

be a passive change secondary to vasoconstriction in other areas. Changes in the NA-induced BF increase by both $\alpha 1$ - and $\alpha 2$ -adrenoceptor antagonists mirroring the changes in the BF-decrease responses supports this passive mechanism hypothesis. This mechanism requires fewer adrenoceptors at the sites of BF increase than at those of BF decrease. McDougall recently reported that adrenergic sensitivity (both $\alpha 1$ - and $\alpha 2$ -) was decreased in the knee joint in monoarthritic rats induced by injection of CFA [16]. If these changes are scattered as patches of pathological tissue changes in polyarthritis induced by CFA, the distribution of adrenoceptors would perhaps not be even. If the site with the BF-increase response was less sensitive to NA, the passive mechanism would be most likely. Our observation that higher doses of NA induced only a BF decrease at the sites with the BF-increase response to SS would support this hypothesis.

The BF-increase responses to SS observed in this experiment might also be a passive response resulting from redistribution of the BF after vasoconstriction in other areas. If this is the case, the redistribution of BF must be a local one, because only a slight and inconsistent increase of MAP was seen after SS in 67.6% of the rats. However, different efficacy of antagonists on SS-induced BF increase from NA-induced BF-increase would suggest redistribution of BF is not the sole cause for vasodilatation: The SS-induced BF-increase response was blocked by a higher dose of prazosin (0.1 mg/kg, I.A.) whereas prazosin suppressed the BF-decrease response to SS at 0.03 and 0.1 mg/kg. In contrast, the SS-induced BF-increase response was not blocked by $\alpha 2$ -adrenoceptor antagonist even at as high as 10 mg/kg. Again the BF-decrease response to SS was suppressed by CH-38083, though at only 1 mg/kg. These differences in effectiveness of antagonists suggest an involvement of other mechanisms additionally to NA-mediation in the SS-induced vascular responses.

A few possible mechanisms can be suggested for this vasodilating response. The first is the β -adrenoceptor. Activation of this receptor induces increase in the contractility of cardiac muscle and relaxation of vascular smooth muscle. Our present result showed ineffectiveness of propranolol on the BF increase response to SS, thus excludes this possibility.

The second is release of vasodilator substances such as nitric oxide (NO) from endothelial cells by $\alpha 2$ -adrenoceptor stimulation, but this action was not found in smaller vessels such as we measured in this experiment and the BF-increase response to SS was not suppressed after CH-38083 injection. Our previ-

ous report demonstrated the BF-increase response was not mediated by the NO system, either [17].

Another possibility for the active mechanism is that some vasodilating substance(s) might be released from sympathetic nerve terminals. The known vasodilating substances released from sympathetic nerves are adenosine 5'-triphosphate (ATP) acting through P2Y purinoceptors [27], acetylcholine (Ach) in certain areas and prostaglandins (PGs) through activation of presynaptic $\alpha 2$ -adrenoceptor on the sympathetic post-ganglionic nerve terminals in pathological states [28]. Our preliminary study showed that a muscarinic antagonist, i.e., atropine, and aspirin, a blocker of cyclo-oxygenase, had no effect on the BF-increase response (data not shown), and that CH-38083 alone had no effect, either. Therefore, Ach and PGs would not seem to be involved. ATP has vasodilating action through P2Y purinoceptor, thus the possible involvement of ATP in the BF-increase response to SS should be studied.

The fourth possible mechanism for active vasodilatation is vasodilatation mediated by neuropeptide(s). CP-96345 (1 mg/kg), a substance P (SP) antagonist, tended to reduce the BF-increase response [19]; thus, SP might have been involved in this phenomenon. Furthermore, since our preliminary experiment has shown up-regulation of SP expression in sympathetic ganglia (unpublished observation), SP co-released with NA from sympathetic terminals might mediate the BF-increase response to SS.

In conclusion, involvements of both adrenergic-passive and non-adrenergic-active mechanisms are suggested in this BF-increase response to SS.

Pathophysiological meaning of the present study. Along the same line as the present observation, McDougall *et al.* reported the vasoconstriction response to electrical stimulation of sympathetic or saphenous nerves was diminished in rats with CFA-induced knee joint inflammation [13, 14]. The vasoconstriction response to SS was reduced in rats with carrageenan inflammation as well [15]. These findings might be the result of altered regulation by postjunctional α -adrenoceptors in blood vessels and release of vasodilators from inflamed tissues.

Changed vascular response to sympathetic nerve stimulation was not limited to inflamed condition: Lundberg *et al.* reported that the electrical stimulation of the sympathetic nerve caused cutaneous vasodilatation in patients with obstructive vascular disease [29]. We also measured BF in rats with chronic constriction injury using the same methods as in the present experiment, and found a BF-increase response to SS similar to that in AI rats (data not shown). In that experimen-

tal series none of the sham-operated control rats showed a vasodilatation response.

The smooth muscles in resistance arteries regulate the vasoconstrictor tone, but in the present observations as well as others this vascular regulation was altered in pathological conditions. These changes in the response to sympathetic activation in pathological conditions might play some role in tissue changes in these conditions. In addition, these changes in vascular responses should be taken into consideration in the therapeutic treatment of patients and their everyday care.

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Effect of Indomethacin on the Development of Eccentric Exercise–Induced Localized Sensitive Region in the Fascia of the Rabbit

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Abstract: The effect of indomethacin on the development of delayed onset muscle soreness (DOMS) and localized sensitive region produced by eccentric exercise was examined in lightly anesthetized rabbits ($n=12$, 2.0–3.3 kg). Repeated eccentric contractions of the gastrocnemius (GS) muscle were made by manual extensions during the tetanic contractions induced by electrical stimulation of the tibial nerve. The development of DOMS was confirmed by evoked reflex EMG in the biceps femoris (BF) muscle elicited by a quantitative manual extension of the GS muscle. The distribution of thresholds for the evoked BF EMG was measured by focal electrical stimulations of the GS muscle. Indomethacin (5 mg/kg in 2% sodium bicarbonate) or a vehicle was injected subcutaneously before, during, and

after the exercise (a total of 60 mg/kg in 12 doses). A clear ropy taut band was palpated at the GS muscle on the second day after the exercise and a localized sensitive region for evoked BF EMG was detected at the depth of the fascia of the band in the exercise and vehicle groups, whereas no such phenomena appeared in the control and indomethacin groups. The palpable band and sensitive region disappeared on the seventh day after the exercise. That indomethacin inhibits the development of DOMS and the localized sensitive region suggests that a sensitization of polymodal-type nociceptors in the fascia mediated by prostaglandins is a possible mechanism for the development of DOMS and the localized sensitive region. [Japanese Journal of Physiology, 52, 173–180, 2002]

Key words: localized sensitive region, eccentric exercise, rabbit, palpable band, indomethacin.

Delayed onset muscle soreness (DOMS) is a common myogenic condition with principal symptoms of tenderness or soreness of affected muscles accompanied by a reduction in their range of movement. It usually occurs 24 to 48 h after unaccustomed exercise or eccentric contractions [1, 2]. However, its underlying mechanisms are not clearly understood. DOMS is considered to be related to a complex set of reactions involving micro-injury of the muscle fibers and connective tissues [3], and the participation of an inflammatory response is suggested [4]. A cellular infiltration of neutrophils, macrophages, and inflammatory mediators has also been demonstrated [5]. In fact, a repetitive administration of relatively high doses of indomethacin, an inhibitor of cyclooxygenase, could

protect against the histological and biochemical changes of DOMS in mice [6]. Thus mechanical and biochemical injuries might be responsible for the development of DOMS [7].

On the other hand, most reports on the experimental DOMS described only the appearance of muscle soreness [8, 9]. A few studies reported the distribution of areas of tenderness [10, 11] and pointed out that the region of the muscle-tendon attachment was the main site of tenderness [10]. In our previous study in human subjects, eccentric exercise of the extensor muscle of the forearm was found to produce a localized tender region on the palpable band accompanying the development of DOMS [12]. The taut band was palpated in the extensor muscle of the forearm near

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the muscle-tendon attachment. The measurement of pain thresholds in deep tissues clearly demonstrated a decrease in pain threshold at the fascia [12]. These results suggested that the sensitization of muscular nociceptors by an inflammatory mediator might be important for the development of DOMS and the focal tender region on the palpable band.

The aim of this study was to examine the effect of indomethacin on the localized sensitive region, which appears after the DOMS procedures, and to clarify the participation of the inflammation in its pathogenic mechanisms.

