# Myofascial Trigger Points

## Comprehensive diagnosis and treatment

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Chapter 21

# Trigger point infiltration

*Hans-Joachim Schmitt, Dominik Irnich*

The infiltration of trigger points is one of the most significant and highly efficient techniques in the treatment of myofascial pain and restricted function. Optimum success is assured by using a precise procedure, suitable materials (thin needles, only as long as necessary) and appropriate medication in as low concentrations as possible. The more chronic the mTrP, the more restrained the infiltration techniques should be, so that the chronic complex problems are not made worse when the attitude of many patients is limited to the somatic (iatrogenic chronification). A different attitude is indispensable for optimum application of this technique. There is an obvious difference between treating a young athlete with mTrPs and a patient with chronic back pain.

## 21.1 INDICATIONS AND CONTRAINDICATIONS

### 21.1.1 Indications

There is an indication for infiltration of a trigger point if:

- manual trigger point therapy or stretching has had an inadequate effect,
- manual trigger point therapy or stretching cannot be adequately carried out because of severe pain,
- acute severe pain and functional problems are to the fore,
- there are additional diagnoses for which manual therapy is unsuitable or may be contraindicated (hypermobility, calcinosis, etc.),
- the post-treatment soreness that occasionally occurs with dry needling is undesirable.

### 21.1.2 Contraindications

- Anticoagulant therapy.
- Taking acetylsalicyclic acid in the last 3 days.
- Anxiety.
- Known drug intolerance.

The number of injection treatments depends on:

- the selection of trigger points which determine the symptoms (5–10 points possible in one session),
- the precision of the injection,
- concomitant/instructed stretching of the affected muscles by the patient or therapist.

We recommend at least 3 days between invasive treatments, or better still 7–8 days, and exercise can be intensified during this time.

## 21.2 INJECTANTS

The choice of injectant depends on a phased plan, whereby local anaesthetic (LA) is the first choice. When choosing the substance, first assess the duration of effect and the myotoxicity (i.e. the potential cause damage to the skeletal muscle tissue, leading to myonecrosis). A rule of thumb is that the shorter the duration of effect and the lower the concentration, the lesser the myotoxicity and cell infiltration into the tissue.

Avoid the addition of adrenaline (epinephrine) because of the occurrence of muscle necrosis.


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The injection of bacteriostatic (alcohol) or physiological saline is very effective compared to LA.

For trigger points at a muscle insertion (with inflammatory components) we recommend the admixture of a crystalline corticoid, but only to a maximum of three times. Infiltration is performed around the tendon.

If non-invasive (manual trigger point therapy) and invasive methods of dry needling and injection as well as combinations of them do not lead to the desired result, the injection of botulinum toxin A is indicated for severe distress in exceptional cases.

## 21.2.1 Local anaesthetics

### Basic principles

LAs work by binding to the tension-dependent sodium channels in the nerve membranes. The LAs interact in their ionised form with the amino acids phenylalanine and tyrosine, which leads to a blockade of sodium inflow. At high concentrations other ion channels are also blocked. The blockade of sodium permeability leads to both depolarisation and the spread of the excitation wave being suppressed. This interrupts the conduction of pain signals.

The following factors affect the length of time the LA remains at the site of application and the effect:

- the pH value determines the level of lipid solubility: the further from the physiological pH value of 7.4, the less effective is the LA (e.g. inflammation),
- local perfusion,
- enzymatic hydrolysis with ester-type LAs.

In the bloodstream ester-type LAs such as procaine and tetracaine are split by plasma cholinesterase. The split products are ineffective as LAs and are non-toxic in their resulting concentrations. Breakdown in the liver is of secondary significance.

Amide-type LAs such as lidocaine are mainly oxidatively dealkylated or hydroxylated and enzymatically hydrolysed by carboxyesterase, located in the endoplasmic reticulum. Amide-type LAs are more slowly metabolised (half-life of between 1.5 and 3.5 h) compared to ester-type LAs (e.g. procaine has a half-life of 0.5–1 h) in the plasma and tissue (Table 21.1).

