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Using computer vision to quantify mirror movements in children with unilateral cerebral palsy

Graduate thesis in Medicine
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Abstract

BACKGROUND: Mirror movements (MM) are common in children with unilateral spastic cerebral palsy (USCP). In the clinic and in research, MM are most often assessed qualitatively using the scoring system proposed by Woods and Teuber (W&T), whereas quantification of MM is more rarely reported - probably since it has been dependent on special equipment.

AIM: This study investigated, whether computer based video analysis could be used to quantify MM.

METHOD: 37 children and adolescents with CP, and 22 typically developing (TD) children and adolescents were instructed to perform hand movements while their hands were video-recorded. The films were used to assess MM qualitatively using the score according to W&T. In addition, the movements of the passive hand were quantified using computer based video analysis.

RESULTS: 25 (68%) of the participants with USCP had MMs in their non-affected, and 22 (59%) had MM in their affected hand when movements were performed at slow speed. At fast speed, 26 (70%) had MM in their non-affected, and 27 (73%) had MM in their affected hand as assessed with W&T. Among the TD participants, 6 (27%) had MM in their dominant hand, and 4 (18%) had MM in their non-dominant had assessed with the score according to W&T when movements were performed at fast speed. One (5%) had MM in the non-dominant hand, and none had MM in the dominant hand when movements were perfomed at slow speed. Boxplots and correlation analyses suggested that quantity of motion (QoM) mean was the variable from the computer based analysis that was most suitable as a proxy for MM. In the total population including TD participants the correlation of QoM mean with W&T scores was moderate to good as indicated by correlation coeffcients between 0.59-0.74 ($p < 0.01$). Within the group of participants with USCP, correlation coeffcients ranged between 0.70 and 0.88 ($p < 0.01$). Particular high correlation coefficients (0.70 - 0.88) were observed among participants where the setup of the video-recordings was strictly adhered to. The correlations were lower in the subsample of participants with USCP, where deviations from this setup were observed. Height and width of motion (HoM and WoM) standard deviation had correlation coefficients 0.63 -0.76 ($p < 0.01$). For the above mentioned subgroup of participants, the correlation coefficients were 0.72-0.84 ($p < 0.01$).

CONCLUSION: We found a moderate to good correlation between the computer based video analysis and the clinical assessment, suggesting that this method may be used to assess MM quantitatively. However, the method seems to be sensitive to deviations in setup of the video recordings.

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1 Introduction

Cerebral palsy (CP) is an umbrella term, describing persistent, non-progressive disturbances of movement and posture, that were caused by damage or dysfunction in the immature central nervous system (CNS) early in life. The condition manifests itself in early childhood and persists throughout the lifespan [1]. A consensus group agreed on the following definition in 2005:

> *CP describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication and behaviour, by epilepsy and by secondary musculoskeletal problems.* [2]

These impairments can significantly affect the childrens activity and participation [1]. Cerebral palsy is the most common motor impairment of childhood, with a prevalence of 1,5-3,0 per 1000 live births [3].

Cerebral palsy is a clinical diagnosis. There are no distinct physical signs for CP; clusters of symptoms or abnormal movement patterns are indicative of the condition. Early physical signs are abnormal muscle tone or the persistence of primitive reflexes. A common presenting symptom is delay in reaching motor milestones [4]. Many children, who later will be diagnosed with CP, have received medical attention for neonatal difficulties such as feeding problems, even before their motor function becomes apparently deviant [1]. At six to 12 months of age, the infant may exhibit spastic hemiplegic or hypertonic movement patterns, leading to the diagnosis during the first 12 to 18 months of life. In a Norwegian study by Andersen et al [3], median age at diagnosis was 15 months. However, if the impairment is mild, the diagnosis may not be definite until the child has grown older, which can be illustrated by the fact that the oldest children were more than eight years old at diagnosis in the same study [3].

One must always verify that the motor impairment is neither progressing nor resolving. A definite diagnosis is often made at four years of age, when transitory symptoms or progressive disorders become more unlikely [5].

1.1 Development of the human brain

The genesis of the brain is mainly made up by three, subsequent stages: cell proliferation, cell differentiation and cell migration [6]. While neurogenesis mainly takes place in the first and second trimester of pregnancy, growth and differentiation dominate in the third trimester and the postnatal life [7]. Any disturbance in one or more of these steps can have consequences of varying severity, depending on the time and location of the deviation.

The differentiation of cell types in the CNS begins in the fourth week, spreading outwards both cranially and caudally from the middle section of the neural tube. The so-called **ventricular zone** is an area that surrounds the central lumen of the neural tube, giving rise to the neurons [8, 6]. Those neurons then migrate peripherally, producing the so-called **mantle zone**, which later will become grey matter. Axons extending from these neurons form the **marginal zone**, later to become white matter. [8] The first maximum in cell differentiation is reached between the 15th and the 25th week, followed by a second maximum in differentiation around the 25th week. When no longer needed for production of new cells, the ventricular zone becomes ependymal tissue [6]. The architecture of the brain can be considered to be complete in the third trimester[7].

The cerebral hemispheres first appear in the fifth week. Here, the process of cell differentiation is more complex than in other parts of the brain as the neurons migrate in clusters, depositing one layer at a time. The neurons of each wave migrate through the preceding layers and establish a more peripheral layer on top of them. As the growing hemispheres press against the thalami, these structures fuse. The former border in between is eventually crossed by the internal capsule. At the end of the ninth week, the corpus callosum starts to form [8]. Corticospinal fibres have completed their caudal extension into lumbrosacral cord by week 29 [9].

At birth, the brain is about 25% of its adult volume [8] and accounts for 20% of the body weight. Most of the structures are formed, but not fully developed. The most advanced part of the brain at birth is the brainstem, which is critical for survival of the infant [10]. Basal ganglia, thalamus and the central regions of the brain show the highest glucose metabolism during the neonatal period [7]. There is an initial burst of synaptogenesis during the first two years of life, followed by a process of elimination. These two processes are highly dependent on each other and there seems to be a threshold after which a synapsis becomes permanent. This is thought to be closely connected to the act of learning [10].

The corpus callosum is the part of the brain connecting the cerebral cortices of both hemispheres. It keeps growing and maturing during the early years of childhood, more

so than in any other period of life. Following the growth of the corpus callosum is the functional specialization of the left and right cerebral cortices. This process is called lateralization and relates to the development of unilateral brain dominance [10], which starts to manifest itself close to the timing of the first spoken words. Myelination is considered to show mature patterns after the age of two years [7]. The brain reaches its final size at around 7-10 years of age, most of the growth resulting from the myelination of nerve fibers [8, 10]. Brain plasticity can compensate for the loss of function following insults to the developing brain. The compensatory potensial is different for different functions of the brain. According to Krägeloh-Mann, there is some evidence for higher plasticity in the motor system. Maintenance of ipsilateral tracts seems to play an incomplete functional role after unilateral lesions in early and mid gestation. There is superior brain plasticity for language function, but the visual system seems to have only limited compensatory potential [7].

The brain continues to develop for at least 20 years and in the adult, it accounts for about 2% of the body weight [10].

1.2 Motor function

Motor control is a highly complex matter. In the following, a simplified illustration of the morphologic organisation is given.

The soma (body) of the primary motoneuron is placed in the primary motor cortex, its axon (tail) follows the corticospinal tract down to the spinal cord, where it forms a synapse with the secondary motoneuron. These are called the pyramidal tracts [11]. About 80% of the axons of the primary motoneuron cross to the opposite side at the height of the medulla oblongata, the remaining 20% form the anterior corticospinal tract. Most of the fibres in this tract cross to the opposite side at the height of the segment they innervate, but not all do [12].

The soma of the secondary motoneuron is placed within the grey matter of the ventral horn of the spinal cord [11]. There are different types of secondary motoneurons, α-motoneurons innervate skeletal muscles. The somata of the secondary motoneurons are organised somatotopicly, meaning that neurons that innervate medial muscles, are placed medially in the spine; whereas neurons that innervate distant muscles are placed laterally [12]. Each α-motoneuron forms synapses with a group of muscle fibres, in a so-called neuromuscular junction [11].

If the primary motoneuron is damaged, conscious control of the skeletal muscle is dis-

rupted. As long as the second motoneuron and sensory fibres are intact, this will lead to a spastic palsy. If the secondary motoneuron is damaged, the muscle will no longer receive any kind of stimuli. This leads to flaccid paralysis [11].

1.3 Causes of CP

The leading causes of CP are related to congenital malformations of the CNS and vascular disturbances within the brain [4]. Malformations occur early in pregnancy, while vascular disturbances lead to brain lesions in the third trimester [4, 7]. However, any damage or disturbance in the developing fetus og infant brain can cause CP.

The various brain regions are vulnerable to insults at different stages in the fetal brain development [4]. Insults occurung before the 20th week of gestation, can result in neuromigrational defects [4]. Between the 28th and 34th week of gestation, the periventricular regions of the brain are especially vulnerable [3, 4], whereas the cortex, basal ganglia and brainstem are most vulnurable close to term [4]. According to Krägeloh-Mann and Bax, the same patterns tend to occur at the same gestational age, meaning that the same insults can occur both pre- and postnatally, depending on the date of birth in relation to gestational age [13]. Both the timing of an insult and perinatal stress play significant roles in determining the severity of consequent impairments [3].

1.3.1 Risk factors for developing CP

Prenatal risk factors for CP are intrauterine growth deviations, intrauterine infections, multiple pregnancies, congenital malformations and congenital stroke. Intrapartum asphyxia is a **perinatal factor** that can lead to CP. Children born at term with evidence of a peri- or neonatal hypoxic ischemic event are at risk of more extensive brain injury including the grey matter, cortex and central nuclei and thus leading to a more severe CP involving both upper and lower limbs [3]. **Postneonatally** acquired CP can be caused by any damage og dysfunction in the brain of the infant [4].

Prematurity and low birth weight are major risk factors of CP, and the risk increases as the gestational age at delivery decreases. However, it is not clear whether complications to prematurity are the sole cause of CP, or if both the premature birth and the CP are caused by a, so far unknown, common factor or combination of factors, [4] such as i.e. intrauterine infection.

A Swedish study showed that in infants born at gestational week 28 or earlier, about

70 per 1000 live births would develop CP. In infants born between gestational week 28 and 31, about 40 out of 1000 live births would develop CP [14]. In comparison, the incidence in the general population is about 2 per 1000 live births, including 1 per 1000 for children born at term [3]. Although children born before week 28 of gestation had a substantially increased risk of getting CP, they comprise only 12% of the total CP population [3]. Thus, the majority of children with CP are born at term. Andersen et al found no differences in the distribution of associated impairments by gestational age except for epilepsy being present in a higher proportion of children born at term [3].

1.3.2 Periventricular lesions

Periventricular lesions are the predominant type of brain damage in preterm babies [13]. There is a selective vulnerability of the periventricular regions of the brain between weeks 24-34 of gestation [3, 7, 15]. A part of the children with CP born prematurely have an injury limited to these areas of the brain [3]. Such lesions include complications to intraventricular hemorrhage (IVH) and periventricular leukomalacia (PVL) [7].

