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## REFERENCES

1. Abel, O., Siebert, W. J. & Earp, R.: Fibrositis. J Missouri Med Ass *36:* 435, 1939.
2. Adler, I.: Muscular rheumatism. Med Rec *57:* 529, 1900.
3. Award, E. A.: Interstitial myofibrositis. Hypothesis of the mechanism. Arch Phys Med & Rehab *54:* 449, 1973.
4. Bonica, J. J.: Management of myofascial pain syndromes in general practice. JAMA *164:* 732, 1957.
5. Brendstrup, P., Jespersen, K. & Asboe-Hansen, G.: Morphological and chemical connective tissue changes in fibrositic muscles. Ann Rheum Dis *16:* 438, 1957.
6. Collins, D. H.: Fibrositis and infection. Ann Rheum Dis *2:* 114, 1940.
7. Cooper, A. L.: Trigger-point injection: its place in physical medicine. Arch Phys Med & Rehab *42:* 704, 1961.
8. Gorrell, R. L.: Musculofascial pain. JAMA *142:* 557, 1950.
9. Fassbender, H. G. & Wegner, K.: Morphologie und Pathogenese des Weichteilrheumatismus. Z Rheumaforsch *32:* 355, 1973.
10. Gowers, W. R.: Lumbago: its lessons and analogues. Br Med J *1:* 117, 1904.
11. Hench, P. S.: Fibrositis. Ann Int Med *10:* 878, 1936.
12. Kagen, L. J.: Myoglobin: Methods and diagnostic uses. CRC Criti Rev Clin Sci *9*(4): 273, 1978.
13. Kraft, G. H., Johnson, E. W. & LaBan, M. M.: The fibrositis syndrome. Arch Phys Med & Rehab *49:* 155, 1968.
14. Kraft, J., Aastrup, H. & Schrøder, P.: Diagnostic value for acute myocardial infarction of creatine kinase and lactate dehydrogenase isoenzymes compared with total enzymes. Acta Med Scand *203:* 167, 1978.
15. Krusen, F. H., Kottke, F. J. & Ellwood, P. M: Handbook of Physical Medicine and Rehabilitation, W. B. Saunders Co., Philadelphia, 1965.
16. Miehlke, K., Schulze, G. & Eger, W.: Klinische und experimentelle Untersuchungen zum Fibrositis Syndrom. Z Rheumaforsch *19:* 310, 1960.
17. Recommended Methods for the Determination of Four Enzymes in Blood. Scand J Clin Lab Invest *33* (4): 287, 1974.
18. Simons, D. G.: Muscle pain syndroms Parts 1 and 2. Special review. Am J Phys Med *54:* 289, 1975; 55: 15, 1976.
19. Smythe, H. A.: Nonarticular rheumatism and psychogenic musculoskeletal syndromes. *In* Arthritis and Allied Conditions (ed. Daniel J. McCarty), pp. 881–891. Lea & Febiger, 1979.
20. Sola, A. E., Rodenberger, M. L. & Gettys, B. B.: Incidence of hypersensitive areas in posterior shoulder muscles. Am J Phys Med *34:* 585, 1955.
21. Stockman, R.: Rheumatism and Arthritis (ed. W. Green). Edinburgh, 1920.
22. Travell, J., Rinzler, S. & Herman, M.: Pain and disability of the shoulder and arm. JAMA *120:* 417, 1942.
23. Valentine, M.: Aetiology of fibrositis: a review. Ann Rheum Dis *6:* 241, 1947.
24. Wittenberg, J. B.: Myoglobin-facilitated oxygen diffusion: Role of myoglobin in oxygen entry into muscle. Phys Rev *50:* 559, 1970.