METHODS

Animal preparation. Twelve rabbits (2.0–3.3 kg, 24 legs) were anesthetized with sodium pentobarbital (30 mg/kg, I.V.). A polyethylene catheter (PE50: outer diameter of 1.0 mm) was then chronically implanted into the auricularis caudalis vein, allowing supplemental doses or continuous infusion of anesthetics (20 mg·kg⁻¹·h⁻¹) during the experimental period.

Rectal temperature was monitored and maintained at 39.0±0.3°C with the use of a heating pad (MK-900, Muromachi Kikai Co., Tokyo, Japan). Heart rate was monitored continuously.

The protocol for this study was approved by the Ethics Committee of the Meiji University of Oriental Medicine.

Eccentric exercise procedure. A lightly anesthetized rabbit (20 mg/kg sodium pentobarbital, I.V.) was set on a frame where its hind leg was fixed on a board to regulate the direction of tetanic muscular contraction. Repetitive eccentric contractions of the gastrocnemius (GS) muscle were performed by manual extensions during tetanic contraction produced by electrical stimulation of the tibial nerve (50 Hz, 1 ms duration, 500 pulses at 0.067 Hz, bipolar insulated needle electrode).

A threshold current for the contraction of GS muscle of less than 0.3 mA was assumed to reflect an adequate electrode placement. Eccentric contractions were repetitively applied for 20 min (80 times) and repeated 3 times with 5 min resting periods (a total of 240 times).

Measurements. Rabbits were lightly anesthetized by sodium pentobarbital (20 mg/kg, I.V.) every time for measurement, then set on a frame.

Measurement of DOMS. The amplitude of the EMG of the biceps femoris (BF) muscle elicited by an extension of the GS muscle was used as an indicator of DOMS. A surface electrode was attached to the

skin over the BF muscle. The plantar was fixed on a board and the knee joint firmly held; then plantar was pushed up about 15° in a dorsal direction by a constant force of about 1 kg to extend the GS muscle. The peak-to-peak amplitudes of the BF EMG were measured three times, and the median value was used for statistical analysis. Each reflex EMG was monitored on an oscilloscope (VC-11, Nihon Kohden, Tokyo, Japan), and recorded on a thermal array recorder (RTA-1200M, Nihon Kohden) and data recorder (RD-1235T, TEAC, Tokyo, Japan).

Distribution of the thresholds and amplitude for reflex EMG activity. The distribution of thresholds of BF EMG evoked by focal electrical stimulation of various tissues of the GS (RT: reflex thresholds) was examined. A stainless steel needle electrode insulated with acrylic resin (180 µm diameter, impedance 391±30 kΩ at 1 kHz; Nishin Medical Institute, Osaka, Japan) was used as a cathodal monopolar stimulating electrode.

A anodal metal surface electrode was attached to the skin 10 mm distant to the needle insertion site, and the insulated needle was inserted manually in 0.5–1.0 mm steps from the skin surface and held in a guide tube attached to the skin with adhesive tape. A train of 5 pulses (100 µs in duration) was applied once every 5 s. The threshold of evoked BF EMG was determined when the reflex responses appeared with a probability over 70%. The cutoff current intensity was 3.0 mA.

The amplitudes of the reflex EMG of the BF muscle (RA: reflex amplitude) evoked by focal electrical stimulation of the tissues on or around the fascia of the GS muscle at a constant intensity (0.5 mA) were also recorded. They were averaged 10 times at intervals of 5 s (Averager, DAT 1100, Nihon Kohden), and the averages were recorded on a thermal array recorder (RTA-1200M, Nihon Kohden) and data recorder (RD-1235T, TEAC). The peak-to-peak amplitudes of the BF EMG were measured and used for statistical analysis.

Experimental schedules.

Experiment 1. Six rabbits (12 legs) were allocated to the control group ($n=6$, no exercise) and exercise group ($n=6$, exercise only) in a crossover manner at intervals of about 1 month. The measurements of BF EMG evoked by the extension of the GS muscle were made before, immediately after, and 1, 2, 3, and 7 d after the exercise.

On the second day after exercise, the GS muscle was carefully palpated and the skin over the detected rosy band was marked. Our previous study in human subjects demonstrated that the minimum pain thresh-

old region (tender point) could be detected on a taut band in the eccentric exercised muscle. The distribution of the RT and RA over the taut band were then measured systematically at 2 mm intervals in the exercise group. Similar measurements were made on the seventh day after exercise.

Experiment 2. Six rabbits (12 legs) were allocated to the indomethacin group ($n=6$, exercise+indomethacin) and the vehicle group ($n=6$, exercise+vehicle) in a crossover manner at intervals of about 1 month. Indomethacin (Wako Pure Chem. Ind., Ltd., Osaka, Japan) was dissolved in 2% sodium bicarbonate (1 ml) and titrated to pH 7.4 with sodium monophosphate. Two percent sodium bicarbonate was used as the vehicle. Indomethacin (5 mg/kg) or the vehicle was injected subcutaneously before, during, immediately after, and 0.5, 1, 1.5, 2, 3, 4, 5, 6, and 7 d after the exercise (a total of 60 mg/kg indomethacin).

Measurements of the development of DOMS were made before, immediately after, and daily on days 1 through 7 after the exercise. The distribution of RTs and RAs was examined on the second day after the exercise. The area corresponding to where the palpable band appeared in experiment 1 was also examined.

Statistics. The data are reported as mean \pm standard deviation (mean \pm SD). The nonparametric multiple test of Tukey and Dunnett's multiple test (StatView ver. 5; SAS Institute Inc., NC, USA) was used for the statistical analysis. A $p < 0.05$ was defined as statistically significant.

RESULTS

Exercise-induced muscle soreness and localized sensitive region

In the exercise group, the exercised legs were kept in a slightly flexed position, and withdrawal response in several cases occurred when the GS muscle was palpated on the 2nd day after the exercise. Increased stiffness of the GS muscle was frequently observed, and these phenomena disappeared on the seventh day. Clear EMG activities were evoked by manual extension of the GS muscle immediately after the exercise and increased to a maximum on the second day (Fig. 1A). They decreased to baseline on the seventh day after the exercise (Fig. 1B). The baseline EMG amplitude of $7.50 \pm 4.37 \mu\text{V}$ increased to $166.1 \pm 66.0 \mu\text{V}$, and this increase was found to be statistically significant ($p < 0.01$, Dunnett's multiple test). The EMG activities of the BF muscle were usually brief bursting discharges. However, prolonged EMG activities were also observed in several cases. The spontaneous EMG activity was also increased to $23.33 \pm 15.68 \mu\text{V}$, from

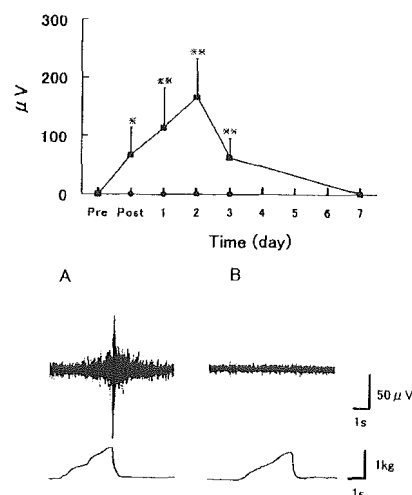


Fig. 1. Evoked EMG activity of the biceps femoris muscle elicited by the extension of the gastrocnemius muscle. The graph shows the changes in amplitude of evoked EMGs of the biceps femoris. Solid circles and solid squares indicate the control and experimental groups, respectively. Asterisks show significant differences compared with the baseline amplitude (pre) (Dunnett's multiple test: * $p < 0.05$, ** $p < 0.01$). The examples of reflex EMGs (upper traces) were those on the second (A) and seventh (B) days after exercise. The monitored force to extend the GS muscle were shown in the lower traces. Pre, before exercise; Post, immediately after.

$7.50 \pm 4.37 \mu\text{V}$ (pre-exercise), on the second day after exercise in the exercise group (Dunnett's multiple test, $p < 0.05$), the change in spontaneous activity had disappeared on the seventh day after the exercise. On the other hand, in the control group (no exercise), extension of the GS muscle evoked no reflex activities during the 7-d experimental period.

