the spinal cord.

Under the finding, laminectomy was performed in two cases (C2-C7, C1-Th2). After operation, some clinical symptoms disappeared or reduced. Heaviness of head, dizziness, blurred vision, syncope attack disappeared and sensory-motor disturbances, ataxia, headache, tinnitus reduced. Stiffness of neck and shoulder remained.

Postoperative air myelogram showed filling of the air anterior and posterior to the cervical cord. This finding may reveal the decompression of the cord. This result seems to suggest that the relative narrowing of the cervical subarachnoid space may cause an intermittent compression and/or an intermittent hyperelongation of the spinal cord due to compression during neck movement. This mechanism may produce some clinical syndrome of whiplash injury. The relative narrowing of the subarachnoid space may result from two factors, congenital and/or acquired. 1) congenital relative or absolute narrowing of the spinal canal to the spinal cord and congenital inequality of the growth in length between spinal cord and canal. 2) subarachnoid narrowing due to traumatic changes of the epidural soft tissue in the spinal canal; bleeding, edema, adhesion, venous congestion, hypertrophy of ligamenta flava or posterior longitudinal ligament, disc hernia, intrathecal pressure change, etc.

It seems that the individual difference of the clinical course of whiplash injury can be explained from this mechanism.

k-20. Cerebrovascular Insufficiency at the Chronic Stage of Traumatic Cervical Syndrome

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Cerebrovascular insufficiency is not infrequently observed at the chronic stage of traumatic cervical syndrome. Any type of the insufficient cerebrovascular disorders could be incident. They are conventionally divided into three types: 1) vertebral basilar insufficiency, 2) carotid basilar insufficiency, and 3) carotid insufficiency. Cervical hemodynamics still remain problematical in the cases of the traumatic cervical syndrome with these insufficiencies.

Fifty seven patients in our series of the chronic traumatic cervical syndrome with such cerebrovascular insufficiencies were studied by means of our technique of the subclavian or brachioaxillar catheterization angiography. Especially, dynamic movements of the head and neck were analyzed angiographically. It
was the most common pattern of cervicocranial hemodynamics in these cases that the vertebrobasilar circulation was dominant in ipsilateral hyper-rotation of the head and neck and alternated with the carotid dominancy in contralateral hyper-rotation. Although this dominancy was competitive and compensative in the cases of incomplete vascular blocking, it could be decompensative in the complete blockade. These dynamo-cervical blockades combined with static potency of the vessels were demonstrated in parallel with the clinical manifestations of cerebrovascular insufficiency. The mechanisms of the vascular blockade were: 1) distributive, 2) microcirculatory, and 3) systemic. Distributive insufficiencies include: a) arterial, b) venous, and c) collateral. One hundred and eighty nine lesions of arterial stenosis and/or occlusion were demonstrated in our series of 62 angiographies. They were: vasospasm, thrombo-embolism, vasocompression (osteo-vertebral, dynamo-vertebral and myotonic), vasostretch-torsion, arterial kink, and hypoplasia. Dynamo-vertebral vasocompression without osteophytic spurs and myotonic arterio-venous compression without organic vascular lesions represented the characteristically common patterns of the lesions. Dynamo-vertebral stenosis and/or occlusion included not only the vertebral arteries but the carotid. The lesions of the vertebral artery were predominantly restricted in the V-3 and V-4 portions. This suggested the important relationships between the patterns of the vertebral artery (atlanto-epistropheal loops, intracranial penetration, and vertebro-basilar fusion) and tonic neck reflex in upper cervical injuries. On the contrary, osteophytic arterial compressions were demonstrated in far small incidence. Special case showed an anomalous infra-epistropheal penetration of the vertebral artery into the vertebral canal, as reported by J. Rickenbach, and compressed by epistropheus in hyper-rotation. It might compress the upper cord. Myotonic compressions on the cervical collaterals of the vertebral and the carotid arteries were not infrequent. Three cases of the subclavian steal and one case of combination with the patent primitive trigeminal artery were demonstrated. These collateral insufficiencies may play an important role in the cerebrovascular disorders of the traumatic cervical syndrome.

One of the most important factors of the microcirculatory disturbance is the rheological property of the blood. It includes the viscosity and the suspension stability of the blood. It has been reported elsewhere that the micro-hemocirculation is predominantly disturbed in severe trauma or shock and diabetes mellitus and hypothermia etc. In these cases, the significant hemoconcentration and denaturation of the blood may occur. Suspension stability of the blood is lowered in denaturation, and results in intravascular aggregation of the blood corpuscles (IVA). Decreased albumin, and increased alpha and beta globulins and lipoproteins, and superimposed variation in fibrinogen are demonstrated characteristic. In our series of 67 cases of the chronic traumatic cervical syndrome, which showed clinically cerebrovascular insufficiency, the deviation of plasma protein fractions was characteristic. It includes: 1) decrease of total protein, 2) abnormal decrease of albumin, 3) abnormal increase of alpha-1 globulin, 4) moderate increase of alpha-2 globulin, and 5) compensative moderate
decrease of beta globulin. Hemoconcentration was not dominant. These could be the causative adjuncts to the microcirculatory insufficiency within the brain and upper cord.

In the dynamo-vertebral and myotonic cerebrovascular insufficiency, stellate ganglion-block or perivertebral infiltration block and epidural infusion block might be effective. In the cases of microcirculatory insufficiency, administration of low molecular weight dextran or other plasma expanders is to be effective. Besides, adequate regulation of fibrinolytic activation system should be effective. Indication of operation must be expanded.

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**k-21. Effect of Stellate Ganglion Block upon Carotid and Vertebral Blood Flow in Dogs and in a Man**

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In previous papers we demonstrated that so-called whiplash injury becomes manifest when an autonomic nervous system loses its normal balance. We have treated 110 whiplash injury patients in three years with a course of stellate ganglion block which has proved to be quite satisfactory.

Stellate ganglion block was employed by many clinicians for the treatment of various cerebrovascular diseases since a known report by Leriche. Forbes and Cobb proved experimentally the regulatory effects of cervical sympathetic system upon intracranial blood flow by microscopic observation of pia-arachnoid blood vessels before and after electric stimulation of the cervical sympathetic chain.

The works of Gibbs, Lennox, Harmel, and Scheinberg were contrary to the results obtained by aforementioned workers. They could find no remarkable changes in cerebral blood flow after stellate ganglion block, while Shenkin, measuring by nitrous oxide technique, suggested an increase of flow after bilateral stellate ganglionectomy.

We studied common carotid and vertebral blood flow in mongrel dogs and internal carotid flow in one patient before and after stellate ganglion block using electromagnetic flowmeter. We knew that it causes relatively constant increase of flow in 20 out of 22 dogs examined. The increase are 12.8% in the common carotid and 35.6% in the vertebral arteries in the average. We have also ascertained that these increases depended neither upon conditions of blood pH or carbon dioxide tension which were variously changed by controlling respiration, nor upon systemic blood pressure changes.

Right internal carotid blood flow changes after stellate ganglion block was measured in a 60 years old man who had bilateral huge parasagittal meningioma. Before stellate ganglion block internal carotid flow was 377.6 ml/m, began to