Intraventricular hemorrhage (IVH) is a relatively common complication seen in preterm infants. When seen in term born children, they are often considered to be of prenatal origin [7]. The risk of IVH corresponds inversely with gestational age [16]. IVH develops usually during the first few days of extrauterine life, especially in very preterm children [7]. Most commonly, it originates from rupture of fragile blood vessels within the germinal matrix, supplying the ependymal tissue surrounding the lateral ventricles. Asphyxia during birth and impaired auto-regulation of cerebral blood flow probably increase the risk of IVH. The fact that intracranial hemorrhages directly relate to the degree of prematurity reflects the importance of the immaturity of the germinal matrix vasculature and alterations of cerebral blood flow seen in premature infants. It appears, however, as if the most severe hemorrhages are caused by ischemic hemorrhagic infarction, presumably as a result of impaired venous drainage [16].

IVH lesions are usually graded on radiologic criteria [17]:

I Hemorrhage limited to the subependymal or germinal matrix region

II a) Intraventricular hemorrhage occupying less then 50% of the lateral ventricles

b) Intraventricular hemorrhage occupying more than 50% of the lateral ventricles, usually with associated ventricular dilation

III Parenchymal hemorrhage in association with IVH

IV Parenchymal hemorrhage in association with IVH, causing a deviation of the midline

A feared complication to intraventricular hemorrhage is a post-hemorrhagic hydrocephalus. Infants with hemorrhages grade III and IV have increased risk of developing CP.

Periventricular leukomalacia (PVL) refers to bilateral necrosis of the white matter adjacent to the lateral ventricles [4]. It is primarily localized in the parietooccipital area [7]. According to Sigurðardóttir, it is the principal ischemic lesion seen in preterm infants and it is strongly related to spastic CP. If the injury is moderate, only the pyramidal tracts supplying the lower limbs will be affected, leading to diplegia [4].. PVL is the most common cause of spastic diplegia [13]. If the upper limbs are affected as well (quadriplegia), the lesion must be more severe and extend laterally. Only the most extensive lesions affect the cortex [4].

1.4 Classification of fine and gross motor function

Gross Motor Function Classification System (GMFCS) is a classification system that can be used to evaluate gross motor function in children with CP [18]. It consists of five levels:

I Walks without limitations

II Walks with limitations (i.e. walking on an uneven surface, inclines, in a crowd or in a confined space)

III Walks using a hand-held mobility device, but might use a wheelchair when going for a long distance or on uneven terrain

IV Self-mobility with limitations. May walk short distances.

V No means of independent mobility

Manual Ability Classification System (MACS) is a classification system that can be used to evaluate overall hand function in everyday situations [19, 20].

I Handles objects easily and successfully. At most limitations in the ease of performing manual tasks requiring speed and accuracy.

II Handles most objects but with somewhat reduced quality and/or speed of achievement. May avoid some tasks or use alternative ways of performance.

III Handles objects with difficulty, needs help to prepare and/or modify activities.

IV Handles a limited selection of easily manageable objects in adapted situations, requires continuos support.

V Does not handle objects and has severely limited ability to perform even simple actions. Requires total assistance.

1.5 Classification of CP by clinical presentation

According to the Surveillance of Cerebral Palsy in Europe (SCPE) network, CP can be divided into **spastic**, **dyskinetic** and **ataxic** subtypes [3, 5]. SCPE further divides the spastic subtype into a unilateral and a bilataral form, depending on whether limbs on one side of the body are affected (hemiplegia), or if limbs on both sides of the body suffer from spasticity. In the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10), the spastic bilateral type is further divided into diplegia or quadriplegia (tetraplegia), determined by whether only the legs are affected, or all four limbs. Lastly, there are unclassified forms. Spastic CP is the most common form, with a prevalence of 82% in the study by Andersen et al, whereas dyskinetic CP (6%) and ataxic CP (5%) are less common [3].

1.6 Unilateral spastic CP

The spastic subtype can be further divided into a bilateral and a unilateral form. In the study of Andersen et al, unilateral spastic CP (USCP) was seen in about a third of all children with CP [3]. In most children with USCP, the upper limb is more severly affected than the lower one [16].

Typical onset of symptoms in USCP is at about 24 months of age. At the same age, there is a rapid development of ipsilateral and contralateral corticospinal projections from the undamaged hemisphere in childen with USCP [21].

Associated impairments such as epilepsy, mental retardation or other disorders are less common in USCP compared both to the bilateral spastic type, but also to the other forms of CP. 45% of the children with USCP had motor impairments only in the study by Andersen et al [3].

The spastic unilateral subtype can be further divided into a right (limbs on the right side of the body affected) and left type. Interestingly, Andersen et al found that fine motor function of the non-affected hand was normal in all children classified as *right* USCP - where one would expect an injury to the left brain hemisphere. "However, in the children classified as left unilateral CP [*damage to the right hemisphere*], the function in the non-affected hand was impaired in six (14%)" [3]. Based on this study, it would thus appear as though impaired hand function on the non-affected side is more common in children

with paresis on the left side.

1.6.1 Pathogenesis

USCP is a common clinical feature in children who suffered from a proliferation disorder in early pregnancy, known as *hemimegalencephaly*. In these cases, abnormal proliferation of neuronal cells takes place in the first or second trimester of pregnancy [7]. Unilateral PVL and IVH (1.3.2) also lead to USCP. Severe PVL is associated with cerebral visual impaiment, severe spastic CP and mental retardation [7].

The most common cause of USCP is a prenatal or perinatal cerebral infarction, that in most cases affected the middle cerebral artery. Possible etiologies include coagulopathy, congenital heart disease or infectious processes [16]. Infarcts of the middle cerebral artery are reported mainly in term born or near term born infants. However, they also occur in very preterm born children [7]. Andersen et al suggest that especially children who are born prematurely, have an injury to the brain limited to the so-called watershed areas supplied by the middle cerebral artery [3]. These are the periventricular regions where motoneurons pass on their way from the cortex to the spine.

1.7 Mirror movements

Mirror movements are involuntary movements of one body part that mirror the voluntary movement of the contralateral homologous part [22, 23, 24]. They are more often seen in the upper limbs and their intensity increases with increasing task complexity or fatigue [26]. They are more pronounced during distal limb movement [23] and are related to intentional movements rather than reflexes or passive movements [22].

Mirror movements may be observed in infants, but in typically developing (TD) children they decrease between 5 and 8 years of age and have usually disappeared completely by the age of 10 [24]. However, Koerte et al found that in healthy adults, the occurrence of mirror movements increases again gradually after the mid-thirties [25].

Mirror movements are common in children with USCP even above the age of 10. They are more often seen in the non-paretic hand when the paretic hand is performing voluntary movements, but occur on both sides [22, 24]. If the paretic hand is completely paralysed, there are normally no mirror movements in the non-paretic hand when movement is attempted with the affected hand [22].

1.7.1 Pathophysiology

The pathophysiology of mirror movements in USCP is not yet fully understood. Various models have been proposed, and possibly the processes in TD children differ from those in children with CP. Further, different processes might lead to similar mirror movements in children with CP, depending on the type and timing of the lesion leading to the CP [26].

Woods and Teuber studied mirror movements in 1978 and assumed them to be the result of a compensatory reorganisation after damage to the immature nervous system. They found that children who had a complete paralysis of their affected hand, normally had no mirror movements. But complete paralysis was more common among children who had suffered from a lesion to the brain after the age of one year [22], thus, they found mirror movements to be more common after early lesions.

Several studies have shown an ipsilateral reorganisation of corticospinal fibres from the undamaged motor cortex to the affected limb(s) [7, 9, 27]. Carr et al studied this phenomenon in 1993 [9] and were able to describe the reorganization of the CNS in individuals after early, unilateral brain lesions. They were able to show a reorganisation of motor pathways, leading to ipsilateral innervation of the affected limb from the non-lesioned motor cortex. Their findings suggest that different mechanisms occur following damage to the brain at different stages of its development, and they were able to demonstrate two different manners of ipsilateral reorganisation.

In one group of patients, they found fibres from the undamaged primary motor cortex branching out, innervating homologous motor neuron pools on both sides of the spinal cord. These subjects had the most pronounced mirror movements, and the authors hypothesized that the reorganisation of motor pathways must have taken place early in pregnancy, before the end point of normal axonal elongation.

In another group of patients, they were not able to show any branched fibres. Instead, they found ipsilateral axons as separate projections from the undamaged cortex to the affected limb. These patients had abscent or weak mirror movements, but also poorer fine motor skills. This kind of reorganisation was attributed to a later insult, after the end point of axon elongation. They suggest that these ipsilateral projections may have developed de novo from the unaffected motor cortex [9] Other studies have suggested that the compensatory potensial for motor function is restricted to early in the third trimester [7].

Kuhnke et al [27] examined nine patients with reorganized ipsilateral projections and

seven patients with with crossed (contralateral) corticospinal projections, all of them with USCP. As Carr et al had found [9], Kuhnke et al, too were able to show that the primary somatosensory representation (S1) of the paretic hand always remains in the contralateral hemisphere - independent of a reorganization of the motor representation (M1). Possibly, this phenomenon can be explained by the lesser degree of compensatory plasticity in the somatosensory system, compared to the motor system [7]. For patients with ipsilateral corticospinal projections, this leads to a hemispheric dissociation between the contralateral S1 in the affected hemisphere and the primary motor representation in the contralesional hemisphere [27].

Other possible explanations of the pathophysiology behind mirror movements include motor overflow [23] and incomplete maturation of the corpus callosum [24].

1.7.2 Measuring mirror movements

Woods and Teuber proposed a scale to score mirror movements in 1978: [22]

0. no clear imitative movement
1. barely discernable repetitive movements
2. slight mirror movements or stronger, but briefer repetitive movements
3. strong and sustained repetitive movements
4. movements equal to those expected for the intended hand.

In their study, participants performed a total of three tasks and were scored for each separately, resulting in possible scores ranging from 0 to twelve. This has been the dominating tool for evaluating mirror movements clinically for the last four decades.

As the need for a continuous scale to measure mirror movements has become more apparent, different approaches have been made. In 2000, Kuhtz-Buschbeck et al used a grip object equipped with strain-gauge transducers to measure grip force in one hand, and a rubber bulb connected to a pressure sensor in the other, in order to quantify mirror movements [23].

In 2010, Koerte et al used a force transducer held in a precision grip between thumb and index finger of each hand. Participants were asked to increase and decrease grip force repeatedly for 15 seconds in one hand, while the resting hand had to prevent the force transducer form falling on the floor [25].

1.8 Therapeutic interventions

CP is a non-curable condition. However, there exist multiple therapeutic approaches to enhance the individual's function, participation and quality of life.

1.8.1 Constraint induced movement therapy

Constraint induced movement therapy (CIMT) consists of immobilization of the non-paretic hand, combined with intensive training of the affected hand [15, 27]. The goal is to improve hand function in the affected hand. It was first developed for adults who suffered from a unilateral stroke and was based on the idea of *learned non-use* occuring when one ceases to use a body part. In children with CP, *developmental non-use* might be a more accurate description [21].

It is thought to be effective via the following mechanisms:

- Constraint of the non-paretic hand reduces activity in the unlesioned brain hemisphere
- Intensive training of the paretic hand increases activity in the lesioned brain hemisphere

This combination is thought to modify an existing imbalance of the interhemispheric interaction between the two motor cortices [28].

