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Bilateral alterations in sensorimotor function and altered sensory strategy in individuals with unilateral chronic ankle instability

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ABSTRACT

Objective: This study aimed to evaluate bilateral sensorimotor function in patients with unilateral CAI. Furthermore, sensory reweighting ability and vestibular modulation were assessed.
Methods: Twenty individuals with unilateral CAI and twenty healthy controls participated in this study. All participants executed ankle proprioception, plantar sensation, unilateral stance, Y balance, motor control test (MCT) and sensory organisation test (SOT) assessments. Proprioception assessment included joint position sense and force sense (FS), and plantar sensation evaluation consisted of light-touch, vibration and two-point discrimination (TPD) thresholds at the heel, head of the first metatarsal (1 MF), base of the fifth metatarsal (5 MF), centre of foot and forefoot. MCT and SOT tests were conducted using NeuroCom Balance Manager System. Except for SOT, all tests evaluated bilateral limbs, and the order of limbs was randomly selected. 2 (group) × 2 (limb) mixed model analyses of variance were performed for outcome measures of unilateral stance, Y balance and MCT, and independent *t*-test was used to analyse the outcomes of SOT between two groups. Mann–Whitney U and Wilcoxon test were applied to examine the differences in plantar sensation between groups and limbs.
Results: For plantar sensation, increased light-touch threshold at heel and 1 MF and the TPD threshold at 1 MF were observed bilaterally in CAI group ($p < 0.05$). No differences were observed in joint position sense (JPS), but bilateral deficit was found in plantarflexor FS with moderate effect size (uninjured side: ES = 0.67; injured side: ES = 0.61) in CAI group. For unilateral stance with eyes closed, moderate postural instability was displayed bilaterally in the anteroposterior direction (uninjured side: ES = 0.71; injured side: ES = 0.86). The delayed latency of MCT with medium-backward translation was also observed in both sides of unilateral CAI (uninjured: ES = 0.74; injured: ES = 0.92). Compared with healthy controls, higher visual reliance was shown moderately in the injured and uninjured sides of unilateral CAI (uninjured: ES = 0.78; injured: ES = 0.91). Sensory analysis of SOT displayed decreased use of visual ($p = 0.001$) and vestibular information ($p < 0.000$) in CAI group.
Conclusion: Unilateral CAI presented impaired plantar sensation and ankle proprioception on both sides. Higher visual reliance, delayed motor response and postural instability under unreliable visual clues were also displayed bilaterally. Except for bilateral sensorimotor alterations, reduced ability of sensory reweighting and fixed sensory strategy also presented in CAI group, but the somatosensory clue still served as the main sensory source in CAI.

1. Introduction

Even for basic tasks, postural control is a plastic process that undergoes continuous evaluation and modification through the integration and analysis of sensory input, efferent motor commands and resulting movements.[1] Multisensory inputs, including somatosensation, vision and vestibulum, play a crucial role in detecting and modifying posture.[2] Somatosensation is the most critical sensation within the sensory system for postural modulation, including tactile, proprioception, pain and temperature.[2,3] The proprioceptive information stemming from peripheral mechanoreceptors in every segment of body, especially in lower extremities, is a key source of somatosensory information and automatic

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responses to unexpected perturbations.[1,3,4] Amongst these, ankle proprioception is the integral component.[4] The ankle–foot complex is the only part of the body interfacing with the ground during movement, and various mechanoreceptors around the ankle–foot complex encode sensory information for modulating posture.[1,5] Ankle injuries usually result in muscle and tendon disruption and damage to inherent receptors, which can adversely alter the quality of proprioceptive information required for postural control.[1,4] When one of the sensory cues is absent or modified due to diseases, postural stability may be impaired, with increased risk for falls and injuries.[6,7] Each sensory source is associated with unique roles that may not be compensated by other sources.[1]

Lateral ankle sprains are one of the most common musculoskeletal injuries in general population, with incidence rates ranging from 2.1/1000 per year to 26.6/1000 per year.[8] However, considering that many injured individuals may not present an emergency department or seek medical care, this is likely a significance underestimation. Approximately 40 % of individuals who experience a lateral ankle sprain may develop into chronic ankle instability (CAI) within one year, presenting with lifelong recurrent ankle sprains, feeling of 'giving way' and instability.[8–10] CAI has been extensively studied and has been found to present with decreased sensorimotor function in the injured limb,[11–16] including proprioceptive deficit,[11,12] postural instability,[16] and decreased muscle strength.[15]

The sensorimotor system, which plays a crucial role in maintaining postural stability, encompasses various sensory, motor and central integration components involved in preserving joint homeostasis during bodily movements.[3] However, an engaging but paradigm-challenging phenomenon observed in recent studies targeting the bilateral assessment of unilateral CAI was that proprioception impairments in patients with CAI could be observed not only in the injured ankle but also in their contralateral ankle.[17,18] This occurrence seems universal in musculoskeletal diseases, as decreased sensory or motor functions also could be observed in the uninjured side in unilateral anterior cruciate rupture,[19] Achilles tendon[20] and acute lateral ankle sprain.[21] These similar findings suggest sensorimotor alterations in the injured limb of unilateral injuries may influence the functions of the contralateral side, which implies a centrally mediated process may be involved in the development of unilateral CAI, which contributes to activity-related functions and disability. However, sensorimotor alterations between different diseases may vary, and the limited and controversial evidence for bilateral assessment of unilateral CAI prompt us to investigate more sensorimotor aspects to confirm the presence of bilateral alteration.

Furthermore, individuals with CAI perform increased visual reliance during single-leg stance.[22] Sensory information is interactive, and diminished input from one sensory source may result in dynamic alterations from multisensory sources.[2,7] Those with CAI reassigned the weight of sensation more on vision, which was seen as a more reliable sensory information for postural stability, because repeated ankle trauma impairs the peripheral mechanoreceptors, resulting in disrupted proprioceptive input.[22] This process is defined as sensory reweighting, which dynamically reassigns sensory clues according to tasks and environment, ensuring an appropriate sensory allocation ratio for postural control.[2] However, such ability appears to be diminished in CAI, and they may not overcome the removal of two sources of sensory information successfully (i.e. constrained somatosensory and visual clues).[23] Excellent sensory reweighting ability is key to response to perturbations and is an integral process for postural modulation.[2] Kim et al.[24] indicated that higher visual reliance occurred bilaterally following acute lateral ankle instability, which let us wonder whether CAI also would display similar visual modulation strategy (i.e. bilateral visual reliance) because CAI develops from the acute lateral ankle sprains, these two diseases share similar risk factors and injury history.[8]

Therefore, the primary purpose of this study was to investigate bilateral sensorimotor characteristics, including bilateral proprioception, plantar sensation, visual modulation and postural control in unilateral CAI. The secondary objective was to assess sensory reweighting ability in unilateral CAI quantitatively and investigate whether vestibular modulation is increased. Based on the existing literature regarding on bilateral assessment of unilateral CAI and sensory integration theory, we hypothesised that unilateral CAI would display bilateral sensorimotor impairments, diminished ability of sensory reweighting and higher vestibular reliance.

2. Methods

2.1. Study design and participants

A cross-sectional study was conducted from May to August 2023 after receiving approval from the Human Ethics Committee of the Shanghai University of Sport (No: 102772023RT073). The sample size was calculated using G-Power software version 3.1.9.7 with effect size $= 0.25$, power $= 0.8$ and $\alpha = 0.05$, which revealed at least 34 participants were required. Finally, 20 patients with unilateral CAI (10 male and 10 female) and 20 (10 male and 10 female) healthy controls matched based on age, sex, height and mass participated in this study.

