Temporal analysis of regional anaesthesia-induced sensorimotor dysfunction: a model for understanding phantom limb

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Key points

- Regional anaesthesia (RA) provides a unique model of selective and reversible transient deafferentation.
- We have described a model which can be used for understanding phantom limb phenomenon.
- We individualized during RA a set of kinesthetic distortions with respect to other phantom limb experiences.
- Further studies could lead to development of interventions to minimize phantom limb phenomenon.

Background. The peripheral deafferentation induced by regional anaesthesia (RA) results in misperception of size-shape (S) and posture (P) of the anesthetized limb. During RA, most patients seem to describe motionless `phantom limbs' fixed in stereotyped illusory positions, suggesting that RA could unmask stable postural patterns. The question of whether movement illusions exist or not after anaesthesia needs a prospective study. This study aimed to describe the phenomenology of RA-induced kinesthetic illusions (K illusions).

Methods. We examined prospectively the body image alteration during infraclavicular blocks in 20 patients. Multimodal sensory testing (pinprick, heat-cold, pallesthesia, and arthrokinesia) and assessment of motor function were performed every 5 min for 60 min after administration of the local anaesthetics. Meanwhile, patients described phantom limb sensations (S, P, and K illusions).

Results. We individualized the occurrence of K illusions [44 (8) min] with respect to S illusions [7 (3) min; $P<0.005$] and P illusions [22 (4) min; $P<0.001$]. A close relationship between the onset of K illusions and proprioceptive impairment (arthrokinesia: $r=0.92$, $P<0.001$; pallesthesia: $r=0.89$, $P<0.0001$) and abolishment of motor activity ($r=0.83$, $P<0.001$) was identified. Finally, a principal component analysis showed that S and P illusions were essentially related to the proprioceptive impairment.

Conclusions. This study analyses for the first time the temporal evolution of sensorimotor dysfunction and the onset of K illusions during RA. Our results suggest the involvement of an alteration of proprioception and motor functions in the origin of this phenomenon. These data agree with the motor awareness theory.

Keywords: anaesthetic techniques, regional, brachial plexus; brain, cerebral cortex; recovery, cognitive; recovery, psychomotor

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The peripheral deafferentation induced by regional anaesthesia (RA) results in misperception of size, shape,1 2 and posture3 4 of the anaesthetized limb. All of these perceptual distortions are known under the term of ‘phantom limb’ experiences, in analogy with the clinical picture described in amputees.5 6 In the latter, the phantom limb phenomena generally come along with kinesthetic illusions7 (i.e. illusion of limb movement), probably related to brain plasticity mechanisms.8 Conversely, during the acute and transient deafferentation induced by RA, previous works have suggested that RA could unmask a stable postural pattern and not induce a change in movement representation (i.e. motionless phantom limbs).5

Our hypothesis was that kinesthetic illusions exist after transient and short-term deafferentation (under RA) and

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also after chronic deafferentation (in amputees) and under-
line the same mechanisms of brain plasticity. This study
aimed to fully describe the phenomenology of RA-induced
kinesthetic illusions, which are not routinely evaluated after
regional block. We examined prospectively the characteris-
tics of body image alteration during RA of the upper extremity.
Multimodal sensory testing and assessment of motor function
were performed at regular intervals after administration of the
local anaesthetics, and the temporal relationship between the
subjective reports of the patients and the progression of
sensory and motor impairment was analysed. In addition,
since visual input interferes with body representation10 and
posture illusions,3 4 we sought to determine the influence of
visual information from the anaesthetized limb on these
different perceptual illusions including kinesthetic illusions.

Methods

The patients gave their consent in accordance with the prin-
ciples of the Helsinki convention. This work was approved by
the local review committee (Comité Consultatif pour la Pro-
tection des Personnes, CHU Toulouse, France; Ref.
2007-A01225-48). Twenty patients undergoing orthopaedic
surgery of the upper limb under RA participated in the
study. The exclusion criteria were the existence of a neuro-
logical or psychiatric disease, diabetes mellitus, and
cutaneous infection at the site of the needle puncture.

