INTRODUCTION: The dorsal root ganglion (DRG) should not be overlooked when considering the mechanism of low back pain and sciatica, so it is important to understand the morphologic and functional changes that occur as a result of DRG compression. A large number of sensory neurons are present in the DRG and the DRG is reported to have an abundant vascular network compared with other nerve tissues. It is generally considered that the genesis of radiculopathy associated with the degenerative conditions of the spine may result from both mechanical compression and circulatory disturbance.\(^1\) However, the basic pathophysiology of circulatory disturbance induced by ischemia and congestion is not fully understood. This study is to investigate the effect of ischemia and congestion on the DRG using an in vivo model.

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. Sixth and seventh lumbar laminae were removed and seventh lumbar DRG 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 (hydrogen injection washout method, \(N=14\)), partial oxygen pressure (polarographical method, \(N=14\)) and action potentials (electro-myographic meter, \(N=14\)) in the DRG were repeated over a period of one hour after release of clamping. Finally, we examine the status of the intraganglionic blood-vessel 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 DRG (\(N=14\)).

RESULTS: Immediately after Aorta clamping, blood pressure in the femoral artery dropped to 20-34 mmHg in the meantime (Fig.1A), central venous pressure was slightly elevated (Fig.1B). When the vena cava was clamped, central venous pressure increased to about 3-4 times of the pressure before clamping (Fig.1B) and blood pressure in the femoral artery was reduced by half (Fig.1A). After release of clamping, both arterial and venous pressures quickly returned to the pressure before clamping.

The blood flow in the DRG due to Aorta and vena cava clamping fell to 55 to 85% of the blood flow before clamping in the ischemic model and to about 15-30% in the congestion model. When the clamp was released, the blood flow in the ischemic model was restored with in 1 hour, while blood flow in the congestion model, however, did not recover, stayed at the reduced level (Fig.2A). The changes of PO\(_2\) indicated a similar tendency to blood flow, 60 to 80% drop in the ischemic model and 30 to 40% drop in the congestion model. After release of clamping, PO\(_2\) recovered completely in both models (Fig.2B). The amplitude diminished by 78.6% in the ischemia model and 30.3% in the congestion model. This drop of amplitude returned almost completely within one hour after release of clamping.

After intravenous injection of EBA, the vascular permeability was maintained in the DRG bilaterally after clamping of the Aorta (Fig.3A) as the control group. After clamping of the vena cava, however, a large amount of EBA was seen outside the microvessels in the DRG bilaterally and extensively through out the intercellular space (Fig.3B).

DISCUSSION: Disc protrusion and lateral stenosis may lead to compression of epidual veins and dilatation of non-compressed veins. Cooper et al.\(^1\) noted a significant relationship between evidence of venous obstruction, intraneural and perineural fibrosis, and neural atrophy. Fibrosis may further impede nutrient transfer to endoneurial fibers, as well as predisposing to nerve stretch injury. The experimental work done by Olmarker et al.\(^1\) showed that the average minimum pressure in the inflated balloon compressing the nerve roots of the pig cauda equina required to stop the flow in the capillaries was 40 mmHg and in the venules was 30 mmHg. In this study, it was confirmed that DRG ischemia had a more serious influence on blood flow, PO\(_2\), and action potential than DRG congestion. After 30 min of DRG ischemia, recovery occurred with reperfusion, but longer ischemic periods will cause a permanent effect on the function of sensory neurons due to oxygen deficiency. When changes in the femoral arterial and central venous pressures were monitored after obstruction of blood flow, both the arterial and venous pressures decreased after aortic blockade and the arterial pressure increased slightly after obstruction of the inferior vena cava. However, the central venous pressure showed an approximately 4-fold increase immediately after obstruction of the inferior vena cava, and this sudden increase in venous pressure could have a marked influence on the capillary blood pressure and blood permeability in the DRG. Venous congestion may be a preceding and essential factor of circulatory disturbance in the compressed DRG inducing radiculopathy.

CONCLUSION: Intragnanglionic edema formation may be the earlier phenomenon inducing the dysfunction of the DRG rather than the arterial ischemia in the clinical point of view.