The age of participants should be between 18 and 30 years old, and the dominant limb must be the right side. The dominant limb was determined as the preferred limb of those who were required to kick a ball.[17] The inclusive criteria recommended by the International Ankle Consortium were used to screen patients with CAI.[25] All individuals with CAI should meet the following criteria: (1) history of at least one significant lateral ankle sprain that happened 12 months before participating in this study, resulting in inflammatory syndrome and at least one day of interruption of desired physical activity; (2) occurrence of the most recent injury more than three months ago; (3) history of previously injured ankle joint 'giving way', recurrent sprains or 'feelings of instability'; (4) self-reported ankle instability confirmed by the Ankle Instability Instrument (AII answer 'yes' to question one along with 'yes' to at least four questions of this questionnaire) and the Cumberland Ankle Instability Tool (CAIT score ≤ 24).[25] Individuals with injuries in both ankles or medial sprains or those who experienced fractures, musculoskeletal disorders, nervous and vestibular system diseases, or other conditions that could affect postural control and sensory function were excluded. Moreover, individuals who participated in any associated intervention were removed. Healthy participants were selected according to the same exclusive criteria used for unilateral CAI and were also removed if they experienced ankle sprains. In addition, the score of CAIT in healthy individuals should be equal to or higher than 28[26]. To exclude the participants with flat feet, navicular drop was used to evaluate the foot condition.

Limbs were categorised into 'injured' and 'uninjured' according to history of ankle sprain in CAI group, whilst the 'matched injured' and 'matched uninjured' limbs were matched to the dominant and nondominant limbs in control group, respectively.[27] Although previous studies have shown no discernible differences between the dominant and the nondominant limbs of healthy subjects,[17,27] both limbs were evaluated in current study.

The purpose of this study was briefly explained to participants. All participants voluntarily participated, and their written informed consent were obtained prior to enrolment. Basic demographic characteristics were noted.

2.2. Procedures and outcome measures

Before the tests, all participants were requested to finish the CAIT and AII questionnaires and provide the total number of lateral ankle sprain occurrences. The severity of ankle sprain determined by diagnosis of clinicians was self-reported. Data were collected during a single session, and most tests assessed bilateral limbs. The evaluations were assigned randomly to participants. The participants and the testers were blind to group allocation.

2.2.1. *Plantar sensation*

All participants were examined prone in a quiet room whilst wearing noise-cancelling headphones. They were further advised to keep their feet suspended outside the bed throughout the whole evaluation. Semmes–Weinstein filament (Baseline, White Plains, New York, NY, USA) with values ranging from 1.65 to 6.65 was applied to evaluate light-touch threshold on soles.[28] Specific sites, including the heel, base of fifth metatarsal (5 MT), head of first metatarsal (1 MT), centre of foot and forefoot, were palpated and marked (Fig. 1).[14,28] Semmes–Weinstein Monofilaments were applied perpendicular to the skin until a 'C' shape was formed and held for 1 s.[28] In addition, all participants were allowed three practice trials on the thenar eminence of the right hand to familiarise with the light-touch sensation and procedure.[28] They were instructed to say 'yes' if they perceived a stimulus on the soles and reported the specific sites verbally or nonverbally (using their hands). To determine the threshold, a 4-2-1 stepping algorithm with 4.74 as the starting point was used.[28] More information about the light-touch evaluation followed previous studies.[13,28]

The instrument of VPT-I (Beijing Huatai Healthcare Technology Co., Ltd, China) was used to assess the vibration threshold at frequency of 50 Hz, which was a reliable frequency that could detect vibratory deficits in CAI.[14,29] The vibrating head was positioned perpendicularly on the designated sites, and vibration intensity was gradually escalated from zero, with a maximum value of 50[29]. Participants were also instructed to say 'yes' if they initially perceived vibration, and the corresponding intensity was recorded as the vibration threshold.[29]

Two-point discrimination (TPD) was assessed by applying Dellon Discriminator (Baseline Discrim-A-Gon Discriminator), which has been demonstrated to be reliable in evaluating TPD.[30,31] The device included two discs, each containing several pins separated by varying distances ranging from 1 mm to 15 mm[30]. A two-point discriminator was applied perpendicularly to the sole with equal pressure exerted on both ends. Participants were required to indicate promptly whether they perceived one or two ends. The measurement commenced at the maximum distance and was gradually reduced until participants could no longer distinguish if two distinct points were presented on their soles.[30] For those who reported perceiving two points as one in two out of three trials, the corresponding distance was recorded as the TPD threshold.[32] The higher values of light-touch, vibration and TPD threshold were referred to as decreased sensitivity.

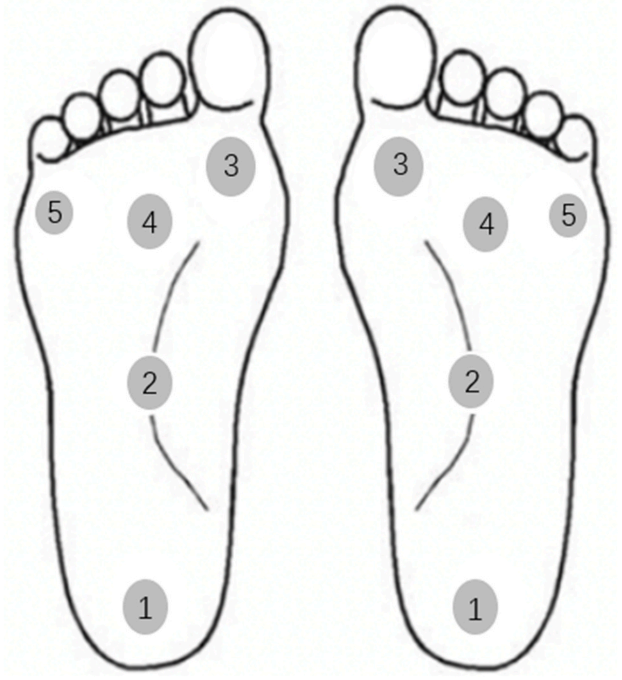

Fig. 1. Test sites of plantar sensation. 1, heel; 2, centre of foot; 3, head of first metatarsal; 4, centre of forefoot; 5, base of fifth metatarsal.

2.2.2. *Proprioception*

The proprioceptive measurements, including ankle joint position sense (JPS) and force sense (FS) assessments, were evaluated by CON-TREX isokinetic dynamometer (PHYSIOMED CON-TREX TP1000, Germany).[33] Passive JPS was evaluated in the following angles: 10° and 15° of inversion, 10° and 15° of eversion, 15° and 30° of plantarflexion, and 15° of dorsiflexion.[11] Initially, participants were required to remove their shoes and socks, and lie on the test bed. For inversion and eversion evaluation, the test limb was positioned at 60° of the knee and hip flexion and 15°of plantarflexion.[17] For the evaluation of plantarflexion and dorsiflexion JPS, the lower extremity was placed at 0° of the ankle, knee and hip.[34] The test foot was positioned on the footplate, and fixation around the ankle was minimised to provide additional sensory clues. Participants were blindfolded and wore earplugs to reduce visual and auditory information. The tester provided participants with detailed instructions regarding the test procedure. Then, the ankle was passively manipulated by the system, moving it from the initial position to one of the target angles randomly. The ankle was maintained at the angle for 10 s. Participants were instructed to memorise the position and repeated this process twice.[33] Subsequently, they were required to hold a button and move their ankle from the neutral position to the target angle. When perceiving that the ankle had reached the target position, the participants pressed a button and the tester recorded the actual angle. Three trials were presented in each target angle with no feedback provided to participants.[17]

