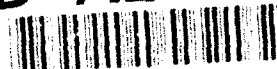


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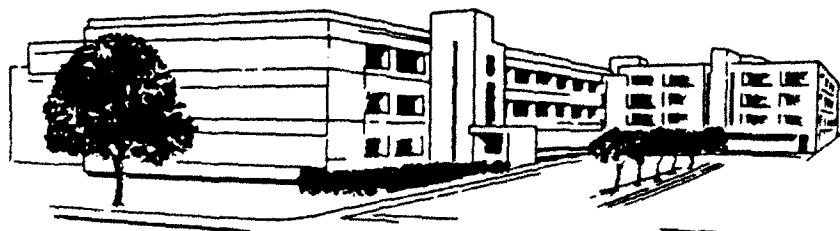
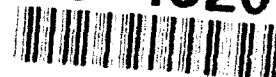
The Dac Tran
Gregory Hanson
Juergen W. Pfeiffer
Michael A. Dubick
X.Q. Yuan

Division of Military Trauma Research

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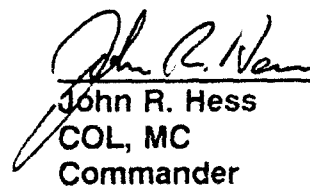
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John R. Hess
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The cardiovascular performances of 10 male Yorkshire swine were investigated following moderate to severe brain trauma that was induced by a fluid percussion device. Arterial pressure, cardiac output, heart rate, and pulmonary arterial pressure reached their highest values 20 seconds after brain trauma. Except for the heart rate, these cardiovascular variables dropped below their baseline values 2 hours after the trauma. These data indicate that brain trauma may significantly suppress cardiovascular performance.					
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ABSTRACT

The cardiovascular performances of 10 male Yorkshire swine were investigated following moderate to severe brain trauma that was induced by a fluid percussion device. Arterial pressure, cardiac output, heart rate, and pulmonary arterial pressure reached their highest values 20 seconds after brain trauma. Except for the heart rate, these cardiovascular variables dropped below their baseline values 2 hours after the trauma. These data indicate that brain trauma may significantly suppress cardiovascular performance.

PREFACE

The study reported here was accomplished during The Dac Tran's tenure in the Division of Military Trauma Research as a Summer Student Fellow of the American Heart Association, California Affiliate.

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Traumatic Brain Injury Generates Biphasic Hemodynamic Responses in The Swine- TD Tran, G Hanson, JW Pfeiffer, MA Dubick, X-Q Yuan

According to one estimate, each year almost 1 million people in the United States suffer from head injuries and more than 400,000 of these are admitted to hospitals [1]. There are about 100,000 deaths of traumatic head injuries each year, many of them die before reaching a hospital [1]. In conventional war, trauma to the central nervous system causes about one-third of all deaths [2]. Among Vietnam combat casualties, head trauma was the most common cause of hospital deaths (42%) [3]. Another study showed that the incidence of head injuries increased markedly from 17% in the Korean conflict to 30% in the 1973 Middle East war [4].

Previous studies showed that in a rat model, head trauma not only suppressed the spontaneous hemodynamic recovery from hemorrhage, but also suppressed the efficacy of fluid resuscitation [5,6]. However, the exclusive effects of traumatic brain injury on cardiovascular function in non-hemorrhaged animals have not been precisely examined. In the present study, we used a swine model to characterize the hemodynamic responses to brain trauma.

MATERIALS AND METHODS

Male Yorkshire swine (25-35 Kg) were fasted overnight. Anesthesia was induced in each animal through a mask by isoflurane and maintained by a balanced anesthesia, which consisted of an intramuscular injection of Innovar-vet (0.11 ml/kg), inhalation of isoflurane (0.5%) and nitrous oxide (50%), and intravenous infusion of fentanyl (0.6 ug/kg/min) and succinylcholine (0.2 mg/kg/min). Polyethylene catheters were placed in the femoral arteries to monitor blood pressure, and to collect blood samples. A Swan-Ganz catheter (7.5 F, American Edward Labs, Irvine, CA) was inserted into the pulmonary artery through the left femoral vein to determine central venous, pulmonary arterial and wedge

