Effects of Electrical Stimulation of Cervical Sympathetic Trunks on Microcirculation in the Facial Nerve

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Abstract This study evaluates the circulatory effects of electrical stimulation of the cervical sympathetic trunks on blood flow in the common carotid artery and facial nerve tissue in dogs. Marked increases in arterial pressure and heart rate were observed due to electrical stimulation of the cervical sympathetic trunks, while blood flow volume in the common carotid artery and in the facial nerve tissue decreased markedly. It was assumed that microcirculation of the facial nerve is definitely impaired by electrical stimulation of the cervical sympathetic trunks, and the tonicity of the sympathetic nervous system appears to be a major factor in changes in the microcirculation of the facial nerve. It is well known that impaired circulation in the nutrient vessels of the facial nerve has an important effect on the pathogenesis of facial palsy. The hypertonicity of the sympathetic nervous system is closely involved in the onset of facial palsy.

Key words: facial palsy, stellate ganglion block, cervical sympathetic trunks, electrical stimulation, microcirculation in facial nerve.

Idiopathic facial palsy, known as Bell’s palsy, is an acute unilateral weakness or paralysis of the face resulting from peripheral facial nerve dysfunction. While there is no readily identifiable cause for Bell’s palsy, the prevailing theory about acute paralysis is that of combined primary and secondary ischemia. It can be summarized as follows: a local arteriolar constriction (the primary ischemia) provokes an increase in permeability of the arteriolar wall due to hypoxemia; the ensuing transudate then compresses the facial nerve fibers; an impairment of the venous and lymphatic return of the circulation causes more edema, compressing the nerve

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fibers further, and causing ischemia of a secondary nature [1]. A vicious circle is created in this way. The main disease state related to the onset of Bell’s palsy is impairment of microcirculation of the facial nerve in the temporal bone. The cause of the arteriolar constriction is not known. An unequivocal explanation might never be found, since various factors are capable of triggering the same pathogenetic mechanism. The ischemia is generally accepted as the triggering factor in this paralysis. On the other hand, it is well known that stimulation of sympathetic nerves does not cause vasoconstriction. It was demonstrated that the sympathetic nerve activity influenced the blood flow in a sciatic nerve [2]. The tonicity of the sympathetic nervous system is assumed to be an important factor in these ischemic changes in the facial nerve. Therefore, in this study the tonicity of cervical sympathetic trunks, which send peripheral branches to the feeding vessels of the facial nerves, was altered by electrical stimulation. Changes in the blood flow volume of the common carotid artery and the facial nerve tissue were determined, and the circulatory effects of electrical stimulation of the cervical sympathetic trunks on the microcirculation of the facial nerve were studied.

MATERIALS AND METHODS

All experiments were performed on thirty mongrel dogs of either sex, weighing 9.5–14 kg. All animal were anesthetized with 100 mg of pentobarbital and 4 mg of pancuronium infused into a femoral vein. The animals’ tracheas were intubated and artificially ventilated under constant conditions with room air so that $P_{\text{aCO}_2}$ was maintained at approximately 37±2 mmHg. The femoral vein was cannulated with polyethylene tubing for continuous blood pressure recording and for the collection of blood samples. A skin incision was made on the frontal cervical region to expose the common carotid artery, and an electromagnetic flowmeter (Nihon Koden, MFV-3200) was connected to continuously measure the common carotid arterial blood flow volume. Skin incisions were also made on the lateral cervical region, the facial nerves were exposed at the ectopic region from the stylo mastoid foramen, a probe for the measurement of tissue blood flow was implanted, a laser Doppler tissue blood flowmeter (Advance, ALF2100) was connected, and the tissue blood flow volume in the facial nerve was continuously measured. Bipolar needle electrodes for electrical stimulation were implanted by puncturing the cervicothoracic ganglion. After attaching the measuring and stimulating devices, the stability of the hemodynamic parameters was confirmed. In the first series of experiments (A), the circulatory effects were determined during cervical sympathetic trunk stimulation by 10 and 20 Hz square waves of 0.5 ms width and intensity of 15 V for 2 min. In the second series of experiments (B), electrical stimulation was applied for 60 min using 20 Hz square waves of 15 V. The effects of these changes on the tone of the sympathetic nervous system, the blood flow volume in the common carotid artery and the facial nerve tissue were evaluated.

Statistical analysis with the two-tailed Student’s t-test for paired data was
performed by comparison of baseline values. $p$ values less than 0.05 were considered statistically significant. All values are given as mean±SE.