A ropy taut band was palpated and a localized sensitive region detected on the band on the second day after exercise. The taut ropy band ($1.3 \pm 0.5 \text{ mm}$ wide, $17.7 \pm 4.7 \text{ mm}$ long, $n=6$, mean \pm SD) was palpated in the GS muscle near the muscle-tendon attachment. The low RT region on the palpable band usually appeared at the restricted area to 4–8 mm distal to the external condyle of the tibia.

The spatial distributions of RT on and around the palpable band are summarized in Fig. 2. On the second day after the exercise, the RTs were remarkably decreased at the depth of the fascia on the palpable band (Fig. 2A). A significant difference between the minimum RT region and other measured points was detected on the second day (Tukey, $p < 0.05$). No localized low RT region was detected on the seventh day (Fig. 2B). On the second day after the exercise, the latency of the evoked BF EMG was $47.7 \pm 4.4 \text{ ms}$

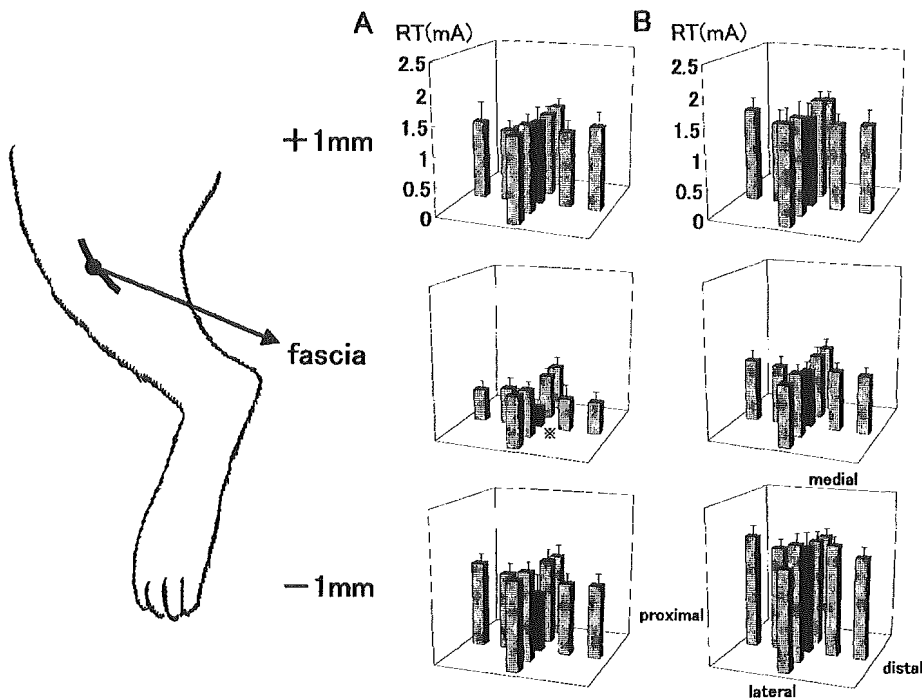


Fig. 2. Distribution of thresholds for the reflex EMG (RT) of the biceps femoris evoked by focal electrical stimulation of the GS muscle at different depths on the second (A) and seventh (B) days after exercise. The column center were adjusted to superimpose the minimum threshold region in each rabbit. The asterisk in fascia (A) shows a significant difference between the center column and the surrounding eight (nonparametric Tukey's multiple test, $p < 0.05$).

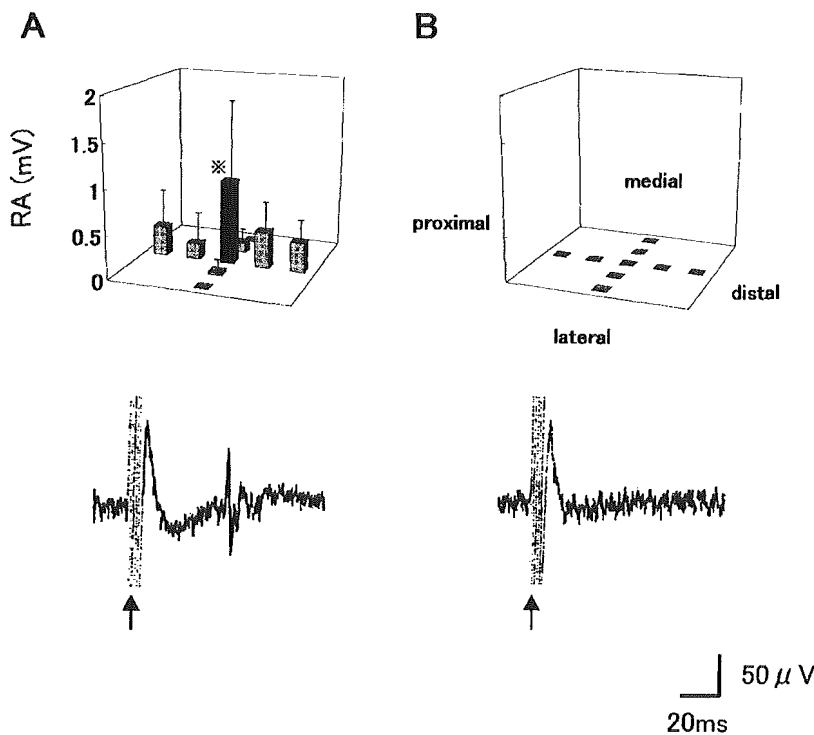


Fig. 3. Distribution of the amplitude of reflex EMG (RA) of the biceps femoris elicited by the constant electrical stimulation (0.5 mA) of the fascia in the GS muscle on the second (A) and seventh (B) days after exercise. The locations of the measuring site were the same as in Fig. 2. The asterisk in the fascia shows a significant difference between the center column and the surrounding eight (nonparametric Tukey's multiple test, $p < 0.05$). The lower trace shows examples of the reflex EMG recorded when the center column (black) was stimulated on the second and seventh days after exercise (↑: electrical stimulation).

(mean \pm SD, $n = 6$), and this had not changed significantly on the seventh day (46.8 ± 2.1 ms).

Figure 3 shows the spatial distributions of RA elicited by a constant current stimulus intensity (upper columns) and representative examples of evoked EMGs (lower traces). At the center of the columns is the largest EMG activity provoked by the stimulation

of the fascia, and no EMG activity was elicited by stimulation 1 mm above or below the fascia in the exercised muscle on the second day after exercise. The evoked EMG amplitude at the most sensitive region on the palpable band was 0.95 ± 1.01 mV (central column), and the other eight measured RAs were lower than in the central region ($0 - 0.41 \pm 0.35$ mV). On the

seventh day (Fig. 3B), the restricted high RA disappeared.

Effect of indomethacin on exercise-induced muscle soreness and localized sensitive region

The amplitude of the BF EMG elicited by the extension of the GS muscle is shown in Fig. 4. In the vehicle group, the evoked EMG was not recorded except for the spontaneous EMG before the exercise, and it appeared on the second day after exercise. It increased to $197.3 \pm 83.3 \mu\text{V}$, from $8.8 \pm 8.1 \mu\text{V}$. The evoked EMG was usually brief bursting discharges (Fig. 4A). On the other hand, in the indomethacin group, the amplitude of evoked EMG was slightly increased on the second day after exercise (Fig. 4B), though the difference was not significant (Dunnett's multiple test, $p=0.281$).

On the second day after exercise, spontaneous EMG activity had also increased to $15.3 \pm 9.2 \mu\text{V}$, from $8.8 \pm 8.1 \mu\text{V}$, in the vehicle group, and to $14.8 \pm 8.8 \mu\text{V}$, from $11.2 \pm 7.9 \mu\text{V}$, in the indomethacin group. These differences, however, were insignificant.

In the exercise group, a ropy taut band was palpated over the GS muscle at the muscle-tendon attachment about 1.4–1.6 mm lateral to the tibia. The minimum threshold region was restricted to 4–8 mm distal to the external condyle of the tibia. The RT and RA were measured 10–22 mm lateral to the tibia and 0–14 mm distal to the external condyle of the tibia.

