tially, to both the habit of general practitioners in Italy to treat pneumonias with penicillin-amoxicillin (73% of cases), together with the high frequency of serious, chronically ill patients, leading to selection of unusual and emerging pathogens. In fact, the incidence of C. pneumoniae infection was not related to any particular associated pre-existing disease. These results point-out the importance of C. pneumoniae infection even in critical hospital settings.

161
Trauma Care in Accident and Emergency Departments—A Critical Analysis

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In most European accident and emergency (A&E) departments, more than 70% of the patients can be discharged home after treatment, and only 5% are major trauma victims or seriously sick in need of qualified emergency care.

Despite vigorous efforts to guide patients with slighter conditions to attend their general practitioners, they still queue-up for treatment. Another group of patients attending A&E departments are those with complaints related to alcoholism, drug addiction, battering, and other types of social misadjustment. The wide spectrum of patients at the A&E departments, with complaints varying from non-urgent banalities to life-threatening conditions, constitutes a great problem.

To handle this situation and to increase the quality of care in the A&E departments, Emergency Medicine was created as a new specialty. Doctors recruited to this specialty were specially trained in handling a variety of emergency conditions.

In several studies, however, avoidable deaths still were noticed among seriously injured after attending emergency hospitals. These deaths most often were due to bad management with a delay in diagnostics and definitive treatment. When trauma centers first were established, it was hoped that better results could be achieved.

To improve emergency care, it has to be centralized to fewer specialized hospitals, where the organization is adjusted to manage a great number of casualties. Thereby, those with major injuries requiring advanced trauma life support, as well as patients with sociomedical problems in need of fast and skillful care by emergency medicine physicians, can be properly handled.

164
Hyperbaric Oxygen Treatment of Smoke Inhalation and Other Acute Carbon Monoxide Poisonings

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Introduction: This is a preliminary report from an ongoing study based on 70 consecutive patients referred to the Karolinska Hospital for hyperbaric oxygen (HBO) treatment of acute carbon monoxide (CO) poisoning. Hyperbaric oxygen (HBO), i.e., 100% O_2 breathing at 2.5–2.8 times normal barometric pressure reoxygenates ischemic tissues, hastens the elimination of CO from heme proteins such as hemoglobin, myoglobin, and cytochrome systems, and reduces cerebral edema.

In animal experiments, HBO antagonizes co-mediated brain lipid peroxidation, speeds up the recovery of energy metabolism and ameliorates the prolonged intracellular acidosis in the brain after CO-induced hypoxia.

Methods: Twenty-one women and 58 men were treated with one or repetitive (mean = 2.8) HBO sessions in multi- or monoplace chambers. No patients were excluded. Mean age was 42 years (range 3–88 years). Forty-two (53%) of the 70 cases were accidental CO exposures, 36 were due to smoke inhalations (15 with burns). Thirty-seven (47%) cases were attempted suicides, in 33 cases from automobile exhaust (3 women, 30 men).

Results: History of unconsciousness at the scene was reported in 73 patients and 41 still were unconscious on admission to the emergency department of the nearest hospital. Mechanically assisted ventilation was given to 47 patients. The delay from rescue to start of HBO averaged 7.7 hours. Four deaths (5%) occurred due to anoxic encephalopathy. Three of these were due to smoke inhalation, of which two were burned severely and required initial CPR. Four patients (5%) had evidence of brain damage on discharge.

Conclusion: These data are in agreement with many previous reports from the past 30 years, and indicate that HBO reduces mortality and morbidity beyond that expected with pure normobaric oxygen. The history of CO intoxication/smoke inhalation and unconsciousness (even transient) in a patient justifies rapid transfer to the nearest hyperbaric center with facilities for critical care and suitably qualified personnel. Any delay to await laboratory results is inappropriate. During transport to such a facility, the patient should receive 100% oxygen. The argument that normobaric oxygen always is satisfactory for severe CO poisoning no longer can be sustained.

167
Carbon Monoxide and Jogging in the City

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Aim of the Study: Taking into account the increase of the minute volume of ventilation during the effort and the carbon monoxide (CO) content of air in the cities, does city jogging result in a rise of blood CO?

Joggers and methods: Twenty-seven, non-smoking, well-trained joggers (age 37.0–7.6 years) participating in the "20 km de Bruxelles" (20,000 runners) had a determination of venous CO the day before the race and just after completion of the run. The CO concentration in the environment was measured every kilometer and more often in the tunnels.

Results: CO content was 2 ppm up to km 7 and between 0 and 1 ppm afterward. Only in the tunnels was the content much higher (between 10 and 20 ppm). Venous CO content before

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the race was 1.47–0.13%; after 20 km it was 1.40–0.10% ($p<0.001$).

Conclusion: In spite of running through the tunnels, a rise in blood CO was not detected; to the contrary, a decrease was measured. The very low CO level in the last kilometers of the run and the increased minute ventilation during jogging explains this small but significant reduction.