**RESULTS**

1. **Short time stimulation group**

   1) **Circulatory changes with electrical stimulation at 10 Hz.** Electrical stimulation produced significant increases in arterial blood pressure and heart rate

   **Table 1.** Hemodynamic effects of electrical stimulation of cervicothoracic ganglion in dogs.

<table>
<thead>
<tr>
<th>Time (s)</th>
<th>10 Hz</th>
<th>20 Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean A. P. (%)</td>
<td>Heart rate (%)</td>
</tr>
<tr>
<td>Pre-stim.</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>10</td>
<td>117.3±2.4*</td>
<td>103.3±2.7</td>
</tr>
<tr>
<td>20</td>
<td>121.6±2.6*</td>
<td>108.1±5.6</td>
</tr>
<tr>
<td>30</td>
<td>120.0±1.6*</td>
<td>108.7±5.8</td>
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<tr>
<td>60</td>
<td>120.3±2.1*</td>
<td>109.3±5.3</td>
</tr>
<tr>
<td>90</td>
<td>120.6±4.2*</td>
<td>109.9±5.4</td>
</tr>
<tr>
<td>120*</td>
<td>118.4±3.8*</td>
<td>111.7±5.2*</td>
</tr>
<tr>
<td>130</td>
<td>108.3±4.7</td>
<td>109.8±5.9</td>
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<td>140</td>
<td>100.3±5.1</td>
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<td>150</td>
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<tr>
<td>180</td>
<td>92.8±3.1</td>
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<tr>
<td>210</td>
<td>92.2±1.9</td>
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<tr>
<td>240</td>
<td>91.5±1.9</td>
<td>100.6±1.7</td>
</tr>
</tbody>
</table>

Values are mean±SE. *Stop stimulation; *$p<0.05$ vs. pre-stimulation level.

![Graph](image)

**Fig. 1.** Changes in the blood flow volume in the common carotid artery and facial nerve tissue during electrical stimulation of cervical sympathetic trunks by 10 Hz square waves for 2 min. ●, common carotid artery; ○, facial nerve tissue. *$p<0.05$ vs. pre-stimulation level.

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throughout the period of stimulation, and significant decreases in the common carotid arterial blood flow volume. A significant decrease persisted 30 s after the stimulation stopped. There was no significant decrease in the tissue blood flow volume of the facial nerves (Table 1 and Fig. 1).

2) Circulatory responses with electrical stimulation at 20 Hz. Electrical stimulation at 20 Hz also caused significant increases in arterial blood pressure and heart rate throughout the period of stimulation, and significant reduction in the blood flow volume in the common carotid artery and the facial nerve tissue. At the end of stimulation, the common carotid arterial blood flow volume took more than 90 s to return to the pre-stimulation level. A significant reduction of the tissue blood flow persisted throughout the period of the stimulation (Table 1 and Fig. 2).

2. Long duration stimulated group

1) Changes in arterial pressure and heart rate (Fig. 3A and B). When electrical stimulation was applied, there was a significant increase in arterial pressure, followed by a gradual decrease. After 15 min, the value had basically returned to the pre-stimulation level and reached a plateau. When the stimulation was stopped, the arterial pressure showed a transient decrease but it returned to the previous value after 10 min. The heart rate also showed a sharp increase at the same time at the start of electrical stimulation and gradually decreased thereafter, but a significant increase persisted throughout the period of stimulation. When the stimulation was stopped, there was a transient decrease and the value returned to the pre-stimulation level.

2) Changes in blood flow volume in the common carotid artery and facial nerve tissue (Fig. 3C). The blood flow volume in the common carotid artery showed a sharp drop at the same time as electrical stimulation started and it reached the lowest level after 15 min. A significant decrease of 50–60% of pre-stimulus value

* *p < 0.05 vs. pre-stimulation level.

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Fig. 3. A: Changes in heart rate with electrical stimulation of cervical sympathetic trunks by 20 Hz square waves for 60 min. *p < 0.05 vs. pre-stimulation level. B: Changes in mean arterial blood pressure with electrical stimulation of cervical sympathetic trunks by 20 Hz square waves for 60 min. *p < 0.05 vs. pre-stimulation level. C: Changes in the blood flow volume in the common carotid artery and facial nerve tissue with electrical stimulation of cervical sympathetic trunks by 20 Hz square waves for 60 min. ●, common carotid artery; ○, facial nerve tissue. *p < 0.05 vs. pre-stimulation level.
persisted throughout the period of stimulation. When stimulation was stopped, there was a simultaneous sharp increase, followed by a transient slight increase over the pre-stimulation level, and then returned to the prior value. The tissue blood flow volume of the facial nerves also showed a sharp drop when the electrical stimulation was started, with the minimum level appearing after 30 min. Significantly low values of 60–70% of the pre-stimulus value continued during the stimulation. When the stimulation was stopped, there was a sharp increase, but the degree of increase gradually decreased. It took approximately 30 min after stimulation was stopped before the pre-stimulation value was reached.