Eliasson et al found that CIMT could improve bimanual hand function in children with USCP after six months. However, long term effects of CIMT remained uncertain [15]. Sakzewski et al showed in 2011 that CIMT had a better outcome than bimanual training after 26 weeks [29]. Kuhnke et al state that, in their experience "ipsilatterally reorganized patients with corticosubcortical lesions are often too severely impaired to participate in a CIMT program, and contralaterally organized patients with periventricular lesions typically have manual abilities that do not require intensive therapy" [27]. This is supported by the findings of Carr et al, who showed in their study that the ability to evoke EMG responses when the affected motor cortex was stimulated (indicating remaining contralateral projections), was associated with *good* function of the affected hand [9].

In 2008, Kuhnke et al investigated whether the type of corticospinal reorganisation would influence the efficacy of CIMT in patients with USCP. They divided their study group in two - one group in which they found reorganized ipsilateral corticospinal projections, and one group with preserved corticospinal projections. Both groups showed a significant improvement in hand function after CIMT, and in the group with preserved contralateral

projections, they found a significant reduction in the time needed to perform certain tasks. However, this did not persist at follow-up after three months. In the group with ipsilateral projections, they found a "trend towards slower performance", which was persistent also after six months. They concluded that "different types of corticospinal organization in congenital hemiparesis respond differently to CIMT" [27] and hypothesized that this could be associated to the hemispheric dissociation between the somatosensory and the motor representation of the paretic hand in patients with reorganized, ipsilateral projections. They considered an intact sensorimotor loop crucial for effective motor learning [27].

Juenger et al studied the same material further in 2013. In patients with ipsilateral projections, they found a significant decrease in motor evoked potential (MEP) amplitude - both in the ipsilateral projection to the paretic hand, and the crossed projection to the non-paretic hand after CIMT. They found a "decrease in transsynaptic M1 excitability (as measured by transcranial magnetic stimulation) and a decrease in synaptic activity during active movements of the paretic hand (as measured by fMRI) after 12 days of CIMT" [28] and suggest that these findings may be interpreted as indicating that CIMT could be potentially harmfull in individuals with an ipsilateral corticospinal reorganisation [28].

Kuhnke et al found that strong mirror movements in both hands only were present in patients who had ipsilateral corticospinal motorprojections to their paretic hand [27], but they did not differ between individuals with branched fibres or separate projections, as Carr et al did [9]. Carr et al [9] found pronounced mirror movements only in patients with branched fibres, whereas subjects with separate ipsilateral projections had abscent or weak mirror movements.

Quantification of mirror movements could serve as a means to identify patients with ipsilateral projections as opposed to preserved contralateral projections, and branched fibres as opposed to separate projections. This could, in turn, be used to identify patients who are more likely to profit on CIMT, and those in whom other interventions may be applied more successfully. Possibly, a quantification of mirror movements could also be used as an outcome measurment to monitor the effect of therapeutic interventions.

1.9 Aim of the study

The aim of this study was to explore if computer based video analysis could be used to quantify mirror movements. This could hopefully provide a cost-efficient, non-invasive, painless and easily accessible method to identify children who would benefit from better targeted therapeutic approaches.

2 Materials and methods

2.1 Study design and population

Participants eligible for the study were children and teenagers with USCP who were mentally capable of cooperation, and TD children of the same age. Mirror movements were scored according to the scale developed by Woods & Teuber [22] based on video recordings and quantified using a computer-based video analysis program as described by Adde et al [30, 31] and adapted for this purpose. The amount of mirror movements assessed clinically was then correlated to the amount of mirror movements detected by the computer program.

We used the terms non-affected and affected hand in the CP group, whereas we used the terms dominant hand and non-dominant hand in the control group.

2.1.1 Participants

Children and adolescents diagnosed with USCP according to the guidelines proposed by the SCPE, were invited to participate. Participants were recruited through the outpatient clinic at St. Olav University Hospital in Trondheim, Norway (in the period from October to December 2011, as well as January 2016) or through the research project "Cognition and Bimanual Performance in Children with Unilateral Cerebral Palsy" at Monash Children´s Hospital in Melbourne, Australia (autumn 2015). In the Norwegian part of the study the inclusion age was 10 to 20 years, while the inclusion age in Australia was 6 to 14 years.

Exclusion criteria were upper limb surgery within 12 months of assessment and injection of Botulinum toxin-A within 3 months of assessment. Written consent was obtained from all parents and teenagers age 16 or above. Ethics committees in both countries approved the study.

For the Australian part of the study, 21 families were invited to join the study and 19 accepted the invitation and met for the examination. In one case, there were technical problems during the assessement, and this participant had to be excluded from the study. For the Norwegian part of the study, 46 children and adolescents were invited to join the study, 21 agreed to participate in the study and 19 met for the test. One participant who met, had to be excluded because of technical difficulties with the video recordings.

A control group comprising 22 TD children and teenagers within the same age range,

and with a similar sex distribution as the CP group was included as well. One of the children had a sibling with USCP, who was subsequently also included in the study.

In total, 59 children were included this study: 37 with USCP and 22 TD children.

Table 1: Background characteristics of 37 children with cerebral palsy (CP) and 22 typically developing (TD) children who participated in this study.

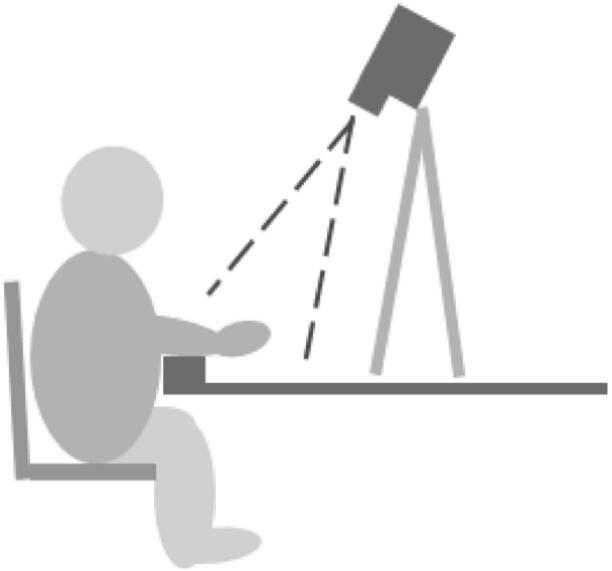
	Children with CP						TD children	
	Trondheim N=19		Melbourne N=18		Both sites N=37		N=22	
Age in years (range)	15.5	(11 - 20)	9.8	(6 - 14)	12.7	(6 - 20)	13.1	(7 - 18)
Female sex	9	(47%)	12	(67%)	21	(57%)	16	(73%)
Left hand affected*	9	(47%)	4	(22%)	13	(35%)	19	(86%)
GMFCS I	10	(53%)	11	(61%)	21	(57%)	-	
GMFCS II	9	(47%)	7	(39%)	16	(43%)	-	
MACS I	2	(10.5%)	3	(17%)	5	(14%)	-	
MACS II	14	(74%)	15	(83%)	29	(78%)	-	
MACS III	2	(10.5%)	-		2	(5%)	-	
unknown	1	(5%)	-		1	(3%)	-	
AHA (range)	66	(37-100)	50	(30-100)	58	(30-100)	-	

* or non-dominant

2.2 Provoking mirror movements

The participants where instructed to execute repeated hand movements with one hand, while possible mirror movements in the other where observed. They were placed in a comfortable sitting position, in a height-adjustable chair and with both forearms resting on a black examination board. The forearms were resting on an elevated rim, providing space to move the hands freely. All participants were allowed to practice the tasks before the recording. They were asked to perform three repetitive tasks: Opening and closing of the fist (task 1), opposition of index finger and thumb (task 2), and tapping their fingers on the examination board (task 3). All tasks were performed separately for each hand with the other hand resting, for the duration of 30 seconds. The Australian group performed each task for 15 seconds. Every task was performed at two speeds: first at a speed coming naturally to the participant, and later as fast as possible. The tasks were performed with eyes opened first, followed by the same procedure with the eyes closed.

Figure 1: Illustration of the set-up for the video recordings.



Due to the enormous amount of data generated, we decided only to include the first task (opening and closing the fist) performed with the eyes opened in this exploratory study.

All tasks were video recorded for later analyses. The video camera was placed above the participant orthogonally to the table surface. These videos were used to score mirror movements qualitatively according to Woods & Teuber [22]. Furthermore, the videos were analysed using software for computer based video analysis, as described by Lars Adde to examine movement patterns in infants [30, 31]. The results from the computer based video analysis were then compared to the clinical score.

2.3 Clinical evaluation of mirror movements

Two observeres, one an occupational therapist and the other holding a master‘s degree in human movement science, scored mirror movements in the non-active hand according to Woods & Teuber [22]. They were blinded to the subjects identity while evaluating the videos that were randomly ordered. They had no knowledge of the other’s score and the computer based video analysis. In the cases in which they had reached different conclusions, the videos were reviewed together and discussed until they arrived at a common conclusion.

In the following, we only used the common score they had agreed on for each partici-

pant. It is provided in table 3. We considered a clinical score of 0 or 1 to represent no certain mirror movements, whereas a clinical score of 2 or more was considered indicative of true mirror movements.

2.4 Computer based video analysis

The videos were recorded using a Sanyo VPC-HD2000 camera, placed orthogonally above the participant.

Some of the participants had performed each task for 30 seconds, whereas others had performed them for 15 seconds. In order to obtain rational results, we attempted to cut equally long film sequences for all participants. We wanted to make sure that the movement had begun and assumed ten seconds to be long enough to capture a representable amount of mirror movements, therefor cutting each film to the duration of ten seconds.

To be able to analyse both hands separately, the four films were further cropped into two approximately equally sized frames, showing only one hand at a time, generating a total of 8 videos per participant. In order to have a standardized approach, we attempted to crop the images to the same size: 636x480 pixels. However, the videos at one site seemed to have been taken from a greater distance. Thus, the hands appeared smaller on film and these films were therefor cropped to the size 400x300 pixels. In some cases, the cropping had to be adjusted so no part of the other hand would appear on the film.

These eight videos showing only one hand each, were further analysed using the computer based video analysis [30].

Motion image The eight cropped films were used as a basis to create *motion images* for every participant. A motion image is a black and white image that is produced by subtracting subsequent frames in the video stream. A pixel displayed in white indicates that no movement happened between the frames, whereas a black pixel indicates movement [31]. Figure 2 shows the motion images of two participants, performing task 1 with the left hand at slow speed. Based on the motion image, several variables were derived.