LAs can also be classified by their duration of effect:

- short duration: procaine 30–60 min,
- average duration: lidocaine, mepivacaine, prilocaine 60–120 min,
- long duration: ropivacaine and bupivacaine up to 400 min.

### Substances

Because of its proven low myotoxicity we recommend lidocaine 0.2–0.5%, mepivacaine 0.2–0.5%, prilocaine 0.2–0.5% and bupivacaine 0.125% or less (a higher dosage is potentially myotoxic). Because of their low lipophilia of the esters procaine and tetracaine have the lowest level of myotoxicity, although esters are estimated to have a higher allergic potential. Low concentrations of LA are usually sufficient to obtain the desired effect. A high dilution also avoids affecting motor fibres.

If this is the first diagnostic injection (i.e. to find out whether the mTrP is actually causing the pathology) for reasons of practicality a short-acting LA should be used (e.g. procaine, lidocaine, prilocaine). The advantages are the fast onset of effect (allowing rapid review) and the shorter duration of effect (meaning safe management in the outpatient setting).

If these are therapeutic injections, short- and long-acting LAs can be combined. This ensures a rapid onset of effect which lasts a long time, with longer desensitisation.

### Side effects

**Myotoxicity:** LAs are indeed the substance of choice for local infiltration; however, they can also be myotoxic. Initially, intracellular membrane systems are affected with subsequent formation of oedema in myocytes. As a consequence this can lead to areas of necrosis, although without sequelae because of regeneration. Basically, all LAs can have myotoxic effects, although the myotoxic potential of the individual LA varies. In animal trials, procaine leads to only minimal structural changes, while the worst damage has been observed with bupivacaine (in high concentrations). Pathophysiologically, myotoxicity is based on a pathologically increased intracellular free calcium ($Ca^{2+}$) concentration in monocytes. Many LAs induce $Ca^{2+}$ release, depending on concentration, from the sarcoplasmic reticulum and at the same time prevent $Ca^{2+}$ reuptake. This effect depends on lipophilia and stereoisomeric configuration. The clinical significance of myotoxicity induced by LAs is controversial, as toxic muscle damage is extremely rarely reported after infiltration of mTrPs. $Ca^{2+}$ deposits can best be avoided with procaine.

LAs can also cause various undesirable reactions and effects. The systemic toxic effects and the very rare allergic reactions play a particular role because of their potentially threatening nature.

**Systemically toxic side effects:** these occur as a result of overdose or inadvertent intravasal injection. They occur primarily as disorders in the CNS or the heart. Cerebral disorders are usually found initially and cardiovascular disorders so not usually occur until the plasma concentrations are higher (Fig. 21.1).

**Table 21.1** Clinical application of LAs (with or without adrenaline). The maximum dosages are only recommendations as different maximum dosages are given in different countries. The duration of effect depends on the blockade technique and the quantity of LA injected (Larsen 2006)

