CHARLES UNIVERSITY IN PRAGUE
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DEPARTMENT OF PHYSIOTHERAPY

Physiotherapy Case-study: Rehabilitation of a Patient after Ischemic Cerebrovascular Accident

BACHELOR THESIS

Author: Giammaria Cattozzo
Academic supervisor: PhDr. Jitka Malá, Ph.D.
Structure supervisor: Bc. Aleš Nesvadba
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ABSTRACT

The aim of the present thesis is to present the case-study of a patient during the rehabilitation from an ischemic cerebrovascular accident (iCVA) in acute and sub-acute state. This document is divided into two parts: theoretical and practical.

The former presents an introduction to the iCVA, its pathophysiology, incidence, main causes, somatic effects and physiotherapeutic treatments.

The latter, the kinesiological examination of the patient (initial and final) and the daily procedures performed during the rehabilitation period.

In the end, a comparison between the initial and conclusive kinesiologic examination is presented, aiming to highlight the beneficial effects of the treatment.

The structure where the practical realisation of the case-study took place is the Rehabilitační nemocnice Beroun. The placement period went form the 18th January until the 12th February 2021.

Keywords: ischemic cerebrovascular accident, iCVA, ischemic stroke, case study, spasticity, physiotherapy.

ABSTRACT

Cílem této diplomové práce je představit případovou studii pacienta během rehabilitace z ischemické cerebrovaskulární příhody (iCVA) v akutním a subakutním stavu. Tento dokument je rozdělen na dvě části: teoretickou a praktickou.

První představuje úvod do iCVA, její patofyziologie, incidence, hlavních příčin, somatických účinků a fyzioterapeutické léčby.

Posledně jmenované, kineziologické vyšetření pacienta (počáteční a konečné) a denní postupy prováděné během rehabilitačního období.

Na závěr je představeno srovnání mezi počátečním a nezvratným kineziologickým vyšetřením s cílem zdůraznit příznivé účinky léčby.

Strukturou, kde k praktické realizaci případové studie došlo, je Rehabili-tační nemocnice Beroun. Období umístění probíhalo od 18. ledna do 12. února 2021.

Klíčová slova: ischemická cerebrovaskulární příhoda, iCVA, ischemická cévní mozková příhoda, případová studie, spasticita, fyzioterapie.

Declaration

I sincerely declare that this bachelor's thesis was managed and fulfilled by myself, under the supervision and instructions of PhDr. Jitka Malá, Ph.D. All information and clinical procedures, which are presented in this thesis, are based on a scientific bibliography and on the competences acquired during my academic studies at The Faculty of Physical Education and Sports of Charles University in Prague. My thesis was performed under the supervision of Bc. Aleš Nesvadba at Rehabilitační nemocnice Beroun, Czech Republic.

Giammaria Cattozzo

Prague, 2021

Abbreviations

1aSN	1st afferent Sensory Neuron
AARoM	Active Assisted RoM
ACA	Anterior Cerebral Artery
AcomA	Anterior communicating Artery
ADL	Activities of Daily Living
APT	Adiponectin
ARoM	Active RoM
BBB	Blood-Brain Barrier
BDNF	Brain-Derived Neurotrophic Factor
C1q	Complement component 1q
CB	Corticobulbar
CBF	Cerebral Blood Flow
ChA	Choroidal Artery
CNS	Central Nervous System
CRP	C-Reactive Protein
CS	Corticospinal
CVA	Cerebrovascular Accident
CVD	Cardiovascular Disease
DN	Dry needling
Ephrin-Eph	Erythropoietin-producing hepatocellular
GRAr	Gait Robotic Assisted rehabilitation
hCVA	Haemorrhagic CVA
ICA	Internal Carotid Artery
iCVA	ischemic CVA
IhCVA	Intracerebral Haemorrhagic CVA
LE(E)	Lower Extremity (-ies)
LMC	Leptomeningeal Collateral
LMN	Lower Motor Neuron
LPT	Leptin
MCA	Middle Cerebral Artery
MLT	Medial Lemniscal Tract