Additionally, FS was assessed by reproducing 20 % and 30 % maximum voluntary isometric contraction (MVIC) torque of plantarflexor and dosiflexor.[12] After warming up involving submaximal isometric contractions, the MVIC torque was obtained for each participant. The lower limb was positioned as in a previous study.[35] The participants contracted the dosiflexor and plantarflexor muscles three times separately.[17] Each trial was maintained for 5 s, and a 2-min rest period was allowed between each trial.[17] Then, the participants were required to look at a screen showing the target torque (i.e. 20 % or 30 % MVIC) and keep the target torque for 10 s. The visual feedback was withdrawn, and they were instructed to reproduce the target torque and maintained it for 5 s. This process was also repeated three times, and a 1-min rest period was provided between each trial.[17]

2.2.3. *Postural control*

Four postural tasks were applied to measure the postural stability in static, dynamic and perturbated conditions. Three tasks were implemented by NeuroCom Balance Manager System (Version 9.3; Natus Medical Incorporated, Middleton, WI, USA) which was demonstrated to be a reliable device sampling at 100 Hz to evaluate postural control.[36] Static and perturbed postural control were measured by applying unilateral stance test (US), motor control test (MCT) and sensory organisation test (SOT). Before the tests, the participants removed their shoes and socks, and stood on force plate. For perturbed tasks (i.e. MCT and SOT), practice trials were not offered to evaluate the automatic postural modulation. Participants were told that perturbations would appear in their surroundings but details were not provided to avoid falls or injuries. Y balance test (YBT), which is reliable test of dynamic performance, was implemented to evaluate dynamic balance bilaterally.[37]

In current study, US was applied to assess the static single-leg balance. Participants were instructed to maintain single-leg stance for 10 s with both eyes open and closed, and three successful trials were performed. The test sequence was fixed by the device as follows: left leg support with eyes open, left leg support with eyes closed, right leg support with eyes open and right leg support with eyes closed. During testing, the participants held the contralateral limb at 90° of knee flexion and 30° of hip flexion whilst looking straight in the eyes-open condition with their hands by their sides.

MCT could measure the ability of individuals to produce effective motor responses when facing surface perturbations.[36] MCT is a part of test module in NeuroCome, which assesses the latency of postural modulation with small (2.8°/s), medium (6.0°/s) and large (8.0°/s) forward and backward translations. Each condition should complete three successful trials, and the average latency (ms) of the three trials would be analysed.[36] Higher values mean delayed motor response.

SOT in six conditions was also applied to assess perturbed postural control combined with different sensory interactions, including vision, vestibular and somatosensation. This test can also measure sensory reweighting ability. Test sequence was fixed according to the system: Condition (Cond) 1– eyes open on stable support, Cond 2 – eyes closed on stable support, Cond 3 – sway-referenced visual surround on stable support, Cond 4 – eyes open on sway-referenced support, Cond 5 – eyes closed on sway-referenced support and Cond 6 – sway-referenced visual surround on sway-referenced support.[36] Three successful trials were captured for each condition, and each trail was performed for 20 s. The outcome measures included equilibrium, composite, strategy scores and sensory ratio. Equilibrium and composite scores with higher values indicate better balance; a value closer to 100 for strategy score indicates a preference for ankle strategy, and the converse is true for the hip strategy.[36] Additionally, the sensory ratio provided insights into the use of vision (VIS), vestibular (VEST) and somatosensation (SOM) in individuals.[36] SOM refers to the ratio of condition 2 to 1 (i.e. $\frac{\text{Cond2}}{\text{Cond1}}$); a low value indicates reduced ability to use somatosensation to resist instability.[38] VIS represents the ratio of conditions 4 and 1(i.e. $\frac{\text{Cond4}}{\text{Cond1}}$), which indicates the ability to apply vision to maintain posture.[38] VEST presents the ratio of conditions 5 to 1 (i.e. $\frac{\text{Cond5}}{\text{Cond1}}$), which indicates the ability to apply vestibulum to resist perturbation.[38] Preference (PREF) is the ratio of the sum of conditions 3 and 6 to the sum of condition 2 and 5 (i.e. $\frac{\text{Cond3+Cond6}}{\text{Cond2+Cond5}}$); a low value means increased visual reliance.[38]

YBT required participants to perform six practice trails in the anterior (A), posteromedial (PM) and posterolateral (PL) directions, followed by three formal trials in each direction and limb.[37] The trial order was counterbalanced and randomised according to the directions and limbs. The average of normalised reach distance was calculated for analysis. The reach distance was normalised by participants' limb length (measured from anterior superior iliac spine to ipsilateral medial malleolus) and multiplied by 100 to calculate the percentage.[39] A higher percentage means better dynamic balance.

2.3. Data reduction

For JPS and FS, the absolute error (AE) of JPS and normalised AE of FS (NAEFS) were calculated for analysis[33] based on the following formulas:

$$\mathrm{AE} = \frac{\sum_{i=1}^{3} |\mathrm{a}_i - \mathrm{a}|}{3} \tag{1}$$

$$\mathrm{NAEFS} = \frac{\mathrm{AE}}{20\%\mathrm{MVIC}} \text{ or } \frac{\mathrm{AE}}{30\%\mathrm{MVIC}} \tag{2}$$

Notes: $\mathrm{a_i}$ represents the actual angle, and a represents the target angle.

In US and SOT, the raw data of the force plate was extracted to calculate the velocity of centre of pressure (COPV) based on a previous study.[40]

$$\text{Anteroposterior COPV} = \frac{1}{\mathrm{T}} \sum_{\mathrm{n}=1}^{\mathrm{N}-1} |\mathrm{AP}[\mathrm{n}+1] - \mathrm{AP}[\mathrm{n}]| \tag{3}$$

$$\text{Mediolateral COPV} = \frac{1}{\mathrm{T}} \sum_{\mathrm{n}=1}^{\mathrm{N}-1} |\mathrm{ML}[\mathrm{n}+1] - \mathrm{ML}[\mathrm{n}]| \tag{4}$$

Notes: T is the period (T = 10 for US and T = 20 for SOT). N is the number of data points (N = 1000 for US and N = 2000 for SOT).

Additionally, sensory modulation was determined by using the raw data of force plate. The percentage of sensory modulation was calculated based on previous studies.[22,24] For visual reliance, raw data of US were used to calculate visual modulation in single-leg stance, data of SOT in Cond 1 and 2 were applied to measure the visual modulation in double-leg stance, and Cond 1 and 3 were computed to estimate visual modulation in visual perturbation. Data in Cond 1 or 2 and 4 or 5 were used to compute vestibular modulation in eyes-open and eyes-close conditions. The percentage represents sensory reliance when the sensory clues are removed or unreliable, and a higher value indicates greater reliance.

2.4. Statistical analysis

The acquired data were analysed using SPSS 25.0 (IBM Corp., Armonk, NY, United States). The data of plantar sensation performed as median and quartiles. For postural control, proprioception and sensory modulation, the data were presented as mean and standard deviation (SD). Shapiro–Wilk test was applied to evaluate the normality of distribution. AE and NAEFS, COPV of US, normalised reach distance of YBT, latency of MCT and visual modulation in single-leg stance were analysed by a series of 2 (group) × 2 (limb) mixed-model ANOVA with repeated measures on the limb variable. Bonferroni adjustment was used to make post hoc comparisons. Independent *t*-test was used to compare the differences in equilibrium, composite and strategy scores, visual modulation in double-leg stance and vestibular modulation between two groups. Mann–Whitney *U* test was used to compare the difference in plantar sensation between two groups. Wilcoxon test was applied to determine the difference between two soles of plantar sensation. Cohen's d estimate was used to estimate the effect size (ES) with a 95 % confidence interval (CI) and interpreted as small ($\mathrm{ES} \geq 0.2$), moderate ($\mathrm{ES} \geq 0.5$) and large ($\mathrm{ES} \geq 0.8$)[24, 41]. The alpha level was set at $p < 0.05$.

3. Results

3.1. Demographic data

No differences were observed between CAI and control group in age, height, mass or BMI ($p > 0.05$, Table 1). The navicular drop test also did not reveal any difference between limbs and groups.