| SUBSTANCE | CONCENTRATION (%) | VOLUME (ML) | ONSET OF EFFECT (MIN) | DURATION OF EFFECT (MIN) | MAXIMUM INDIVIDUAL DOSE (MG) |
|---|---|---|---|---|---|
| **Amide-type LAs** | | | | | |
| Lidocaine | 0.5–1 | – | – | – | 200 without adrenaline, 500 with adrenaline |
| | 1–1.5 | 30–50 | 10–20 | 120–240 | – |
| | 1–2 | 15–30 | 5–15 | 30–90 | – |
| | 5, hyperbaric | 1–2 | 5 | 30–90 | – |
| Prilocaine | 0.5–1 | – | – | 30–90 without adrenaline, 120–360 with adrenaline | 400 without adrenaline, 600 with adrenaline |
| | 1–2 | 30–50 | 10–20 | 180–300 | – |
| | 2 | 15–30 | 5–15 | 150–600 | – |
| Mepivacaine | 0.5–1 | – | – | 45–90 without adrenaline, 120–360 with adrenaline | 300 without adrenaline, 500 with adrenaline |
| | 1–2 | 30–50 | 10–20 | 180–300 | – |
| | 1.5–2 | 15–30 | 5–15 | 60–180 | – |
| | 4, hyperbaric | 1–2 | 5 | – | – |
| Bupivacaine | 0.25–0.5 | – | – | 120–240 without adrenaline, 180–420 with adrenaline | 175 without adrenaline, 225 with adrenaline |
| | 0.25–0.5 | 30–50 | 15–30 | 360–720 | – |
| | 0.25–0.75 | 15–30 | 10–20 | 180–300 | – |
| | 0.5 | 2–4 | 10 | 75–250 | – |
| Levo-bupivacaine | 0.25–0.5 | 1–60 | 1–5 | – | 175 without adrenaline |
| | 0.25–0.5 | 30–50 | – | – | 225 with adrenaline |
| | 0.25–0.75 | 10–30 | 8–20 | – | – |
| | 0.5 | 2–4 | 10 | 75–250 | – |
| Etidocaine | 0.5–1 | 30–50 | 10–20 | 360–720 | 400 with adrenaline |
| | 1 | 15–30 | 5–15 | 180–300 | – |
| Ropivacaine | 0.2–0.5 | 1–100 | 1–5 | 120–360 | 300 |
| | 0.5–1 | 15–30 | 15–30 | 360–720 | – |
| | 0.2–1 | 15–30 | 10–20 | 180–360 | – |
| Ester-type LAs | | | | | |
| Procaine | 0.5–2% | 10–20 | 10–15 | 30–60 | – |

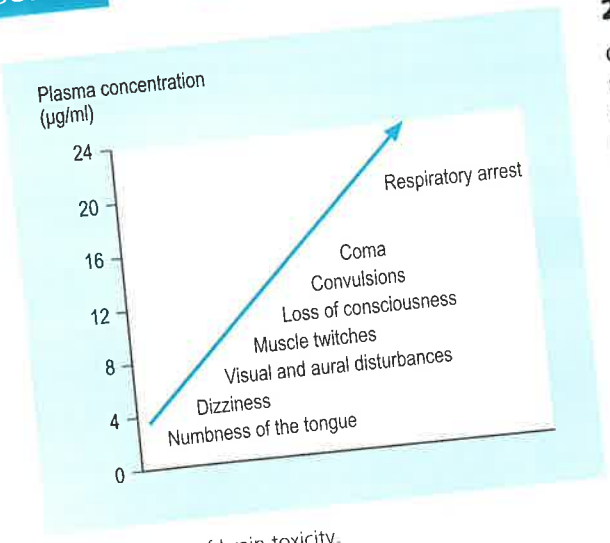


**Fig. 21.1** Signs of brain toxicity. *(From Larsen 2006)*

**Neurotoxicity:** in animal experiments high concentrations of LA can damage nerve tissue. However, this is not to be expected as long as the usual clinical concentrations are used. Individual reports are available of long-term damage as a result of intraneural injection.

**Allergies:** allergies occur extremely rarely. These are more commonly described for LAs from the ester group. With amide-like LAs, allergies to the added stabilisers have been described (e.g. *para*-hydroxybenzoic acid methyl ester). Allergic reactions can occur as allergic dermatitis, asthma attacks or anaphylactic shock.

**Vasovagal reactions:** these can occur with any method of sampling blood or injection and must be distinguished from allergic reactions. As prophylaxis patients should be treated lying down.

### Dosage

A quantity of about 0.2–0.5 ml should be used for infiltrating each mTrP so that even if 10–15 mTrPs are injected there is no danger of exceeding the approved overall dose.

## 21.2.2 Physiological saline solution

This is used for treatment in some studies. The effect is to some extent comparable to a LA but this has not been reflected in all studies. It is probably based on the alcoholic, bacteriostatic mixtures used and the mechanical lesion at the mTrP caused by the traumatising injection needle or the infiltrated volume (dry needling). An injection with physiological saline should always be considered if the patient is known to be intolerant or has an allergy to LAs.