The contour line illustrates the distribution of RT and RA in the vehicle and indomethacin groups. In

the vehicle group (Fig. 5A), at the depth of the fascia, needling stiffness increased on reaching the palpable band, and RTs were decreased. At the center of the measured area (16 mm lateral to the tibia and 6 mm

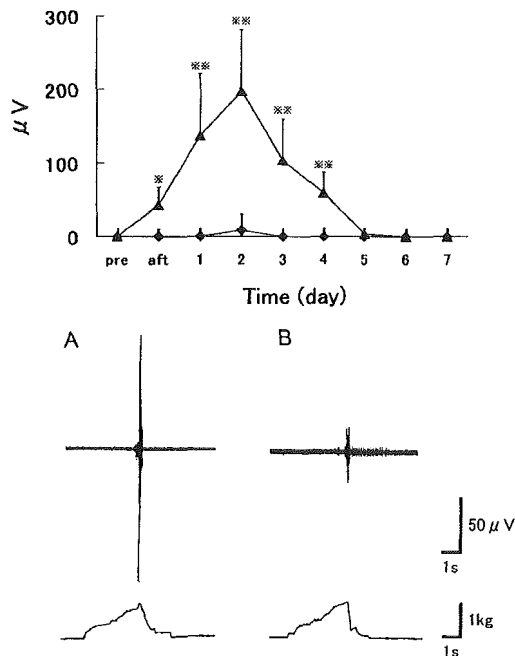


Fig. 4. Effects of indomethacin on the amplitude of the evoked EMG of the biceps femoris elicited by an extension of the gastrocnemius muscle. Solid diamonds and triangles are those of the indomethacin and vehicle groups, respectively. Traces in **A** and **B** are the reflex EMGs of the vehicle and indomethacin groups on the second day after exercise. The notation is the same as in Fig. 1.

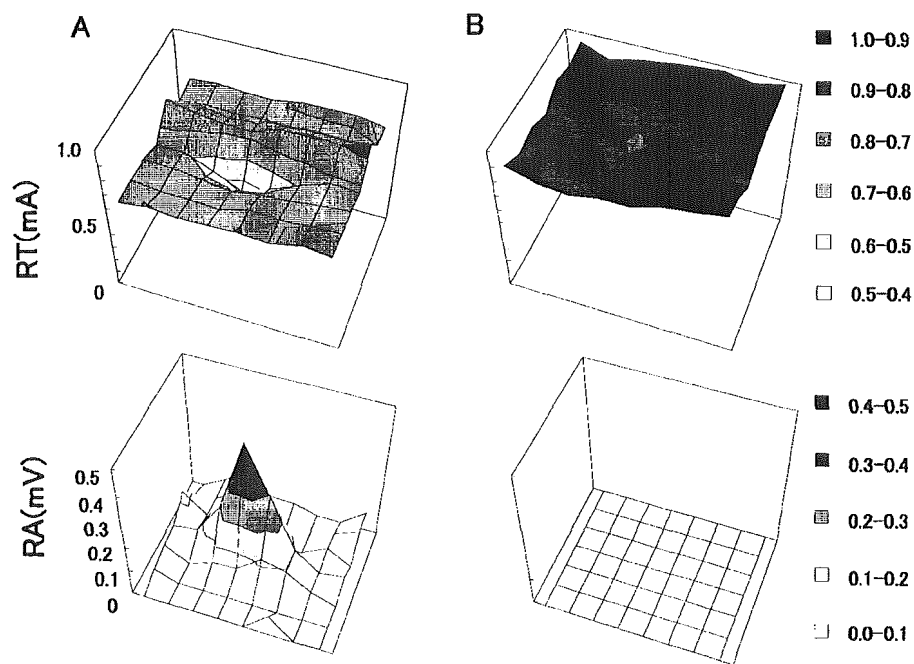


Fig. 5. Distribution of the thresholds (RT) and amplitudes (RA) of reflex EMGs of the biceps femoris muscle elicited by the electrical stimulation of the gastrocnemius muscle. The results of focal stimulation of the fascia in the vehicle (**A**) and indomethacin (**B**) groups are summarized. **The upper traces are the RT and the lower traces RA.** The thresholds were divided into six steps in RT and the amplitudes into five steps in RA, respectively.

distal to the external condyle of the tibia), the RT was lowest (Fig. 5A, upper) and its RA highest of all areas (Fig. 5A, lower). A palpable band could also be detected in the measurement areas of GS muscle, and a localized sensitive region existed on the palpable band. The latency of the reflex EMG was 44.3 ± 5.6 ms. On the other hand, in the indomethacin group at the depth of the fascia, the change of needling stiffness was not detected. The latency of the reflex EMG was 46.7 ± 7.0 ms. The distribution of the RT (Fig. 5B, upper) and RA (Fig. 5B, lower) were almost similar, and the location of the palpable band could not be determined. It is clear that the RTs in the indomethacin group were much higher than those in the vehicle group.

DISCUSSION

In the present study, a localized sensitive region at the depth of the fascia of the palpable band developed after eccentric exercise in rabbits. An administration of indomethacin inhibited the development of the sensitive region and palpable band as well as DOMS. These results suggest that the sensitization of muscular nociceptors by prostaglandins might be important for the development of DOMS and the localized sensitive region on the palpable band.

Experimental DOMS in rabbit. Many investigators have demonstrated that the most effective way of inducing DOMS is through exercise that incorporates eccentric muscular contractions [13]. Eccentric contraction induces greater tension and is considered to be the major cause of mechanical disruption of the muscle fibers and connective tissue [5, 14]. In this study we used the rabbit as an experimental animal. Under light anesthesia, tetanic contractions were provoked by electrical stimulation of the tibial nerve, and the GS muscle was then manually extended during contractions. These procedures could produce tenderness in the exercised muscle with a focal sensitive region on a palpable band in the rabbits. In our previous study in humans, we demonstrated that a localized tender region appears on the palpable band on the second day after eccentric exercise [12]. A similar localized sensitive region on a palpable band developed in the present study.

The amplitude of evoked EMG of the BF muscle was used as an index in this study. The evoked EMG could not be elicited by mechanical distension of the GS muscle in the control group, and it appeared when the affected muscle was extended several days after the exercise. The time course of the changes in the amplitude of evoked EMGs was quite similar to that

measured in subjective soreness [12].

The evoked EMG of the BF muscle elicited by an electrical stimulation of the foot, called withdrawal reflex, has long been used as a useful index of pain in human subjects [15, 16] and of nociception in experimental animals [17–19]. In human psychophysical study, the amplitude of withdrawal reflex was shown to be correlated with the subjective pain magnitude ratings [15, 16]. The responsible afferents for the withdrawal reflex, however, were estimated from its latency in experimental animals [18, 19], and A-delta and C-afferent fibers in the withdrawal reflex has also been strongly suggested [17].

In this study the latency of EMGs evoked by focal electrical stimulation of the GS muscle exceeded 40 ms, and a rough estimation of the conduction velocity of the afferent fibers was less than 10 m/s, which suggested the participation of thin afferents in this reflex circuit. Thus the amplitude of the evoked EMG of the BF muscle might be a reasonable indicator for the measurement of DOMS.

Effect of indomethacin on DOMS. The present study clearly shows that a repetitive administration of indomethacin inhibits the development of a localized sensitive region as well as DOMS, and it also suppresses the formation of a palpable band. We used a relatively large dose of repetitively administered indomethacin, following a protocol in mice that succeeded in preventing the development of DOMS [6]. The adequacy of the dose and its form of administration are undoubtedly important. That the rabbits in the indomethacin group showed no abnormal behavior and had no apparent illness indicated that the dose used and the method of administration were suitable for the experiment. In contrast, several studies have demonstrated that the administration of nonsteroidal anti-inflammatory drugs (NSAIDs) could not protect against the development of DOMS in humans [20, 21], suggesting a role for noninflammatory mechanisms in DOMS. The importance of dose, administration times, and route of administration has been indicated [22], so the negative results of the NSAID studies should be carefully reconsidered.

Thus it is suggested that the development of DOMS and a localized sensitive region might be associated with various changes in the inflammatory process, such as minor tissue injury and sensitization of nociceptors by prostaglandins.

Mechanisms of appearance of a localized sensitive region. It is well known that the activity of group III and IV afferent receptors are related to muscle pain [23] and found mostly within the connective tissue of muscle [2]. In particular, the fascia is

one of the most sensitive among the deep tissues [24, 25]. Most thin afferent fibers in muscle are thought to innervate polymodal-type receptors. They are easily sensitized by various chemical mediators such as prostaglandins [23, 26, 27], which are released in injured muscular tissue [7, 28]. In various inflamed tissues, silent or sleeping nociceptors have been found [23]. Although these receptors have not yet been found in skeletal muscle, they may be one cause of the development of DOMS and the localized sensitive region. Thus the inflammatory process following muscle injury may be important in the production of the muscle soreness [3, 7].