3.2. Plantar sensation

For light-touch threshold, no differences were observed at the centre of the foot ($p > 0.05$, Table 2). Decreased light-touch sensitivity on both soles was observed in CAI group compared with control group at heel (uninjured: $p = 0.026$; injured: $p < 0.001$) and 1 MF (uninjured: $p = 0.001$; injured: $p < 0.001$). Moreover, the injured side of CAI showed

Table 1
Demographic characteristics.

Variable	CAI	Control	*p*
Age	21.45 ± 2.06	22.20 ± 2.61	0.320
Height	172.89 ± 10.54	171.44 ± 9.03	0.642
Mass	61.51 ± 12.26	61.82 ± 11.39	0.935
BMI	20.37 ± 2.30	20.83 ± 1.86	0.498
CAIT	14.30 ± 3.48	28.9 ± 0.91	**< 0.001**
AII	6.82 ± 1.15	0.5 ± 0.69	**< 0.001**
No. of previous ankle sprains	2.9 ± 2.00	NA	
Severity of ankle sprain severe	0	NA	
moderate	14	NA	
mild	6	NA	
Time since last sprain	13.00 ± 11.23	NA	

AII, ankle instability instrument; BMI, body mass index; CAI, chronic ankle instability; CAIT, Cumberland ankle instability tool; NA, not applicable.

Table 2
Plantar sensation thresholds [median (first, third quartile)] for healthy control and unilateral CAI groups.

Variable	Uninjured	Injured	p
Light touch at heel			
CAI	3.96(3.84, 4.17)	4.13(4.07, 4.31)	**0.014**
Control	3.61(3.22, 3.84)	3.22(3.22, 3.84)	0.168
p	**0.026**	**< 0.001**	
Light touch at 1 MF			
CAI	3.84(3.22, 3.84)	3.96(3.22, 4.08)	**0.041**
Control	3.22(2.54, 3.22)	3.22(3.22, 3.84)	0.247
p	**0.001**	**<0.001**	
Light touch at 5 MF			
CAI	3.84(3.22, 4.56)	4.07(3.22,4.28)	**0.036**
Control	3.22(3.22, 3.84)	3.22(3.22, 3.22)	0.172
p	0.072	**0.001**	
Light touch at the centre of forefoot			
CAI	3.61(3.22, 3.84)	3.84(3.32, 4.31)	**0.001**
Control	3.22(2.83, 3.78)	3.22(3.22, 3.69)	0.917
p	0.308	**0.001**	
Light touch at the centre of foot			
CAI	3.22(2.83, 3.51)	3.22(3.22, 3.61)	0.295
Control	3.22(2.83, 3.22)	3.22(2.83, 3.69)	0.109
p	0.345	0.446	
Vibration at heel			
CAI	5.50(5.00, 7.00)	7.00 (6.00, 8.75)	0.067
Control	5.00 (5.00, 7.00)	6.00 (4.00, 7.75)	0.295
p	0.697	0.098	
Vibration at 1 MF			
CAI	6.50(5.00, 7.75)	7.00(6.00, 8.00)	**0.025**
Control	5.50(4.25, 7.00)	5.50(4.00, 7.75)	0.860
p	0.226	**0.043**	
Vibration at 5 MF			
FAI	6.00(5.00, 6.5)	6.00(5.00, 6.00)	0.417
Control	5.00(3.00, 6.00)	5.00(3.25, 6.75)	0.858
p	0.242	0.205	
Vibration at the centre of forefoot			
CAI	5.00(4.00, 6.75)	5.00(5.00, 6.75)	0.977
Control	5.00(4.00, 7.00)	5.00(4.00, 7.00)	0.253
p	0.659	0.515	
Vibration at the centre of foot			
CAI	6.00(5.00, 7.00)	6.00(5.00, 7.75)	0.724
Control	5.00 (4.00, 7.00)	5.50(4.00, 6.75)	0.582
p	0.326	0.137	
TPD at heel (mm)			
CAI	12.00(11.00, 13.00)	13.00(10.25, 15.00)	0.405
Control	11.00(10.25, 12.75)	12.00(10.25, 13.00)	0.781
p	0.258	0.119	
TPD at 1 MF (mm)			
CAI	10.00(9.00, 12.00)	10.50(9.00, 13.75)	0.306
Control	8.50(7.00, 11.00)	7.50(6.00, 9.75)	0.131
p	**0.048**	**0.001**	
TPD at 5 MF (mm)			
CAI	10.00(7.50, 11.00)	11.50(10.00, 13.00)	0.025
Control	9.00(7.00, 11.00)	10.00(7.25, 12.75)	0.120
p	0.293	0.147	
TPD at the centre of forefoot (mm)			
CAI	12.50(9.25, 13.00)	13.00(12.00, 14.75)	**0.019**
Control	12.50(10.00, 13.00)	12.00(10.25, 14.00)	0.915
p	1.000	0.081	
TPD at the centre of foot (mm)			
CAI	14.00(12.25, 15.00)	13.00(12.25, 15.00)	0.248
Control	12.50(8.25, 14.75)	13.00(10.25, 14.00)	0.440
p	0.070	0.151	

CAI, chronic ankle instability; 1 MF, head of the first metatarsal; 5 MF, base of the fifth metatarsal; TPD, two-point discrimination.

greater thresholds at 5 MF ($p = 0.001$) and centre of forefoot ($p = 0.001$) compared with the matched injured side of healthy control. Thresholds of the injured side were higher than those of the uninjured side at heel ($p = 0.014$), 1 MF ($p = 0.041$), 5 MF ($p = 0.036$) and centre of forefoot ($p = 0.001$) in CAI group.

For vibration threshold, no significant differences were presented at heel, 5 MF, centre of foot and forefoot ($p > 0.05$). Only reduced vibration sensitivity was observed in the injured side of CAI compared with the matched injured side of healthy control ($p = 0.043$) and the uninjured side of CAI ($p = 0.025$). Compared with healthy controls, increased TPD thresholds on both soles of CAI were revealed at 1 MF (uninjured: $p = 0.048$; injured: $p = 0.001$). Additionally, the injured side of CAI showed significantly greater TPD threshold at the centre of forefoot ($p = 0.019$) than the uninjured side of CAI.

3.3. Proprioception

For passive AE and NAEFS, no significant group-by-limb interactions were observed for all measures ($p > 0.05$). Only group main effects were found in 20 % plantarflexor, 20 % and 30 % dorsiflexor NAEFS (20 % plantarflexor: F = 6.269, $p = 0.017$; 20 % dorsiflexor: F = 4.323, $p = 0.044$; 30 % dorsiflexor: F = 4.878, $p = 0.033$; Table 3), which meant no side-to-side differences in either CAI group or control group, and the groups were different after pooling data from both limbs. The group differences were moderate (ES = 0.57 to 0.68), and associated 95 % CIs did not cross zero. Only 20 % plantarflexor NAEFS displayed bilateral decrease with moderate ES (uninjured: ES = 0.67, 95 % CI = 0.03 to 1.30; injured: ES = 0.61, 95 % CI = 0.03 to 1.30).

3.4. Postural control

For US, no significant group-by-limb interactions were observed, but the group main effects were shown in eyes-closed condition (AP: F = 7.242, $p = 0.011$; ML: F = 5.125, $p = 0.029$; Table 4). Pairwise comparisons revealed CAI group on average showed larger COPV in the AP (ES = 0.79, 95 % CI = 0.33 to 1.24) and ML (ES = 0.65, 95 % CI = 0.20 to 1.10) directions compared with the control group. However, the AP direction showed larger velocity on both sides (uninjured: ES = 0.71, 95 % CI = 0.06 to 1.34; injured: ES = 0.86, 95 % CI = 0.20 to 1.51).

