2025 年度　東京都立大学大学院　人間健康科学研究科
博士後期課程　入学試験問題（冬季）理学療法科学域　筆記

I . Explain which statistical methods are appropriate for which types of data.
以下の統計手法はどのようなデータに用いるのが適切か説明せよ。

I -1. Spearman's rank correlation coefficient
スピアマンの順位相関係数

I -2. chi-square test
カイ二乗検定

I -3. Mann-Whitney U test
マン・ホイットニーの U 検定

Ⅱ. Read the following paper and answer the following questions.
下記の論文を読み、後の問いに答えよ。

Title: Passive muscle properties are altered in children with cerebral palsy before the age of 3 years and are difficult to distinguish clinically from spasticity

Discussion

The clinical choice of antispasticity treatment requires that it is possible to determine correctly the contribution of reflex activation to muscle stiffness. We have demonstrated in this study that pathologically enhanced stretch reflexes only contributed to muscle stiffness in a minority of the investigated children with spastic CP, whereas change in passive muscle properties was a much more frequent problem. This was shown to be the case irrespective of ankle joint position within the range of 30° that we were able to study. Dietz and Berger were the first to show that muscle properties other than reflex stiffness may be altered in children with spastic CP. Several studies since then have documented that alterations in passive muscle properties occur quickly after brain lesions. These are difficult to distinguish from reflex-mediated contributions to the stiffness without the aid of electrophysiological and biomechanical evaluation. This was confirmed for children with CP in the present study by the observation that only seven out of 35 of those with CP determined as having spasticity in the clinical examination showed reflex stiffness that exceeded the range observed in healthy children.

Similar to other studies, no correlation was found between the neurological assessment and the biomechanical evaluation, emphasizing the inadequacy of the clinical scoring systems in distinguishing and quantifying the different contributions to muscle stiffness.

It should be pointed out that one limitation of the present study is that all children were in the three best functional groups according to the GMFCS and that none of them scored above 3 on the MAS. There is no reason to believe that our results would have been fundamentally different in functionally more severely affected children, but it may be argued that it would be easier to distinguish passive and reflex-mediated stiffness in children with a higher score on the MAS. What speaks against this is that the children were distributed throughout the Tardieu Scale except for the highest possible score (immobile joint). In addition, no correlation was found for the MAS score 1–3 and it is unlikely that the inclusion of score 4–5 would have affected the result.

It has been assumed for a long time that changes in muscle properties that eventually develop into contractures are caused by the pathological muscle tension from abnormal reflex activity. Indeed, much of the rationale of antispasticity treatment is based on this assumption. However,

the data supporting this assumption are scarce and not convincing. Pierce et al. found that passive stiffness in muscles around the knee in children with CP increased with age from 7 to 14 years. However, changes in body size were not taken into account and their observations are, therefore, difficult to interpret. The observation by Hägglund and Wagner of an association between spasticity in early childhood as determined from the MAS and reduced ankle movement in young spastic adults is not surprising because the MAS is not an efficient way of distinguishing reflex and non-reflex contributions to stiffness. It should also be noted that a 10-year follow-up of the effect of selective dorsal rhizotomy showed no effect on development of contractures, making it unlikely that abnormal reflex activity plays a pathophysiological role.

In the present study no or a weak negative correlation was found between age and both measures of stiffness. This is in line with the study by Nordmark et al., who observed no age-related change in passive ankle joint movement for children with CP older than 4 to 5 years. In the age group 2 to 4 years, however, a significant decline in movement range was observed, suggesting that the changes in passive muscle properties take place before the age of 4 years and thus for children younger than the youngest included in this study. Barber et al. have similarly observed reduced passive ankle movement in children with CP in the age group 2 to 5 years. Thus, our data do not support the idea that spasticity during childhood leads to contractures, but rather that contractures begin to develop before the age of 3 years. This emphasizes the importance of making an early diagnosis of CP to implement treatment to prevent contractures before the age of 3 years.

Our data suggest that there is reason to pay more attention to changes in passive muscle properties than pathologically increased reflex-mediated stiffness in children with spastic CP both in research and in the clinic. There is an overwhelming amount of knowledge about the pathophysiological changes leading to increased reflex excitability, but comparatively little knowledge of the pathophysiology of changes in passive muscle properties. There are emerging data showing changes in specific intra- and extra-cellular components in muscles, tendons, and connective tissue, which may be related to the alterations in the elastic properties of the tissue, but we are still far from a full mechanistic understanding of pathological changes in the elastic properties of muscles and tendons. The pathophysiological role of altered muscle tension (spasticity) and muscle inactivity in a developmental perspective especially remains unclear. Clinically, it is worrying that we do not have any effective interventions that can prevent or treat the development of pathologically increased passive stiffness of the tissue that leads eventually to contractures. Stretching, splinting, and casting have been shown to have no clinically significant effects.

All measurements in this study were made when the children were at rest. Several studies have demonstrated that reflexes are greatly modulated during functional motor tasks.

Observations of increased reflex excitability at rest are, therefore, not necessarily also manifested during voluntary activation of the muscles. Increased reflex excitability, if any, is therefore likely to be of even less significance during functional motor tasks in children with spastic CP than we have found here, but an answer to this question requires specific experiments.