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# REDUCED BLOOD FLOW IN FIBROMYOTIC MUSCLES DURING ULTRASOUND THERAPY

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**ABSTRACT. The muscle blood flow (MBF) in m. trapezius was studied in 7 subjects with fibromyotic pain syndrome before and during treatment with ultrasound (1 Watt/cm²) and during placebo treatment, using the local $^{133}$Xe-washout technique. MBF in the fibromyotic muscles was significantly reduced during ultrasound treatment ($p<0.05$) compared to the blood flow before the treatment and during placebo treatment 1.57± SEM 0.52 and 2.51± SEM 0.43 ml/100 g/min, respectively. In 6 normal trapezius muscles the mean MBF was 2.30± SEM 0.44 ml/100 g/min before ultrasound treatment and 2.31± SEM 0.41 ml/100 g/min during ultrasound therapy. MBF decreased in a lidocaine blocked fibromyotic muscle during ultrasound treatment while no effect on MBF was detectable during ultrasonating a normal lidocaine pretreated muscle. It is concluded that ultrasound treatment decreases MBF in fibromyotic muscles and that this is paradoxical effect of ultrasound might be due to a direct effect on the vessels or a local release of vasoactive substances in the fibromyotic muscles.**

*Key words:* Muscle blood flow, $^{133}$xenon clearance, fibromyotic pain syndrome, myofascial pain syndrome, trigger points, ultrasound therapy

It is generally believed that treatment of fibromyotic muscles with ultrasound induces an increase in muscle blood flow. Recently we have shown that blood flow in fibromyotic muscles is not reduced compared to normal muscles (2).

In this study we have measured the blood flow in fibromyotic muscles before, during and after treatment with ultrasound, using the $^{133}$xenon clearance method.

## METHODS

The study was carried out in 7 volunteers all suffering from chronic, localized changes in the consistency of m. trapezius (trigger points), these being the points from which the actual pain could be provoked.

Muscle blood flow was measured in the supine position after at least 30 min rest at a constant room temperature of 22–23°C, using the local $^{133}$Xe-clearance method as described in details elsewhere (4, 6). After injection of 0.1 mCi $^{133}$Xe dissolved in isotonic saline (0.1 ml) into the trigger point a thin layer of glycerine was applicated on the dorsal side of the m. trapezius over the depot. A NaI scintillation detector was placed 15 cm over the radioactive field on the ventral side of the muscle. This procedure made it possible to measure the MBF during ultrasound therapy.

15–20 min after the injection the disappearance of $^{133}$xenon was monitored at 20 sec intervals and followed for at least 6 min before the ultrasound treatment. The ultrasound was then applied using a Siemens Sonostat 623 with continuous output, 1 Watt/cm². The sound head was moved in circles at a constant rate over the trigger point. When the washout of the tracer was monoexponential for at least 6 min the subjects were told that the treatment with ultrasound was changed to another intensity and the procedure was continued with 0 Watt/cm² until the washout of the tracer was monoexponential in at least another 6 min. Each experiment was terminated by a series of isometric contractions of the m. trapezius in order to check that $^{133}$xenon washout could be increased when exercise hyperemia occurred. Thus we were sure that the tracer depot had been injected into the muscle and not into subcutaneous tissue.

Six experiments were performed in which MBF was measured before and during ultrasounding normal trapezius muscles. In one subject MBF was examined before and during ultrasound treatment 75 min after injection of lidocaine into the trigger point (4 ml, 1% lidocaine). The same procedure was performed in one subject without any symptoms from the trapezius muscle.

Muscle blood flow was calculated from the slopes of the washout curves using the equation:

$$f = K_{xe} \cdot \lambda \cdot 100 \text{ (ml/100 g/min)}$$

where $K_{xe}$ is the rate constant and $\lambda$=0.7 the tissue to blood partition coefficient.

Linear correlation by the least squares' method, and Wilcoxon rank sum test was used to analyse the data. As limit of significance was chosen $p$=0.05. Informed consent was obtained after the subjects had been explained the purpose and risks by the examination.

Table 1. *Muscle blood flow (MBF ±1 SE, ml/100 g/min) before and during treatment with ultrasound and during placebo treatment in 7 subjects with myofasciel pain syndrome*

| | MBF before ($ref_1$) | MBF ultrasound | MBF placebo ($ref_2$) | MBF ($ref_1$+$ref_2$)/2 |
|---|---|---|---|---|
| A | 3.60±0.08 | 1.16±0.06 | 2.26±0.06 | 2.93±0.01 |
| B | 1.90±0.04 | 0.65±0.06 | 1.71±0.08 | 1.81±0.09 |
| C | 0.85±0.03 | 0.33±0.04 | 0.94±0.03 | 0.90±0.04 |
| D | 5.25±0.05 | 4.46±0.04 | 3.65±0.16 | 4.45±0.17 |
| E | 2.34±0.06 | 2.02±0.04 | 1.90±0.09 | 2.12±0.11 |
| F | 2.74±0.06 | 0.99±0.06 | 3.83±0.28 | 3.29±0.29 |
| G | 2.06±0.03 | 1.40±0.04 | 2.13±0.05 | 2.10±0.06 |
| Mean | 2.68 | 1.57 | 2.35 | 2.51 |
| ±1 SE | 0.53 | 0.52 | 0.39 | 0.43 |
| $p<$ | 0.05[a] | | | |

[a] $Ref_1$ + $ref_2$/2 compared to MBF during ultrasound treatment.

