

Pain Relief by Bone Drilling and Osseous Pain Sensitization: A Hypothesis

Sumihisa Aida^{1*} and Gentaro Takahashi²

¹Pain Medicine, Nippori Jyogu Hospital, Tokyo 116-0014, Japan

²Takahashi Pain Clinic, Tokyo 167-0032, Japan

*Corresponding author: Sumihisa A. Pain Medicine, Nippori Jyogu Hospital, Tokyo 116-0014, Japan, Tel: 03-5827-0176; Fax: 03-5827-0176; E-mail: aida.sum@gmail.com

Rec date: Mar 05, 2014, Acc date: Apr 09, 2014, Pub date: Apr 11, 2014

Copyright: © 2014 Sumihisa A, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Bone drilling (BD) is reconsidered to be a novel treatment for pain including vertebral compression fracture (VCF), vertebral spondylosis, osteoarthritis (OA) and osteonecrosis of the knee and hip, and neuropathic pain. BD is simple and harmless. The technique of BD is the similar to that of bone marrow puncture. BD is conducted under X-ray fluoroscopy and regional anesthesia. Exact penetration of bone cortex is required for intramedullary decompression, and sufficient aspiration of medullary blood is important to eliminate intramedullary inflammatory cells (ICs) and inflammatory chemical mediators (ICMs). Onset of the effect is immediate, and the effect lasts several months or a few years. However, precise of the underlying mechanisms of BD is remained unclear. On the other hand, effectiveness of BD was also shown in neuropathic pain, a representative of complex regional pain syndrome (CRPS.) Neuropathic pain was induced after the nerve root injury, and the bone is generally intact. However, BD alleviates a foot ache that is produced thereafter. This fact may indicate that subsequent medullary inflammation occurs and bone tissue is involved in CRPS. Therefore, a certain change within the bone is induced by CRPS. The fact suggests a hypothesis that a self-facilitating mechanism of pain is provided independently within bone marrow. We term this mechanism 'osseous pain sensitization'. Also medullary ICs cause bone resorption, and the ultimate bone dystrophy due to CRPS may be 'Sudeck dystrophe'.

Keywords: Bone drilling; Pain relief; Intramedullary decompression; Neurogenic inflammation; Dorsal root reflex; Osseous sensitization

Introduction

Bone drilling (BD) had been undergone for treatment of pain in knee and hip osteoarthritis (OA) by Makenzie in 1936 [1]. Thereafter BD has been kept barely for treatment of pain in osteonecrosis of the femur head [2]. Kohashi et al. [3] found in 2004 that the backache due to vertebral compression fracture (VCF) is alleviated by BD alone: bone cement injection was failed in percutaneous vertebroplasty (PVP). They focused on this phenomenon. They performed BD of the vertebra for the treatment of backache in vertebral compression fracture (VCF). On the other hand, Shinjo [4] have shown effectiveness of treatment on arthralgia of knee OA in 2005. They did give the BD an important post for pain relief. Recently, the pain relief effect of BD in VCF is ensured to be nearly equivalent between BD and PVP [5].

Thereafter, BD has been considered to be one of the important treatments for pain, and the indication of BD has been extended to various pains including neuropathic pain in Japan [6]. The simple method, immediate onset, long-lasting effect, and harmlessness are the appreciable characteristics [1-7]. The effect becomes evident immediately after BD, and develops further for a few days. Thereafter steady pain relief is obtained [7]. The effect lasts for several months [6] or a few years [7].

Effectiveness of BD is shown in backache due to VCF [3,5,7,8] and vertebral spondylosis [7,8], arthralgia due to OA of the knee, hip and shoulder [1,4,8] as well as osteonecrosis of the hip [2,8], and neuropathic pain [6]. In acute pain due to VCF, BD is more effective

[7]. Usually, an effect of BD on most pain due to malignant tumors may be insufficient, and BD has been scarcely attempted in pain due to trauma (a bruise, a sprain or fracture) except for VCF (mentioned above). As a matter of course, the underlying disorder should be treated first.