Additionally, significant group-by-limb interactions were found in the anterior direction (F = 4.312, $p = 0.045$) and the composite score (F = 6.016, $p = 0.019$) of YBT. Post hoc analysis showed the normalised reach distance of the injured side was significantly smaller than that of the uninjured side (F = 7.583, $p = 0.009$) and the matched injured side (F = 4.816, $p = 0.034$) in the anterior direction. The injured side showed reduced reach distance of composite score compared with the matched injured side (F = 9.074, $p = 0.005$). Group main effects were demonstrated in the PL (F = 5.481, $p = 0.025$) and PM (F = 4.500, $p = 0.040$) directions and the composite score (F = 6.151, $p = 0.018$). The group differences were moderate (ES = 0.65 to 0.78) with 95 % CIs not crossing zero.

No significant group-by-limb interactions were noted for MCT. Group main effect was observed in medium-backward translation (F = 9.263, $p = 0.004$), which indicated the CAI group on average presented significantly delayed latency when resisting postural perturbation compared with healthy controls. The group difference was moderate (ES = 0.72, 95 % CI = 0.26 to 1.17), and 95 % CI also did not cross zero. Both sides showed moderate ES without crossing 95 % CIs (uninjured: ES = 0.74, 95 % CI = 0.09 to 1.38; injured: ES = 0.92, 95 % CI = 0.26 to 1.56). Limb main effects displayed in medium-forward (F = 6.264, $p = 0.017$, ES = 0.40, 95 % CI = 0.07 to 0.72) and large-forward (F = 6.135, $p = 0.018$, ES = 0.40, 95 % CI = 0.07 to 0.72) translation, which implies the uninjured side was different with injured side after pooling data from both groups. The composite score of MCT, which pooled bilateral scores based on the system calculation, also showed the CAI group demonstrated significantly later motor response ($p = 0.015$, ES = 0.81, 95 % CI = 0.16 to 1.45).

For equilibrium score of SOT (Fig. 2a), the scores of CAI group significantly decreased in Cond 3 ($p = 0.008$, ES = −0.89, 95 % CI = −1.53 to −0.23), 4 ($p = 0.003$, ES = −1.02, 95 % CI = −1.68 to −0.36), 5 ($p < 0.001$, ES = −1.08, 95 % CI = −1.74, −0.41), 6 ($p = 0.001$, ES = −1.08, 95 % CI = −1.74 to −0.41) and composite ($p = 0.006$, ES = −0.91, 95 % CI = −1.56 to −0.25) compared with healthy controls, which indicated patients with unilateral CAI presented postural

Table 3
Normalised absolute error of force sense.

Variable	Group-by-Limb Interaction	Group main effect	Side	CAI	Control	Effect size (95 % CI)
20 % plantarflexor	F = 0.115 p = 0.736	F = 6.269 p = **0.017**	Uninjured	0.22 ± 0.09	0.16 ± 0.09	0.67 (0.03, 1.30)
			Injured	0.22 ± 0.12	0.15 ± 0.11	0.61(0.03, 1.30)
			Combined	0.22 ± 0.10	0.15 ± 0.10	0.68(0.22, 1.12)
30 % plantarflexor	F = 1.166 p = 0.287	F = 0.030 p = 0.864	Uninjured	0.19 ± 0.08	0.16 ± 0.09	0.39(-0.24, 1.01)
			Injured	0.20 ± 0.13	0.22 ± 0.20	−0.14(-0.76, 0.49)
			Combined	0.20 ± 0.11	0.19 ± 0.15	0.04(-0.40, 0.48)
20 % dorsiflexor	F = 1.391 p = 0.246	F = 4.323 p = **0.044**	Uninjured	0.31 ± 0.27	0.20 ± 0.17	0.49(-0.14, 1.12)
			Injured	0.42 ± 0.39	0.22 ± 0.20	0.67(0.03, 1.30)
			Combined	0.37 ± 0.34	0.21 ± 0.18	0.59 (0.14, 1.03)
30 % dorsiflexor	F = 0.346 p = 0.560	F = 4.878 p = **0.033**	Uninjured	0.34 ± 0.32	0.22 ± 0.18	0.45(-0.18, 1.07)
			Injured	0.37 ± 0.31	0.20 ± 0.18	0.69(0.05, 1.33)
			Combined	0.36 ± 0.31	0.21 ± 0.18	0.57(0.12, 1.02)

CAI, chronic ankle instability; CI, confidence interval.

Table 4
Velocity of centre of pressure with eyes closed during single-leg stance, normalised reach distance of Y balance test and latency of motor control test.

Variable	Group-by-Limb Interaction	Group main effect	Side	CAI	Control	Effect size (95 % CI)
Unilateral stance						
AP COPV with eyes close (cm/s)	F = 0.123 p = 0.728	F = 7.242 p = **0.011**	Uninjured	6.38 ± 1.85	5.15 ± 1.63	0.71(0.06, 1.34)
			Injured	6.56 ± 1.72	5.18 ± 1.47	0.86(0.20, 1.51)
			Combined	6.47 ± 1.77	5.17 ± 1.53	0.79 (0.33, 1.24)
ML COPV with eyes close (cm/s)	F = 0.428 p = 0.517	F = 5.125 p = **0.029**	Uninjured	5.88 ± 1.57	5.18 ± 1.44	0.46(-0.17, 1.09)
			Injured	5.80 ± 1.06	4.87 ± 0.87	0.96(0.20, 1.61)
			Combined	5.84 ± 1.32	5.03 ± 1.19	0.65(0.20, 1,10)
Y balance test						
A reach distance (%)	F = 4.312 p = **0.045**	F = 3.395 p = 0.073	Uninjured	65.28 ± 7.83	67.99 ± 5.72	−0.40(-1.02, 0.23)
			Injured	62.48 ± 8.80	68.17 ± 7.57	−0.69(-1.33, −0.05)
			Combined	63.88 ± 8.34	68.08 ± 6.62	−0.56(-1.00, −0.11)
PL reach distance (%)	F = 2.206 p = 0.146	F = 5.481 p = **0.025**	Uninjured	106.94 ± 12.51	113.52 ± 10.77	−0.64(-1.27, 0.01)
			Injured	105.93 ± 12.67	114.76 ± 8.94	−0.81(-1.45, −0.15)
			Combined	106.44 ± 12.44	114.14 ± 8.25	−0.73(-1.18, −0.28)
PM reach distance (%)	F = 1.321 p = 0.258	F = 4.500 p = **0.040**	Uninjured	108.17 ± 12.49	114.36 ± 13.54	−0.48(-1.10, 0.16)
			Injured	107.42 ± 10.98	116.18 ± 9.29	−0.86(-1.51, −0.21)
			Combined	107.79 ± 11.62	115.27 ± 11.50	−0.65(-1.10, −0.20)
Composite reach distance (%)	F = 6.016 p = **0.019**	F = 6.151 p = **0.018**	Uninjured	93.46 ± 10.11	98.62 ± 6.90	−0.60(-1.23, 0.04)
			Injured	91.94 ± 9.52	99.70 ± 6.49	−0.95(-1.60, −0.29)
			Combined	92.70 ± 9.73	99.16 ± 6.64	−0.78(-1.23, −0.32)
Motor control test						
Small-forward latency (ms)	F = 3.313 p = 0.077	F = 0.399 p = 0.531	Uninjured	158.00 ± 34.72	144.00 ± 14.29	0.53(-0.11, 1.16)
			Injured	139.50 ± 38.18	144.00 ± 20.62	−0.15(-0.77, 0.48)
			Combined	148.75 ± 37.22	144.00 ± 17.51	0.16(-0.28, 0.60)
Medium-forward latency (ms)	F = 0.850 p = 0.362	F = 2.937 p = 0.095	Uninjured	148.50 ± 17.85	140.50 ± 11.46	0.53(-0.10, 1.16)
			Injured	142.00 ± 10.56	137.50 ± 10.70	0.42(-0.21, 1.05)
			Combined	145.25 ± 14.85	139.00 ± 11.05	0.48(0.03, 0.92)
Large-forward latency (ms)	F = 0.170 p = 0.682	F = 0.305 p = 0.584	Uninjured	132.00 ± 12.40	129.50 ± 12.76	0.20(-0.42, 0.82)
			Injured	128.50 ± 10.40	127.00 ± 12.61	0.13(-0.49, 0.75)
			Combined	130.25 ± 11.43	128.25 ± 12.59	0.17(-0.27, 0.61)
Small-backward latency (ms)	F = 0.760 p = 0.389	F = 0.979 p = 0.329	Uninjured	144.00 ± 19.57	138.50 ± 10.40	0.35(-0.28, 0.97)
			Injured	138.00 ± 11.52	136.50 ± 9.33	0.14(-0.48, 0.76)
			Combined	141.00 ± 16.14	137.50 ± 9.81	0.26(-0.18, 0.70)
Medium-backward latency (ms)	F = 1.368 p = 0.250	F = 9.263 p = **0.004**	Uninjured	143.00 ± 31.64	126.00 ± 7.54	0.74(0.09, 1.38)
			Injured	131.50 ± 10.40	123.00 ± 8.01	0.92(0.26, 1.56)
			Combined	137.25 ± 23.96	124.50 ± 7.83	0.72(0.26, 1.17)
Large-backward latency (ms)	F = 0.672 p = 0.418	F = 1.343 p = 0.254	Uninjured	128.00 ± 11.52	125.50 ± 8.26	0.25(-0.37, 0.87)
			Injured	126.50 ± 14.24	121.50 ± 10.40	0.40(-0.23, 1.03)
			Combined	127.25 ± 12.81	123.50 ± 9.49	0.33(-0.11, 0.77)