Standard monitoring was applied to all patients under-
going nerve blocks. All patients were anaesthetized using
the same infraclavicular brachial plexus technique.11 All
blocks were performed with the arm and forearm placed
alongside the body. Immediately after anaesthesia, the
limb was placed in abduction (arm 90° with respect to the
body) with the elbow in extension (180°) to avoid contact
of the studied limb with non-anaesthetized parts of the
body. The anaesthetized limb was hidden from the patient’s
sight just before the block was performed. After cutaneous
landmarks had been drawn on the skin and after disinfection,
a 22 G 100 mm insulated needle, connected to a peripheral
nerve stimulator was used to identify one of the following
nerves according to their specific motor-evoked responses
as follows: median, ulnar, radial, and musculocutaneous.
Specific motor responses were sought with the nerve stimu-
lator set at 1 Hz frequency, 100 μs, and a current of 1.5 mA,
then progressively reduced to 0.5 mA before injection of local
anaesthetic solution (0.75% ropivacaine). A single injection
protocol was used in all cases. The total volume of ropiva-
caine administered was 30 ml.

Assessment of sensory and motor functions

The nerve block was defined as the abolition of sensory
(heat-cold, arthrokinesia, pallesthesia, pinprick) or motor
function (voluntary movement) in one of the main distri-
butions of the brachial plexus (musculocutaneous, median,
radial, and ulnar nerves) 60 min after the initial adminis-
tration of local anaesthetics. Patients could drop out of the
study in the case of a level of pain >30 on a visual analogue
scale ranging from 0 (no pain) and 100 (maximal imaginable
pain) or failure of the nerve block initially performed. No
patient received any sedative or opioid drug before or
during the study period. The following sensory and motor
function testing were assessed immediately after the end
of block placement and then every 5 min for 60 min in the
territory of the last blocked nerve to check on the quality of
the anaesthesia: (i) the sensations elicited by pinprick, cold
(glass tube containing water at 16°C), heat (glass tube con-
taining water at 42°C); (ii) the accuracy of proprioception
assessed by arthrokinesis (perception of the mobilization of
a joint within the deafferented area) and pallesthesia (per-
ception of vibration applied at the level of a joint using a
tuning fork within the deafferented area); and (iii) the volun-
tary movement. For all the sensory and motor assessments,
a scale was used to facilitate assessment.1 2 4 Normal sen-
sation or movement was scored a 2, blunted sensation or
moderately impaired movement was a 1, and an absence
of sensation or movement was scored zero. Furthermore,
sensory and motor dysfunctions were also assessed in all
other territories of the anaesthetized limb 15, 45, and 60
min after the end of the block placement, to evaluate the
overall quality of the block before the surgery. The same
scale as before was used.

Assessment of perceptual illusions

Patients spontaneously reported perceptual distortions,
before they were encouraged every 5 min for 60 min to
describe their sensations.1 2 4 Finally, the visual mask was
removed 60 min after the block placement and perceptual
distortions were analysed immediately and then every
5 min for 15 min.

Statistical analysis

The data (timings of the occurrence of illusions or onset
times) are expressed as mean (SD). The normality of the
data was verified using a Kolmogorov–Smirnov test. The
two means were compared using Student’s t-test. A principal
component analysis (PCA) with Varimax rotation12 (only for
the axis with initial eigenvalue >1) was applied to all the
standardized onset times of perceptual distortions and
sensory dysfunctions. All P-values were two-sided and
values of <0.05 were considered as significant. The analyses
were performed using Statistica© 7.0.

Results

Twenty consecutive patients (no refusals) were included in
the study (Table 1). At the end of the study, all the subjects
underwent the scheduled surgery, without requiring
additional nerve blocks, nor perioperative injections of seda-
tive or opioid drugs.

Perceptual illusions of size, shape, and posture

Misperception of size or shape (swelling illusion or S illusion)
and posture (postural illusion or P illusion) were identified in
all subjects (Fig. 1). The mean onset times were 7 (3) and 22 (4) min for S and P illusions, respectively (P<0.005). In all cases, S illusions preceded P illusions (Fig. 1). During the P illusions, the anaesthetized limb was perceived in adduction, incomplete abduction, or elbow flexion by 8 (40%), 6 (30%), and 6 patients (30%), respectively. In the majority of cases, the phantom limb was raised with respect to the actual posture of the limb (80%). PCA allowed us to study the temporal relationships between the onset of perceptual illusions (S and P illusions), and the progression of sensory impairments. The two first Varimax components uncover the fact that the occurrence of sensitive impairment (heat-cold, pinprick) correlated well to principal component 1 and was only very slightly related to the occurrence of perceptual illusions (component 2). The occurrence of perceptual illusions (S and P illusions) seems essentially related to the proprioceptive impairment evaluated by kinesthesia and pallesthesia, as is suggested by their correlations with principal component 2 (Fig. 2).

