catastrophic maternal trauma discussed include prehospital management, emergency department maternal evaluation, fetal evaluation, outcome, pregnancy-related injuries, fetal injuries, and management of injuries. Emergency department perimortem cesarean section as a resuscitation procedure in the third semester is stressed.

Controversy exists on the management of non-catastrophic trauma. The primary goal is diagnosis and treatment of premature labor and occult placental abruption. Based on a careful review of the existing literature, a four-hour period of cardiotocographic monitoring is recommended for appropriate patient care. The burn literature on pregnant patients suggest that complications correlate with the extent of body surface burned. Avoidance of maternal hypoxia, shock, and sepsis is critical because all are associated with increased fetal mortality.

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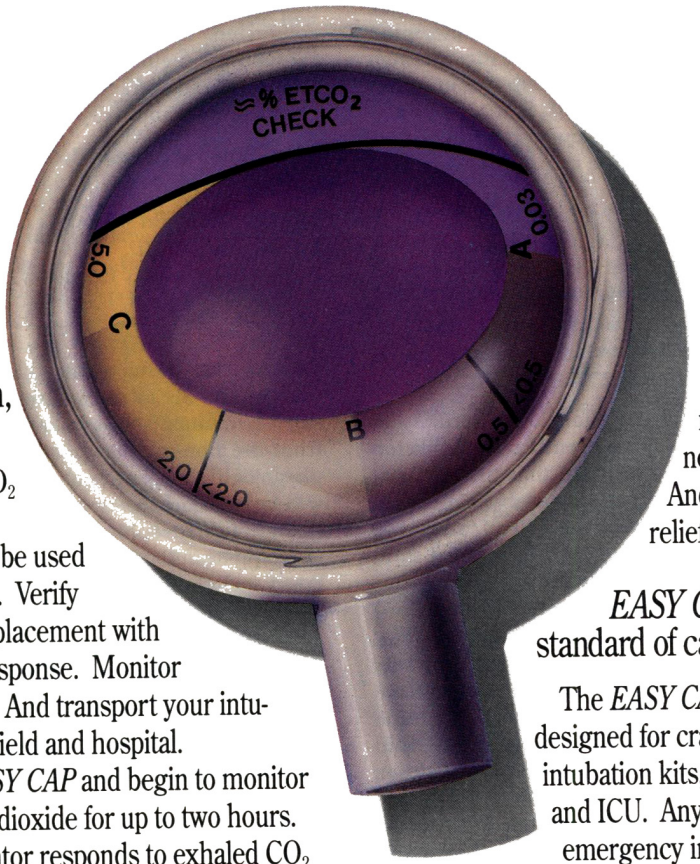

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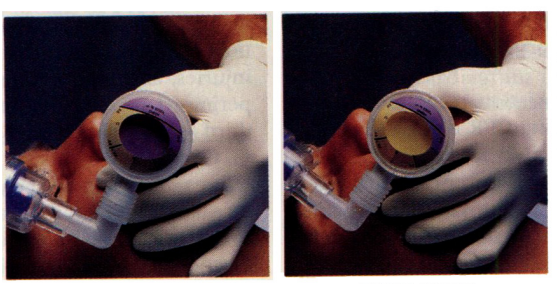

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Finally, the surprising sensitivity of the developing uterus to minor electrical shocks sustained in the home setting is discussed. Emergency physicians particularly must be wary of this mechanism of injury because of the high rate of fetal complications and demise. [Editor's Note: This article has been published in *Emerg Med Clin N America* 1991;9: 549–561.]

Prehospital Care in Paroxysmal Supraventricular Tachycardia: Adenosine-Triphosphate versus Verapamil

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To reverse sinus rhythm in paroxysmal supraventricular tachycardia (PST), mobile cardiac care units (MCCU) have used intravenous (IV) administration of verapamil or adenosine-triphosphate (ATP). A one-year experience was analyzed to compare the effectiveness and safety of these two drugs. Verapamil (5–25mg IV) was administered in 63 cases of PST (60 patients); ATP (10–40mg IV) was administered in 49 cases (45 patients). The difference between these two groups in age, sex, previous history of PST, duration of paroxysm, and pre-treatment of heart rate, blood pressure was not significant (NS, p>.05). Verapamil terminated PST in 58 (92.1%) cases, or in 55 (91.7%) patients; ATP was effective in 44 (89.8%) cases, or in 40 (88.9%) patients (NS). Hemodynamically significant heart blocks took place in two cases (3.2%) after verapamil, and in one case (2.0%) after ATP administration (NS). No other complications were registered. Three cases of PST recidivism within the first 30 minutes after sinus rhythm reversion occurred in the ATP group, in patients who got verapamil not a single case of re-paroxysm was described (p<.05), It is concluded that IV ATP in PST is neither more dangerous nor less effective than verapamil, which traditionally is used in prehospital conditions.