Because current antispasticity treatment is primarily directed at diminishing reflex activation of muscles rather than passive muscle properties, there is reason to be concerned that our findings may indicate that many children with CP do not receive adequate treatment. To guide antispasticity treatment there is clearly a need for more optimal evaluation of the different components of muscle stiffness than the current clinical examinations afford.

[Abbreviation: CP; cerebral palsy, GMFCS; Gross Motor Function Classification System, MAS; Modified Ashworth Scale]

Source of reference（出典）
Willerslev-Olsen, M., Lorentzen, J., Sinkjær, T. and Nielsen, J.B. (2013), Passive muscle properties are altered in children with cerebral palsy before the age of 3 years and are difficult to distinguish clinically from spasticity. Dev Med Child Neurol, 55: 617-623.）
Partial excerpt（一部抜粋）

II -1. For the following sentences A, B, C, and D, circle “T” if the sentence is true and circle “F” if the sentence is false.
下記の A、B、C、D の文章についてその内容が正しければ T を、誤っていれば F を○で囲いなさい。

A. This study was performed on children with spastic cerebral palsy who had a mild GMFCS level and a relatively high motor function level.
本研究は、GMFCS レベルが軽度で運動機能レベルが比較的高い痙直型脳性麻痺児を対象として行われた。

B. In most children with spastic cerebral palsy who were the subjects of this study, the pathologically enhanced stretch reflex contributed to muscle stiffness.
本研究の調査対象となった痙直型脳性麻痺児では、そのほとんどで病理学的に亢進した伸張反射が筋の stiffness に寄与していた。

C. MAS is the most efficient way to distinguish between the reflex and non-reflex contributions to muscle stiffness.

MASは筋のstiffnessに対する反射性と非反射性の寄与を区別する最も効率的な方法である。

D. Stretching, splinting and casting have been reported to be clinically effective in preventing the progression of pathological passive stiffening that leads to contractures.

拘縮につながる病的な受動的硬化の進行予防に、ストレッチング、スプリント、ギプス包帯は臨床的に効果があると報告されている。

Ⅱ-2. Explain what is considered important for the antispasticity treatment in children with spastic cerebral palsy, based on the results of this study.

本研究から示唆される、痙直型脳性麻痺児に対する抗痙縮治療に重要と考えられることを説明しなさい。

Ⅲ. Read the following paper and answer the following questions.
以下の文章を読んで後の問いに答えよ。

Introduction

Chronic pain affects 20% of people in the US, with an estimated annual cost of more than $600 billion. The most common type is chronic back pain (CBP). In approximately 85% of cases, definitive peripheral causes of CBP cannot be identified, and central nervous system processes are thought to maintain pain. For people with this type of CBP— often referred to as primary, nonspecific, ① **nociplastic,** or centralized pain—psychological and behavioral treatments are recommended. Although these treatments can improve functioning, reductions in pain intensity are limited and better treatments are needed.

(This part is omitted)

Clinical Measures

The primary outcome was average pain over the last week on a numerical rating scale from 0 to 10 from the Brief Pain Inventory Short Form, assessed at the 1-month postbaseline session. We also calculated the proportion of participants reporting pain reduction of 30% or more, pain reduction of 50% or more, and a pain score of 0 or 1, indicating a pain-free or nearly pain-free state. Secondary outcomes included pain interference (Oswestry Disability Index); Patient-Reported Outcome Measurement Information System (PROMIS) short forms for depression, anxiety, anger, and sleep quality; and the Positive and Negative Affect Scale.

We considered 3 measures of pain beliefs as potential mediators: (1) the Tampa Scale of Kinesiophobia (TSK-11), assessing belief that pain indicates injury and fear of movement; (2) the Pain Catastrophizing Scale (PCS); and (3) the Survey of Pain Attitudes Emotion subscale (SOPA-Emotion), assessing beliefs that stress and negative emotion increase pain. Adverse events were recorded when participants spontaneously reported them to study personnel. Baseline pain was computed as the average score from 2 prerandomization assessments (eligibility session and pretreatment fMRI session).

Neuroimaging Measures

Structural T1 and multiband blood oxygenation level-dependent functional imaging was conducted on a 3-T Siemens Prisma Fit MRI scanner with standard fMRI preprocessing. During fMRI, participants completed (1) an evoked back pain task with a series of randomly ordered trials distending the back to 1 of 4 intensity levels and (2) a

spontaneous pain scan in which participants rested and rated ongoing pain once per minute. Participants rated pain during scanning on a visual analog scale from 0 (no pain) to 100 (worst pain imaginable).

