

Comments on Padawer and Levine, *PAIN*, 48 (1992) 132–135

Padawer and Levine (1992) discuss whether exercise-induced analgesia is a fact or artifact. They suggest that the exercise-induced analgesic effect may be an artifact of reactivity to pain tests. They found that pain intensity ratings for the cold pressor test were significantly reduced after pre-exposure to the same pain test. However, in the experimental design chosen by these authors, they were unable to detect an exercise effect on pain intensity rating.

It is important to note that an experimental pain test itself can activate endogenous pain modulation. It follows that in order to determine an analgesic effect of any intervention a control group should be introduced into the experimental design. The fact that Padawer and Levine could measure a reduction in pain intensity and unpleasantness ratings in a repetition of the cold pressor test is not unexpected and is most likely due to the novelty of the first trial. Among the many different experimental pain tests, the cold pressor test is strongly influenced by cognitive coping processes, which is one reason why it has been often used to study psychological interventions in pain.

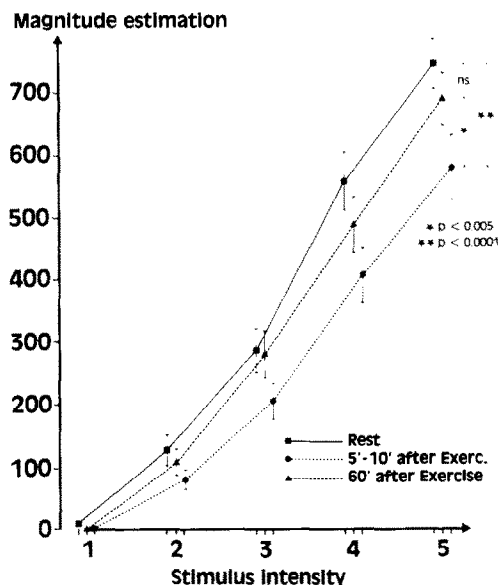


Fig. 1. Mean subjective magnitude estimations (range: 0–1000, arbitrary units) of 5 suprathreshold stimulus levels ($0.8\text{--}4 \times$ detection threshold). Mean values of 10 healthy male subjects (± 1 S.E.M.) are shown. The different symbols present the results for measurements made before (\blacksquare) directly after (\bullet) and 60 min after (\blacktriangle) short-term exhaustive physical exercise on cycle ergometer. Significance levels are based on analysis of covariance (before exercise–directly after exercise, $P < 0.0001$; before exercise–60 min after exercise, n.s.; directly after exercise–60 min after exercise, $P < 0.005$). Electrical intracutaneous finger stimulation was applied, each stimulus intensity was delivered 10 times in random order, yielding a total of 50 trials/subject for each time of measurement. (Printed with permission of Williams and Wilkins, Baltimore, MD).

In the Padawer and Levine study there is no mention whether the water was stirred during the cold pressor test. Our own investigations (Droste 1983) have indicated that during the cold pressor test a layer of warm water can form around the skin if the water is not properly stirred, which clearly affects the resultant pain ratings. Physical exercise influences body temperature regulation, which according to our experience makes the cold pressor test for the analysis of pain regulation after exercise less useful.

It remains open whether the experimental design in the study of Padawer and Levine was appropriate to assess exercise-induced analgesia. The exercise intensity selected by the authors was such that it remains questionable whether it was sufficient to provoke the expected analgesic effect. The release of pituitary hormones like beta-endorphin is not only dependent on $\%VO_{2\max}$ but also dependent on surpassing the anaerobic threshold. Heart rate is only a vague index of $\%VO_{2\max}$ and blood lactate, both of which were not measured in the study. In addition, exercise-induced analgesia is most likely more dependent on 'central command' than on peripheral markers of exercise intensity (cf., the review in Droste 1992).

We would like draw the readers' attention to a study that was published recently (Droste et al. 1991). The results of this investigation strongly suggest the existence of exercise-induced analgesia. A significant pain threshold elevation was found during exercise for electrical intracutaneous finger and dental pulp stimulation. Several extensive psychophysical pain measurement techniques were employed (e.g., 2-interval forced-choice method). A series of control measurements were also conducted to rule out the influence of repeated measurements. Not only the thresholds were significantly increased during exercise but also the subjective magnitude estimation of suprathreshold stimuli was significantly reduced after exercise (see Fig. 1). The finding that these differences were no longer evident for the retest conducted 1 h after exercise speaks against the argumentation of Padawer and Levine.

References

- Droste, C., Schmerz bei koronarer Herzkrankung. Eine Literaturübersicht und psychophysiologische Untersuchung an symptomatischen und asymptomatischen Patienten mit Myokardischämie. Minerva Publication, Munich, 1983.
- Droste, C., Transient hypoalgesia under physical exercise—relation to silent ischaemia and implications for cardiac rehabilitation, *Ann. Acad. Med. Singapore* 1992, *Ann. Acad. Med. Singapore*, 21 (1992) 23–33.
- Droste, C., Greenlee, M.W., Schreck, M. and Roskamm, H., Experimental pain thresholds and plasma beta-endorphin levels during exercise, *Med. Sci. Sports Exerc.*, 23 (1991) 334–342.
- Padawer, W.J. and F.M. Levine, Exercise-induced analgesia: fact or artifact? *Pain*, 48 (1992) 134–135.

Conrad Droste

Benedikt Kreutz Cardiovascular Rehabilitation Center
D-7812 Bad Krozingen, Germany

Mark W. Greenlee

Neurological Clinic
Department of Neurophysiology
University of Freiburg,
D-7800 Freiburg, Germany