MMP	Matrix Metalloproteinase
NDC	Neural-death Signal
NDMAR	N-methyl-d-aspartate receptor
NVU	Neurovascular Unit
PCA	Posterior Cerebral Artery
PcomA	Posterior Communicating Artery
PNF	Proprioceptive Neuromuscular Facilitation
PNS	Peripheral Nervous System
PRoM	Passive RoM
PT	Physiotherapy
RoM	Range of Motion
ShCVA	Subarachnoid Haemorrhagic CVA
STT	Spinothalamic Tract
TIA	Transient Ischemic Attack
TNF-α	Tumor Necrosis Factor Alpha
UE(E)	Upper Extremity (-ies)
UMN	Upper Motor Neuron
VEGF	Vascular Endothelial Growth Factor
VPL	Ventro-postero-lateral nucleus of Thalamus
VR	Virtual Reality

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1. Introduction

The aim of the present thesis is to highlight the main physiotherapeutic approaches involved in the rehabilitation of an Ischemic Cerebrovascular Accident (iCVA), also named Ischemic Stroke or Brain Attack. The initial part of this written production will present the main tracts of the central nervous system from an anatomical and functional point of view. Furthermore, I will move to the etiology and pathogenesis of the iCVA establishing a correlation within the affected artery, the affected district of the encephalon and the clinical sings. Another part is dedicated to the pathologies predisposing to the iCVA and the markers which should be observed for assessing its predisposition. In the conclusive chapter of the theoretical part, I will introduce different physiotherapeutic methods employed for the iCVA rehabilitation.

The second part is destinated to the Case Study of a patient that I followed during the rehabilitation process after a recent iCVA. The rehabilitation was held in the period going from the second half of January until the beginning of February (present year).
The placement took place in "Rehabilitační nemocnice Beroun". During the period going from the 18th January until the 12th February 2021, the patient underwent 9 physiotherapeutic session with me, under the direct supervision of Bc. Aleš Nesvadba. In this part, I present the kinesiological examination (initial and final) and the resumption of the daily examination of the patient, highlighting subjective and objective condition, going through an assessment and proposing a plan on the short and long term.

2. General part

Functionally, the CNS can be divided into different pathways: efferent (motoric pathway) and afferent (somatosensory pathways). Their function is respectively to transmit motoric orders from the CNS to the target muscles and to carry the perceptive information from the periphery to the superior centres.

2.1. Central Nervous System

2.1.1. Motoric Pathway

The motor pathway is composed by different structures. Underneath, the components belonging exclusively to the CNS are be listed in cranio-caudal order.

The highest centre is the cerebral cortex, which specific function depends on the location. The pyramidal tract is composed of corticospinal (CS) and corticobulbar (CB) tracts. The former controls limbs and trunk's motion, while the latter, head motion and facial mimic (11). The pyramidal pathway originates in the frontal motor area more exactly in the posterior limb's posterior part (internal capsule), and its function is connected with prefrontal, premotor and supplementary cortex. The former is involved in movement planification and commencement; the second and the latter in movement modulation.

Despite the common opinion, the parietal lobe is not only involved in perception but also in movement guidance together with association areas which role is to reunite information respectively belonging to the sensory system and to the motoric system, aiming to perform precise and sharp movements.
The subcortical centres (*basal ganglia* and *cerebellum*) which role is to ensure adequate muscle tonus, posture and movement coordination follow the superior cortex centres. Furthermore, CS and CB motor impulses travel through the brainstem entering the *pons* through the cerebral peduncle's feet. The *truncus encephalicus* mainly controls automatic actions (e.g. breathing, heart rate) (11; 51).