Bone drilling is possible repeatedly, when the effect become faint.

Methods and Complication of BD

The technique of BD has been published elsewhere [8]. Briefly, BD is performed under X-ray fluoroscopy and regional anesthesia at a puncture point. Procedure is similar to bone marrow puncture. Bone marrow puncture needle (11-14 G, with a notched tip) are usually used for BD, while for small bones a more slender needle (a conventional or nerve block needle, 18-22 G) is also used. For BD of vertebral body, the same route as PVP, transpedicular approach is adequate [9]. For arthralgia, one side or both sides of juxta-bones is drilled [4]. In complex regional pain syndrome (CRPS), multiple tender points are selected for the targets of BD [6].

To verify the location of a needle tip, radio-opaque contrast medium, 1-10 ml, is injected into bone marrow for osteophlebography. Two important actions, penetration of bone cortex and aspiration of medullary blood, should be important. The penetration may lower intramedullary pressure (IP), while the aspiration may remove inflammatory cells (ICs) and inflammatory chemical mediators (ICMs), as mentioned below. Medullary blood, occasionally tens ml or more, is aspirated into an attached syringe.

Complications (infection, hematoma, and ectopic puncture) associating with BD are also similar to those with bone marrow puncture. In rare cases, bone fracture and breaking a needle may be

probable. Antibiotic medication to prevent infection, especially osteomyelitis, is required in some case. BD to patients undergoing anti-coagulant therapy should be conducted with caution. To avoid an incorrect or accidental tap, practice under X-ray fluoroscopy should be inevitable.

Discussion

Intramedullary Pressure (IP)

Most authors have ever considered that arthralgia and backache are caused by intramedullary hypertension, and pain relief after BD is brought about by intramedullary decompression [1-7]. IP was measured by some investigators. According to their studies, IP fluctuates easily receiving various inputs from circumstances. Vertebral IP fluctuates parallel with cerebrospinal fluid (CSF) pressure [10]. Femoral IP varies among animals [11]. An obstacle of venous return elevated IP in the tibia medulla [12], while IP and systemic pressure were linearly related [13]. Intramedullary hypertension followed after intra-articular hypertension of juxta-joint of the metacarpal bone [14].

Thus bone marrow receives incessantly various inputs from circumstances. When these inputs are accumulated all together, the fluctuations may become a potent stress to the bone marrow. Therefore, in the pathological conditions with hyperalgesia, pain sensitization, and complex regional pain syndrome (CRPS) [15], the bone marrow may sensitively react to the stress, and may transmit nociceptive impulses to the central nervous system (CNS). Probably, the bone hole opened by BD may release from intramedullary hypertension. Considering these, therefore, the reason of immediate alleviation of pain is explainable by intramedullary decompression.