Usually the inflammatory process provoked by the tissue injury takes several minutes to hours, but not days. However, the time courses of serum prostaglandin E₂ (PGE₂) level changes and the degree of muscle soreness are closely related [4, 29]. It is hypothesized that elevated muscle calcium stimulates macrophages to synthesize PGE₂ [29]. In muscle, high [Ca²⁺] stimulates the production of PGE₂ [30]. Moreover, the macrophage is the predominant cell type at the site of injury 2–3 d after eccentric contraction and is capable of releasing large quantities of PGE₂ [31].

Indomethacin is a kind of NSAID and a potent inhibitor of prostaglandin synthesis, which suppresses peripheral inflammation, and inflammation-induced pain [32]. Because indomethacin inhibits the development of a localized sensitive region, the sensitization of polymodal-type nociceptors in the fascia mediated by prostaglandins might be one of the possible mechanisms.

In the present study, the musculotendinous attachment area was the most sensitive region, and a sensitive region appeared on the palpable taut band. The mechanisms of the formation of a palpable band and the sensory region on it are not clear. However, the inflammatory process elicited by tissue injury may be closely related to their appearance. The musculotendinous attachment might be the area sensitive to the eccentric contraction, and an application of intense tension here might easily cause a minor tissue injury.

Possibility of central sensitization in DOMS. Recent ischemic conduction block experiments demonstrate the participation of thick afferent fibers in the development of a muscle soreness sensation and suggest central sensitization (allodynia) in DOMS [33]. A decrease in the thresholds of nociceptive dorsal horn neurons in the acute inflammation is well known [34]. An expansion of receptive fields and the appearance of new receptive fields were induced by the intramuscular injection of chemical algescic agents [35]. The data from these animal experiments

also suggest that central changes occur when muscle tissue is injured by DOMS. The possibility of central sensitization in DOMS cannot therefore be excluded. However, the appearance in the present study of a very localized sensitive region in the fascia on the palpable band is difficult to explain in regard to central sensitizing mechanisms.

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 総説

トリガーポイントに関する研究の現状と諸問題

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Recent Studies on the Trigger Point and Issues to be Solved

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【要旨】 トリガーポイントは筋筋膜性疼痛症候群の患者の診断・治療にきわめて重要なものである。それは、局所の疼痛、索状硬結上の圧痛部位、特定のパターンを示す関連痛、患者の痛みの再現、局所単収縮反応、ジャンプ・サイン、筋力低下、可動域の制限などの特徴をもっている。このトリガーポイントの成因に関して、従来の筋拘縮説を修正した、運動終板の機能不全とする作業仮説が最近提唱されている。その根拠として電気的活動が機能異常になった運動終板から限局して記録され、それが筋の持続的拘縮によるエネルギー危機をもたらし、侵害受容器の感作が生じるために圧痛部位ができるとしている。この仮説は、筋のトリガーポイントには適用できる可能性があるが、筋膜、靭帯、骨膜に現れるトリガーポイントに関して説明ができない。

一方、われわれの作成した実験的トリガーポイントモデルは、索状硬結上に限局して圧痛部位が生じ、それが筋膜の痛覚閾値の変化と関連していること、また記録電極の刺入によって、筋膜の深さにおいてのみ、電気活動が得られ、それは被験者の重だるい痛み感覚と対応することを明らかにした。さらに、ウサギの実験において、このトリガーポイント様部位の発現は、抗炎症薬の投与で抑制された。以上のことから、筋の他、筋膜、骨膜、靭帯の組織損傷によって局所炎症が生じ、それに伴う深部組織の局所的な浮腫が索状硬結として検出される可能性があること、またポリマー・タイプ受容器の感作によって圧痛部位、関連痛パターン、局所単収縮反応が生じることは説明可能と考えた。トリガーポイントと鍼灸医学の密接な関連に着目した今後の研究の発展が強く望まれる。

I. はじめに

慢性的な筋痛を主訴とする患者には、血液検査やX線撮影などの臨床検査で異常がみられず、原因不明のために心因性のものと診断されることも多い。歯科領域における顎関節症も筋由来の疼痛症候群として捉えられており、国際疼痛学会 (IASP) では、原発性線維性疼痛症候群 (PFM)、筋筋膜性疼痛症候群 (MPS) とともに、顎関節痛機能異常症候群 (TMPDS) を取り上げている¹⁾。この3つの症候群には、それぞれ鑑別診断の基準が設けられているが、共通するところも多い。同一患者を MPS

と PFM の専門家が独自の診断基準に従って診断した結果、患者の多くは両診断名を重複して与えられている²⁾。この事実は、MPS と FM の症状に多くの共通点があることを示唆している。また TMPDS も、トリガーポイントと密接に関連していることがよく知られている³⁾。

MPS に関しては、Travell と Simons の精力的な努力によって、体系的な成書がまとめられており、そこにはトリガーポイントをキーワードとする MPS の病因論やその治療法が具体的に紹介されている⁴⁾。そのため、多くの臨床家にとってトリガーポイントは、原因不明の筋痛患者の治療にあたっては、一つの大きな手がかりとされているのが現状である。そこで本稿では、MPS の基本概念であるトリガーポイントに焦点をあて、その成因に関する最近の研究を Simons の主張を中心にして概述する⁵⁾。さらに、われわれが検討しているトリガーポイントの実験モデルについて紹介し、トリガーポイントの研究

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表1 トリガーポイントの特徴

1. 限局した圧痛部位
2. 触知できるロープ状のバンドの存在 (索状硬結)
3. 局所単収縮反応
4. ジャンプ・サイン
5. 特徴的な関連痛パターン
6. 患者の痛みの再現

をめぐる問題点とわれわれの提唱しているポリモーダル受容器仮説との接点について解説する。

II. MPSの概要とトリガーポイントの特徴

MPSは限局した特定筋の疼痛を主訴とする症候群とされるが、その原因はトリガーポイントと呼ばれる特殊な部位にあることがその特徴である⁴⁵⁾。トリガーポイントは、患者が疼痛を訴えるところから離れた部位に存在していることが多い。そのため、患者が示す疼痛部位の検査だけでは、触診やX線像に異常所見は認められないことになる。また睡眠障害や疲労感を訴える患者も多いため、心因性の痛みとして扱われることが多い。また、トリガーポイントが原因となって自律神経症状が出たり、トリガーポイントの発生した筋の筋力低下や可動域の制限が生じることも多い。そこで、MPSは疼痛専門医の治療対象であるばかりではなく、運動障害をもたらすことから徒手療法家の治療対象にもなっており、冷却スプレーとストレッチの組合せなど種々の治療法が開発されている⁶⁾。

このMPSの的確な診断にはトリガーポイントを正確にみつけることが重要とされている。TravellとSimonsは筋筋膜トリガーポイントを、骨格筋内の刺激に敏感な部位で、索状硬結中に触知できる過敏な小結節と定義している。また、その部位を圧迫すると強い痛みがあり、特有な関連痛パターンが生じる他、それに関連して運動機能障害や自律神経症状を生じるとしている⁴⁵⁾。

このトリガーポイントの特徴についてまとめたものが表1である。

1. 患者の疼痛再現と特徴的な関連痛パターン

MPS患者の筋筋膜トリガーポイントを圧迫したり、そこに鍼を刺入すると、その患者がこれまで主な症状として訴えていた疼痛と同じ性質のものが再現されることがある。TravellがMPSの概念を思いついたのは、自分自身の症状が遠隔部位の圧迫で再現された体験によることはよく知られている。この患者の疼痛再現の現象は、筋筋膜トリガーポイントの診断上最も重要な規準の一つである。

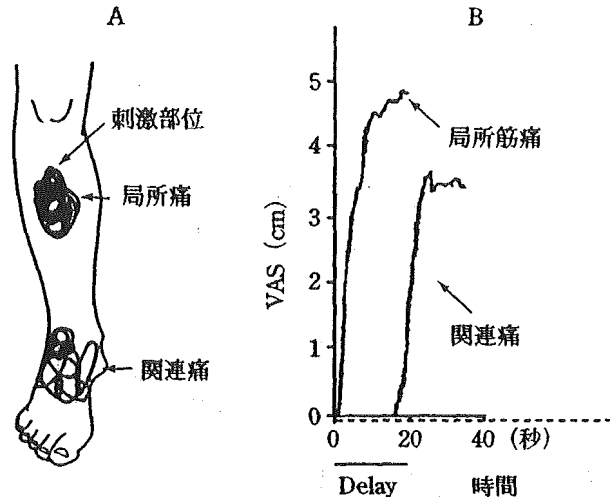


図1 ヒトの筋への電気刺激で生じる関連痛
A: 10 Hz, 10 秒の電気刺激より生じた、局所の筋痛と遠隔部位に関連痛
いずれも 10 例の結果を重ね書きしたもの。
B: 局所筋痛と関連痛の発現の時間的遅延を示す
関連痛は局所筋痛に比べて約 20 秒ほどの遅れがある (Arendt-Nielsen, 2001 より改図)。