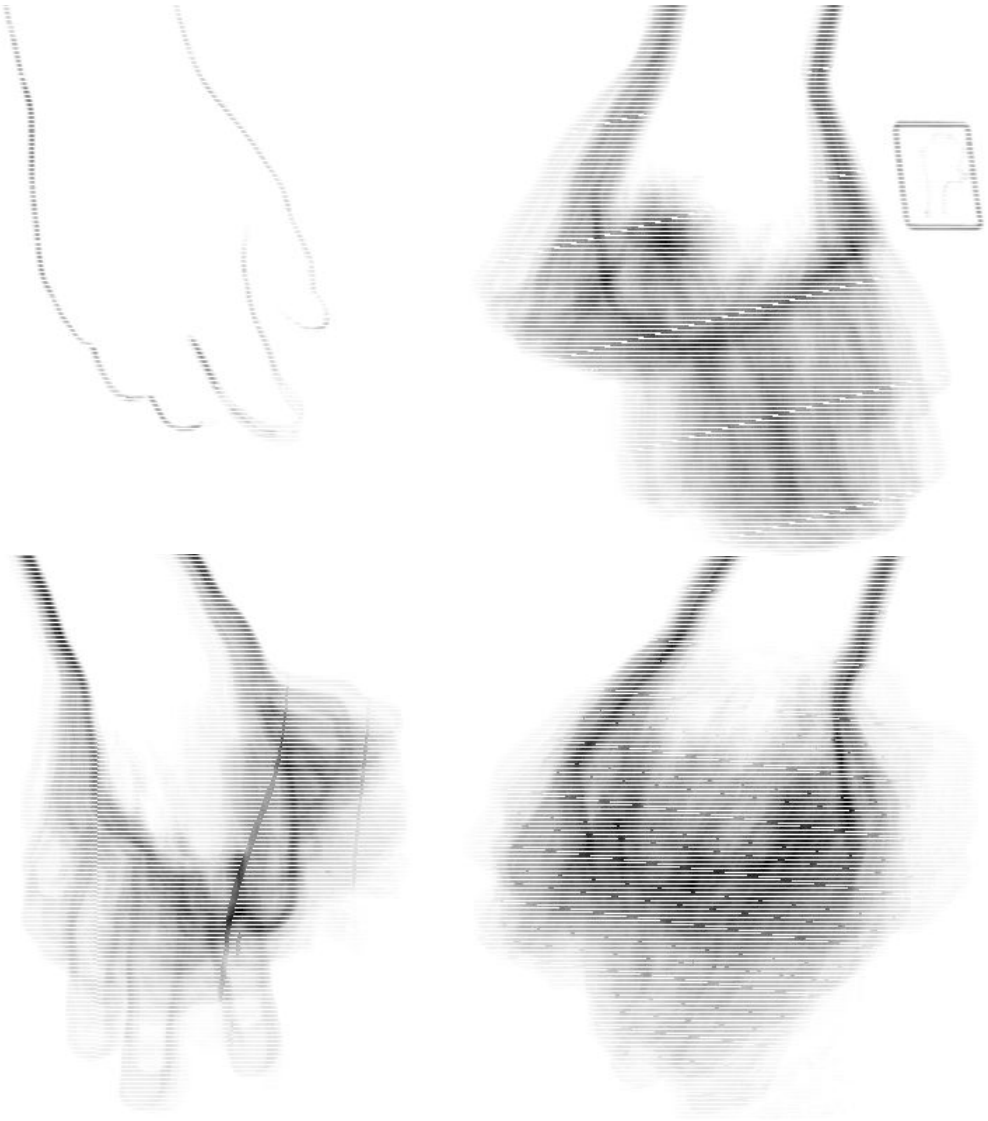


Figure 2: Motion images of two participants: Both were performing task 1 with the left hand at slow speed and were scored with respectively 0 (above) and 2 (below) when mirror movements were assessed clinically according to Woods & Teuber.

Quantity of motion Quantity of motion (QoM) is calculated as the sum of all black pixels in a motion image, divided by the total amount of pixels in the image [31, 32]. This will result in a number between 0 and 1, where 0 implies no movement at all, whereas 1 represents movement over the entire area of the film [32]. This is calculated for every frame in the film and subsequently presented as a mean value and a standard deviation (SD). QoM can be viewed as a amount of all the movement in the film. The mean value represents the average movement from frame to frame, whereas the SD gives information about the variability in the amount of movement [31].

Height and width of motion Height of motion (HoM) describes height of the area in which motion takes place, measured as the distance between the highest and the lowest changing pixel along the y-axis for each frame [31, 32]. This is subsequently presented as a mean value and a SD. Width of motion (WoM) is a measurement of the distance between the outermost changing pixels along the x-axis, and is also presented with a mean value and a SD [31, 32].

In addition to the above mentioned variables, other variables are calculated as well. These were of no importance to the present study, and will therefore not be explained here.

2.4.1 Outcome variables of the video analysis as means and standard deviations

For each participant, different variables were calculated based on the motion image, including QoM, HoM and WoM. They were calculated based on the pixels that changed from one frame to the next, and this was done for *every* frame. Meaning that with 25 frames per second, it was done approximately 250 times in a film lasting for about 10 seconds. For QoM, the mean value describes the mean amount of moving pixels between each frame for *one* participant.

If the hand is lying totally still for some duration of the film, this will generate few moving pixels, and thus a QoM close to the number 0. Now, if the hand suddenly performs a big movement over the entire screen, this will generate a QoM close to the number 1. Thus, a hand that lies still for half of the duration of the film, and then starts moving a lot, will generate a mean QoM close to 0.5.

The SD is defined as the square root of the variance, which is a measurement of the dispersion of values about their mean [36, 35]. If there are values with a big distribution among them, the standard deviation will be bigger than in the opposite case, with the different values lying close to each other. If a hand is lying still for most of the film and then suddenly moves a lot, one would expect a greater distribution among the calculated variables. This would generate a larger SD. If the hand is slightly moving the entire time, there will be no big difference from frame to frame, leading to a smaller standard deviation for this participant.

In other words, one would expect that a participant whose hand is slightly moving the entire time, and a participant who's hand is lying still most of the time before it moves a lot, might both end up with the same *mean* value for QoM. But in the first case, there will be a much smaller SD than in the latter. A hand that is lying perfectly still the entire

time will also generate a small SD, combined with a small mean value for QoM.

The same principles can be applied for the other variables HoM and WoM.

2.5 Statistical analysis

All analyses were performed using IBM SPSS version 23 (IBM, Armonk, NY, USA). Unweighted kappa as a measurement of concordance between the two observers scoring mirror movements according to Woods & Teuber, was calculated using SPSS, while weighted kappa was calculated with the help of VassarStats: Website for Statistical Computation [33].

Normal distribution was assessed by inspection of histograms and linearity was explored through scatter plots. The relationship between the clinical assessment (score according to Woods & Teuber [22]) and the computer based video analysis was assessed by calculating Spearman's rho correlation coefficient (ρ_S). As proposed by Portney and Watkins, [34] correlation coefficients between 0 and 0.25 may be considered to indicate little or no relationship, between 0.25 and 0.50 low, between 0.50 and 0.75 moderate to good, and above 0.75 may be considered to indicate a good to excellent relationship - although stressing that these limits should be viewed as guidelines rather than strict cut-off lines. As proposed by Douglas G. Altman, a kappa coefficient between 0.6 and 0.8 suggests *good* agreement, whereas $\kappa > 0.8$ suggests *excellent* agreement [35]. Two-sided p values <0.05 were considered significant.

We investigated the correlation between the clinical assessment and the variables provided by the computer program. This was done for each hand and each speed separately, resulting in four different correlation matrices, which are provided in the appendix. For each of the tasks, we chose the variables that had a statistically significant correlation coefficient $|\rho| > 0.5$. These variables were again compared to the clinical score, but only for the CP population. This resulted in a smaller group (N=37), but with a bigger distribution of values within in the material. Again, we chose the variables with a correlation coefficient $|\rho| > 0.5$ and performed a partial correlation analysis, adjusting for the participants' age. Those variables that still had a correlation coefficient $|\rho| > 0.5$ were used to create boxplots, in order to consider if a discrimination between participants with and without mirror movements was possible.

To control for the potential confounders age and hand size, we calculated the partial correlation coefficient. We assumed that hand size could be a possible confounder, as QoM is calculated as the sum of all white pixels in a motion image, divided by the to-

tal amount of pixels in the image. Since all the images were cropped to the same sizes (636x480 pixels and 400x300 pixels), a larger hand would generate a bigger amount of white pixels in the motion picture. However, we had no objective measurement of the area of the participants' hands. Therefore, we measured the width of the wrist in the non-affected hand. We found it difficult to get an accurate measurement of the wrist on the affected side in some of the subjects, given the various angles of hand-positioning in relation to the camera, but assumed that there would be a strong correlation in the hand size of both hands in the same individual. We found a strong correlation between the participants' age and width of the wrist on the non-affected side (Spearman's rho 0.664, $p < 0.001$). The confounders were entered into the analysis one at a time, and as none of them changed the result significantly, only age was included in the final analysis as it was considered more certain.

3 Results

3.1 Clinical assessment

Table 2 shows the agreement between the two observers in their independent scoring of mirror movements according to Woods & Teuber. As there were a total of four films for each participant (affected and non-affected hand active at fast and slow speed), the two observers evaluated a total of 236 videos.

Table 2: Agreement of clinical score according to W&T between two raters.

		Observer 1					
		0	1	2	3	4	Total
Observer 2	0	59	4	0	0	0	63
	1	8	48	15	0	0	71
	2	0	1	61	6	0	68
	3	0	0	3	13	5	21
	4	0	0	1	1	11	13
	Total	67	53	80	20	16	236

The resulting unweighted kappa coefficient was $\kappa = 0.75$ (95% confidence interval (CI) 0,682-0,813), while weighted kappa was $\kappa = 0.85$ (95% CI 0.8076 - 0.8912).

Table 3 shows that at slow speed, 22 (59%) of the participants with CP had mirror movements in their affected hand, and 25 (68%) in their non-affected hand. For the TD participants, none had mirror movements in their dominant hand, and one (5%) had mirror movements in the non-dominant hand at slow speed. The proportion of children with mirror movements increased when the task was performed at fast speed, both for children with CP and for TD children. The highest proportion of mirror movements was seen in the non-affected hand when task 1 was performed at fast speed. In this situation, 27 of the children with CP (73%) showed some degree of mirror movements. For the same task, 6 of the TD children (27%) had mirror movements.

In general, mirror movements were more common in the non-affected hand, and more common at fast speed. More details are provided in table 6 in the appendix.

Table 3: Proportions of participants with mirror movements (Woods % Teuber score ≥ 2) and without mirror movements (Woods & Teuber score < 2) in their affected (or non-dominant) and non-affected (or dominant) hand at fast and slow speed

		MM in the affected hand*		MM in the non-affected hand**	
	W&T score	Slow speed	Fast speed	Slow speed	Fast speed
CP	0 - 1	15 (41%)	11 (30%)	12 (32%)	10 (27%)
	2 - 4	22 (59%)	26 (70%)	25 (68%)	27 (73%)
TD	0 - 1	21 (95%)	18 (82%)	22 (100%)	16 (73%)
	2	1 (5%)	4 (18%)	0	6 (27%)

* while performing task 1 with the non-affected (or dominant) hand
** while performing task 1 with the affected (or non-dominant) hand

3.2 Computer based video analysis

When cutting the films, it proved to be quite challenging to achieve the exact same duration, and mean duration turned out to be 9.6 seconds (min 4.4, max 10.8). Mean number of frames was 288 (range: 131 - 323).

Following the procedure described in 2.5, we found the highest correlation coefficients between the clinical score and the variable QoM, as well as the standard deviation of HoM and WoM, albeit not with the mean values of these variables. Correlation matrices and boxplots are provided in the appendix, table 7 - 10 and figure 8 - 9.

3.2.1 Quantity of motion, mean

In the entire study population, the correlation coefficient between the clinical score and QoM mean was >0.74 ($p < 0.001$), and this was the computer based variable with the highest correlation with the clinical score for mirror movements in the *non-affected* hand. Figure 3 shows the individual values of QoM mean according to the clinical classification score underlying these correlation coefficients.