Medullary edema and inflammation

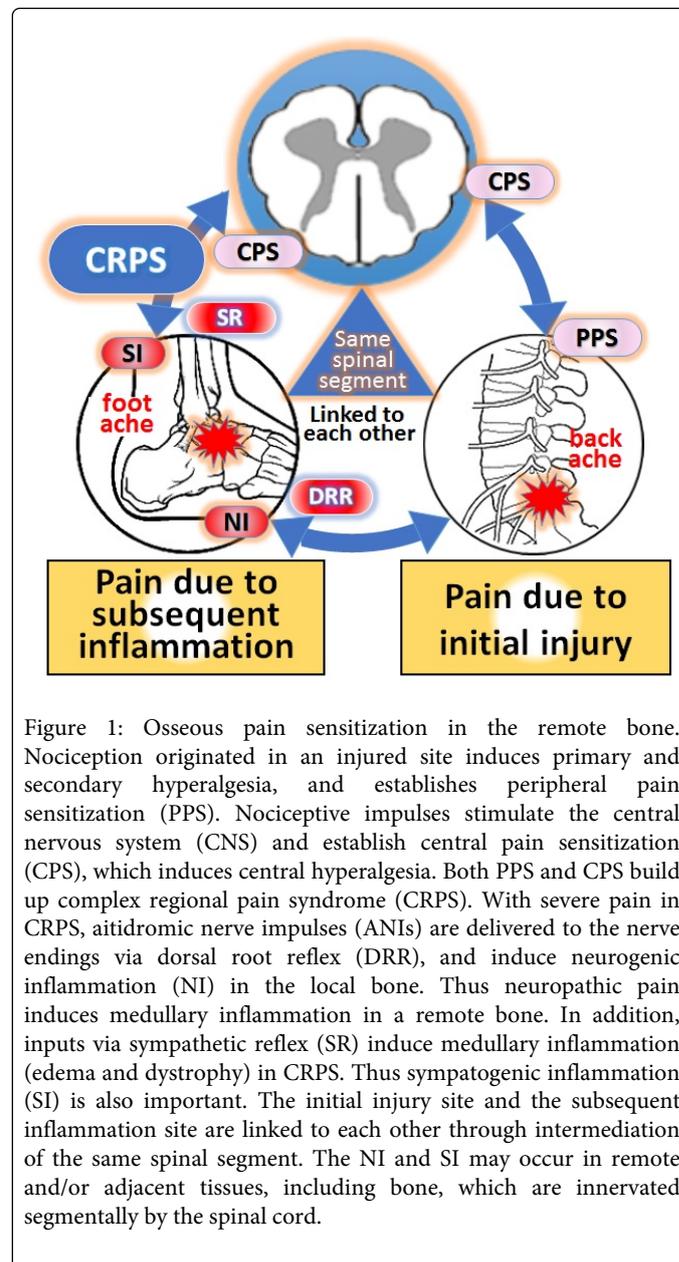
Medullary edema of the vertebra with compression fracture [16], and of the tibia with OA [17,18] is shown. The edema is a potent risk factor for bone destruction and arthritis [19,20]. After aspiration of medullary blood, however, the edema does not consider to vanish immediately. Therefore, the immediate pain relief effect may not relate to intramedullary decompression, but the later (a few days after) effect may be brought about by disappearance of the edema following elimination of ICs and ICMs. Thus, to eliminate ICs and ICMs may also be very important for the pain relief.

Remote induction of medullary inflammation

Effectiveness of BD was also shown in neuropathic pain that is a representative of CRPS [6]. Neuropathic pain occurred initially at the nerve root after back bone surgery (failed back surgery syndrome). Thereafter, a severe foot ache occurred in spite of absence of any history of trauma or injury. BD of the foot bones was undergone, and BD alleviated the foot ache. This fact shows that the foot ache is induced remotely by nerve root CRPS. Consequently, the foot bones are involved in CRPS associating with severe pain and inflammation.

First, one of the factors causing the phenomenon may be neurogenic inflammation [21], which is brought about by antidromic nerve impulses (ANIs) to sensory nerve endings via dorsal root reflex (DRR) [22,23]. By receiving ANIs, neurogenic inflammation (NI) occurs in the bone innervated segmentally by the spinal cord. Next, inflammation (edema and dystrophy) of the bone is also induced by stimulation via autonomic (including sympathetic) reflex

(sympathogenic inflammation; SI) [14]. Thus the foot bones are involved in the CRPS. Furthermore, the initial and subsequent injuries and inflammations may be linked to each other through intermediation of the same spinal segment (Figure 1).