From this point, CB and CS divide their path. CB will project their information into the cranial nerves, not descending lower than the *medulla oblongata*. CS instead, will descent more along the spinal cord only after decussating (90% of the total amount of

fibres) at the level of the spinal cord's cranial entrance. In the spinal cord, the upper motor neuron (UMN) impulses travel in the white matter (lateral and anterior corticospinal tract), ending in the anterior horn of the spinal cord at a specific level. In this location, it will get in contact with the lower motor neuron (LMN) which will exit the spinal cord aiming for the targeted muscle (11).

2.1.2. Somatosensory Pathways

Somatosensory perceptions are transmitted through the afferent spinothalamic tract (STT) and medial lemniscal tract (MLT) pathways. STT is responsible for the transmission of pressure, temperature, pain and crude touch information from the contralateral part of the body. MLT instead, transmits information concerning proprioception (body segments perception in the space) and discrimination (17; 33; 35; 36).

Along the spinal cord's white matter, the STT information travel along the anterolateral column whether MLT's along the posterior column (the latter larger than the former). The MLT path along the spinal cord is divided into two tracts: *cuneatus* and *gracilis* (respectively more lateral and more medial). Given that the *cuneatus* brings information from the upper extremities and the thorax, it is only observable in the cervical spine. The *gracilis* instead, is present all along the spinal cord as it carries information from the lower extremities (17).

The caudal origin of the MLT's 1st afferent sensory neuron (1aSN) is in the receptor and accesses the spinal cord through the ipsilateral posterior horn of the grey matter. The info is transmitted along the homolateral posterior column, until the *nuclei cuneatus* and *gracilis* which are located in the *medulla oblongata*. Here, after the synapsis with the 2aSN, the decussation takes place. The 2aSN is then directed to the contralateral VPL nucleus of the thalamus, passing by the contralateral medial lemniscus. This information is than projected to the primary somatosensory cortex by the 3aSN (17;36).

Along the spinal cord, the STT path is divided in ventral and lateral *funiculi*. The former is responsible for crude touch, pressure and information's transmission. The latter, pain and temperature. The 1aSN, at his level of entrance in the spinal cord, immediately forms a synapsis with the 2aSN in the dorsal horn of the grey matter. The second order

sensory neuron, after an immediate decussation, starts climbing to higher structures, ending in the contralateral VPL nucleus of the thalamus. Through the 3aSN, the information passes by the posterior limb of the internal capsule and the posterior part of the *corona radiata*; it is then transferred to the primary somatosensory cortex (located in the postcentral *sulcus*) (17; 33; 35; 36).

Despite the fact that both STT and MLT pathways transmit the information to the primary somatosensory cortex, Jang et al. (2012) underlines that the MLT path projects information in the precentral gyrus (motoric cortex) whether STT in the postcentral gyrus (sensory cortex) (36).

2.1.3. Encephalic Vascularization

The cranial vascularisation arises directly form the aorta. Intracranially, it composes the Willis' circle which function is to reunite the cranial circulations (anterior, posterior, right and left) (Figure 1) (8; 49).
The cardinal vessels are the anterior, middle and posterior cerebral arteries (ACA, MCA and PCA). The first two, as well as the choroidal arteries (ChA), belong to a ramification of the internal carotid artery (ICA). The PCA originates form a division of the basilar artery, but is communication with ACA and MCA through the posterior communicating artery (PcomA). At the cortical level, the ACA divides again into 4 branches: Orbitofrontal, Frontopolar, Callosomarginal and Pericallosal. Furthermore, the MCA instead divides into superior and inferior branch (8; 49).

In the following part I will introduce which artery irrorates a specific part of the encephalon. The hemispheres vascularisation is performed through two typologies of arteries: perforating (arising from the Willis' circle) and cortical (arising from ACA, MCA and PCA, forming a web on the lobes' surfaces). The perforating arteries are the following: ACA, MCA, PCA, PcomA, AcomA, ICA and ChA (71).