A, anterior; AP, anteroposterior, CAI, chronic ankle instability; CI, confidence interval; ML, mediolateral.

instability during sensory perturbations. The strategy scores (Fig. 2b) demonstrated that in Cond 3 ($p = 0.003$, ES = −0.99, 95 % CI = −1.64 to −0.33), 5 ($p = 0.014$, ES = −0.82, 95 % CI = −1.46 to −0.17) and 6 ($p = 0.032$, ES = −0.71, 95 % CI = −1.34 to −0.06), the CAI group preferred to use hip strategy to maintain balance.

3.5. Sensory strategy analysis

The sensory ratio (Fig. 3a) revealed the CAI group presented decreased ability using vision ($p = 0.001$, ES = −1.12, 95 % CI = −1.78 to −0.45) and vestibular ($p < 0.001$, ES = −1.24, 95 % CI = −1.91 to −0.55). Moreover, value of PREF significantly differed between CAI group and control group ($p = 0.010$, ES = −0.86, 95 % CI = −1.50 to −0.20). The results indicated that CAI presented decreased ability of sensory reweighting and increased visual reliance.

There was no significant difference was observed in visual modulation of double-leg stance (eyes open and eyes closed) between two groups. However, a significant difference was observed in the visual modulation in the AP direction ($p = 0.022$, ES = 0.76, 95 % CI = 0.11 to 1.40; Fig. 3b) when vision input was perturbed, which meant patients

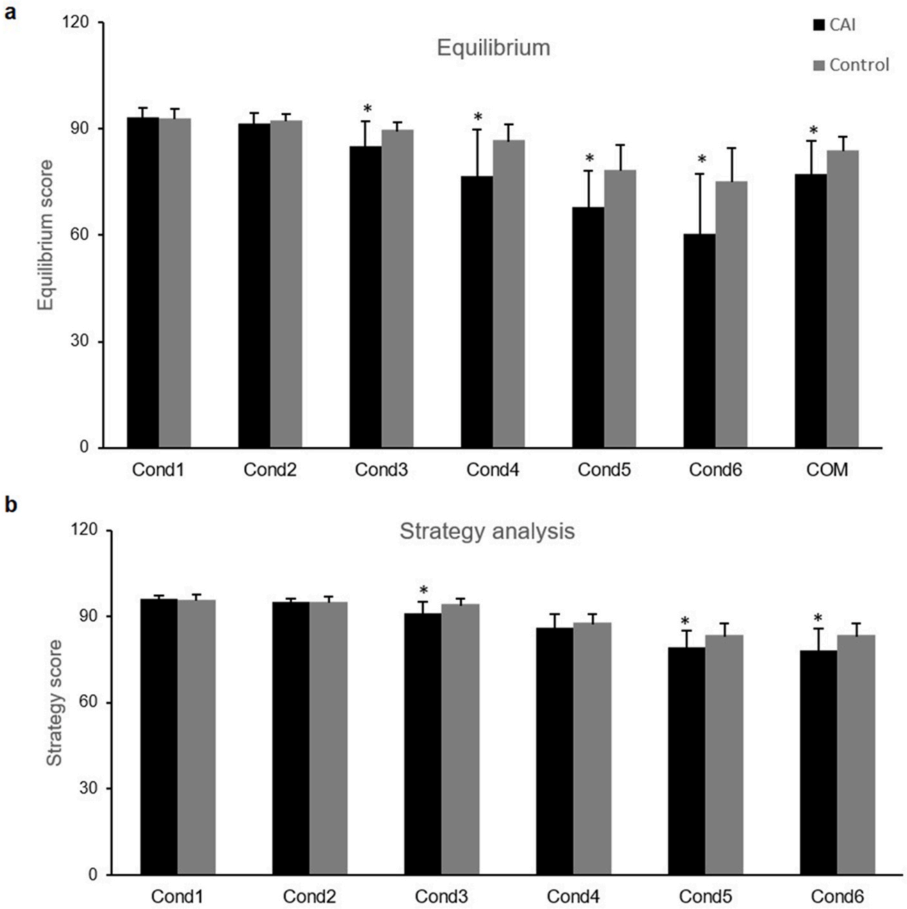

Fig. 2. Equilibrium and strategy score of sensory organisation test. a, Equilibrium score; b, Strategy score. CAI, chronic ankle instability; Cond, condition; COM, composite. * denotes significantly different than control group.

with CAI may present reduced ability to ignore perturbed visual information. The increased vestibular modulation was also found in CAI group during eyes-open (AP: $p = 0.035$, ES = 0.69, 95 % CI = 0.05 to 1.33; ML: $p = 0.026$, ES = 0.74, 95 % CI = 0.09 to 1.37; Fig. 3c) and eyes-closed conditions (AP: $p = 0.012$, ES = 0.84, 95 % CI = 0.18 to 1.48; Fig. 3d).

For visual modulation of US, no group-by-limb interactions were observed. Group main effect was observed in the AP direction (F = 9.192, $p = 0.004$; Table 5). The visual modulation on the injured side did not differ from that on the uninjured side in individuals with unilateral CAI. After pooling the data from both limbs, the CAI group showed significantly higher decline in the single-leg stance in the AP direction than control group, implying greater reliance on visual information (ES = 0.85, 95 % CI = 0.39 to 1.31). The bilateral sides of CAI group indicated higher visual reliance in the AP direction (uninjured: ES = 0.78, 95 % CI = 0.13 to 1.42; injured: ES = 0.91, 95 % CI = 0.26 to 1.56).

4. Discussion

The main purpose of current study was to evaluate the bilateral sensorimotor function related to unilateral CAI. A series of evaluations on unilateral CAI indicated higher light-touch threshold at the heel and 1 MF, higher TPD threshold at 1 MF, reduced plantarflexor FS, increased postural instability during US with eyes closed, delayed latency in the medium-backward translation of MCT and higher visual reliance presented on not only the injured side of patients with unilateral CAI but also the uninjured side. Furthermore, restricted ability to reweight sensory information and resist visual perturbation was observed in CAI group.

