responsibilities of a trauma surgeon are not limited to the mechanical repair of the various injuries, but include physiological support of the patient, organization of the team and hospital resources, coordination of pre-hospital care, research and education.

Harlem Hospital Center was established in 1887, serving a population that is depressed. People in the area are poor and deprived and suffer from severe illnesses and an extraordinary high number of interpersonal injuries. Starting as a 20 bed hospital 100 years ago, it has grown into a relatively modern 800 bed institution. Its experience in trauma management has been unique. It has contributed a great deal to the management of various trauma which has brought this center into international focus. This also led to the development of an organized trauma service as a separate surgical division and was later designated as one of the four certified centers for the treatment of trauma in New York City. Our overall experience over the past five years in trauma management, as well as special types of injuries encountered and their management, will be presented. Planning, teaching and organization of a trauma service will also be discussed so as to share the information and experience with others whose population and type of injuries are similar to ours.

PATTERNS OF DYING FROM SEVERE BLOOD LOSS IN AWAKE RATS

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The RRC has established volume controlled hemorrhagic models in awake rats (McGlew et al, Circ Shock 16:35, 1985) and lightly anesthetized monkeys (Bar-Joseph et al, JWAEDM 1, SI:169, 1985). In this study, we determined in awake spontaneously breathing rats, responses and dying patterns in the same model over 3 h post-hemorrhage (H), while monitoring art. and CV pressures, breathing movements, EEG, and art. blood gases. Four groups of 10 rats each were studied. After cannulation under light anesthesia, and awakening, the rats were bled over 20 min: Gr I, 2 ml/100 g H; Gr II, 2.5 ml/100 g H; Gr III, 3 ml/100 g H; Gr IV, 3.5 ml/100 g H. Death was defined as syst. BP $\geq$25 mmHg. *Results*: Survival rates were 90% after H 2 ml/100 g; 80% after H 2.5 ml/100 g; 40% after H 3 ml/100 g with mean survival time 73 (7-162) min; and 0% after H 3.5 ml/100 g with mean survival time 32 (1-122) min. Art. press. decreased at end of H to a minimum of 30 mmHg, rose transiently in all groups (attempted self-resuscitation), and then either recovered to near normotension or declined to death. Art. PO_2 increased. Art. PCO_2, pH, Hct decreased. Hyperventilation changed to gasping until pulselessness. EEG depression correlated with hypotension. *Conclusions*: The H 3.5 ml/100 g model is suitable (and will be used next) for the study of responses to field resuscitation potentials suitable for use by laymen, with the aim to delay secondary cardiac arrest from severe hemorrhage while waiting for transport (e.g., external stimuli, rectal fluids, hypothermia, O_2 inhalation). (Assisted by Lisa Porter. Supported by A.S. Laerdal Foundation.)

EFFECTS OF SODIUM BICARBONATE IN CANINE HEMORRHAGIC SHOCK

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We studied the use of sodium bicarbonate ($NaHCO_3$) administration in a canine model of hemorrhagic shock to determine its effect on lactic acidosis, serum bicarbonate, pH, and cardiac output. Thirteen dogs were anesthetized, paralyzed, mechanically ventilated and hemodynamically monitored. Hypotension was induced and maintained at a MAP of 40-45 mmHg by controlled hemorrhage and reinfusion. After 2.5 hr of shock, the dogs were randomized in 2 groups: A=6 control dogs received NaCl infusion; B=7 dogs received $NaHCO_3$ 1 mEq/kg followed by a continuous infusion of 2.5 mEq/kg/hr for 2.5 hr.

VARIABLES (MEAN ± SD)		BASELINE	HEMORRHAGE ONLY	HEMORRHAGE + TREATMENT
Cl	A	4.3 ± 0.9	1.1 ± 0.1	1.2 ± 0.3
Cl	B	4.2 ± 1.3	1.0 ± 0.2	1.3 ± 0.3
LACT	A	0.9 ± 0.4	6.8 ± 0.9	5.1 ± 1.2
LACT	B	0.7 ± 0.4	7.1 ± 3.1	10.1 ± 3.2
HCO_3^-	A	20.1 ± 1.8	13.4 ± 1.6	13.6 ± 2.0
HCO_3^-	B	20.1 ± 1.8	11.1 ± 3.0	13.3 ± 2.3
pH	A	7.34 ± .03	7.09 ± .07	7.17 ± .04
pH	B	7.37 ± .02	7.03 ± .10	7.16 ± .10

Results: *=$p<0.05$

As in other models of lactic acidosis (hypoxic and phenformin induced), this model demonstrates an increase in lactic acid associated with $NaHCO_3$ administration. Although the pathophysiologic