The ACA mostly irrigates the frontal lobes and the *corpus callosum*. In particular it irrorates the precentral gyrus (primary motor cortex), the structure whose function is to control the voluntary movement. His representation is called "motor homunculus" and

shows the inequality of the control quality of the different body parts (Figure 3) (8; 49; 71; 77).

The MCA supplies part of the frontal lobe, parietal and temporal (cranial half) lobes. Between these are included Broca's and Wernicke's areas, constituting the centres of language control on the dominant brain hemisphere. Another important structure irrorated by the MCA is the primary sensory area whose representation is called "sensory homunculus" and reflects the representation of the sensory areas of the body (Figure 3) (8; 49; 71).

The PCA irrorates the caudal part of the temporal lobe, occipital lobe and extends until the posterior area of the parietal lobe (8; 49; 71).

The brain territories suppliance for each vessel is presented in Figure 2.

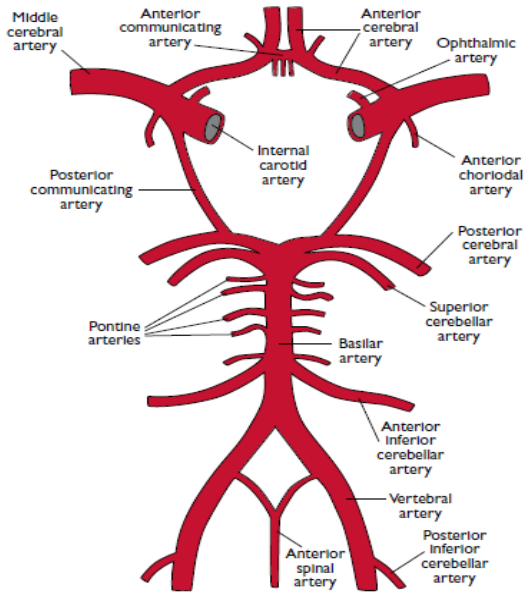

Figure 1: Willis' Circle (49)

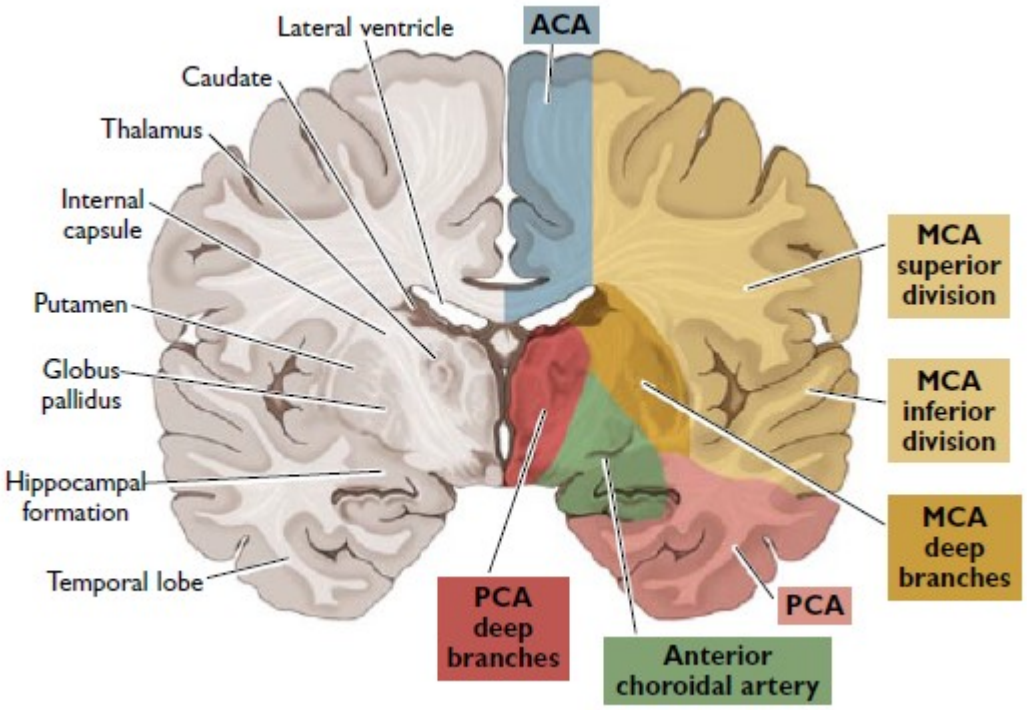

Figure 2: Brain territories' vascularisation (49)