ヒトにおいて実際に関連痛が筋組織の刺激で生じることを見いだしたのは Kellgren である⁷⁾。彼は筋に高張食塩水を微量注入すると、注入局所以外の遠隔部位にそれぞれ特有の関連痛パターンが現れることを実験的に明らかにした。この研究は、トリガーポイントの診断上の役割を、関連痛パターンとして解析することの妥当性を示したものである。図1は、近年行われた電気刺激を用いて関連痛のパターンと発現の時間経過を調べた成績を示している。

関連痛が生じるメカニズムに関して、現在一般的に認められているのは、収束・投射説である。この収束・投射説では、中枢神経系において、内臓からの入力や皮膚からの入力と誤認された結果としている⁸⁾。しかし、実際に証明されているのは、脊髄後角において両組織からの入力収束しているニューロンがあるという事実のみであり、特定の関連痛パターンの説明には十分ではない。

一方、筋に強力な発痛物質を注入すると、麻酔下ラットの脊髄後角ニューロンの受容野が拡大したり、新しい受容野を発現したりすることが実験的に知られている⁹⁾。この現象は、単なる収束・投射説とは異なり、これまでは潜在していたシナプス結合が活性化した結果と考えられている¹⁰⁾。この仮説が現在トリガーポイントによる関連痛パターンの説明に用いられているが、新しい受容野の発現には、発痛物質注入後少なくとも数分を要することから、実際のヒトでの生じる関連痛の潜時(数十秒)¹¹⁾との違いはまだ説明できていない(図1-B)。

また末梢神経の分枝説も関連痛の機序として考えられ

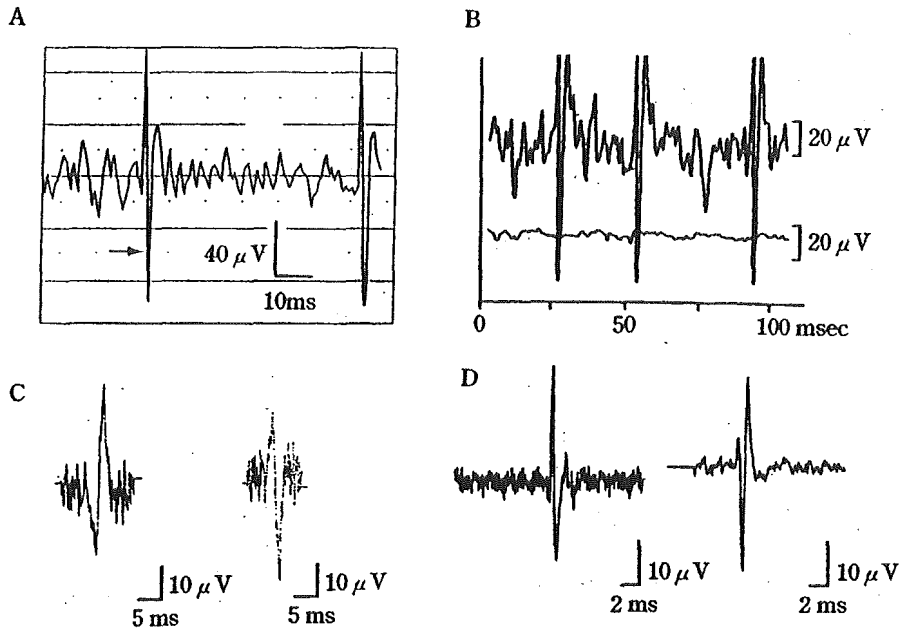


図2 トリガーポイントから記録される各種の電気活動

- A: ヒトのトリガーポイントから記録された筋電図
著者は筋紡錘の錘内筋の筋電図としている (Hubbardら, 1993より改図)。
- B: ウサギの索状硬結から記録された電気活動
著者は運動終板からの記録としている (Simonsら, 1995より改図)。
- C: ヒトの実験的トリガーポイントモデルで記録された電気活動
著者は筋膜の刺激による反射性の電位としている。
- D: ウサギの実験的トリガーポイントモデルで記録された電気活動
著者は筋膜の刺激による反射性の電位としている。

ている¹²⁾。内臓疾患に関連して、特定の部位に局限してさまざまな反応が起こることや、関連痛部位に局所麻酔を投与することで関連痛が軽減することも知られている。そこで、関連痛に関連する領域において、ポリモーダルタイプの受容器が分枝していると仮定すると、それらの現象の説明はきわめて容易になる。しかし、解剖学的な研究の結果は、実際の分枝の割合はきわめて少なく¹³⁾、また分枝説では、関連痛の発現が数十秒程度遅れることの説明も困難である。

以上簡単に述べてきたように、関連痛の発現機序をめぐっては、中枢神経系におけるニューロンの回路の活動を反映しているものと考えられるが、まだ確定したメカニズムはわかっていない。

2. 索状硬結と圧痛部位

トリガーポイントを見つける最初のステップは、筋の注意深い触診によって筋にあるロープ状の索状硬結を探しあてることである。索状硬結を圧迫すると全体的に痛みがあるが、その中でも特に敏感な部位を見いだすことができる。それがトリガーポイントであり、強い圧痛があると同時に、その部位をさらに強く圧迫すると関連痛

が生じ、ときには患者のもっている疼痛などの症状が再現することがある。

この索状硬結の成因に関しては、種々の可能性が示唆されている。Travellらは、筋の損傷による筋線維の部分的な拘縮 (contracture) であるとし、筋の損傷をきっかけとして、筋小胞体からカルシウムイオンが筋細胞内に漏出し、その結果アクチンとミオシンの収縮タンパクの相互作用によって筋が拘縮するというものである。この筋拘縮モデルは広く認められていたが、その後、トリガーポイント部位から電気活動が記録されることがいくつか報告され修正が必要となった。

HubbardらはTMP患者の筋筋膜トリガーポイントから、自発的な電気活動が記録できることを示した (図2-A)。さらに、その筋電図活動の記録部位の生検によって筋紡錘が認められ、交感神経遮断剤であるフェントラミンの投与によってその電気活動が消失したことから、この電気活動は、筋紡錘の錘内筋の活動電位であるとした¹⁴⁾。これに対して、トリガーポイント・マニュアルの著者の一人であるSimonsらの研究グループは、筋電図活動がトリガーポイントから記録できることを、ウサギを用いた実験において確認し¹⁵⁾、得られた電位の波形はき

わめて限局した部位のみで記録され、その波形が陰性から始まる2相性のものであることから、運動終板に由来するスパイク電位としている(図2-B)。

そして、改訂されたトリガーポイント・マニュアル⁴⁾では、トリガーポイントは筋の運動終板が機能異常になって、アセチルコリンの過剰分泌によって終板周囲の限局した筋線維が過度に短縮している部位(収縮ノット)としている(図3)。そして、収縮ノットがいくつか集まっているエリアをトリガーポイント複合体と呼んでいる⁵⁾。

筋線維の一部が局所的に拘縮してノット状になると、その部位が圧迫に対して非常に敏感な圧痛部位であることは直接的結びつかない。そこで、終板の付近には血管や神経が分布しており、アセチルコリンの過剰分泌や筋の拘縮によって、組織のエネルギー危機がきっかけで侵害受容器の感作をもたらすとしているが、十分に説得力があるとは考えにくい⁶⁾。

3. 局所単収縮反応

局所単収縮反応(LTR)は、索状硬結を指で強く弾いたり、鍼をトリガーポイントに刺入することで生じる反応であり、トリガーポイントの診断的な意義がきわめて高いものとされているものの、その反応を誘発するのは技術的に最も難しいとされている。

