INTRODUCTION: In the spinal lesions, the thoracic and lumbar spinal cord are often involved in disease process and injuries, such as spinal canal stenosis, disc herniation, ossification of posterior longitudinal ligament (OPLL), tumor and vertebral fracture. It is generally considered that the genesis of myelopathy associated with the pathological conditions of the spine, may result from mechanical compression of the spinal cord. This factor may change the intraparenchymal circulation and produce spinal cord dysfunction. The mechanical compression may also lead to a series of intraneural reactions, including edema formation, dymyelination, motor neuron dysfunction and fibrosis. In general, the development of myelopathy is thought to be closely related to intraparenchymal edema resulting from compression. Basic research investigations in this aspect, however, are quite few and details of circulatory problems in the spinal cord are not so well understood. Especially, the basic pathophysiology of circulatory disturbance induced by ischemia and congestion is not fully understood. The aim of the present experimental investigation was to examine the effect of ischemia and congestion on the spinal cord.

METHODS: The mongrel adult dogs, weighing 15 to 20 kg, were ventilated on a respirator under general anesthesia. The femoral artery and vein were cannulated, and arterial blood pressure and central venous pressure were monitored in all animals throughout the experiment. At first, the third lumbar laminae were removed, and the dural tube was exposed widely. Aorta was clamped as an ischemia model of the nerve root and inferior vena cava was clamped as a congestion model at the 6th costal level for 30 minutes using forceps transpleurally. Measurements of blood flow (electrolytic hydrogen washout method, N=28), partial oxygen pressure (baro-grafographic method, N=20) and spinal evoked potentials (electro-myographic meter, N=24) in the 3rd lumbar cord were repeated over a period of one hour after release of clamping. Finally, we examine the status of the blood-spinal cord barrier under fluorescence microscope after injection of Evans blue albumin (EBA) into the cephalic vein to find out what sort of circulatory disturbance occurred in the lumbar cord (N=10). Comparison values were performed using a repeated-measures analysis of variance and post hoc (Scheffe) compared before and after clamping of the Aorta or inferior vena cava. Data were entered into a database and analyzed by using SPSS statistical soft ware, version 14.0J (SPSS Inc, Chicago, IL). A probability of 5% was considered statistically significant.

RESULTS: The femoral arterial pressure and CVP before clamping was 112.5 ± 5.6 mmHg (average ± S.E.M) and 5.0 ± 2.5 cmH2O, respectively. Immediately after Aorta clamping, the arterial pressure dropped to 20-34 mmHg in the meantime, central venous pressure was slightly elevated. When the vena cava was clamped, central venous pressure increased to about 4-5 times of the pressure before clamping and arterial pressure was reduced by half. After release of clamping, both arterial and venous pressures quickly returned to the pressure before clamping.

The absolute blood flow volume in the gray matter and white matter was 38.8 ± 5.6 and 15.3 ± 2.5 ml/min/100g, respectively. The blood flow in the gray matter due to Aorta and vena cava clamping fell to 59 to 90% of the blood flow before clamping in the ischemic model (p<0.05) and to about 19-59% in the congestion model (p<0.05). The blood flow in the white matter fell to 54 to 85% of the blood flow before clamping in the ischemic model (p<0.05) and to about 21-44% in the congestion model (p<0.05). When the clamp was released, the blood flow in the ischemic model was restored within 1 hour. The blood flow in the congestion model, however, did not recover and stayed at the reduced level in the gray (p<0.05) and white matter (p<0.05).

The partial oxygen pressure (PO2) in the gray matter and white matter was 26.0 ± 2.1 and 12.4 ± 2.3 mmHg, respectively. The changes of PO2 in the lumbar cord indicated a similar tendency to blood flow, 75 to 87% and 72 to 83% drop in the gray (p<0.05) and white matter (p<0.05) of the ischemic model (p<0.05), respectively. In the congestion model, 23 to 38% and 28 to 38% drop in the gray (p<0.05) and white matter (p<0.05), respectively. After release of clamping, PO2 in the gray and white matter recovered completely in both models.

In the electrophysiological study, the latency of the ascending and descending SEP prolonged by 122.4% (p<0.05) and 118.8% (p<0.05) in the ischemia model, respectively, and 111.3% (p<0.05) and 113.1% (p<0.05) in the congestion model, respectively. When the clamp was released, the latency in the ischemic model was restored within 1 hour. The latency of the ascending and descending SEP in the congestion model, however, did not recover and stayed at the prolonged level (p<0.05). The amplitude of the ascending and descending SEP diminished by 8.4% (p<0.05) and 11.9% (p<0.05) in the ischemia model, respectively. In the congestion model, however, the amplitude of the ascending and descending SEP only fell to 16.6% (p<0.05) and 17.8% (p<0.05), respectively. These dropped amplitudes returned almost completely to base line within one hour after release of clamping.

After intravenous injection of EBA, marked extravasation of EBA in the lumbar cord was evidenced after a 30 minutes clamping of the vena cava (Fig.1B), but not by Aorta clamping in all the animals, where there was no extravasation of EBA and the blood-spinal cord barrier was preserved (Fig.1A). In the venous congestion model, EBA diffused throughout the intramedullar space and stained in the motor neuron.

SIGNIFICANCE: Intraparenchymal edema formation may be the earlier phenomenon inducing the dysfunction of the spinal cord rather than the arterial ischemia. Venous congestion may be a preceding and essential factor of circulatory disturbance inducing myelopathy.

REFERENCES: