ABSTRACTS

Editorial Comment: Material for this section is not abstracted in a uniform style. Many employ direct quotations only. Others are written in the more conventional form. At times there may be included a few opinions, personal to the abstractor, which, where they appear, will be bracketed or labeled "Comment." The Editorial Office continues in its desire to receive correspondence from readers relative to the management of this section.

CHESS, STEPHEN; CHESS, DOROTHY, AND COLE, W. H.: *Experimental Tourniquet Shock with Particular Reference to the Toxic Factor; a Method of Production Eliminating the Influence of General Anesthesia and Nervous Impulses.* Arch. Surg. **49**: 147–155 (Sept.) 1944.

"Tourniquet shock can be produced consistently in animals and is therefore particularly adaptable for study, but the extreme pain produced by the tourniquet makes it necessary to utilize some type of anesthesia. Prolonged anesthesia, whether produced by a barbital compound or a general anesthetic, is undesirable. Moreover, since nervous impulses are obviously so intensive, this factor might alter the data derived from the experiment. To obviate these disadvantages we have adopted the procedure of cutting the spinal cord [of dogs] at the level of the lowest dorsal or the uppermost lumbar vertebra, two to four days before the experiment is to be performed. It is unwise to wait much longer, since the anesthesia induced in the lower extremities, which are dragged over the floor of the cage, may allow development of ulcers, infection, etc. Manipulation or operation may be conducted without pain on the lower extremities of the animal with no more anesthetic than a moderate dose of morphine. . . . In the experiments in which shock was produced by release of the tourniquet death always occurred if the tourniquet was left in place for at least nine hours. In 10 animals studied by this method, death occurred after an average of two hours and thirty-four minutes following release of the tourniquet. The average loss of plasma into the extremity before and following release of the tourniquet, as determined by the method of Blalock, was only 2.1 per cent of the total body weight, which is insufficient in itself to explain death. To determine whether or not a toxin may have developed in the constricted limb and may have been an important factor in the pathogenesis of shock and death, we performed cross transfusion, injecting blood obtained from the distal portion of the femoral vein of the constricted limb into a normal (control) animal. To prevent increase in shock in the animal subjected to the application of the tourniquet through loss of blood, an equal amount of blood was removed from the recipient (control) dog and injected into the shocked animal. Because of exigencies of war, work on the problem was interrupted; only 5 such experiments could be performed. Four of the 5 animals receiving blood from the constricted limb after release of the tourniquet died two to twelve hours after the transfusion was begun. The only dog to survive transfusion of blood which had circulated through the constricted limb was 1 which did not receive any blood from the constricted limb until ten minutes following release of the

tourniquet. If a toxin were present in the constricted limb it would supposedly be more concentrated in the blood draining from the limb during the first few minutes following release of the tourniquet. If this were true, survival of this animal, which, however, did go into shock during the transfusion (but recovered afterward), might be explained." 33 references.

J. C. M. C.

BLALOCK, ALFRED: *Utilization of Oxygen by the Brain in Traumatic Shock.* Arch. Surg. **49**: 167–169 (Sept.) 1944.

Dogs were used in all experiments. General anesthesia was produced approximately one hour before the control studies were performed by the intravenous injection of pentobarbital sodium, 25 to 30 mg. per kilogram of body weight. Subsequent subcutaneous injections were given as indicated. . . . All of the four experimental procedures for producing shock, hemorrhage, trauma, tourniquets and burns were associated with an increase in the arteriovenous difference of both the cerebral sinus blood and the mixed venous blood. The oxygen utilization of the cerebral sinus blood and that of the mixed venous blood in general paralleled each other closely. Particular attention is directed to the fact that the difference in oxygen content of the arterial blood and that of the sinus blood increased in the early stages of shock and this difference usually became more marked as shock developed. The early increase in the arterial-venous sinus oxygen difference was due in some instances to an increase in the oxygen content of the arterial blood rather than to a decrease in oxygen content of the venous blood. . . . The arterial-venous sinus oxygen difference usually increased before there was a significant decrease in the arterial blood pressure. Alterations in the total oxygen consumption of the body throughout the course of the experiments were not great. If one could assume that the oxygen consumed by the brain also remained essentially constant, the finding of an increased utilization of oxygen would mean that the cerebral blood flow was considerably reduced. Such an assumption is not warranted. . . . It appears that under conditions of decreasing blood flow the brain, unlike the kidneys, can maintain its oxygen consumption at least partially by extracting increased proportions of oxygen from the arterial blood." 4 references.

J. C. M. C.

ENGEL, D.: *Sympathetic Block: Proposed Therapy in Traumatic Shock.* Brit. M. J. **2**: 434–435 (Sept. 30) 1944.

"The purpose of this paper is to draw practical conclusions from recent experiments . . . and to propose regional sympathetic block as a preventive and curative method in cases of extensive crush injuries. Though my proposal is based on animal experiments there is enough clinical evidence to support my views. The result of the experimental work can briefly be summarized as follows: In 'traumatic shock' the rate of filtration through the capillaries in tissue adjacent to trauma, and probably also in the traumatized tissue itself, is greatly increased in the first 1 to 5 hours following trauma, and reduced afterwards. The increased rate of filtration, suggesting increased capillary permeability, is thus restricted to the traumatized area and is not generalized over the whole body. It was further shown that by regional sympathectomy it is possible to reduce considerably the increased rate of filtration caused by trauma. . . . The practical significance of these experiments is that if it is true that capillary permeability is one of the most im-