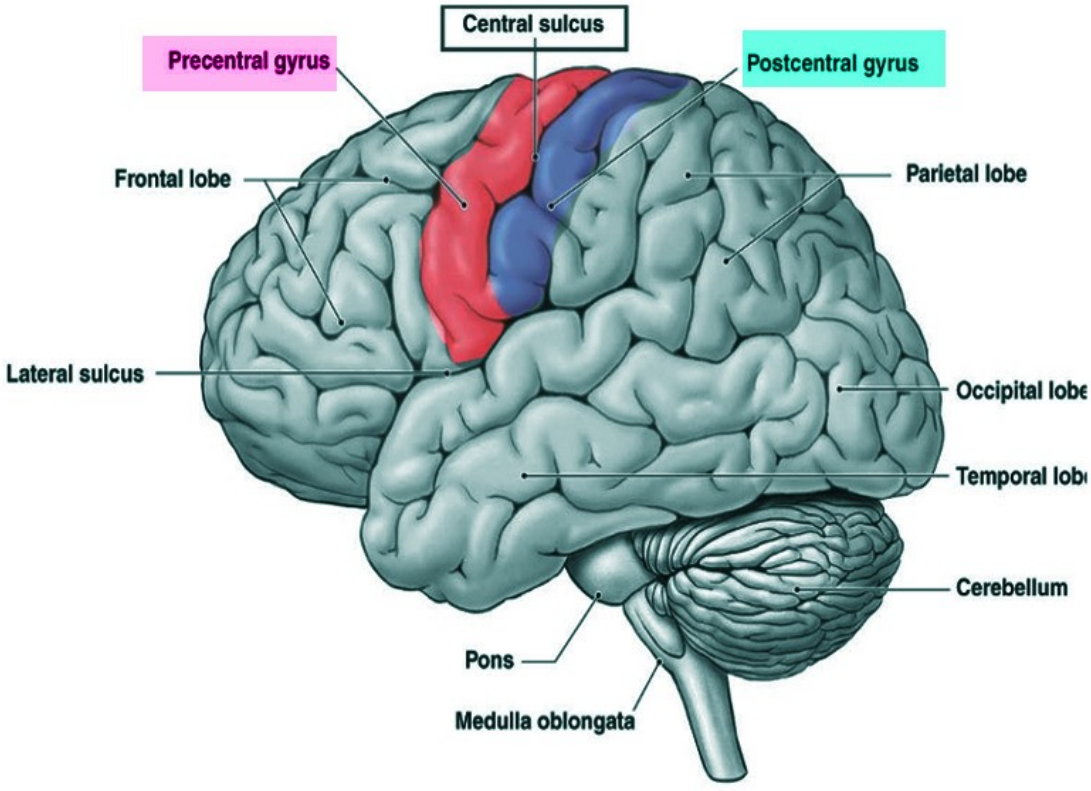

Figure 3: Pre (in red) and Post (in blue) central gyrus. The Motor and Sensory cortex (20)

2.2. CVA

In the following chapter, I will introduce the Cerebrovascular Accident (CVA) also known as stroke.

2.2.1. Historical background of CVA and TIA's definitions

The first historical signs refer to Hippocrates era (about 400 years BC) where "Apoplexia" was the name attributed to CVA, until 1689, when William Cole introduced the first CVA medical definition. In 1970, the World Health Organisation (WHO) introduced the latest CVA definition (below-mentioned) (62).