このような反応の成因として、Hongらは、ウサギ下肢筋の索状硬結の最も感受性の高い部位にプローブによる機械的刺激を加え、LTRの出現率と誘発される筋電図活動を調べ、脊髄切断後一時的に消失するものの、その後回復したことから、この反応を脊髄反射としている¹⁰⁾。このような侵害性の入力による脊髄反射性の筋収縮反応は、Simonsらの主張するように機能不全におちいって過剰に興奮している運動終板の近傍の刺激のみで起こる特殊な反応ではない。筋膜や骨膜などさまざまな組織に対する侵害的な刺激で起こりうる一般的な現象で、トリガーポイントの鍼治療に際して、鍼を刺入したときにLTRが現れることはよく知られている⁹⁾。また鍼の臨床家にとってもツボや圧痛点に鍼を刺入した際の局所的な筋収縮は、手下感とも呼ばれ治療上の指標とされている。このような反応は筋膜の刺激によって生じることが多いとされており、機能不全の運動終板のみに関連づけることには問題がある。

III. トリガーポイントの実験モデルについて

トリガーポイントの成因に、筋の損傷が関連していることは多くの研究者が認めている。しかし、実験的にそのような現象について検討を加えた研究は皆無であっ

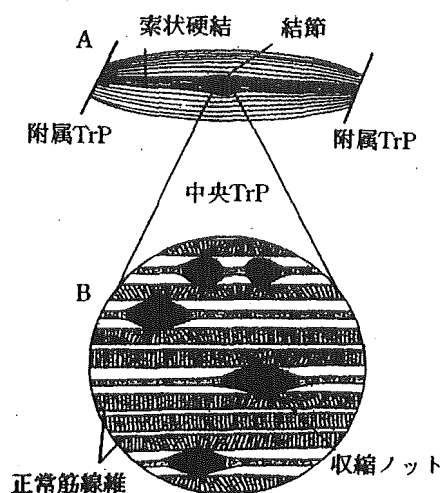


図3 トリガーポイントでみられる索状硬結部位の模式図
詳細は本文参照のこと(Simons, 1999より改図)。

た。そこでわれわれは、筋の損傷を起こしやすい運動として知られる伸張性収縮を特定の筋に負荷し、遅発性筋痛と呼ばれる運動後2~3日で現れる筋痛モデルを作成し、その筋痛部位について詳細に検討した。その結果、伸張性収縮運動負荷の2日後に、運動負荷を行った筋にトリガーポイント様の索状硬結と限局した圧痛部位が出現することをヒトの実験で見いだした。そして、その索状硬結に見いだされる圧痛閾値の低下部位は、筋膜の閾値低下に由来することを、絶縁鍼電極を用いた深部痛覚の測定によって明らかにしてきた¹⁰⁾。さらに、鍼の刺入によって、トリガーポイント・マニュアルに描かれているような特定の関連痛パターンが生じることも確認された(図4-A)。また、その部位から限局して筋電図活動が記録されること(図2-C)、その際電極は筋膜に接している状況においてのみ記録され(図4-C)、被験者は必ず重だるい深部痛覚様感覚を訴えた。また、このようなトリガーポイント様の部位は、ヒトばかりでなくウサギを用いた実験においても、同様の伸張性運動負荷後に出現することが確認された¹¹⁾。また侵害性の反射性誘発筋電図の閾値を指標とした解析の結果、ヒトと同様に筋膜における誘発閾値の低下と局所の電気活動を記録することができた(図2-D)。さらには、抗炎症薬であるインドメタシンの繰り返し投与によって、同様の運動負荷後のトリガーポイント様部位の発生は抑制され、局所的な電気活動もみられなかった。

これらの実験結果から、われわれの作成したトリガーポイントの実験モデルは、伸張性運動負荷によって、筋膜もしくは筋細胞膜が損傷した結果、その修復過程で産生されたプロスタグランジンによってポリモーダル・

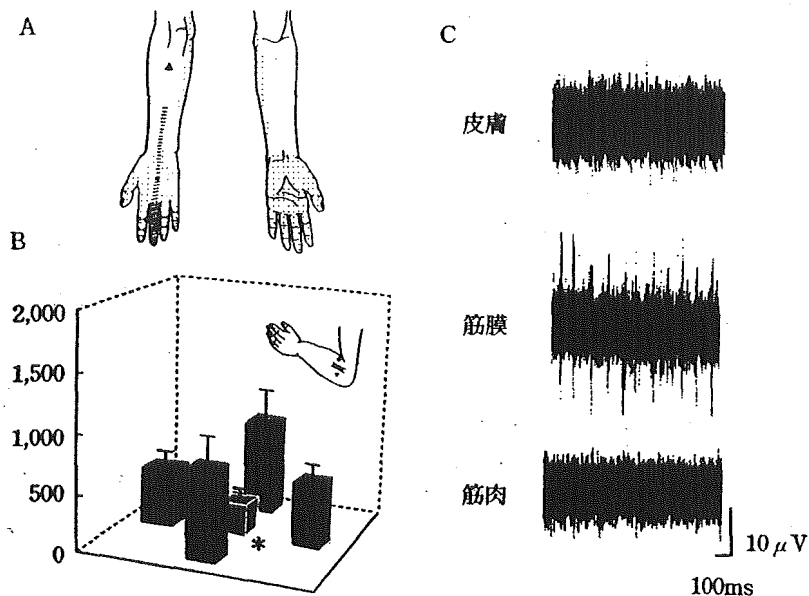


図4 ヒトの実験的トリガーポイントモデル

- A: 運動負荷後2日目にみられるトリガーポイント様部位の圧迫で出現する関連痛のパターン
 B: 索状硬結とその周囲の圧痛閾値の分布
 縦軸は任意のスケール、硬結の中央部が有意に閾値が低い。
 C: トリガーポイント部位から得られる電気活動
 筋膜の深さに限局して深部痛覚の発生とともに電気活動が記録される。

タイプの受容器が感作(sensitization)によってもたらされたものと考えられた。また記録された電気活動は、筋膜のポリモーダル・タイプの受容器が記録用の針電極の刺入によって機械的に刺激された結果、脊髄反射性に誘発された筋電図と推察した。このモデルにおいては、トリガーポイント様の特徴は運動負荷後7日でほぼ消失することから、今回のモデルはトリガーポイント発現の初期の状態を示しており、それになんらかの持続因子(血流不全、持続的筋収縮)が加わることによって、より臨床像に近いトリガーポイントモデルになるものと考えている。

IV. トリガーポイント研究に関する問題点

これまで、トリガーポイントの成因に関する研究の現状について、主に Simons らの主張を中心に紹介してきた。その提唱するところの統合仮説(図5)には多くの問題が残されている。その一つが、正常ウサギの筋への触診によって検出された硬結部位で記録できた電気活動やLTRから、その部位をヒトのトリガーポイントと同一とみなすことの妥当性である。単に麻酔をしたウサギの下腿の筋を触診して索状硬結を見つけ、その部位に電極を刺入して得られる電位の波形の特徴から、それを終板電位とみなし、逆にヒトのトリガーポイントから得られた

電気活動を終板由来の電位としている。しかし、われわれの実験モデルから得られた電位は多様であり、彼らの主張する終板電位に近い波形も観察されたが、それ以外の波形も混在することが多く、記録電極と電気現象の生じている部位との相対的な位置関係による記録波形の違いの可能性も考えられた。仮に終板電位を一つの特徴とみなしても、次に、その記録部位とLTRが誘発されることとの関連性については、明確な説明はない。自発放電が記録され終板に特有の波形を示すことを強調しておきながら、トリガーポイントでは限局した結節部位で強い圧痛がみられることに関しては、終板の解剖学的な構造から血管と神経がありその中に侵害性の求心性神経があることを挙げているのみで、アセチルコリンが過剰分泌している部位(終板の活動亢進部位)の侵害受容器が感作する理由はみあたらない。そこでエネルギー危機による感作物質の産性が考えられているものの、終板から発生したスパイク電位は伝導性をもつ以上、その筋線維の収縮が起こることが予想されるにもかかわらず、その点に関しては一切言及されていない。

トリガーポイントの運動終板説に関するもう一つの問題は、Travell や Simons 自身が認めているように、トリガーポイントは筋だけでなく、皮膚、筋膜、骨膜、靭帯などにも存在することである。そして、筋以外の組織への高張食塩水の注入が関連痛を生じることも、すでに