Thermometry of Expired Air: A New Method of Noninvasisve Control of Patient's Condition in Traumatic Shock

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Introduction: Thermometry of expired air (TEA) is a new, noninvasive method available for patient observation. The use of TEA allows detection of the development of pulmonary insufficiency and also other systematic disorders. **Methods:** Analysis of thermometric curve of the total volume of expired air enables one to obtain the following indices: Maximal temperature of expiratory gas (T-Max); Mean quantity of expiratory gas temperature (T-Mean); Quantity of heat in the unit of time (QT). **Results:** The quantity T-Max corresponds to the temperature of pulmonary arterial blood, which in turn is prognostic for the outcome of traumatic shock. The quantities T-Mean and QT correspond to functional lung area, these indices may serve as criteria for severity of lung injury. The combination of thermometric indices correlates inversely with the magnitude of cardiac output. **Conclusion:** TEA may be useful in clinical monitoring in trauma shock patients.

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Immunological and Metabolic Consequences of Extracorporeal Joining of Pig Spleens

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Experience of 245 extracorporeal pig spleen joining (EPSJ) to patients with pyo-septic and tumor diseases established that this procedure has a high treatment effectiveness. In order to define the possible EPSJ mechanisms, the influence on blood ex vivo and in vivo was investigated. It has been demonstrated that the level of middle molecular mass substance (MMS), free hemoglobin concentration (FHb), and osmolarity essentially were increased in blood after its transfusion through the pig spleen. These changes provide evidence of the functions of pig spleens.

Simultaneously, EPSJ decreased the number of circulating immune complexes (CIC), granulocyte, and thrombocyte quantity, and myoglobin level. Filtration pressure was decreased. This demonstrated the adsorption level of EPSJ.

The resorption effect of EPSJ consisted of a decreased of MMS, FHb, fibrinogen, CIC, and urea levels. Fractional composition of blood proteins was normalized and the concentration of superoxide substances was decreased. There also was an influence of EPSJ on the immune system: one hour after the procedure was finished, granulocyte and thrombolcyte quantity was restored and the new leucocyte pool was distinguished from the initial one by a normal level of chemotactic and bactericidal activity. In 18–20 hours, there occurred an increase of T-active and T-1,5 lymphocytes and decrease of T-zero lymphocytes.

Thus, the effect of EPSJ is multifactorial and connected with toxic metabolites elimination, with CIC and corpuscular particles adsorption simultaneously with stimulation of the immune system.

Intravenous Laser Radiation in the Prophylaxis of Reinfarction in Acute Myocardial Infarction

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Reinfarction complicates approximately 10% of the cases of acute myocardial infarction (AMI) and significantly worsens its prognosis. Systemic thrombolytic therapy (STT) is known to increase the risk of reinfarction. Intravenous (IV) laser radiation of the circulating blood has been reported to improve myocardial metabolism, decrease the incidence of arrhythmias, and to possess a moderate hypocoagulation effect in AMI. In the present study, the effect of IV laser radiation (V=632.8nm, total dose=1.0 J/kg/course of treatment) on the risk of reinfarction, has been studied in 102 patients with AMI.

Three identical groups of patients were determined based on age, sex, and site and size of the infarction: 1) 35 patients with AMI and STT (streptodebase, APSAC, and streptokinase); 2) 34 patients with AMI, STT, IV laser radiation; and 3) 33 patients with AMI, but without both STT and IV laser radiation.

Retrospective analysis of clinical, ECG, and laboratory data showed that 2–3 weeks after SMI reinfarction developed: in the first group in the patients treated with APSAC,—in 24.5±5.7%; in

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the second group—8.8±4.9%; and in the third group—in 18.2±6.7% (p1–p2<.05; p2–p3<.05). Thus, intravenous laser radiation significantly decreased the risk of reinfarction in patients with AMI after STT.

Endogenous Digoxin-like Factor (EDLF): Evident Role in Pathogenesis of Early Myocardial Ischemia-Induced Arrhythmias

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Endogenous digoxin-like- factor (EDLF) is a circulating chemical compound, as yet unidentified, with the biological properties and immunoreactivity similar to digitalis glycosides. Based on previous clinical and experimental observations demonstrating supersensitivity of the myocardium to the arrhythmogenic action of digitalis, inhibition of myocardial Na,K-ATPase activity, and loss of the specific digitalis receptor sites in the myocardium in acute myocardial infarction/ischemia (MI) it recently was suggested that increased plasma concentrations of EDLF may participate in the arrhythmogenesis in MI.

In accordance with this suggestion, it was shown that the increase of plasma Na,K-ATPase-inhibitory potency on patients and experimental animals with MI. Administration of antidigoxin antiserum significantly decreased the incidence of ventricular arrhythmias in acutely coronary-ligated rats.

An increase of plasma digoxin-like immunoreactivity has been observed in 45 patients in the first day after a first transmural MI (1.34±0.31 ng/ml) as compared with six healthy controls (0.31±0.1, p<.05) and with 16 patients with unstable angina pectoris after an episode of chest pain (0.40±0.07, p<.05). In six patients with MI complicated by ventricular fibrillation, plasma concentrations of EDLF were significantly higher (2.8±0.67 ng/ml) than in the other 39 patients with uncomplicated MI (1.1–0.03, p<.05).