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pressures, and to inject the cold saline to measure cardiac output by the thermodilution method using an American Edwards Cardiac Output computer. All catheters were attached to pressure transducers (Gould) which were connected to a Gould ES-2000 multi-channel monitor and recorder for continuous monitoring and recording of the pressures and electrocardiogram. After completing the instrumentation, at least one hour was allowed for hemodynamic stabilization. Cardiovascular variables listed above were measured before head trauma and at intervals over a 2 hr period after injury. In addition, systemic vascular resistance was calculated by subtracting central venous pressure from mean arterial pressure and dividing by the cardiac output. The results were expressed as resistance units (RU). Cardiac index was calculated by dividing cardiac output by body weight and the results expressed as L/min/kg. During the experiment, mechanical ventilation was maintained, and only an intravenous infusion of fentanyl (0.2 ug/kg/min) and succinylcholine (0.07 ug/kg/min) was continued [7].

A fluid percussion brain trauma model was used in this study. Fluid percussion brain injury models have been used in many species, including dog, cat and rat [8-11]. This type of model was produced with a fluid percussion device to simulate a closed head injury that often occurs in a motor vehicle accident or battlefield casualty. The main part of the trauma device is a 60 cm long Plexiglas cylinder filled with isotonic saline. The injury is induced by a metal pendulum, which strikes the piston at one end of the cylinder from a predetermined height. The resulting impulse is measured extracranially at the time of strike via the transducer mounted at the other end of the cylinder and recorded on a storage oscilloscope. The oscilloscope is triggered photoelectrically by the descent of the pendulum. The impulse generated is transmitted through the fluid in the cylinder into the cranial cavity of the animal. The impact level we implemented ranged from 4.09 to 5.79 atmospheres.

RESULTS

All data are expressed in mean $\pm$ standard error of mean.

Mean Arterial Pressure (mmHg) (Fig 1A)

After the brain had been injured, the mean arterial pressure increased from the baseline value of 123 ± 4 mmHg to 179 ± 7 mmHg at 10 seconds, and it continued to increase to 186 ± 7 mmHg at 20 seconds. At 2 hours after trauma the mean arterial pressure began to decrease to 102 ± 11 mmHg.

Heart rate (beats/min) (Fig 1B)

The heart rate increased from 112 ± 7 beats per minute at the baseline to 190 ± 15 beats per minute 10 seconds after the injury. It continued to increase to 224 ± 16 beats per minute at 20 seconds after the injury. The heart rate then decreased, but at 2 hours after brain trauma, the heart rate was 152 ± 20 beats per minute, which remained higher than the baseline value.

Cardiac output (L/min) (Fig 2A)

The cardiac output increased from 6.1 ± 0.7 L/min at the baseline to 7.7 ± 0.8 L/min at 1 minute after the brain trauma, then decreased to 5.5 ± 0.4 L/min at 2 hours after the trauma. Cardiac index followed a trend similar to that observed for cardiac output (Fig 2B).

Mean pulmonary arterial pressure (mmHg) (Fig 3A)

The mean pulmonary arterial pressure increased from a baseline of 15.6 ± 1.3 mmHg to 26.4 ± 3.3 mmHg at 10 seconds after the trauma. At 20 seconds after trauma, it increased to 28.7 ± 4.0 mmHg, and then rapidly declined to 13.3 ± 0.9 at 15 min after the injury. At 2 hours after the trauma mean pulmonary arterial pressure was 13.1 ± 1.7 mmHg.

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Systemic vascular resistance (RU) (Fig 3B)

Systemic vascular resistance rose about 10% 1 min after head trauma, fell below baseline at 9 min, and slowly rose toward baseline levels over the 2 hr experimental period.

DISCUSSION

Our data showed that mechanical brain injury induced rapid increases in arterial pressure, cardiac output, heart rate, and pulmonary arterial pressure. This immediate hyperdynamic response may be caused by a sympathoadrenal activation [5,11]. Despite the apparent elevated sympathoadrenal activity indicated by persistent tachycardia, a hypodynamic profile ensued as indicated by prompt decreases in arterial pressure and cardiac output. By 20 min after the injury, arterial pressure dropped below baseline. A substantial decline in cardiac output without a simultaneous significant change in systemic vascular resistance suggests that the cardiac function may have been damaged following severe brain injury [12-15]. It is important to keep this in mind when resuscitating a multiple trauma patient with head injury or managing a donor heart from a traffic accident victim.