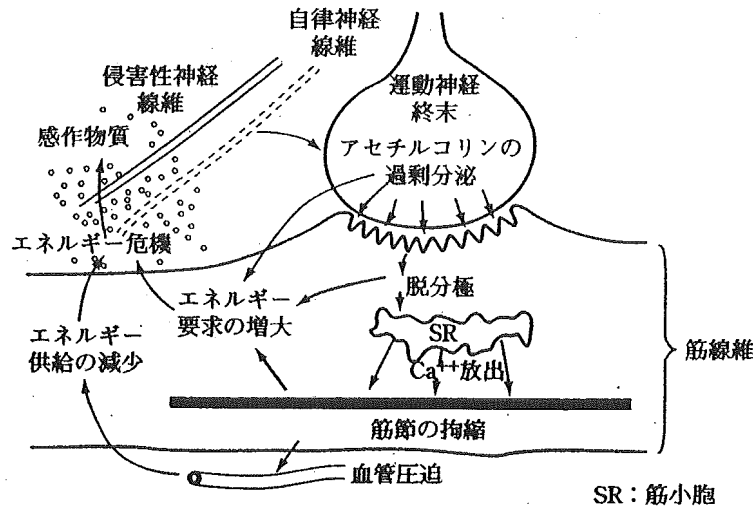


図5 トリガーポイントを説明する統合仮説
詳細は本文参照のこと (Simons, 1999より改図)。

Kellgren や Inman らによって報告されている^{7,10)}。現在、提唱されている筋筋膜トリガーポイントの成因に関する、運動終板の機能異常説は、あくまでも、筋のトリガーポイントを説明できるのみであり、それ以外の組織のトリガーポイントを説明できないことは明白である。

この点に関しては、われわれの実験結果を参考にしながら局所炎症の面から考えると、トリガーポイントの特徴の多くを説明することが可能である。トリガーポイントの成因として、なんらかの原因から筋・筋膜、靭帯、骨膜等の組織に微小損傷が起こり、その部位に局所炎症が生じることを仮定する。その結果、炎症関連の発痛物質によってその部位に分布するポリモーダル・タイプの受容器が興奮し、軸索反射によって局所に浮腫を生じる。また、その後の組織修復の過程で生じるプロスタグランジンなどの生理活性物質が、ポリモーダル・タイプの受容器を感作させる。このために、圧痛閾値の低下が起こり圧痛部位を形成する。また、そのような感作部位への発痛物質の注入はより容易に関連痛を生じることができる。また、深部組織に生じた浮腫は触診によって索状硬結のように検出される可能性がある。われわれの実験性トリガーポイントモデルで触診された索状硬結は、抗炎症薬の連続投与によって発現しなかったため、筋膜において生じた浮腫の可能性は高いと考えられる。また、交感神経とトリガーポイントとの関連については、ポリモーダル受容器が炎症の過程で交感神経の α 受容体を発現することが知られており、病態においては交感神経系の亢進がトリガーポイントを活性化することを裏づける可能性は十分考えられる。以上の仮説に関してまだ十分な実験的データがないので、今後の大切な課題と考えている。

V. 鍼灸医学におけるトリガーポイントのもつ意味

われわれはこれまで、鍼灸刺激がポリモーダル受容器を刺激して、さまざまな効果をもたらすという作業仮説を提唱している。その詳細や論拠についてはすでに発表した拙稿¹⁹⁾を参考にさせていただき、ここではトリガーポイントと鍼灸の関連について述べてみたい。

トリガーポイントとツボが高い一致率(71%)をもって存在することは、ツボというものを考える際の大きなヒントになるものである²⁰⁾。しかし、臨床的にはトリガーポイントの治療はそれを不活性化することが重要であり、そのために局所麻酔薬の注入が繁用されている一方、鍼灸医学においては、ツボになんらかの刺激を与えることで、生体の調整機能を高めるとする点において大きな違いがある。しかし、トリガーポイントの治療に鍼の刺入が有用であることが多くの研究から明らかになっており²¹⁾、鍼治療においても硬結や圧痛点を指標に治療を行って効果を上げている臨床家も多い。

トリガーポイントの特徴としての索状硬結は、鍼灸の臨床家が治療部位として用いる部位でもある。われわれが鍼灸の臨床家に対して行ったアンケート調査では、硬結そのものにも痛みがあるものとならないもの、鍼治療によって即座に消失するもの、なかなか消えないものなどがあり、また硬結の部位は固定したものでなく、可動性があるという意見もあり、硬結の実体を把握するにはその多様性を踏まえる必要がある²²⁾。これらのアンケート調査結果を踏まえ、硬結そのものはダイナミックに変化するものという観点から、それは硬結が微小な組織損傷により局所炎症反応の結果生じた細胞外への血漿漏出に始まる、一連の炎症・組織修復過程を反映している可能

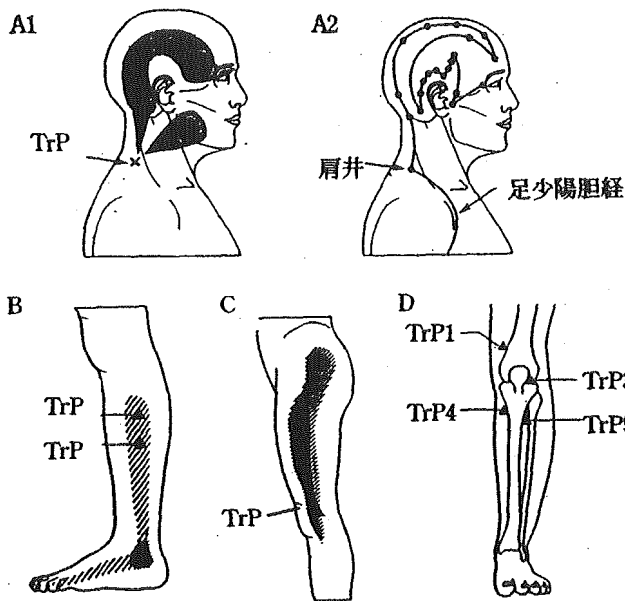


図6 トリガーポイントとツボの類似性

- A1: 僧帽筋のトリガーポイントの刺激で生じる関連痛パターン
 A2: 同一部位のツボ(肩井)と経絡の走行の図(Filshieら, 1999より改図)
 B: 下肢のトリガーポイントの刺激で誘発される関連痛パターン(末梢へ放散)
 C: 下肢のトリガーポイントの刺激で誘発される関連痛パターン(中枢へ放散)
 D: 膝周辺に頻出するトリガーポイントの分布図
 微妙な違いはあるものの、多くのトリガーポイントはツボに一致している。
 TrP1: 血海, TrP3: 犢鼻, TrP4: 陰陵泉, TrP5: 足三里(Baldry, 1989より改図)

性が考えられた¹⁹⁾。

一方、MPSと並ぶ筋痛症候群として知られているFMにおいて、患者の鑑別診断に用いる全身18カ所の圧痛測定部位は、その多くは鍼灸医学で用いるツボに一致している²⁰⁾。このことは、筋痛を訴える患者においては、圧痛の発現する部位が、鍼灸医学でいうツボと共通して現れやすいことを意味しており、生体に備わった一つのパターンないし、ヒトとしての生活・活動様式がもたらす共通する生体のひずみを反映していることをうかがわせるものである。

トリガーポイントに特有の関連痛パターンに関連して、その多くは刺激部位から末梢側へ投射するものが一般的であるが、一部では中枢側へ投射することが知られている²¹⁾。この投射パターンをみていくと、部分的には経絡現象を想像させるような軌跡を描いていることから²⁰⁾、西洋医学の、経絡とは全く無縁と考えられているトリガーポイントによる関連痛パターンが、経絡現象を解く糸口になる可能性は考えられる(図6)。

鍼灸医学の基本概念である経絡・経穴と、トリガーポイントはまったくその背景は異なるものの、両者のあいだには、出現部位、関連痛パターン以外にもLTR、治療部位としての有効性など多くの共通性をみることができよう。鍼灸の研究としてもトリガーポイントに関する実験モデルの重要性を確認することができよう。

VI. おわりに

トリガーポイントは原因不明の筋痛の診断・治療の中で、重要な一つの指標となっているのは事実である。しかし、体系化されたトリガーポイントに関する仮説を支えている実験事実は十分ではなく、本総説では、Simonsらの提唱している作業仮説についていくつかの批判を加え、それに代わるものとして、ポリモーダル・タイプ受容器の感作と深部組織の浮腫の可能性を紹介した。トリガーポイントは鍼灸の研究にとって一つの糸口を提供しており、今後の研究の発展が強く望まれるものである。

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