In acute MI in rats, pretreatment of the animals with purified antidigoxin antibody resulted in a five-fold reduction of the incidence of ventricular arrhythmias. However, in these experiments, acute coronary ligation alone did not cause the changes in the activity of myocardial Na,K-ATPase and Na/K-pump. At the same time, pretreatment of coronary ligated animals with 1 mg/Kg propranolol led to occurrence of effects resembling some of the symptoms of acute digitalis overdose, significant inhibition of myocardial Na,K/pump, and pronounced increase of plasma potassium concentration. Administration of antidigoxin antibody to propranolol-pretreated rats exposed to coronary ligation in parallel to anti-arrhythmic effect prevented both inhibition of myocardial Na/K pump and development of hyperkalemia.

These results provide further evidence that elevated plasma concentrations of EDLF contribute to genesis of the early ventricular arrhythmia in MI. The fact that acute blockade of adrenoceptors unmasked the effects of EDLF points out the importance of EDLF-adrenergic counter-reactions and may be useful in the explanation of the effect of high doses of epinephrine in cardiac arrest and ventricular fibrillation in patients with MI.

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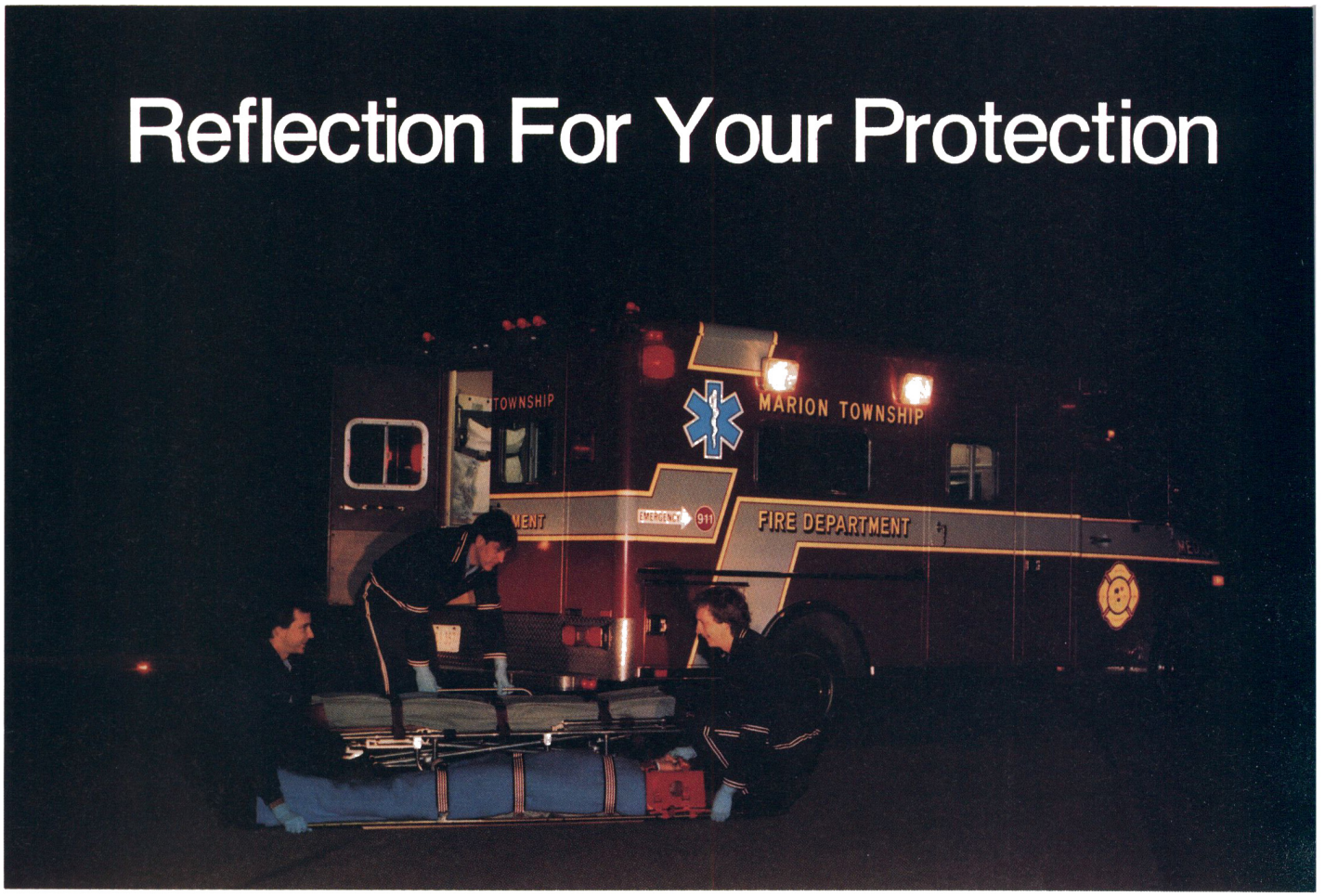

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