DISCUSSION

The mechanism of Bell's palsy is considered to be combined primary and secondary ischemia. Impairment of microcirculation of the facial nerves inside the facial canal is thought to be the main pathophysiologic condition of the acute state of Bell's palsy. Edema and compression due to ischemic changes in the nerves appear to be the mechanism for the development of palsy. There is no readily identifiable cause for the primary ischemia of the facial nerves. Anatomically the main feeding vessel of the facial nerves in the temporal bone is the external carotid artery. Several reports have concentrated on a close relationship between circulatory disorders in the external carotid arterial system and the onset of Bell's palsy. In clinical cases, it has been reported that facial palsy occurred due to circulatory disorders in the external carotid arterial system associated with therapeutic procedures such as plugging of the external carotid arterial system including the middle meningeal artery [3, 4], intra-arterial chemotherapy in the external carotid arterial system [5–7], or trigeminal root decompression by ligation of the middle meningeal artery [8, 9]. There have also been reports on facial palsy produced experimentally by means of circulatory disorders caused by plugging the external carotid artery or its peripheral branches, the internal maxillary artery or the posterior auricular artery [10, 11], and injection of epinephrine into the stylomastoid foramen [12]. It was strongly suggested in all of these cases that ischemic paralysis of facial nerves occurred due to circulatory disorders in the external carotid arterial system. Anatomically, however, the main arteries in the facial neural tubes are the stylomastoid artery which branches from the accessory meningeal artery and the petrosal artery which branches from the middle meningeal artery [13]. A complex vascular network is formed by anastomosis of these arteries which feed the facial nerves [14, 15]. Even if the blood flow in part of this network is impaired, it is difficult for an overall ischemic condition to occur [16], and blockage of the stylomastoid artery had no effect on the function of the facial nerves [17]. Therefore, the mechanism of onset of circulatory disorders in these reports of facial palsy was artificial in all cases and also appeared to cover a very wide range. There seems to be a definite difference between circulatory disorders associated with the mechanism of onset of Bell's palsy occurring due to physiological conditions with
no external factors involved, and the mechanisms of onset in these reports. Since
many patients with Bell's palsy have facial pain and stiff shoulders, as well as pain
resembling "stiff shoulder" from the retroauricular region through the neck [18], it
is readily assumed that many patients with Bell's palsy show an increase of
sympathetic nervous activity on the affected side of the neck and head region. The
existence of a vasoconstrictive sympathetic nerve fiber innervating the vasa nervo-
rum was determined in the sciatic nerve in rats [19]. It is also considered possible
that sympathetic hypertonicity is involved in the onset of circulatory disorders
associated with the onset of Bell's palsy.

As demonstrated in both experiments, the arterial blood pressure and heart
rate showed marked increases systematically when electrical stimulation was
applied to the cervical sympathetic trunks, but the blood flow volume in the common
carotid artery and in the facial nerve tissue decreased. Nerve fibers from the
cervical sympathetic trunks are found not only in the head and neck region but
sympathetic cardiac branches also run efferently to the heart and stimulation of
them leads to systemic increases in the arterial pressure and heart rate. However,
in the region supplied by the stimulated perivascular sympathetic nerves fibers,
impairment of microcirculation is caused by severe vasoconstriction. Marked
decreases in facial tissue blood flow volume due to the effects of electrical cervical
sympathetic nerve stimulation have been observed using microspheres in rabbits
[20] and cats [21]. Few reports of studies on blood flow in the facial nerves have
appeared to date, but it has been reported to be about one-fourth of the cerebral
blood flow per unit weight in measurements on guinea pigs using microspheres
[22]. In these experiments, changes in the tissue blood flow of the facial nerves
were investigated using a laser Doppler blood flowmeter. This method has been
used in continuous measurements of microcirculation by analysis of the Doppler
shift which is caused by that portion of light scattered by the red cells. It gives the
velocity of flow for those cells and is also useful for continuous evaluation of
microcirculation locally in the facial nerves [23]. Electrical stimulation of the
cervical sympathetic trunks caused a decrease in the facial nerve tissue blood flow
volume, parallel with the decrease in blood flow volume in the common carotid
artery, and there was definite impairment of the microcirculation of the facial
nerves. However, further studies will be required in the future to determine if this
impairment of microcirculation is the direct cause of Bell's palsy.

In conclusion, the findings in the present study indicate that electrical stimula-
tion of the cervical sympathetic trunks caused marked decreases in blood flow
volume in the common carotid artery and in the facial nerve tissue, which resulted
in impairment of microcirculation of the facial nerve itself, and it was assumed that
hypertonicity of the sympathetic nervous system is closely involved in the onset of
Bell's palsy.

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