Restricted to children with CP, the correlation coefficients were essentially unchanged; slightly lower in the non-affected hand, but slightly higher in the affected hand (Table 7 - table 10). When we analysed the data for participants with CP from respectively Trondheim and Melbourne separately, we found a marked difference between the two

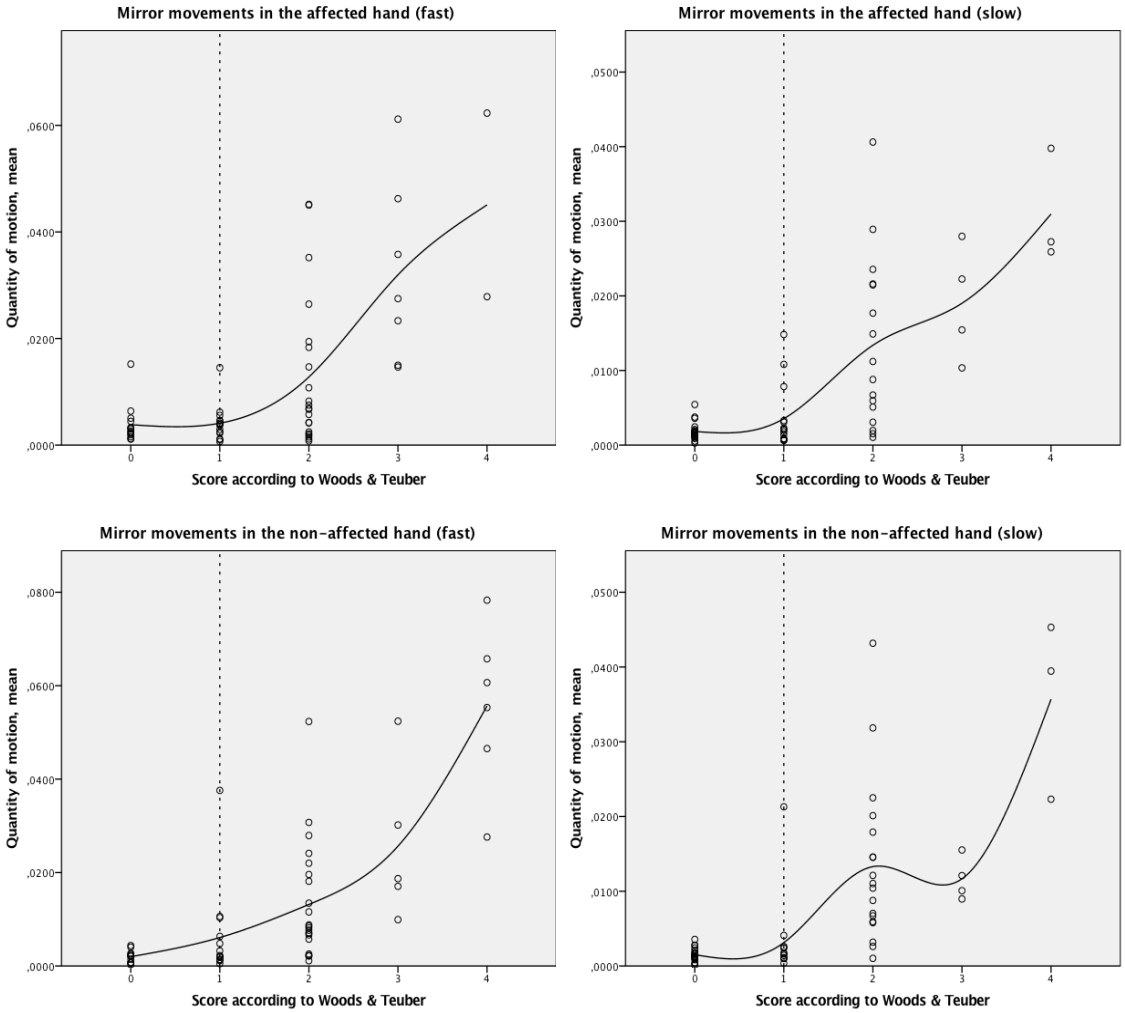
groups. While the correlation coefficients between mirror movements according to Woods & Teuber and QoM mean in the Norwegian population ranged between 0.70 and 0.89 ($p < 0.001$), the correlation coefficients among the 18 participants from Melbourne varied between 0.33 and 0.55. In general, there were only minor differences between hand movements performed at slow and at fast speed.

Figure 5 shows that regardless of speed, QoM mean was able to separate participants with mirror movements from participants without mirror movements, although the figure suggests slightly more overlap between the two groups when the movements are performed at fast speed.

Table 4: Correlations between the clinical score (W&T) and quantity of motion, mean, for Trondheim (N=19) and Melbourne (N=18) separately.

	Affected hand		Non-affected hand	
Fast	Trondheim	r 0.701 $p = 0.001$	Trondheim	r 0.879 $p < 0.001$
	Melbourne	r 0.635 $p < 0.05$	Melbourne	r 0.418 $p > 0.05$
Slow	Trondheim	r 0.709 $p = 0.001$	Trondheim	r 0.827 $p < 0.001$
	Melbourne	r 0.539 $p < 0.05$	Melbourne	r 0.330 $p > 0.05$

Figure 3: Scatterplots of quantity of motion, mean and the clinical assessment according to W&T for the entire population (N=59)



We only considered a W&T score >1 to represent true mirror movements. The reader is advised to pay attention to the different scales along the y-axis.

Figure 4: Boxplots of quantity of motion, mean, for the affected or non-dominant (upper panel) and the non-affected or dominant hand (lower panel) at fast (left) and slow speed (right) among 37 participants with CP and 22 TD participants.

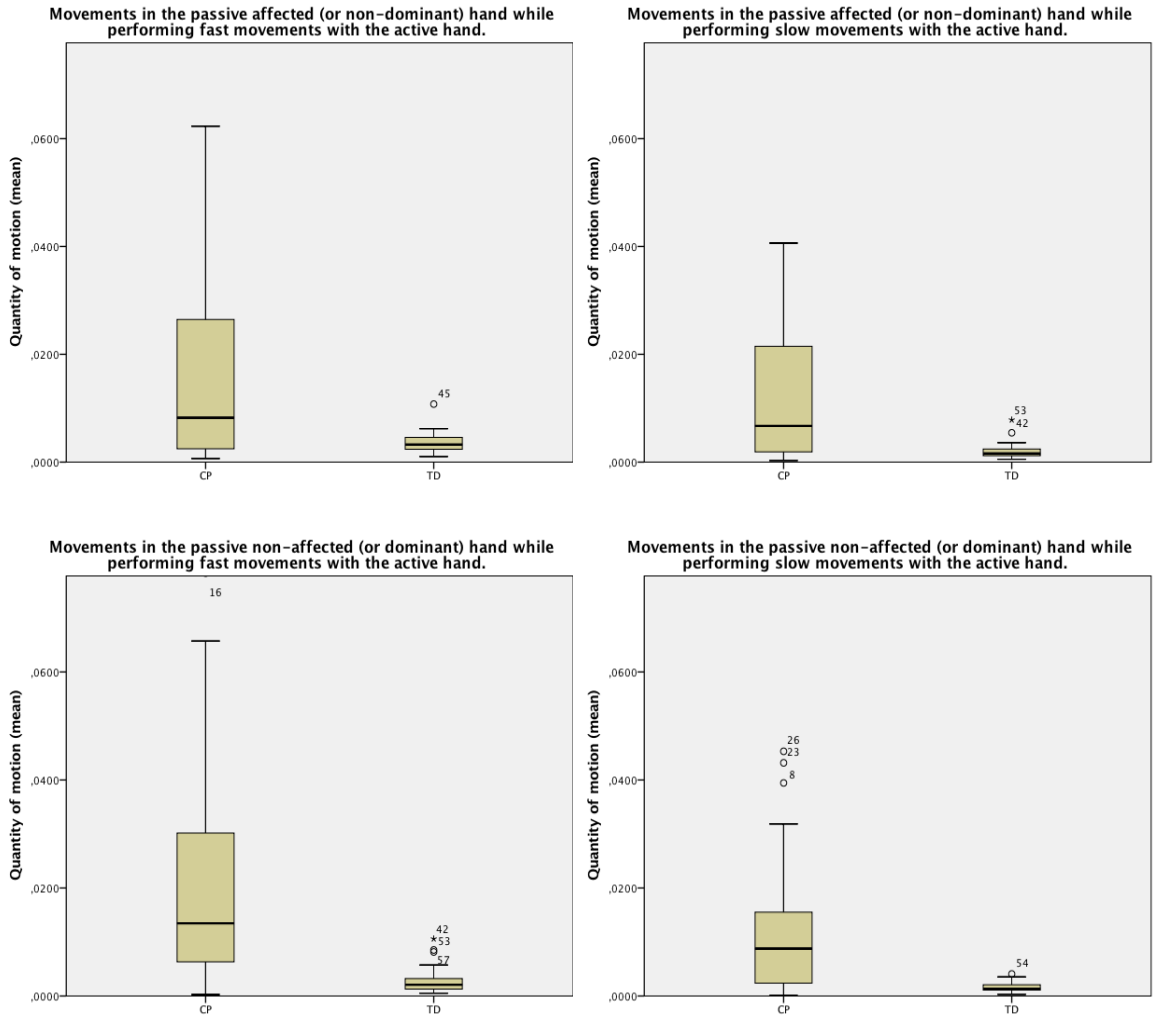
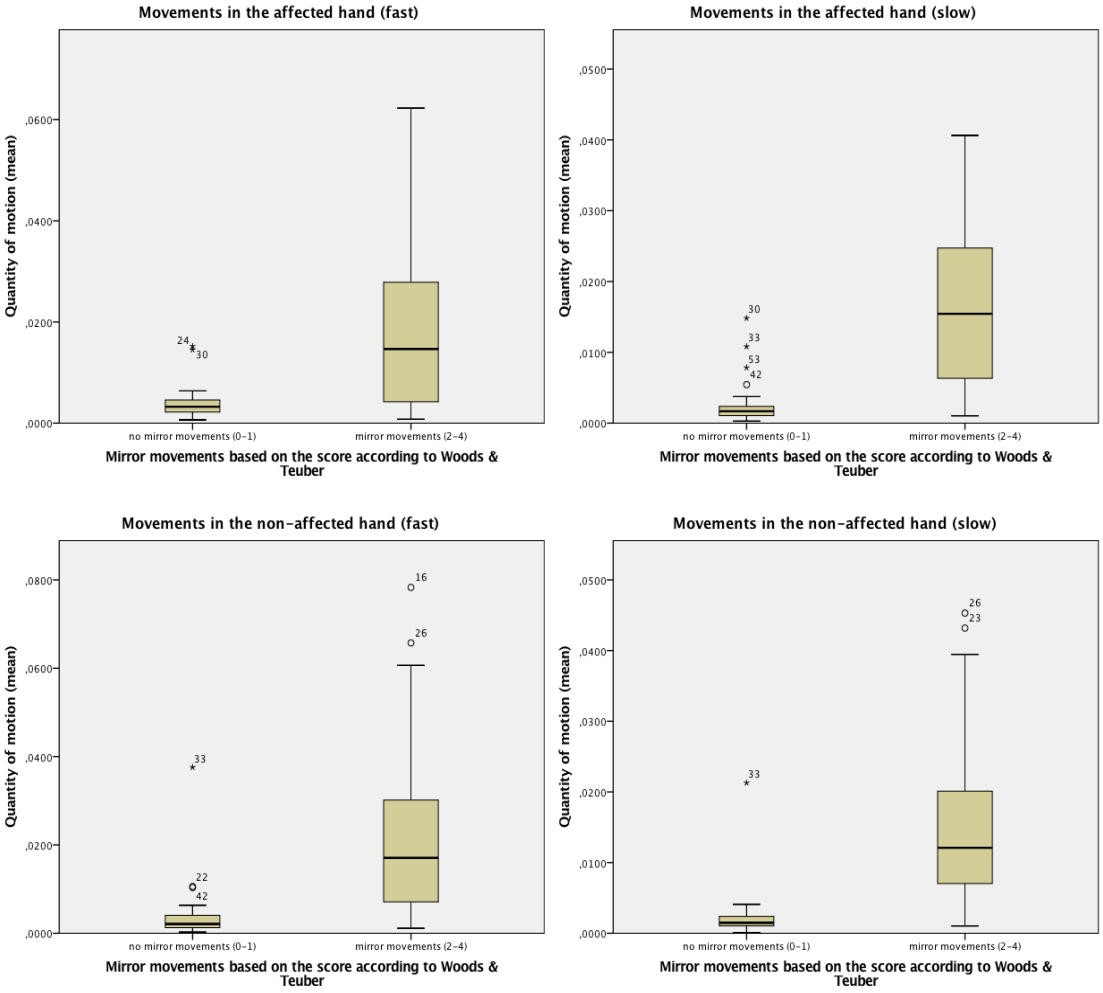