More studies are needed to define the effects of head trauma on cardiac contractility and other functions and to examine therapeutic means to avoid or reduce head trauma-induced heart dysfunction.

CONCLUSION

Severe traumatic brain injury creates an immediate hyperdynamic response followed by a hypodynamic state, which may be caused by a compromised cardiac function.

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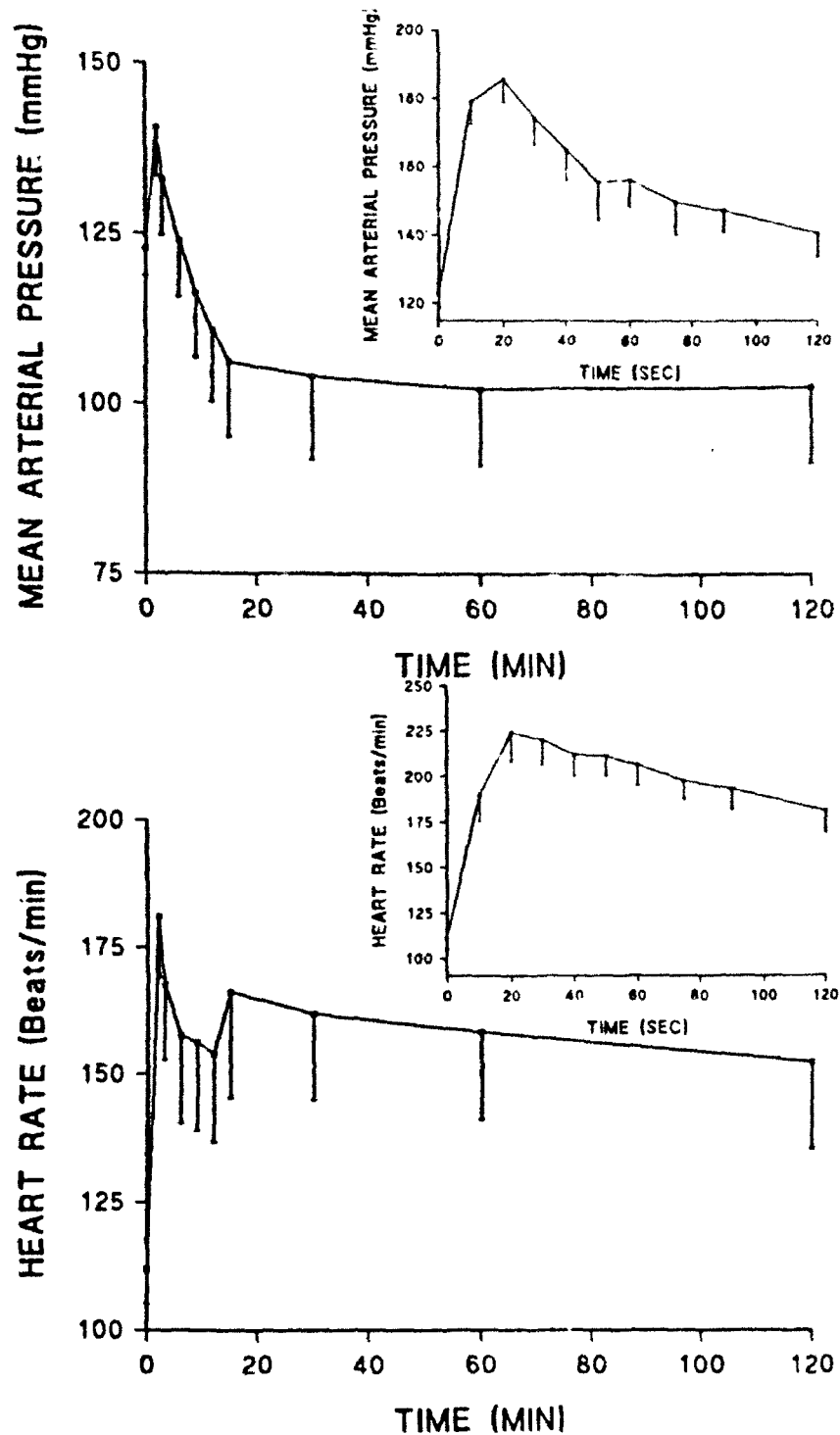


Fig. 1. (A) Mean arterial pressure and B) Heart Rate before and following head trauma. Data expressed as mean $\pm$ S.E. from 10 pigs. Small figures depict events over first 120 sec following head injury.

