Blood Pressure Responses in Acute Compression of the Spinal Cord*

SHEDDEN ALEXANDER, M.D., AND FREDERICK W. L. KERR, M.D.

Section of Neurologic Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

In experiments on the effects of acute compression of the spinal cord, a marked transient rise in blood pressure was observed coincident with the application of pressure. Since this was to us an unexpected phenomenon, we reviewed the literature and performed a number of experiments to elucidate the mechanism of this response.

Material and Methods

Thirty-six adult cats were studied. Two monkeys were also used to determine whether significant variations of species were present. Pentobarbital sodium (30 mg./kg.) was administered intraperitoneally and supplemented as required, and an endotracheal or tracheostomy tube was inserted. A catheter was placed in the abdominal aorta via the femoral artery and connected to a Statham strain gauge and in turn to an amplifier and pen-writing system. Laminectomies of varying lengths were performed at all levels. A slender, elongated latex balloon was introduced into the epidural space, covering a longitudinal extent of one or occasionally two or three segments; one or more laminae were left intact at the level where compression was to be applied, to afford counter resistance. The balloon was inflated by a syringe connected to a mercury manometer. Compression of the cord was obtained by raising the pressure in the balloon to levels of 50, 100, 200, and 450 mm. of mercury for periods ranging from 5 to 25 sec. and on occasion up to 2 min. The responsiveness of the cord at different levels was studied. Various procedures were carried out to try to determine the factors responsible for the pressor activity and the role of certain vasomotor reflexes in this response. These included bilateral denervation of the carotid sinus and aortic nerves, bilateral division of the vagus nerve high in the neck, bilateral (chronic) division of the dorsal roots at T1 to T3, bilateral adrenalectomy, decerebration at the level of the superior colliculus performed under ether anesthesia (following which anesthesia was discontinued), transection of the cord rostral to the area to be compressed, and perfusion of upper thoracic segments of the cord with autologous blood or saline under pressure via a corresponding intercostal artery. Succinylcholine was given intravenously in small and repeated doses as required to rule out changes in blood pressure which might be associated with movement resulting from compression of the cord.

Results

Spinal Cord Pressor Response. The response to compression of a segment of the upper portion of the thoracic cord consisted in a rapid increase in mean systemic arterial pressure after a latent period of 2 to 6 sec.; the increase in pressure was sometimes as much as 100 mm. of mercury above the resting level (Figs. 1, 2 and 4). Following decompression of the cord a fairly rapid fall in blood pressure occurred in most instances and was often interrupted by a momentary rebound rise or a brief plateau. Bradycardic pulses of large amplitude occurred at the peak of the pressor response and during the fall in pressure; they were characteristically observed at upper thoracic levels and began between 10 and 15 sec. from the onset of compression. This increase in pulse pressure was a variable but frequently seen phenomenon; Fig. 1 (a and b) illustrates a particularly pronounced response of this type. The persistent increase in blood pressure seen in this instance was also more marked than was usual. The effect of sinu-aortic denervation on this response will be discussed subsequently. No difference was observed between intradural and extradural compression of the cord.

Cord Levels. Pressor responses were elicited from all levels of the spinal cord, but were of significantly different magnitude and pattern at various levels. Fig. 2 shows responses evoked by compressing single segments of the cord at L1, T11, T9, T7, T5, and T3, each at 200 mm. of mercury for 10 sec. It will

Received for publication September 23, 1963.

* This investigation was supported in part by Research Grant B-39296 from the National Institutes of Health, Public Health Service.
be seen that compression at L1 evoked a moderate pressor response with slightly increased pulse-pressure amplitude. Compression at T11 and T9 evoked a negligible response. At T7 compression again produced a moderate rise in pressure, and pulse-pressure amplitude increased moderately during the response. Compression at T5 and T3 elicited a marked rise in blood pressure and a marked rise in pulse-pressure amplitude, and slowing of the heart rate occurred; a small secondary rise was noted at the end of the falling phase of the pressor response. It is of interest to note that the increase in pulse pressure was obtained between T2 and T6 (level of sympathetic outflow to the heart) but not at T1 or above, while at T7 and below it was absent or much less pronounced. The rise in mean arterial pressure, however, as noted before, was elicited from all levels of the cord between C4 and L4, but with variable intensity.

Repeated compression of the cord at pressures of 100 to 200 mm. of mercury in the same animal caused signs of deterioration of the response; for this reason, it was not possible in any single experiment to obtain entirely valid comparisons between any two segments. The relative amplitude of the responses obtained from compression of various segments of the cord is illustrated in Fig. 3 based on results obtained in 9 animals.

**Varying Degrees of Compression.** The rise in mean blood pressure tended to be proportional to the degree of compression of the cord up to approximately 200 or 250 mm. of mercury of compression (Fig. 4a). These ex-