Statistical Analyses

② Intent-to-treat analyses (including all randomized patients) were performed for the primary outcome with a mixed-effects model (*fitlme*, MATLAB 2020a), including 2 group × time interactions (PRT vs placebo × posttreatment vs pretreatment and PRT vs usual care × posttreatment vs pretreatment), covariates for age and sex, and a random intercept per participant. Treatment response rates for 30% or greater reduction in pain, 50% or greater reduction in pain, and a pain-free or nearly pain-free state at posttreatment and 1-year follow-up were based on all randomized patients; those missing data were considered nonresponders. For follow-up time points and secondary outcomes, we calculated ③ Hedges g for the PRT vs placebo and PRT vs usual care comparisons. Follow-up time points were analyzed individually, testing group differences in change from baseline to each time points. The placebo vs usual care comparison will be reported elsewhere.

To investigate psychological treatment mechanisms, we (1) correlated pretreatment to posttreatment changes in pain intensity with pretreatment to posttreatment changes in pain beliefs (TSK-11, PCS, and SOPA-Emotion) within each group and (2) tested pretreatment to posttreatment changes in pain beliefs as mediators of treatment effects on pain at follow-up timepoints (1 through 12 months posttreatment), controlling for baseline pain. PRT vs placebo and PRT vs usual care were tested in separate models. We also tested the reverse: whether pretreatment to posttreatment pain reductions mediated treatment effects on pain beliefs at follow-up, controlling for baseline pain beliefs. Correlational and mediation analyses were not prespecified in the trial protocol.

(This part is omitted)

Discussion

PRT yielded large reductions in CBP intensity relative to open-label placebo and usual care control conditions in a community sample, with nearly two-thirds of randomized patients and 73% of those initiating PRT reporting they were pain-free or nearly pain-free at posttreatment. Large effects of PRT on pain continued at 1-year follow-up. PRT also reduced experimentally evoked back pain and spontaneous pain during fMRI with large effect sizes, and several secondary outcomes (eg, disability and anger) also improved for

PRT relative to the control groups.

PRT targets primary (nociplastic) pain by shifting patients' beliefs about the causes and threat value of pain. It presents pain as a reversible, brain-generated phenomenon not indicative of peripheral pathology, consistent with active inference and constructionist accounts of interoception and pain. PRT builds on and extends existing psychological treatment models. Cognitive-behavioral, acceptance-based, and mindfulness-based interventions typically aim to improve functioning by decreasing pain catastrophizing, enhancing pain coping or acceptance, and promoting engagement in valued life activities. Exposure-based treatments share with PRT an emphasis that painful activities are not injurious, but do not emphasize reappraising pain sensations and reattributing the causes of pain. Some pain neuroscience education interventions present pain in a similar way as PRT, though they typically lack guided exposure and reappraisal exercises.

(This part is omitted)

PRT reduced evoked pain-related activity in aPFC, aMCC, and aIns. The aPFC and adjacent dorsolateral prefrontal cortex (dlPFC) are implicated in the detection and inhibition of pain. aPFC reductions following PRT suggest a potential reduction of pain-related signals or decreased prioritization of pain control. The aMCC and aIns are cortical convergence zones in the construction of negative affect in pain and other domains. Cognitive pain regulation strategies, including mindful acceptance and placebo analgesia, have been found to reduce aMCC and aIns responses to pain, demonstrating parallels between experimental findings and our clinical findings. The aIns reductions in our study were not specific to PRT vs placebo and may reflect processes common to both these interventions.

Source of reference（出典）
Ashar YK, Gordon A, Schubiner H, et al. Effect of Pain Reprocessing Therapy vs Placebo and Usual Care for Patients With Chronic Back Pain: A Randomized Clinical Trial. JAMA Psychiatry. 2022;79(1):13–23. doi:10.1001/jamapsychiatry.2021.2669)
Partial excerpt（一部抜粋）

Ⅲ-1. Explain “Nociplastic pain” in underlined part ①.
下線部①の Nociplastic pain とはどのようなものか説明せよ。

Ⅲ-2. The International Association for the Study of Pain (IASP) defines pain as being of two types in addition to “Nociceptive pain.” Please write these names.
世界疼痛学会（IASP）が定義している疼痛は Nociplastic pain の他に 2 つある。それらの名称を記載せよ。

Ⅲ-3. Describe the pain assessment method used by the authors.
筆者らが用いた疼痛の評価方法を記載せよ。

Ⅲ-4. Describe the pain belief assessment battery used by the authors.
筆者らが用いた疼痛に対する信念の評価バッテリーを記載せよ。

Ⅲ-5. Explain what was evaluated in the fMRI.
fMRI では何を評価したのかを説明せよ。

Ⅲ-6. Explain the advantages and disadvantages of Intent-to-treat analyses in underlined part ②.
下線部②の Intent-to-treat analyses の利点と欠点を説明せよ。

Ⅲ-7. Explain what Hedges *g* in ③ means.
③の Hedges *g* とは何か、説明せよ。

Ⅲ-8. What purpose do the authors describe that pain reduction in PRP serves?
PRP における疼痛減少はどのような目的で起こると筆者は説明しているか。

Ⅲ-9. What intervention do the authors describe as responsible for reducing pain through mindfulness?
マインドフルネスによって痛みが軽減するのはどのような介入によるものだと筆者は説明しているか。

Ⅲ-10. Why did brain activity in the prefrontal cortex decrease after PRP?
PRP 後に前頭前野の脳活動が減少したのはなぜか、説明せよ。