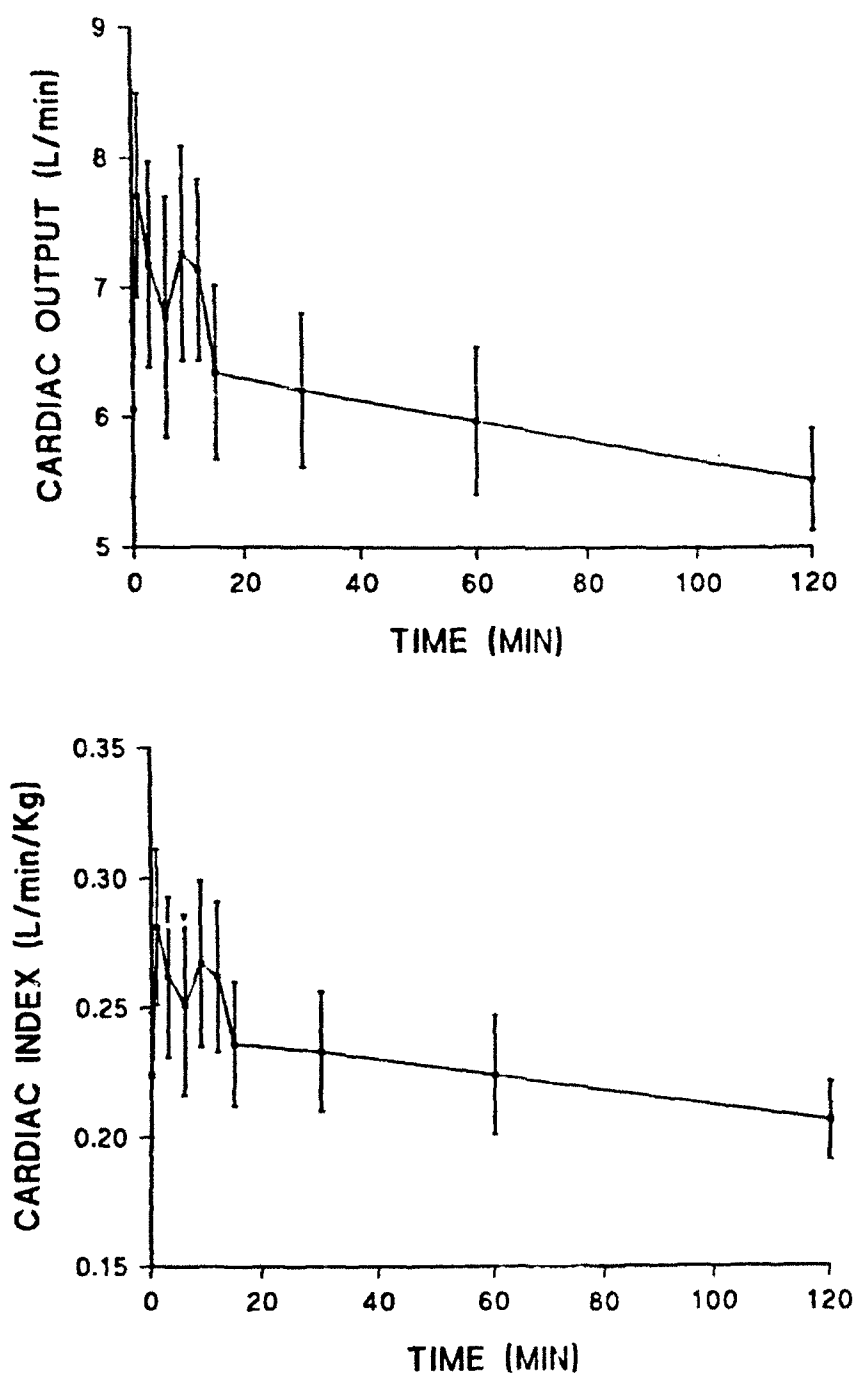


Fig. 2 A) Cardiac output and B) Cardiac Index before and following head trauma. Data expressed as mean $\pm$ S.E. from 10 pigs.