## RESULTS

The mean blood flow in the fibromyotic muscles was 2.68±SEM 0.53 ml/100 g/min before the ultrasound treatment. During treatment the mean blood flow was 1.57±SEM 0.57 ml/100 g/min. After the treatment the mean blood flow increased to 2.35± SEM 0.39 ml/100 g/min. The MBF in the fibromyotic muscles was significantly reduced during ultrasound therapy ($p<0.05$), Table I.

Ultrasounding normal muscles induced an increase in MBF in 3 and a decrease in 1 subject. In two subjects the MBF was practically unaffected by ultrasound treatment (Table II). In one lidocaine pretreated fibromyotic muscle the ultrasound induced a decrease in the blood flow from 0.88±0.03 ml/100 g/min before the treatment to 0.59±0.06 ml/100 g/min during ultrasound treatment.

In one normal lidocaine blocked muscle the blood flow was 2.47±0.07 ml/100 g/min before the ultrasound treatment. During ultrasound therapy MBF was 2.48±0.12 ml/100 g/min.

## DISCUSSION

The ultrasound treatment induced a decrease in the resting blood flow in all the fibromyotic muscles, and during the subsequent placebo ultrasound therapy the MBF increased to values near the values before ultrasounding the fibromyotic muscles. It has previously been shown that ultrasound has no significant effect on MBF immediately after ultrasound treatment of normal muscles (1, 5). Our results show that the MBF during ultrasound therapy is not significantly altered.

The reason for the paradoxical effect of ultrasound on MBF in fibromyotic muscles is unknown.

Ultrasound may alter nerve conduction velocity (3). However, we observed a decrease in blood flow during ultrasound treatment of a lidocaine blocked fibromyotic muscle while no effect of ultrasound on MBF in a normal lidocaine blocked muscle was detectable. This indicates that the paradoxical response of ultrasound on fibromyotic muscle blood flow might be due to either a direct effect on the vessels or a local release of a vasoactive substance maybe from sympathetic nerve endings.

We conclude, therefore, that any beneficial effect on fibromyotic muscle is not due to an increase in MBF since ultrasound therapy decreases the blood flow in fibromyotic muscles.

Table II. *Muscle blood flow before and during ultrasound treatment (1 Watt/cm₂) in 6 nomral subjects*

| | Before | During |
|---|---|---|
| | 2.26±0.11 | 2.40±0.10 |
| | 3.89±0.13 | 2.69±0.29 |
| | 2.67±0.10 | 3.67±0.10 |
| | 2.80±0.09 | 2.84±0.11 |
| | 1.25±0.02 | 1.33±0.05 |
| | 0.95±0.03 | 0.97±0.04 |
| Mean | 2.30 | 2.31 |
| ±1 SE | 0.44 | 0.41 |





## REFERENCES

1. Hansen, T. I. & Kristensen, J. H.: Effect of massage, shortwave diathermy and ultrasound upon $^{133}$Xe disappearance rate from muscle and subcutaneous tissue in the human calf. Scand J Rehab Med *5:* 179, 1973.
2. Klemp, P., Nielsen, H. V., Kosgård, J. & Crone, P.: Blood flow in fibromyotic muscles. Scand J Rehab Med (in press).
3. Madsen, P. W. & Gersten, J. W.: The effect of ultrasound on conduction velocity of peripheral nerve. Arch Phys Med *42:* 645, 1961.
4. Lassen, N. A., Lindbjerg, J. & Munck, O.: Measurements of blood flow through skeletal muscle by intramuscular injection of Xenon-133. Lancet *1:* 686, 1964.
5. Paaske, W., Hovind, H. & Sejrsen, P.: Ultralydbehandling og vævsgennemblødningen. Ugeskr Læg *135:* 2087, 1973.
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