**Perceptual illusions of movement**

Fifteen patients (75%) spontaneously described kinesthetic illusions (K illusions). The median onset time of these perceptual distortions was 44 (8) min (Fig. 3). K illusions appeared after the change of posture and size illusions (K vs P illusions, P<0.005; K vs S illusion, P<0.001). The patients described wrist flexion-extension, hand opening and hand closing, or elbow flexion-extension illusory movements in 8 (40%), 4 (20%), and 3 (15%) patients, respectively. A voluntary control of the ‘phantom limb’ was described in half of the cases. Finally, we demonstrated a significant correlation between the onset of these illusions, the abolition of proprioception assessed by two different approaches (Fig. 4A and B) and the abolition of motor function (Fig. 4C).

**Visual information and body image distortion**

We assessed the effect of incongruent visual information on each component of the phantom limb sensations (S, P, and K illusions). Only one subject (5%) described an effect of the view of the hidden limb on the S illusion. However, the same visual information causes a rapid superposition of the position of the phantom limb with the real posture of the anaesthetized limb (i.e. “fusion phenomenon”) in all subjects. In 18 subjects (90%), the reintroduction of the visual mask came along with the reappearance of the P illusion (“recurrence phenomenon”). Six subjects (30%) described the spontaneous disappearance of phantom movement sensations before the removal of the visual mask [mean duration of phantom movement 12 (5) min]. In the remaining subjects, the vision of their anaesthetized limb caused fusion phenomena followed by recurrence of K illusions in all cases.

### Table 1 Main characteristics of the patients. The values are expressed as mean (range) or mean (SD). ASA, American Society of Anesthesiologists (i.e. score of fitness of patients before surgery)²³

<table>
<thead>
<tr>
<th>Patients (n=20)</th>
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</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>35 (21–46)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69 (19)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 (8)</td>
</tr>
<tr>
<td>Male/female</td>
<td>15 / 5</td>
</tr>
<tr>
<td>ASA physical status (n)</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>14</td>
</tr>
<tr>
<td>II</td>
<td>6</td>
</tr>
<tr>
<td>Site of surgery</td>
<td></td>
</tr>
<tr>
<td>Hand</td>
<td>14</td>
</tr>
<tr>
<td>Wrist</td>
<td>3</td>
</tr>
<tr>
<td>Elbow</td>
<td>3</td>
</tr>
</tbody>
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**Fig 1** Temporal relationship between the occurrence of the perceptual illusion of size (S illusion) and the postural illusions (P illusions). The size or shape misperceptions occurred concomitantly (“swelling sensation”). The onset of S illusions preceded that of P illusions in all patients.
In fact, being conscious of an action would depend essentially on the activation of ‘efference copies’, representations of the prediction of the consequences of the movement to be executed (i.e. sense of agency). Ventral premotor cortex has been shown to be partly involved, but all the cerebral substrates of these processes need to be further described. The efference copies would be related to the integrity of the controllers (motor command) and predictors (estimate of the consequences before the sensory feedback). In the absence of sensory feedback, the perception of movement illusion would be based more on the prediction of sensory consequences of potential movement than on the integration of the actual status of the limb. From these theoretical data, the deafferentation related to the RA and thus the loss of sensory afferents could generate kinesthetic illusions, caused by a persistent motor command and the production of efferent copies.