## 21.2.3 Corticosteroids

Corticosteroids cause inhibition of the release of arachidonic acid and a reduced formation both of thromboxanes and prostaglandins (inflammatory mediators), as well as normalisation of the increased capillary permeability in the inflamed tissue (oedema reduction). This also leads to reduction of fibroblast proliferation and collagen deposits ('scarring') as well as reduced leucocyte infiltration (inflammatory pain).

An immediate injection in the mTrP is not recommended (danger of rupture); the infiltration of the insertion site with a combination of LA and steroid can be balanced for a short time (maximum of three injections) with inflammatory participation.

We tend to recommend crystalline solutions with a mild systemic effect and longer duration of effect, and which are applied in a comparatively low concentration (e.g. 10 mg triamcinolone/ml). An injection directly into a tendon itself should be avoided!

## 21.2.4 Botulinum toxin A

### Basic principles

Published applications of botulinum toxin A (or BTX-A) include spasticity, dystonia, achalasia, tremor (essential), piriformis muscle syndrome and myofascial pain (US Food and Drug Administration). However, according to the current literature there is insufficient evidence that the injection of botulinum toxin A into mTrPs is effective.

### Substances

Botulinum toxin is produced by *Clostridium botulinum*, an anaerobic Gram-positive bacterium. It blocks ACh release at the cholinergic neuromuscular synapse. This block is irreversible and is the cause of a long-term effect lasting about 2–3 months. Re-innervation of the target tissue does not occur until new axons are produced from the peripheral nerves. This process has been observed, according to the literature, up to 40 times so far.

### Indications

Individual indications are spasticity, dystonia and tremor. In the treatment of mTrPs it is only indicated in the rarest of cases and after all other treatment measures have been exhausted.

### Side effects

There is no organ toxicity apart from in the target muscle. Possible side effects are:

- weakness of the target muscle with a reduction of physiological function and the resulting biomechanical consequences,

- a temporary functional disorder in the neighbouring muscle and glandular tissue,
- a systemic feeling of weakness (although because of the low dose this is rather rare), shortness of breath, difficulties swallowing and accommodation disorders,
- occasionally flu-like symptoms (systemic side effect or immunological reaction).

### Dosage

The effective strength is measured as a biological unit in mouse units (MU). One MU corresponds to the $LD_{50}$ of botulinum toxin for a mouse population. An immediate comparison of the effective strength of the products on the market is not available because of the different bioassays of the individual companies producing the toxin. The known highest dose in one session is 800 MU.

## 21.3 TECHNIQUE

- Disinfect the area.
- Palpate the taut band in the muscle: find the mTrP or trigger point region based on the local pain maximum.
- The point or region is isolated by holding it between thumb and index finger (Fig. 21.2a) or using the Y-grip between the index finger and the middle finger, holding it in place by pressing against the underlying tissue (Fig. 21.2b).
- Apply the above grip with pre-tension (extension) of the muscles being sought (this is recommended for deeper-seated muscle layers, e.g. gluteal).
- During the exploratory injection, the support of the hand guiding the cannula provides security.
- The syringe is usually steadied on the webbing between the second and third fingers with support from the fifth finger or the hypothenar eminence. Pressure on the plunger is exercised by the thumb (Fig. 21.3a).
- The syringe can be held for vertical insertion like a dart between the thumb and digits III–V and the therapist supports himself on the hypothenar eminence or the pisiform bone. Pressure on the plunger is from the index finger (Fig. 21.3b).
- Further protection over the thoracic region is provided by a flat almost tangential angle of insertion and at a right angle to the course of the ribs, using the Venetian-blind-like effect of the ribs and support from the hand (Fig. 21.4).

An attempt at aspiration is usually made before injection to rule out intravasal positioning of the cannula. With deep injection close to large vessels, aspiration should be performed in two planes (= double aspiration with turning of the needle tip through 90°).