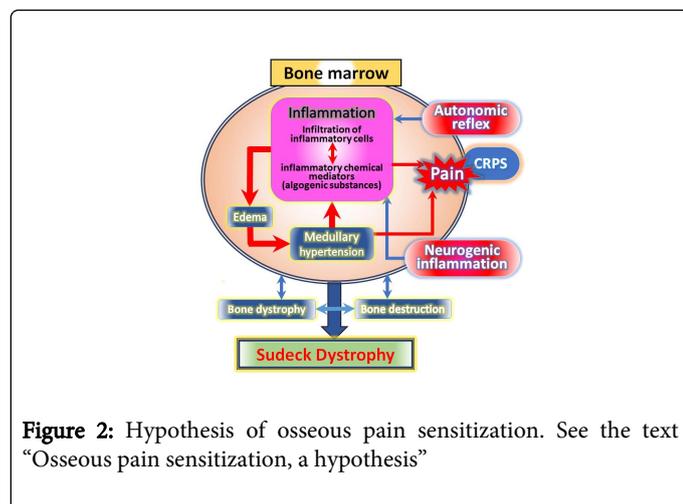


Osseous Pain Sensitization: A Hypothesis

Thus bone inflammation is induced by ANIs via DRR as well as stimulation via autonomic reflex. The edema is a potent risk factor for bone destruction. In the bone marrow with edema, infiltration of ICs and release of ICMs is observed [18,19]. Released ICMs such as TNF α and IL-6 are induced further edema and inflammation [24,25], which exacerbate again the IP and inflammation to the higher level. Therefore, the pain becomes still more intensive. These sequential phenomena form a vicious circle, which rolls independently within the bone marrow. On the other hand, ICs such as macrophages or

osteoclasts work for bone resorption causing bone destruction (if absorbed violently) or dystrophy (if absorbed diffusely).

Thus, the self-facilitating mechanism is provided naturally in the bone marrow. We term this mechanism 'osseous pain sensitization'. The ultimate result of CRPS in the bone tissue may be the 'Sudeck dystrophy' (Figure 2).



Conclusion

We should reconsider the BD for a novel treatment of pain. In spite of the evident effect, the clinical findings concerning BD are inadequately documented. Furthermore, histopathological and cytopathological knowledge concerning pain relief by BD is nonexistent. Therefore, studies on the underlying mechanisms of the pain relieving effect of BD are necessary. Then BD should be given an important post as a novel treatment for pain, and will be used even more widely.