168
Effect of IV Organophosphate Application (Paraoxon [E 600]) on Coagulation in Mini-Pigs

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Introduction: Paraoxon (E-600) can cause thrombosis, embolization, and death in mini-pigs. There is little agreement, however, about the extent and nature of the coagulation abnormality induced by paraoxon.

Purpose: Establish, in a controlled study, the dose-effect curve between the quantity of paraoxon administered and blood coagulation in the mini-pig.

Material and Methods: Animals fasted for 12 h were premedicated with 0.5 mg Fentanyl (orally). After adequate sedation (assessed by clinical impression), 400 mg Ketamine and 2 mg Flunitrazepam were given intramuscularly (IM). The animals were weighed and an ear vein was cannulated (G22). After preoxygenation by mask, muscle relaxation was achieved with 10 mg Alcuronium. Additional intravenous (IV) drugs were given: Lidocaine 2 mg/kg, and Fentanyl 0.15 mg. The animals were intubated with a size 7.0 endotracheal tube and mechanically ventilated to achieve normocapnia [F_iO_2 = 0.5 (N_2O + 0.4% Halothane), tidal volume = 10 ml/kg, 20 cycles/min]. Additional doses of relaxant and opiate were given throughout the procedure as needed. *Monitoring and baseline measurements*: continuous BP (carotid artery), CVP (left jugular), capnometry, arterial and venous blood gases, Hematocrit (Hct); [Hemoglobin], coagulation profile (PT, PTT, Factor V, Factor VIII, fibrinogen, and right jugular. The measurements were carried-out every 10 minutes (nine times), and then every 20 minutes (three times). Fluids were administered through the left jugular vein to maintain the Hct close to baseline value. Paraoxon was infused continuously through an ear vein. The control animals did not receive Paraoxon.

Results: Low-dose Paraoxon activates intrinsic coagulation (PTT) without having any significant influence on the extrinsic coagulation (PT). Increases in dose did not have any additional effect on either the intrinsic or the extrinsic coagulation pathways.

Discussion: The selective influence of Paraoxon on the intrinsic pathway of coagulation is striking. An explanation cannot be offered at this stage. The lack of a dose-dependent relationship and the lack of effect in-vitro implies an indirect effect.

169
Diaspirin Cross-Linked Hemoglobin (DCLHb): An Effective Resuscitation Solution in a Swine Model of Hemorrhagic Shock

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Purpose: DCLHb is an acellular, human hemoglobin-based, oxygen-carrying resuscitation solution that currently is being evaluated in clinical safety studies. This study examined the effects of increasing doses of DCLHb in a pig hemorrhagic shock model. Results were compared to infusion of lactated Ringer's solution (LR, 3x hemorrhage volume) and untreated animals.

Protocol: Eight treatment groups (5 pigs/group) were hemorrhaged 30 ml/kg in 20 minutes (min) (5 min @ 3 ml/kg/min, then @ 1 ml/kg/min) followed immediately by infusion of either 10 g/dl DCLHb (0.5, 1.0, 2.0, 4.0, 10.0, or 30.0 ml/kg infused at 1 ml/kg/min) or LR (90 ml/kg infused at 3 ml/kg/min). The untreated group received no fluids. Mean arterial blood pressure (MAP) was monitored continuously and a variety of clinical chemistry and cardiovascular variables were monitored at predetermined time points for an additional six hours following resuscitation.

Results: The table below presents MAP values (mean—SEM) following hemorrhage and infusion.

Treatment (ml/kg)	Baseline (mmHg)	End Hem (mmHg)	5 min-Post (mmHg)	3 hr-Post (mmHg)
DCLHB (0.5)	119 ±4	45 ±1	102 ±8§	112 ±11†§
DCLHb (1.0)	114 ±5	48 ±7	98 ±10†§	106 ±3†§
DCLHb (2.0)	112 ±3	50 ±6*	121 ±4†§	111 ±5†§
DCLHb (4.0)	114 ±4	43 ±3*	112 ±8†§	122 ±5†#§
DCLHb (10.0)	121 ±1	79 ±5*†#	134 ±7†§	147 ±8†#§
DCLHb (30.0)	129 ±9	53 ±7*	138 ±6†#'	160 ±6†§
LR (90)	118 ±5	47 ±4*	105 ±8†§	103 ±6†§
Untreated	109 ±2	37 ±2*	56 ±7†	79 ±8†§

*p <.05 vs Baseline
§ p <.05 vs End Hemorrhage
†p <.05 vs Untreated
#p <.05 vs Lactate Ringers(LR)

The MAP in all groups decreased from baseline by end of hemorrhage. The DCLHb and LR groups had significantly greater MAP than untreated animals by 5 minutes after the start of infusion. MAP in the DCLHb and LR groups tended to remain greater than untreated animals through the six-hour study.

Conclusion: Infusion of DCLHb produced an immediate and significant increase in MAP when given following hemorrhage in pigs. In addition, DCLHb was as effective as much larger volumes of LR in restoring and maintaining pressure following hemorrhage. DCLHb appears to be an effective fluid for use in resuscitation from hemorrhagic hypovolemic shock.

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