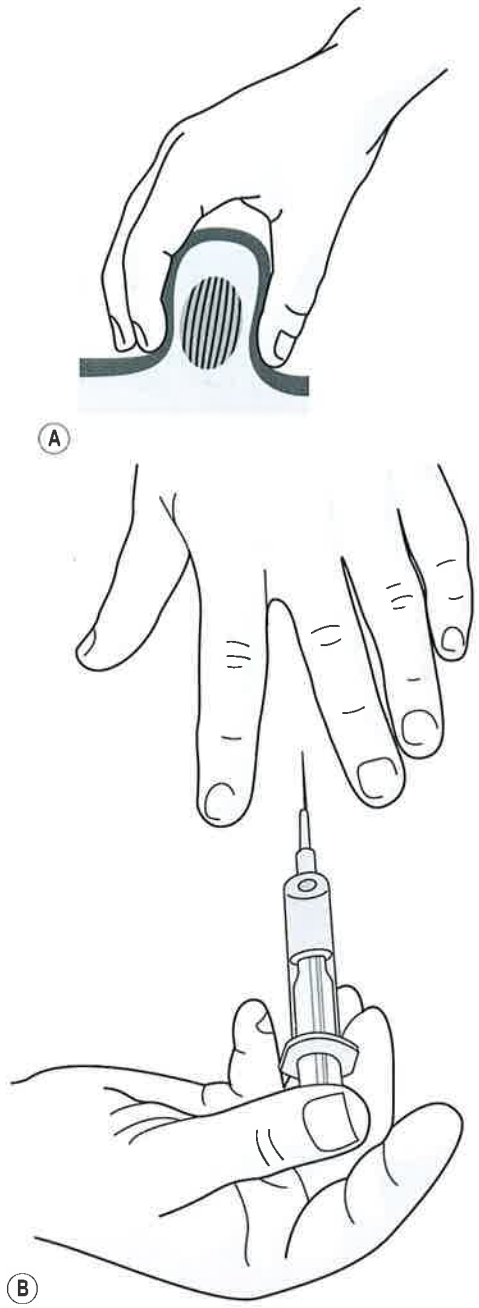


**Fig. 21.2** Fixation of the point or region with a pinch grip between thumb and index finger (a) or the Y-grip between the index finger and the middle finger and with a fixing pressure against the underlying tissue (b).

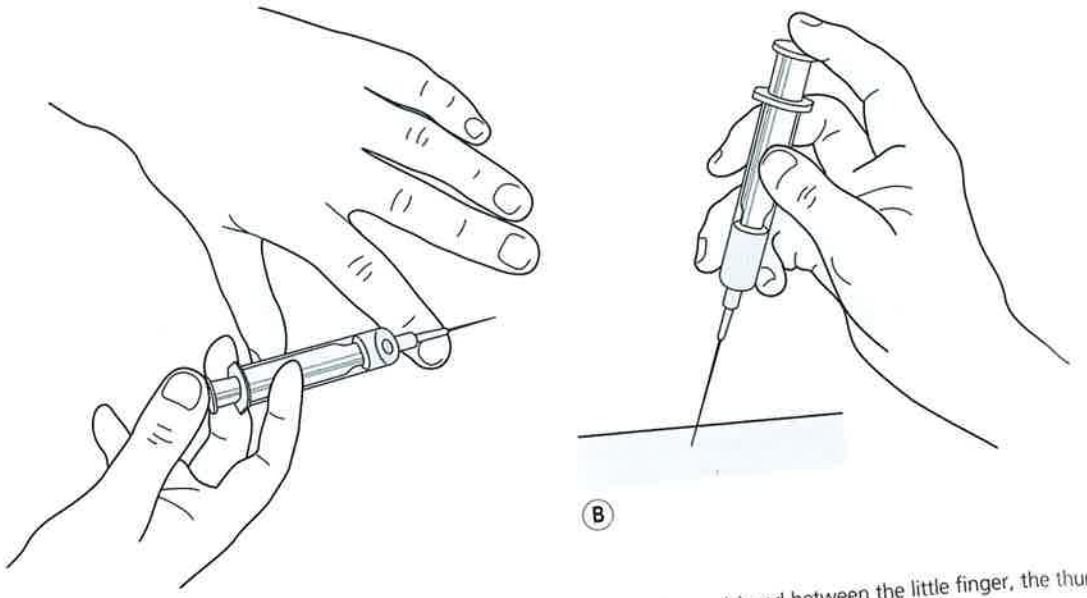


**Fig. 21.3** Holding the syringe between the thumb and the third and fourth fingers (a) and between the little finger, the thumb, and the third, fourth and fifth fingers (b).