Figure 5: Boxplots of quantity of motion, mean, for the affected or non-dominant (upper panel) and the non-affected or dominant hand (lower panel) at fast (left) and slow speed (right) among all 59 participants of the study, divided in whether they had mirror movements clinically (W&T score > 1) or not (W&T score < 2)



3.2.2 Height and width of motion, standard deviation

In the entire study population, the correlation coefficient between the clinical score and HoM SD or WoM was $|\rho| > 0.63$ ($p < 0.001$), and these were the computer based variables with the highest correlation with the clinical score for mirror movements in the *affected* hand. In all cases, there was a negative correlation between HoM SD or WoM SD, and the clinical score.

Table 5: Correlations between the clinical score (W&T) and height and width of motion (standard deviation), for Trondheim (N=19) and Melbourne (N=18) separately.

	Affected hand		Non-affected hand	
Fast	Trondheim	r - 0.719 p = 0.001	Trondheim	r - 0.736 p < 0.001
	Melbourne	r - 0.648 p < 0.05	Melbourne	r - 0.523 p < 0.05
Slow	Trondheim	r - 0.843 p < 0.001	Trondheim	r - 0.720 p = 0.001
	Melbourne	r - 0.594 p < 0.001	Melbourne	r - 0.594 p < 0.05

Figure 6: Boxplots of height of motion, SD, for the affected or non-dominant (upper panel) and the non-affected or dominant hand (lower panel) at fast (left) and slow speed (right) among 37 participants with CP and 22 TD participants

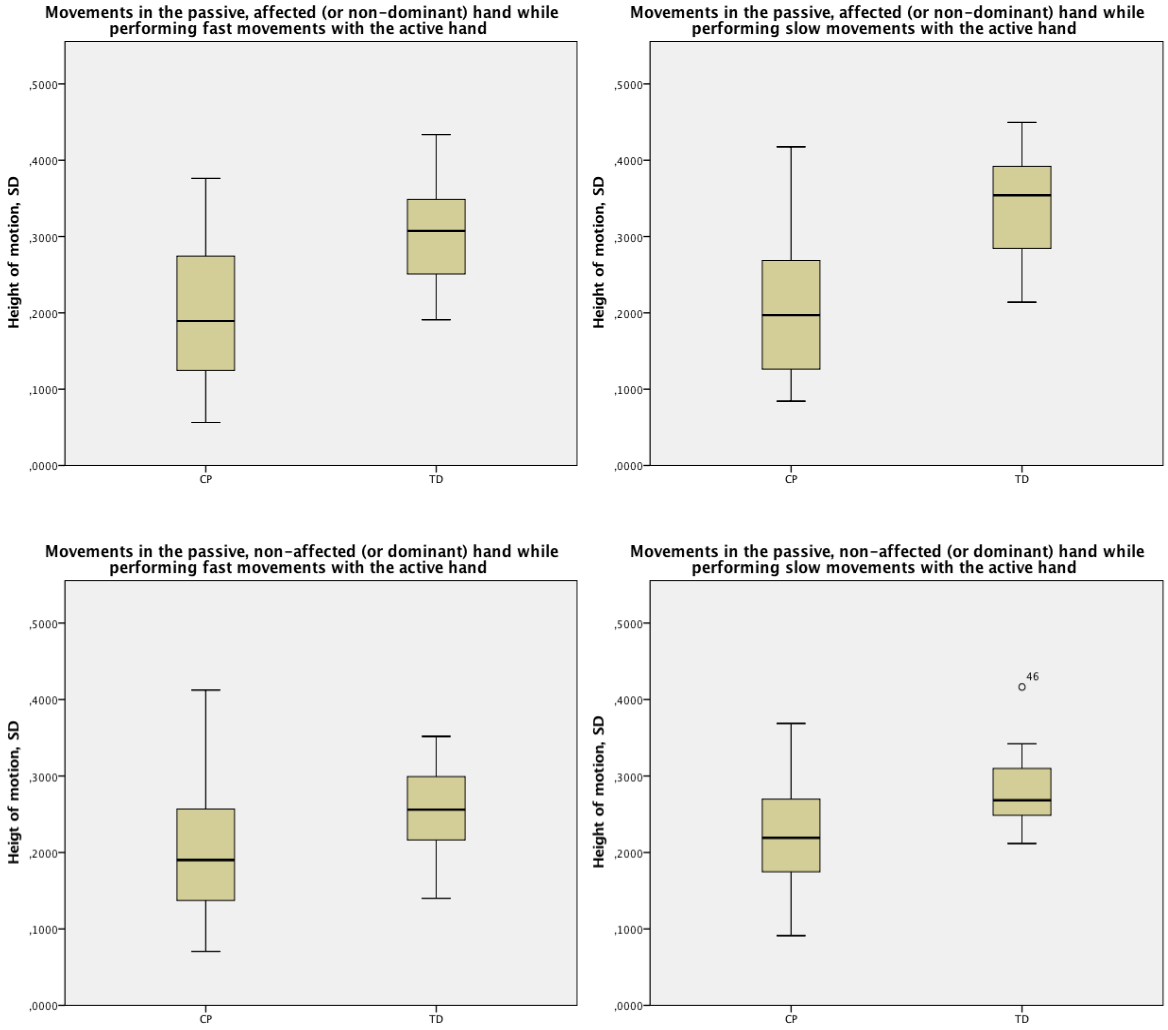
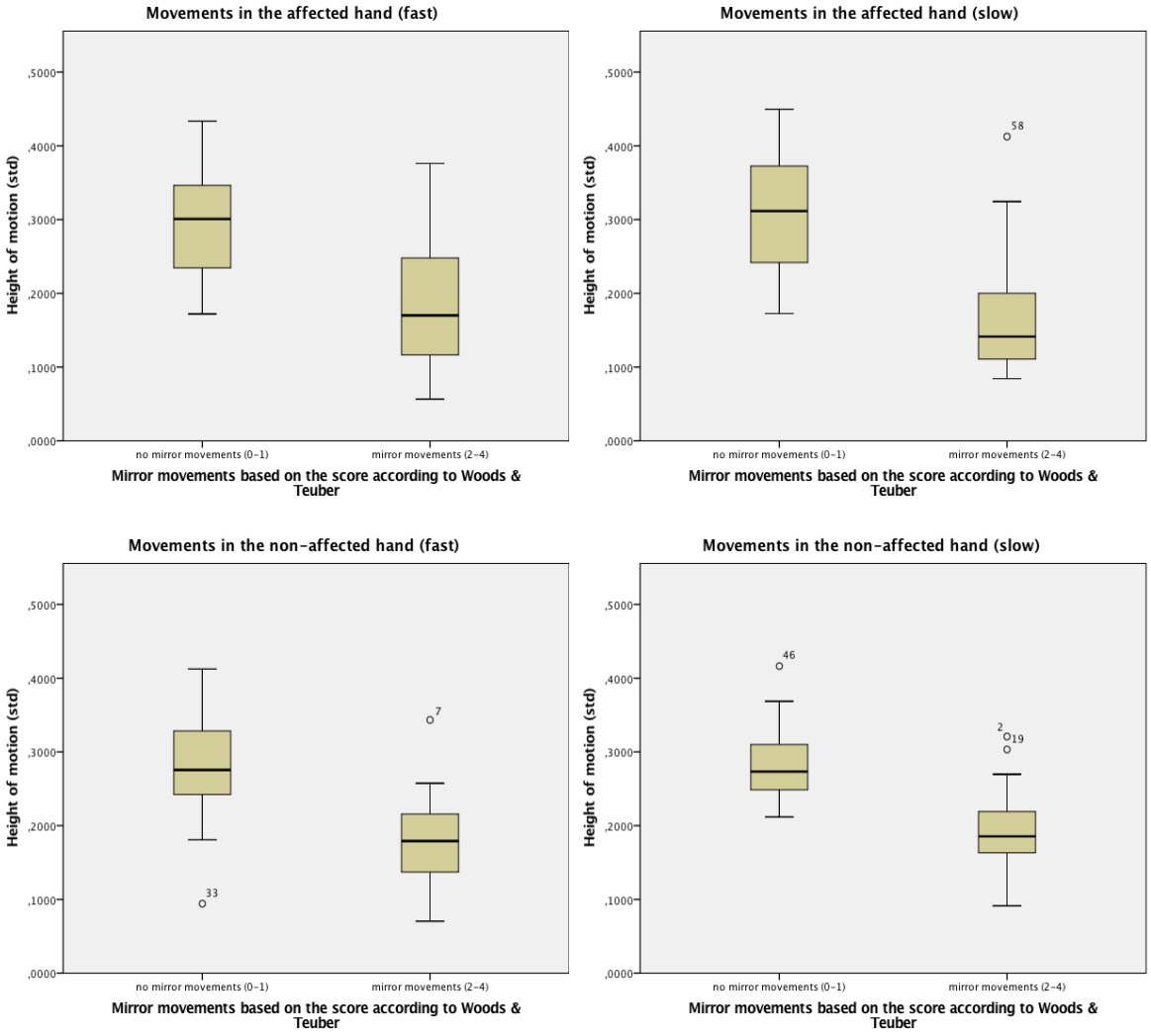


Figure 7: Boxplots of quantity of motion, mean, for the affected or non-dominant (upper panel) and the non-affected or dominant hand (lower panel) at fast (left) and slow speed (right) among all 59 participants of the study, divided in whether they had mirror movements clinically (W&T score > 1) or not (W&T score < 2)



4 Discussion

In this study we found that computer based analyses of movement in the passive hand may be used to quantify mirror movements when voluntary movement are performed by the active hand. Our results indicate that mirror movements are more common in the non-affected hand, and are more marked when the voluntary movements are performed at high speed. However, when the movements were performed at high speed, our results suggest a bigger overlap between between participants with and without mirror movements assessed clinically for the computer based variables.