The increased bilateral FS errors in the CAI group was supported by a previous study.[17] Similarly, no differences in JPS were observed in that study. Although JPS and FS shared the same receptors (i.e. muscle spindles and GTOs) in some specific areas, the muscle spindle, which is mainly responsible for conveying FS information, can be stimulated in entire range of motion.[3] In contrast to FS, JPS is unlikely to be conveyed by midrange of motion at joint capsular.[3] To avoid excessive range of motion that may cause injuries to participants, the midrange of motion as the test angle, which may lead to the inability to detect JPS errors, was selected in this work. Moreover, the test method of JPS may influence the possibility of evaluating the errors in JPS. Due to the settings of instrument, passive JPS was used in the current study, which was in contrast to active JPS activated by a wider range of receptors.[3] The test of JPS was performed under blindfolded condition, and the ankle fixation might have provided additional sensory cues regarding the ankle position.[5] Nevertheless, errors were slightly (nonsignificant) larger in patients with unilateral CAI than in healthy individuals.

Decreased sensitivity of plantar sensation also was observed bilaterally, which provided another insight into the sensory system of individuals with unilateral CAI. Previous research revealed reduced plantar light touch on the injured side of patients with CAI at the heel, 1 MF and 5 MF indicated constraints in CAI were not limited to the ankle or peripheral afferent.[13,14,28] The test sites on soles received the sensory innervations from the sural nerve and branches of the tibial nerve.[42] Although the ankle injuries had limited ability to affect the plantar sensation, the superficial peroneal nerve conducted the sensory information of lateral ankle and dorsum of the foot and tibial nerve, which transmitted plantar sensation originating from the sciatic nerve.[43,44] Impaired proprioceptors may lead to changes in transmission of sensory

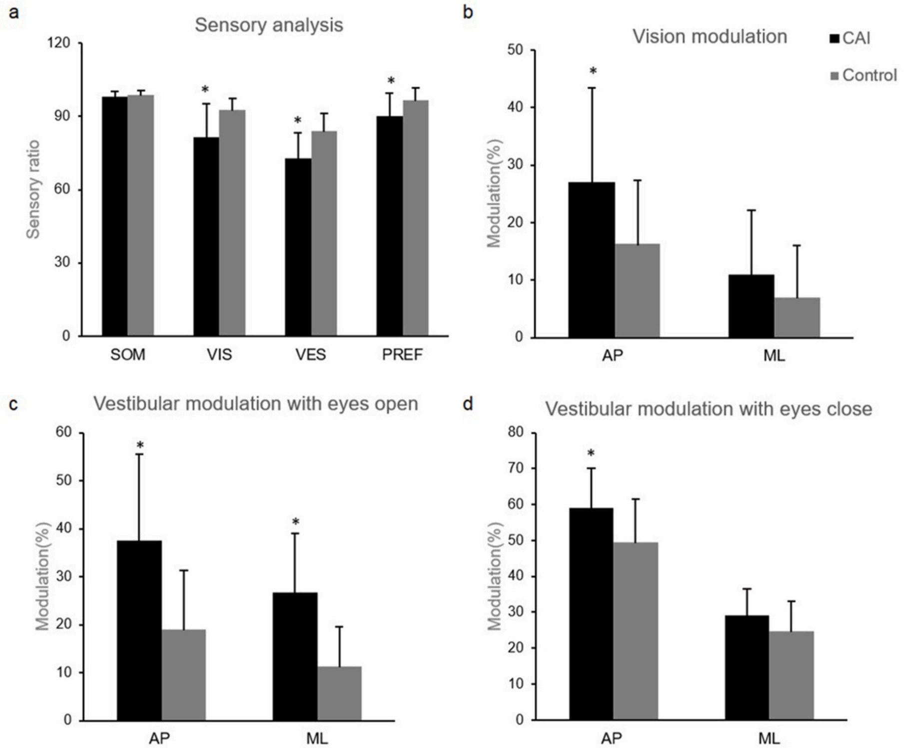

Fig. 3. Sensory ratio and sensory modulation. a, sensory ratio of sensory organisation test; b, visual modulation between conditions 1 and 3 of sensory organisation test; c, vestibular modulation between condition 1 and 4 of sensory organisation test; d, vestibular modulation between conditions 2 and 5 of sensory organisation test. AP, anteroposterior; CAI, chronic ankle instability; ML, mediolateral; PREF, preference; SOM, somatosensory; VES, vestibular; VIS, vision.

Table 5
Visual modulation for unilateral stance.

Variable	Group-by-Limb Interaction	Group main effect	Side	CAI	Control	Effect size (95 % CI)
AP (%)	F = 0.011 *p* = 0.917	F = 9.192 *p* = **0.004**	Uninjured	53.47 ± 11.53	42.86 ± 15.29	0.78 (0.13, 1.42)
			Injured	54.96 ± 9.39	43.95 ± 14.24	0.91 (0.26, 1.56)
			Combined	54.22 ± 10.40	43.41 ± 14.60	0.85 (0.39, 1.31)
ML (%)	F = 1.401 *p* = 0.244	F = 3.744 *p* = 0.060	Uninjured	48.24 ± 12.06	44.20 ± 16.20	0.28 (-0.34, 0.90)
			Injured	51.45 ± 10.82	42.17 ± 12.05	0.81 (0.16, 1.45)
			Combined	49.85 ± 11.42	43.19 ± 14.13	0.52 (0.07, 0.96)

CAI, chronic ankle instability; CI, confidence interval; AP, anteroposterior; ML, mediolateral.

information by sciatic nerve, which may influence the integration and reorganisation of sensory information in spinal or cortical levels,[45] resulting in alterations in behaviour of normal peripheral afferent receptors in nerve branches and may produce a process of bilateral sensory plasticity.[2,3,46] Zhang et al.[46] investigated sensory nerve function around anterior talofibular ligament area in both ankles and found individuals with unilateral CAI presented diminished ability to transmit pain signals bilaterally (both fast and slow pain signals), which further supported our finding of bilateral decreased sensitivity of plantar sensation, receiving sensory innervation from branches of tibial nerve. Although the threshold values of light touch indicated CAI does not lose protective sensation (5.07–6.65),[28] subtle deficits may contribute to sensorimotor impairments and recurrent ankle sprains.[5] Similarly, decreased TPD at 1 MF was observed bilaterally. Although no bilateral deficits were found in plantar vibration, which is another kind of sensation related to somatosensation, the diminished vibration presented on the injured side at 1 MF. Hoch et al.[14] found that the CAI group displayed diminished vibration at the heel, 1 MF and 5 MF at 10, 25 and 50 Hz frequencies compared with healthy controls. The dissimilar findings may be attributed to different devices used in evaluating the vibration threshold.

Moreover, reduced postural control in static and dynamic conditions were observed in CAI group. This study was the first to evaluate the bilateral latency of automatic motor response in unilateral CAI. The delayed motor response in MCT implies patients with unilateral CAI presented reduced ability to produce effective motor responses to resist passive perturbation. The uninjured side displayed later response in medium-forward (ES = 0.51, 95%CI = 0.04 to 0.97) and large-forward (ES = 0.47, 95%CI = 0.00 to 0.93) translation compared with the injured side, which may be attributed to learning effect and protective response. Although NeuroCom system has been demonstrated to be a reliable and valid instrument for evaluating the postural control, small learning effects were evident.[36] As the learning effect resulted in the feedforward response, individuals with CAI may protectively preactivate muscles on the injured side in response to potential perturbation.[47,48] Lin et al.[48] reported the biceps femoris of the injured side activated earlier than the control group regardless of perturbed or unperturbed walking, and the uninjured side activated later during perturbed walking. Similarly, Sousa et al.[47] found the tibialis anterior and soleus of the uninjured side displayed later-onset activation.