**Perceptual illusions and proprioceptive dysfunction**

Previous studies found a correlation between P illusions and impairment of proprioception (arthrokinesia). However, they did not find any correlation between S illusions and any sensory modality which could explain the origin of the phenomenon. In the present study exploring pallesthesia andarthrokinesia, we found both correlations (Fig. 2). During RA, we identified a set of perceptual illusions for which the onset lags with time (S and P illusions, Figs 1 and 3), suggesting the involvement of distinct neuronal substrates. Although selective and progressive alteration of afferent routes has been used to explain this phenomenon, central reorganizational changes could participate in the genesis of anaesthesia-induced perceptual illusions. Perceived changes in the posture, size, and shape of body parts are probably mediated by hierarchically higher-order somatosensory areas in the parietal cortex. In fact, works carried out using functional brain imaging have allowed the identification of distinct brain representations for the senses of size-shape (anterior part of intraparietal sulcus) or posture (premotor cortex and area 5 in monkey parietal cortex, a dorsal premotor-parietal circuit in humans) of the body.

**Influence of visual sensory inputs in phantom sensations**

We identified fusion and recurrence phenomena for K illusions and for P illusions during controlled visual exposure of the deafferented limb. Many studies have suggested that visual information plays a crucial role in the construction and preservation of the body schema. For example, postural or kinesthetic illusions have been recreated in amputees using mirrors that allowed the missing limb to be seen. Recent studies in functional magnetic resonance imaging are in favour of the existence of polymodal regions in the human ventral premotor cortex and the depth of intraparietal sulcus. In contrast, the lack of influence of vision on the S illusions in the present study suggests that the neurophysiological mechanism involved in this type of illusion take place at another, presumably less integrated, level of body image construction.

**Discussion**

RA provides a unique model of selective and reversible transient deafferentation, and constitutes an original way to explore the short-term interactions between peripheral and central nervous systems. We addressed whether RA-induced perceptual illusions could be used as a model of understanding the changes in body schema brain representations, linked to brain or peripheral nerves injuries. Moreover, clinical importance of using these anaesthetic procedures to modulate the brain plasticity was suggested by several studies. For example, RA of the healthy hand in patients with chronic stroke was associated with significant motor and sensory improvement of the affected hand, probably due to interhemispheric plasticity processes.

**Phantom limb movement and the neural basis of the motor awareness**

Our work is the first to describe in detail the onset of kinesthetic illusions during RA. We individualized the set of kinesthetic distortions with respect to other phantom limb experiences (S and P illusions, Fig. 3). Finally, we demonstrated a close relationship between the proprioceptive and motor dysfunction, and the onset of these phenomena (Fig. 4). The theory of ‘motor awareness’ could afford an explanation of the phenomenology of phantom limb movement. Numerous studies have demonstrated that the conscious movement experience is generated between the time when we plan to perform the action and instant of its execution. In fact, being conscious of an action would depend essentially on the activation of ‘efference copies’, representations of the prediction of the consequences of the movement to be executed (i.e. sense of agency). Ventral
Fig 3 Temporal relationship between sensorimotor dysfunction (alteration of motor function and proprioception) and the onset of phantom limb sensations (S, P, and K illusions). The onset time of perceptual illusions and the start of the alteration (score 1) of proprioception (assessed by pallesthesia and kinesthesia) and motor function are represented. S illusion, misperception of size or shape (i.e. ‘swelling illusion’); P illusion, postural illusions; K illusion, kinesthetic illusion. Medians (quartile and range).

Fig 4 Correlation between the onset of kinesthetic illusions (K illusions) and the abolition (score 0) of proprioception or motor function. Proprioception was assessed by pallesthesia (A) and kinesthesia (B). The abolition of motor function corresponds to an absence of voluntary movement (C).
In conclusion, this study comparatively analyses for the first time, the temporal evolution of sensorimotor dysfunction and the onset of kinesthetic illusions during RA. Our results suggest the involvement of an alteration of proprioception and motor functions in the origin of this phenomenon. These data agree with the motor awareness theory. Furthermore, we confirmed the existence of size, shape, and postural misperceptions for which the onset seems strongly related to an alteration in proprioception. Only postural and kinesthetic illusions seem influenced by the sight of the deafferented limb. This element is in favour of the polymodal nature of the underlying neuronal substrate. The phenomenon has been fully characterized and future studies can now focus on its brain correlates. This work could pave the way for future clinical and functional brain-imaging studies linking peripheral anaesthesia and brain plasticity. Thereby, modulation of afferent inputs by RA could become a new tool in neurorehabilitation in the not so distant future.

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Conflict of interest
None declared.

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