4.1 Validity of the findings

4.1.1 Chance

The results of the present study are unlikely to be caused by chance, as indicated by the low p-values. However, in some of the subgroup analyses, the number of individuals is small, and lack of statistically significant findings should be interpreted with caution. This applies for some of the analyses restricted to the participants in Melbourne, where correlation coefficients of 0.33 and 0.42 failed to reach statistical significance. Therefore, more emphasis should be placed on the correlation coefficients than on the p-values when the results of this study are interpreted.

4.1.2 Confounding

Study site The major confounder in the present study is study site. In one of the sites, the recordings seem to have been taken from a greater distance than in the other, and from different angles for different particpants. In general, one could often see other parts of the body of the participant than just the hands, and even other people. It also appeared as though the participants were not seated very comfortably, with their fore-arms stretched across the examination board. In many cases, this led to the hands being very close to each other. This made it challenging to crop the films to a size that showed only one hand at a time, and in a few cases fingers belonging to the other hand would occasionally appear on the cropped film - which the computer program would register as changing pixels, thus including them in the calculated QoM. The different angles for the camera with regard to the source of light also led to more visible shadows. A human eye would not consider the moving shadows a part of mirror movements, but the computer program counts the amount of pixels changing from frame to frame, including shadows.

When we analysed the material separately for each of the sites, we found a consistent better correlation between the computer based variables and the clinical assessment for the Norwegian study population. When restricted to this group, the correlations between the two methods assessing mirror movements were excellent. This could possibly be due to differences in the quality of the recordings. Other possible explanations include the differences between the two groups, as described in 4.1.3.

Hand size We assumed hand size to be a possible confounding factor, as QoM is calculated by the sum of changing pixels, divided by the total amount of pixels in the frame. A bigger hand would generate a bigger amount of changing pixels. As all of the films were cropped to about the same size, a bigger hand would therefor also generate a bigger value for QoM. The size of the hand also effects the height and width of the area in which movement takes place, possibly influencing the HoM SD or WoM SD. However, when we performed partial correlations adjusting for approximate hand size, there were no significant changes in the correlation coefficients.

4.1.3 Bias

It is a strength of the present study that the two observers performing the clinical scoring of mirror movements were unaware of the computer based findings as well as of the identity of the participants or whether they belonged to the group of TD participants or the group of participants with CP. However, it was impossible to blind them to the site from which the participant had been recruited because of visible differences in the recordings. Nonetheless, the independent clinical scoring of mirror movements by the two observers suggested excellent inter-rater reliability of the scoring system proposed by Woods & Teuber, as indicated by a kappa $\kappa = 0.85$.

Selection bias The Norwegian participants with CP are probably representative of the general population with USCP, as all patients attending the out-patient clinic were invited to participate. This group showed a broad variation in mirror movements. However, the data suggested that the population in Melbourne might have been prone to selection- and information bias. This group was recruited through another study program. Possibly, families who are already participating in a study are the ones with children who are more severely affected by their CP - having a greater motivation to participate in research. By recruiting families through another study, it is possible that these participants in general were more affected than those recruited by inviting everyone with CP. Additionally, the Australian study population was significantly younger than the Norwegian population, with several participants under the age of 10. The fact that there were more mirror movements in the non-affected hand compared to the affected hand in this group, can

possibly be explained by their younger age. However, they also appeared to be more severely affected by their CP in comparison to the Norwegian group. This is based on their generally lower AHA-scores, indicating a more severely impaired hand function. Moreover, there was a marked lack of variability of mirror movements assessed clinically in the Australian part of the population, where only one participant had a score of zero; one had a score of three and none had a score of four. Thus, selection bias may have affected our results, resulting in too low correlation coefficients for the correlation between the score according to Woods & Teuber and QoM mean in the total study population.

Information bias (video recordings) In Melbourne, the video recordings were not obtained with strict adherence to the guidelines, thus limiting the quality of the computer based analysis.

Another bias may also be related to the fact that the computer based analysis only describes the *amount* of movement and not the *quality* of these movements - in contrast to the clinical assessment. When voluntary movements are performed at high speed in the active hand, there is a risk for unspecific co-movements in the passive hand, not reflecting mirror movements, which are not differentiated by the computer based analysis. This applies especially to the youngest participants - in whom physiologic mirror movements occur more commonly as well. Thus, the higher correlation coefficients observed at fast speed should be interpreted with caution.

Based on the score according to Woods & Teuber, there were more mirror movements when performing the tasks at fast speed. However, we found the highest correlation for mirror movements for all variables, when the tasks were performed at slow speed - regardless of the hand (affected or non-affected) performing the task. This is strengthened by the impression we got clinically, as especially the younger children had a higher occurrence of associated movements other than mirror movementes when performing the task at high speed. When performing the task at slow speed, this phenomenon disappeared. Possibly, this may explain the bigger overlap of QoM mean between children who had and who did not have mirror movements assessed clinically, when movements were performed at high speed. At inspection of the boxplots created for the different variables, it appeared as though there was the least overlap between the two groups, when the task was performed at slow speed.

We therefore assume that selection bias and information bias (i.e. suboptimal video recordings) may have affected the results obtained in the total population. Nonetheless, the correlation between the clinically assessed mirror movements and computer based

assessment of mirror movements was high ($r > 0.70$). Further, we consider the measurements from the computer based method to be most accurate when tasks are performed at slow speed, and assume the results obtained at slow speed, and in the Norwegian part of the population, to be the most robust findings of the present study.

4.2 Comparison with the literature

Consistent with previous studies, we found that mirror movements in children with USCP are more common in the non-affected hand [22, 23, 37] and more common with increasing fatigue [26], which in the present study is represented by a bigger amount of mirror movements when the tasks were performed at fast speed.

Nass [37] found mirror movements to be more pronounced in the non-affected hand, compared to the affected hand in children below the age of ten. In children and adolescents above this age, mirror movements were equally prominent in the affected and in the non-affected hand [37]. The fact that the Australian study population had more pronounced mirror movements in their non-affected hand could be due to their younger age with several participants under the age of ten.

Somewhat inconsistent with our findings, Koerte et al. found no significant difference in the occurrence of mirror movements in the dominant or non-dominant hand in their study [25]. And in contrast to our findings, they found that mirror movements were more pronounced when movements in the active hand were performed at slow speed. However, since their study population consisted exclusively of healthy TD participants with no history of any neurological disease, one may speculate that mirror movements in TD individuals may have other pathophysiological causes than in individuals with brain injuries, and this may explain the different findings.

To the best of our knowledge, quantification of mirror movements by computer based video analysis has not been attempted earlier. Other attempts to quantify them include grip objects, such as used by Kuhtz-Buschbek et al in 2000 [23]. They used a grip object that was equipped with strain-gauge transducers, being held between the thumb and the index finger by the participant. The participants then were instructed to squeeze an oval rubber bulb connected to a pressure sensor rhythmically with the contralateral hand. The authors concluded that "Recording of isometric fingertip forces during bimanual activities is a practicable sensitive method to quantify mirror activity" [23].

Koerte et al used a pressure sensor being held by the participant between the thumb and the index finger of each hand, being instructed to use the passive hand to prevent

the pressure sensor from falling down while applying pressure to the pressure sensor with the active hand [25].

These methods, like the computer based video analysis of the present study, measure the amount of movement, not the movement quality. They therefore fail to distinguish true mirror movements from other associated movements, and they must therefore be considered as proxies for mirror movements. We speculate that future development of computer based video analyses may be able to identify the quality of the movements, thus measuring "true" mirror movements.

A strength of the computer based video analysis compared to the two above mentioned approaches, is that a single task can be used both for the clinical evaluation and for the computer based assessment. Further, the necessary equipment (a camera, an examination board and a computer) is easily accessible. Lastly, computer based video analysis is not limited to assessing mirror movements of the hands - as the methods relying on precision grip between the index finger and the thumb are. Even though they are less common, mirror movements occur in the lower extremities as well [26], and could possibly be assessed using computer based video analysis.

4.3 Interpretation of results

In this study we found a moderate to good correlation between QoM mean, and the clinical assessment of mirror movements. In the Norwegian subgroup of participants, we found an excellent correlation between QoM mean and the score according to Woods & Teuber. We further found a good to excellent correlation between HoM SD and WoM SD, and the clinical assessment.

4.3.1 Quantity of motion

QoM mean was the video based variable that we found to best reflect mirror movements. This variable had the best correlation of all the computer variables for mirror movements in the non-affected hand for the entire study population. As mirror movements appear to be more common in the non-affected hand, a method that quantifies mirror movements would be required to be reliable especially for this hand. However, we found the overall best correlation for QoM mean in the Norwegian subgroup for mirror movements in the *affected* hand, when task 1 was performed at *fast* speed.

4.3.2 Height and width of motion, SD

Other potential variables of the computer based video analyses that might have been considered to reflect mirror movements were HoM SD and WoM SD. These variables had similar high correlations with the clinical assessment of mirror movements - in particular for mirror movements in the affected hand. However, Tamhane and Dunlop emphasize that the standard deviation should only be used as a measure of dispersion, if the data is symmetrically distributed [36]. Assuming a film in which the changes in HoM or WoM are not symmetrically distributed, this makes it difficult to interpret what the standard deviation represents. Finally, the interpretation of these variables is further complicated by the fact that there was no correlation between clinical scores and the corresponding mean values (HoM mean and WoM mean).

HoM SD and WoM SD had identical correlation coefficients with the clinical assessment in almost all cases. In the present study, task 1 was opening and closing the fist, which is a movement that takes place in both the horizontal and the vertical plane. Thus, those two variables contain similar information.

5 Conclusion

Computer based video analysis of involuntary movements in the passive hand can be used as a proxy to quantify mirror movements in children and adolescents with USCP, aged six to 20 years, when voluntary movements are performed at a speed coming naturally to the participant. However, it is extremely important to adhere strictly to the guidelines for the video recordings.

The method has the potential to become a useful tool in evaluating the effect of therapeutic interventions, but further studies are required to assess its responsiveness.

6 Acknowledgements

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Appendix

Table 6: Descriptive table of mirror movements according to Woods & Teuber

		MM in the affected hand*		MM in the non-affected hand**	
	W&T score	Slow speed	Fast speed	Slow speed	Fast speed
CP Norway	0	4	2	6	3
	1	6	3	3	4
	2	3	7	7	6
	3	3	5	1	2
	4	3	2	2	4
CP Australia	0	1	3	1	1
	1	4	3	2	2
	2	12	10	11	10
	3	1	2	3	3
	4	0	0	1	2
TD Children	0	16	9	13	8
	1	5	9	9	8
	2	1	4	0	6

* while performing task 1 with the non-affected hand

** while performing task 1 with the affected hand

Table 7: Correlations between clinical score (W & T) and computer based variables.

Affected hand, fast*

Entire population (N=59)			Only children with CP (N=37)			Adjusted for age (N=37)		
	r_S	p		r_S	p		r_S	p
qom (mean)	.587	.000	qom (mean)	.637	.000	qom (mean)	.617	.000
qom (std)	.527	.000	qom (std)	.489	.002			
aom (mean)	.135	.310						
aom (std)	−.423	.001						
hom (mean)	.207	.117						
hom (std)	−.628	.000	hom (std)	−.603	.000	hom (std)	−.629	.000
wom (mean)	.260	.046						
wom (std)	−.628	.000	wom (std)	−.603	.000	wom (std)	−.629	.000
com x (mean)	−.072	.587						
com x (std)	−.528	.000	com x (std)	−.467	.004			
com y (mean)	−.212	.108						
com y (std)	−.633	.000	com y (std)	−.554	.000	com y (std)	−.562	.000

*MM in the affected hand while the non-affected hand is moving at fast speed

Table 8: Correlations between clinical score (W & T) and computer based variables.