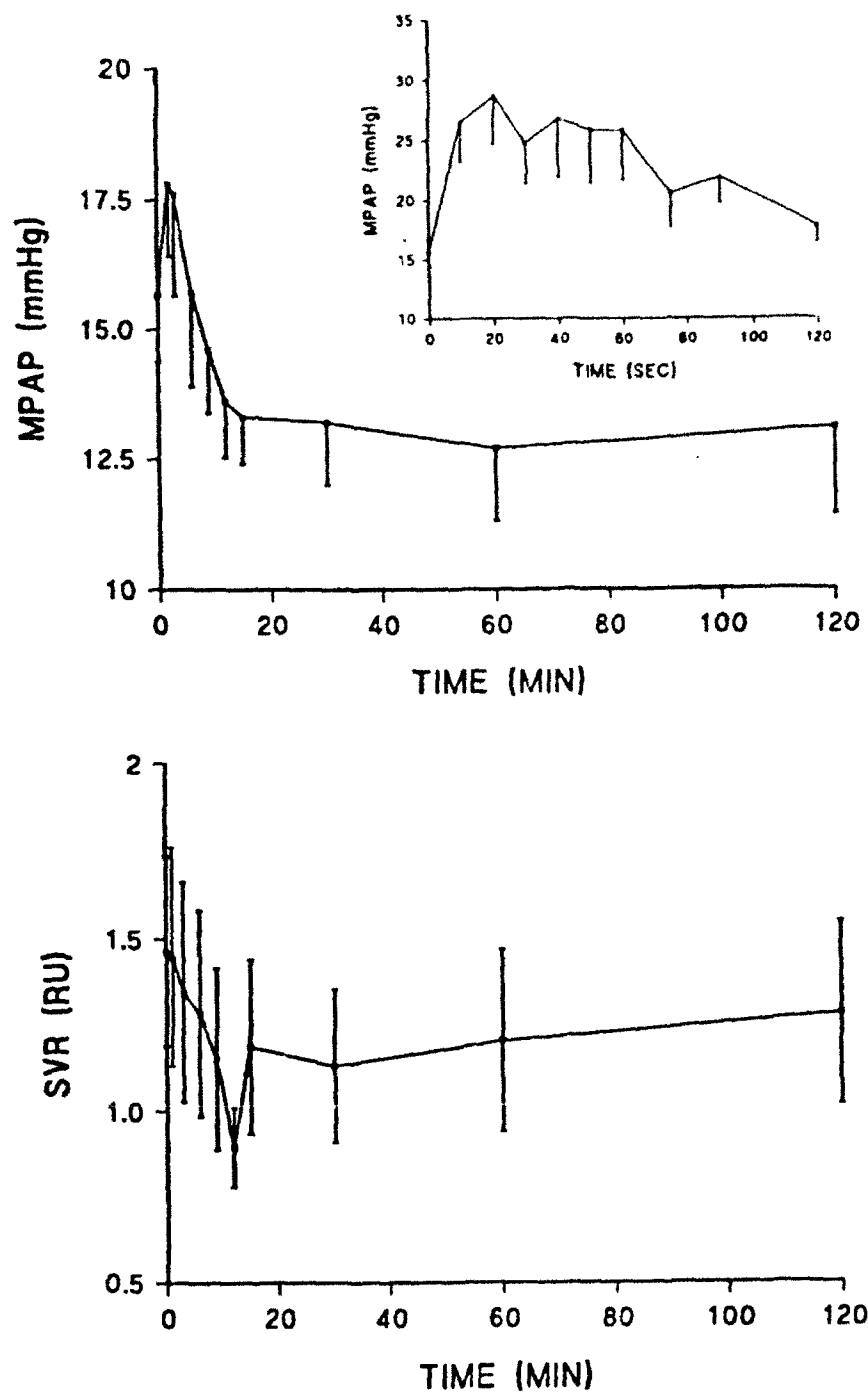


Fig 3 A) Mean pulmonary arterial pressure (MPAP) and B) Systemic Vascular Resistance (SVR) before and following head trauma. Data expressed as mean $\pm$ S.E. from 10 pigs. Small figure depicts changes in MPAP over first 120 sec following head trauma.

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