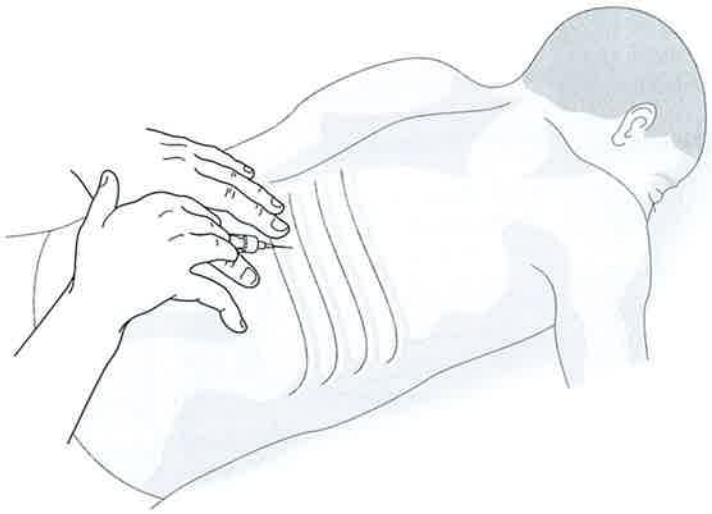

**Fig. 21.4** Further protection over the thoracic region is provided by a flat almost tangential angle of insertion, using a Venetian-blind-like effect of the ribs and by support from the hand.

After prior explanation to the patient and instruction about the twitch reaction that is to be expected, disinfection is followed by insertion of the injection needle (guage 21, 25 or 27). It is quickly pushed through the skin then slowly pushed forward in the direction of the mTrP. A fan-shaped forward movement procedure with the injection needle may be required in the fixed area in order to find the mTrP (Fig. 21.5).

The correct position of the needle in the mTrP is controlled by the visible or tangible twitch response (or also EMG-controlled). The volume to be injected at the mTrP is about 0.2 ml.

It is possible to inject both at the mTrP and at the insertion (possibly infiltrating by using a simplified technique with rather more volume). A disadvantage of infiltration is the lesser effect and the lack of control over the needle position in the T-point.

This probe technique with twitches is an mTrP injection technique that is more painful than usual for patients, it has to be performed with confident verbal

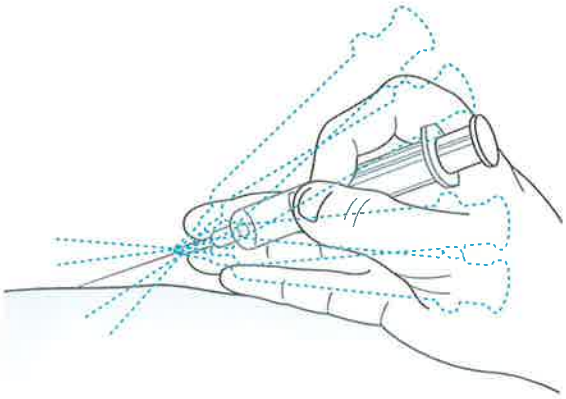


**Fig. 21.5** Superficial probing with the injection needle.

reassurance and breathing instructions (expiration). It is usual for patients to cry out, yawn or laugh. Occasionally, tears are observed as an expression of pain or in memory of pain previously suffered. Seeing this should prompt the therapist to respond and if necessary include it in the discussion of the treatment plan with the patient (psychosomatic).

An absolute prerequisite for injection is knowledge of the precise regional anatomical conditions and knowledge of possible risks. It is imperative to explain about possible complications (infection, bleeding, pneumothorax, haematoma, blood vessel or nerve lesion, allergic reaction, cardiovascular toxicity, sensory deficits), including their documentation.