Affected hand, slow*

Entire population (N=59)			Only children with CP (N=37)			Adjusted for age (N=37)		
	r_S	p		r_S	p		r_S	p
qom (mean)	.674	.000	qom (mean)	.721	.000	qom (mean)	.688	.000
qom (std)	.558	.000	qom (std)	.600	.000	qom (std)	.504	.002
aom (mean)	.010	.943						
aom (std)	−.640	.000	aom (std)	−.380	.020			
hom (mean)	.048	.715						
hom (std)	−.761	.000	hom (std)	−.711	.000	hom (std)	−.680	.000
wom (mean)	.305	.019						
wom (std)	−.761	.000	wom (std)	−.711	.000	wom (std)	−.680	.000
com x (mean)	−.389	.002						
com x (std)	−.551	.000	com x (std)	−.526	.001	com x (std)	−.420	.011
com y (mean)	−.160	.225						
com y (std)	−.729	.000	com y (std)	−.561	.000	com y (std)	−.559	.000

*MM in the affected hand while the non-affected hand is moving at slow speed

Table 9: Correlations between clinical score (W & T) and computer based variables.

Non-affected hand, fast*

Entire population (N=59)			Only children with CP (N=37)			Adjusted for age (N=37)		
	r_S	p		r_S	p		r_S	p
qom (mean)	.744	.000	qom (mean)	.742	.000	qom (mean)	.719	.000
qom (std)	.681	.000	qom (std)	.640	.000	qom (std)	.574	.000
aom (mean)	.137	.299						
aom (std)	−.302	.020						
hom (mean)	.298	.022						
hom (std)	−.704	.000	hom (std)	−.624	.000	hom (std)	−.598	.000
wom (mean)	.327	.012						
wom (std)	−.645	.000	wom (std)	−.624	.000	wom (std)	−.598	.000
com x (mean)	−.039	.767						
com x (std)	−.682	.000	com x (std)	−.666	.000	com x (std)	−.600	.000
com y (mean)	−.028	.835						
com y (std)	−.730	.000	com y (std)	−.654	.000	com y (std)	−.691	.000

*MM in the non-affected hand while the affected hand is moving at fast speed

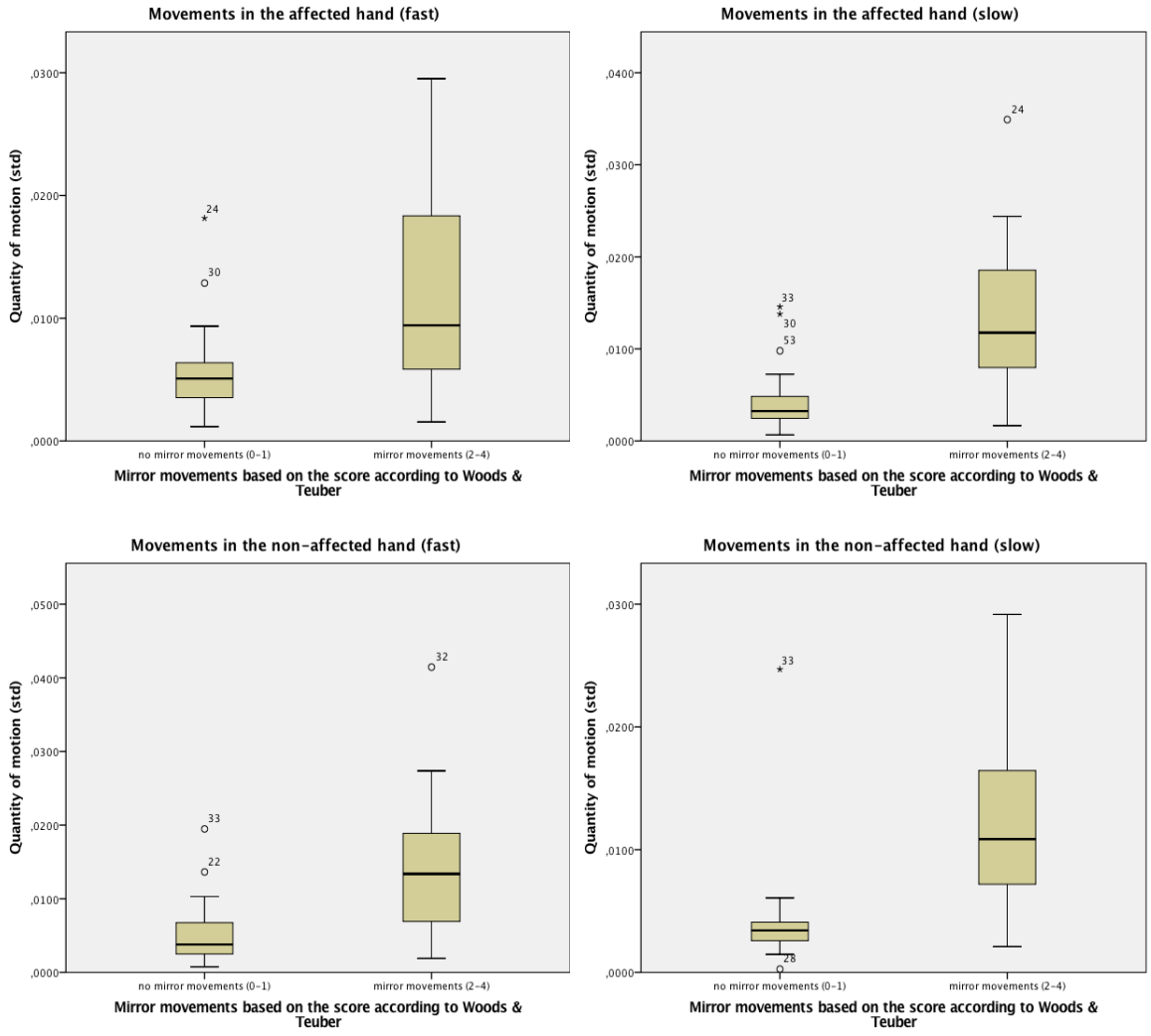
Table 10: Correlations between clinical score (W & T) and computer based variables.

Non-affected hand, slow*

Entire population (N=59)			Only children with CP (N=37)			Adjusted for age (N=37)		
	r_S	p		r_S	p		r_S	p
qom (mean)	.742	.000	qom (mean)	.708	.000	qom (mean)	.609	.000
qom (std)	.720	.000	qom (std)	.661	.000	qom (std)	.552	.000
aom (mean)	.082	.537						
aom (std)	−.494	.000						
hom (mean)	.424	.001						
hom (std)	−.691	.000	hom (std)	−.721	.000	hom (std)	−.665	.000
wom (mean)	.168	.204						
wom (std)	−.691	.000	wom (std)	−.721	.000	wom (std)	−.665	.000
com x (mean)	.181	.170						
com x (std)	−.620	.000	com x (std)	−.574	.000	com x (std)	−.563	.000
com y (mean)	−.523	.000	com y (mean)	−.420	.010			
com y (std)	−.596	.000	com y (std)	−.620	.000	com y (std)	−.509	.002

*MM in the non-affected hand while the affected hand is moving at slow speed

Figure 8: Quantity of motion (std) for both hands at both speeds. Entire study population (N=59)



The reader is advised to pay attention to the different scales along the y-axis

Figure 9: Width of motion, SD, for both hands at both speeds. Entire study population (N=59)

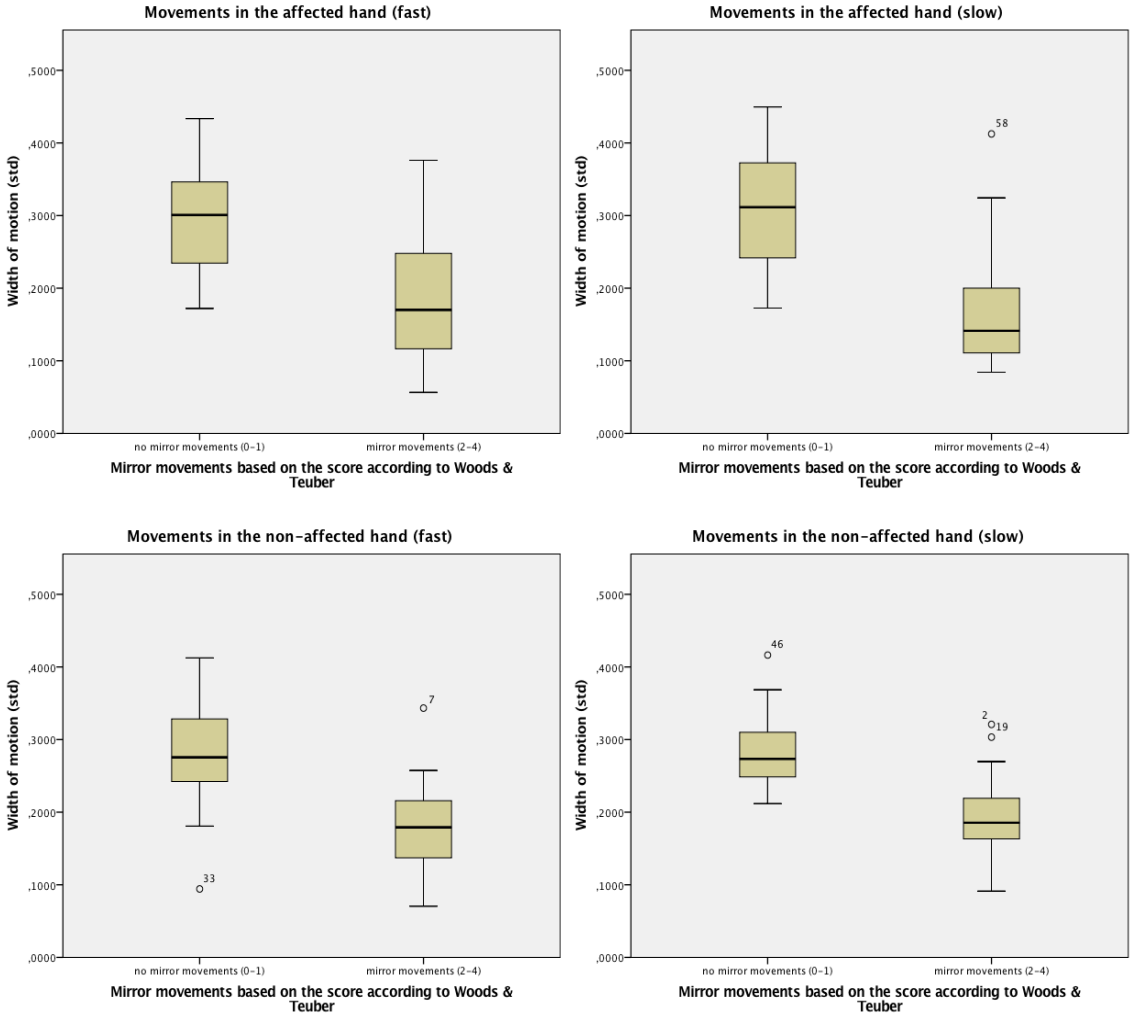


Figure 10: Correlations between quantity of motion, mean, and the clinical assessment for children with CP (N=37)