References

1. Mackenzie JF (1936) Osteo-arthritis of Hip and Knee: Description of a Surgical Treatment. *Br Med J* 1: 306-308.
2. McGrory BJ, York SC, Iorio R, Macaulay W, Pelker RR, et al. (2007) Current practices of AAHKS members in the treatment of adult osteonecrosis of the femoral head. *J Bone Joint Surg Am* 89: 1194-1204.
3. Kohashi Y, Matsuura T, Ishitani E, Shin K, Okada F (2004) The effect of decompression of internal pressure in vertebral body for painful osteoporotic vertebral compression fracture. *Orthoped Surg Traumatol* 47: 1589-1595.
4. Shinjo K (2005) New surgical treatment of osteoarthritis based on its pathogenesis. *J Cent Assoc Orthoped Surg Traumatol* 48: 531-532.
5. Yokoyama K, Kawanishi M, Yamada M, Tanaka H, Ito Y, et al. (2012) Comparative study of percutaneous vertebral body perforation and vertebroplasty for the treatment of painful vertebral compression fractures. *AJNR Am J Neuroradiol* 33: 685-689.
6. Aida SI, Kuratani N, Ohara Y, Amagasa S, Wajima Z (2011) Immediate alleviation of chronic pain by bone drilling and probable involvement of bone tissue in pain sensitization. *BMJ Case Rep* 2011.
7. Sato A, Ogihara M, Akamine T, Wakayama H (2012) Therapeutic outcomes by different times of initiating the vertebral body drilling treatment for osteoporotic vertebral compression fracture. *Pain Clinic*; 33: 984-988.
8. Ogihara M (2010) Marrow decompression, in *The Guideline of Therapeutics for Pain Clinicians Ver 3*, ed by Committee for the Therapeutic Guideline of Japan Society of Pain Clinic Tokyo Sinko-Koeki; 52-54.
9. Layton KF, Thielen KR, Wald JT (2006) A modified vertebroplasty approach for spine biopsies. *AJNR Am J Neuroradiol* 27: 596-597.
10. Hanai K, Kawai K, Itoh Y, Satake T, Fujiyoshi F, et al. (1985) Simultaneous measurement of intraosseous and cerebrospinal fluid pressures in lumbar region. *Spine (Phila Pa 1976)* 10: 64-68.
11. Thomas IH, Gregg PJ, Walder DN (1982) Intra-osseous phlebography and intramedullary pressure in the rabbit femur. *J Bone Joint Surg Br* 64: 239-242.
12. Wilkes CH, Visscher MB (1975) Some physiological aspects of bone marrow pressure. *J Bone Joint Surg Am* 57: 49-57.
13. Kiaer T, Gronlund J, Jensen B, Svalastoga E (1990) Effects of variation in systemic blood pressure on intraosseous pressure, PO₂, and PCO₂. *J Orthop Res* 8: 618-622.
14. Arnoldi CC, Reimann I, Mortensen S, Christensen SB, Kristoffersen J, et al. (1980) The effect of joint position on juxta-articular bone marrow pressure. Relation to intra-articular pressure and joint effusion--an experimental study on horses. *Acta Orthop Scand* 51: 893-897.
15. Treede RD, Meyer RA, Raja SN, Campbell JN (1992) Peripheral and central mechanisms of cutaneous hyperalgesia. *Prog Neurobiol* 38: 397-421.
16. Voormolen MH, van Rooij WJ, Sluzewski M, van der Graaf Y, Lampmann LE, et al. (2006) Pain response in the first trimester after percutaneous vertebroplasty in patients with osteoporotic vertebral compression fractures with or without bone marrow edema. *AJNR Am J Neuroradiol* 27: 1579-1585.
17. Felson DT, Chaisson CE, Hill CL, Totterman SM, Gale ME, et al. (2001) The association of bone marrow lesions with pain in knee osteoarthritis. *Ann Intern Med* 134: 541-549.
18. Felson DT, McLaughlin S, Goggins J, LaValley MP, Gale ME, et al. (2003) Bone marrow edema and its relation to progression of knee osteoarthritis. *Ann Intern Med* 139: 330-336.
19. Shen PC, Lu CS, Shiau AL, Lee CH, Jou IM, et al. (2013) Lentiviral small hairpin RNA knockdown of macrophage inflammatory protein-1 β ameliorates experimentally induced osteoarthritis in mice. *Hum Gene Ther* 24: 871-882.
20. Kawane K, Ohtani M, Miwa K, Kizawa T, Kanbara Y, et al. (2006) Chronic polyarthritis caused by mammalian DNA that escapes from degradation in macrophages. *Nature* 443: 998-1002.
21. Rosa AC, Fantozzi R (2013) The role of histamine in neurogenic inflammation. *Br J Pharmacol* 170: 38-45.
22. Willis WD Jr (1999) Dorsal root potentials and dorsal root reflexes: a double-edged sword. *Exp Brain Res* 124: 395-421.
23. Hagains CE, Trevino LA, He JW, Liu H, Peng YB (2010) Contributions of dorsal root reflex and axonal reflex to formalin-induced inflammation. *Brain Res* 1359: 90-97.
24. Stannus OP, Jones G, Blizzard L, Cicuttini FM, Ding C (2013) Associations between serum levels of inflammatory markers and change in knee pain over 5 years in older adults: a prospective cohort study. *Ann Rheum Dis* 72: 535-540.
25. Theoharides TC, Boucher W, Spear K (2002) Serum interleukin-6 reflects disease severity and osteoporosis in mastocytosis patients. *Int Arch Allergy Immunol* 128: 344-350.