Although the US and MCT displayed diminished postural control on both sides, only those on the injured side were reduced in YBT. Previous studies on YBT in patients with unilateral CAI found bilateral impairments,[49,50] but controversial evidence remains.[51,52] A possible explanation may be visual compensation.[22] In US and MCT, bilateral alterations only were observed in conditions where visual cues were unreliable (i.e. US with eyes closed and MCT with backward translation). In YBT, participants were allowed to use visual cues to finish dynamic tasks, so visual input may compensate for the minor deficits on the uninjured side. The proprioceptive function of modification in feedforward was only partly compensated by visual clues.[1] Song et al.[22] demonstrated that the injured side of CAI presented higher visual use during single-leg stance, and the visual modulation in current study also suggested CAI group bilaterally showed higher visual reliance during US. However, this hypothesis needs to be further clarified by YBT assessment in blindfolded condition. Although the uninjured side showed 95 % CI of ES in YBT crossing zero, the reached distance was smaller in the uninjured side compared with control group.

Based on the sensory analysis of SOT, the diminished somatosensory input from the ankle–foot complex is unable to limit unilateral CAI's use of somatosensory information, as the sensory ratio displayed a similar ratio in SOM between unilateral CAI (98.10 ± 2.22) and healthy individuals (98.9 ± 1.55). Somatosensory information still serves as the primary sensory source of unilateral CAI during postural control, but the use of visual and vestibular cues decreased, which suggests an inability to reweight sensory information.[22] The strategy analysis revealed patients with CAI prefer to use hip strategy for postural adjustment, which implies the postural control strategy of CAI may change, and those with CAI prefer to use somatosensory information in hip or other segments, especially when visual clue is unreliable. The hip modulation strategy only became important in CAI group when visual clue was perturbed (Cond 3, 5 and 6). After resecting the posterior or medial knee articular nerves of cats without altering mechanical stability, in addition to spinal-level motor alterations, changes in supraspinal motor program controlling voluntary movements were observed, and postural adjustments initiated from visual and vestibular sources were altered.[1,53] Although somatosensory constraint in the ankle–foot complex is unable to influence the use of proprioception during double-leg balance in CAI, the minor constraint may play a potential influence on the integration of central nervous system and reweight vestibular and visual clues.

As the difficulty of task increased, the CAI group presented higher reliance on visual and vestibular clues. Visual modulation in the static double-leg stance displayed 8.66 % decline in the AP direction when vision was removed, whereas a 54.22 % decline was noted during US. Vestibular modulation in eyes-open condition decreased by 26.79 % in the AP direction, whereas it was reduced by 49.41 % when vision was removed. Although the proprioceptive information provides motor system with multisegment positional information, the task of determining the muscle tone required for movement becomes extremely complex in more difficult postural tasks involving multiple joints.[1] It requires multisensory sources for postural stability, but the somatosensory constraints on injured side may result in the reorganisation of sensory system, leading to the reliance on a more reliable source.

Additionally, the dissimilar observation on visual reliance reiterates central adaptation or reorganisation is different between CAI and acute lateral ankle sprain. Although these two diseases displayed higher visual reliance bilaterally during single-leg stance,[24] no increased visual use was observed in CAI during static double-leg stance in current study. Increased visual reliance during static double-leg stance displayed in acute lateral ankle sprain may be due to the acute pain effect resulting in inhibitory modulation of pain pathways.[24] However, the modulation mechanism may be more complicated in CAI because it may combine chronic pain,[46] sensorimotor network and interaction of interhemispheric connections.[54] The CAI group showed an inability to ignore perturbed visual clues. The visual modulation displayed a 26.95 % decline in the AP direction during double-leg stance when visually perturbed, which was higher than that during the removal of visual input. Reduced ability in sensory reweighting and fixed strategies in sensory input (increased visual and vestibular reliance) may be the true cause of postural instability in patients with unilateral CAI, but the visual reliance is more likely to influence daily activities because the function of vestibular system is less likely to be evoked by simple postural tasks that lack evident head movement.

Overall, the finding of bilateral decreases in sensorimotor function supports the emerging hypothesis that maladaptive reorganisation of centrally meditated changes occurs in sensorimotor function following unilateral CAI, which may impair the sensorimotor function on the injured and uninjured sides.[17,27,46,55] A possible explanation for this finding is the coupled neural circuits that control both limbs.[56] Edgley et al.[56] reported a group of interneurons that received supraspinal input from the vestibulospinal and reticulospinal pathways and pyramidal tract and bilateral peripheral input from group II fibres and joint afferents. Because the recurrent ankle sprains impair the peripheral mechanoreceptors around the ankle joint,[4] the decreased sensory input from injured ankle may be mediated by interneurons bilaterally resulting in error motor commands.[17] However, this hypothesis is less likely to clarify the exact mechanism due to some bilateral alterations in the supraspinal level (bilaterally increased visual use and delayed motor response), the interneurons serve as the modulation of spinal level.[56]

Additionally, interaction and adaptation of bilateral sensorimotor networks are more likely to prove this central phenomenon. Functional magnetic resonance imaging (fMRI) indicated unilateral activation could result in bilateral activation in the primary motor area, premotor area, secondary somatosensory area and cerebellum, and unilateral training could activate bilateral primary somatosensory area.[57] Recently, Xue et al.[54] discovered the activity of cerebellar lobule VIIIb, which is responsible for ipsilateral limb actions, was affected bilaterally by unilateral CAI. The fMRI observations imply some coactivation networks could influence the bilateral sensorimotor functions in individuals. Although sensorimotor control is hierarchically conducted and modulated at the spinal cord, brain stem and cerebral cortex, several parallel regions and interacting neural networks at each level play an integral role in influencing sensorimotor function.[3] Hale et al.[58] reported that training the uninjured side would enhance bilateral balance in unilateral CAI. The potential mechanism for bilateral intersection would be beneficial for rehabilitating unilateral CAI and contribute to determining the possible mechanisms of how the impaired sensation in injured limb affects the bilateral functions. However, the underlying mechanism remains unclear, and further research regarding combining the central and peripheral systems is required to elucidate this.

Some limitations should be clarified in this study. Firstly, although the bilateral somatosensory in both ankle–foot complexes were assessed comprehensively, the proximal joints were not evaluated. Some studies reported sensation in proximal joints also presented adaptative alterations in CAI.[59,60] Secondly, the perturbations, including force plate movement and environment, mainly took place in the AP direction, which resulted in some significant differences only in the AP direction. Finally, the cross-sectional design hindered evaluating the cause and process of bilateral alteration, so the possible explanations were based on the current hypothesis and observations. However, the findings of current study may promote the improvement of rehabilitation models and facilitate more targeted training methods regarding unilateral CAI, which will result in the reorganisation of sensorimotor function. Further studies combining the peripheral and central nervous system and long-lasting prospective studies are required to detect the underlying mechanism. Moreover, the training on the uninjured side or visual-vestibular training may be a direction for enhancing the bilateral functions and ability of sensory reweighting.

5. Conclusion

Individuals with unilateral CAI presented bilaterally decreased sensorimotor function, which indicated the constraint in the injured ankle may have a bilateral interaction and adaptation mechanism regarding the sensorimotor network. Increased visual modulation and decreased postural control related to visual input suggested the CAI group may display fixed sensory strategy (i.e. visual reliance) during postural modulation, but somatosensory cue was still the primary sensory source. In addition, the ability to ignore perturbed visual clues was reduced in CAI.

CRediT authorship contribution statement

Xiaomei Hu: Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, All authors have read and agreed to the published version of the manuscript. **Xihe Hou:** Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, All authors have read and agreed to the published version of the manuscript. **Lin Wang:** Conceptualization, Methodology, Writing – review & editing, All authors have read and agreed to the published version of the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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