Concerning the TIA (Transient Ischemic Attack) its first definition appeared in the 1950's decade: *"temporary vascular-related episodes of brain dysfunction that would not qualify as strokes"*. The Second Princeton Cerebrovascular Disease Conference modified the TIA definition in *"[…] transient ischemic attack which may last from few seconds up to several hours […]"*.
In 1975 a "Cerebrovascular disease" Committee agreed on defining TIA as follows: *"Transient ischemic attacks are episodes of temporary and focal dysfunction of vascular origin, which are variable in duration, commonly lasting from 2 to 15 minutes, but occasionally lasting as long as a day (24 hours). They leave no persistent neurological deficit"* (62).

In the present days, CVA is defined as follows:

"[…] a neurological deficit attributed to an acute focal injury of the Central Nervous System (CNS), by a vascular cause[...]" (62)

"Rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin." (WHO, 1970; 62)

Scientific and clinical communities present a broad variety of CVA, rendering the WHO's definition obsolete (62). In matter of different typologies of stroke, the literature presents mostly the distinction between Ischemic and Haemorrhagic CVA (iCVA and

hCVA) (38; 62; 85; 87). Secondarily, the hCVA is divided according the haemorrhage location: Intracerebral (IhCVA) and Subarachnoid (ShCVA).
Sacco et al. (2013) study proposes more detailed definitions concerning the different types of CVA and their causes (62):

*"**Definition of ischemic stroke:** An episode of neurological dysfunction caused by focal cerebral, spinal, or retinal infarction."*

*"**Definition of stroke caused by cerebral venous thrombosis:** Infarction or haemorrhage in the brain, spinal cord, or retina because of thrombosis of a cerebral venous structure. Symptoms or signs caused by reversible oedema without infarction or haemorrhage do not qualify as stroke."*

*"**Definition of intracerebral haemorrhage:** A focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma."*

*"**Definition of stroke caused by intracerebral haemorrhage:** Rapidly developing clinical signs of neurological dysfunction attributable to a focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma."*

*"**Definition of silent cerebral haemorrhage:** A focal collection of chronic blood products within the brain parenchyma, subarachnoid space, or ventricular system on neuroimaging or neuropathological examination that is not caused by trauma and without a history of acute neurological dysfunction attributable to the lesion."*

*"**Definition of subarachnoid haemorrhage:** Bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord)."*

*"**Definition of stroke caused by subarachnoid haemorrhage:** Rapidly developing signs of neurological dysfunction and/or headache because of bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord), which is not caused by a trauma."*

Every year 15 million people are affected by CVA (≥65 years old); 5 million of them dies and other 5 million will see the rest of their life affected by its clinical signs (38; 67).

The epidemiologic analysis highlights that CVAs are the second most common cause of death and the third cause of disability, mostly affecting countries with a low or middle income. In the USA, CVA's death incidence comes after heart diseases and cancers (9; 24; 38; 49; 62; 67; 74; 79). In the EU, the above-presented data should increase of about 30% between 2020 and 2025 (32).

Truelsen et al. (2006) study reports that iCVA incidence in Caucasians is higher (80%) than IhCVA (10 to 15%) and ShCVA (5%). In Asian population these data differ, as they report a substantial higher incidence of hCVA (20 to 30%) (75).

Despite the incidence of this pathology and the amount of CVA-dead related, few scientific definitions can be found as brain vascularisation and neurophysiology knowledge only developed during the last 2 centuries, particularly during the last 50 years. Furthermore, the medical investigation means permitted to observe closely this disease only during the last 25 years (75).

During the course of this thesis, I will focus on causes, predisposing factors, signs and treatment of the iCVA.

The causes, which can directly lead to an iCVA, belong to four factors: thrombosis, embolism, hypoperfusion and arterial lumen obliteration. The thrombus formation into a vessel obstructs the blood flow and does not fully allow some cells to receive nourishments and oxygen. The embolism is the formation of a corpuscle (can be composed by different materials) in some other structures different from directly in the brain (e.g. in the heart); with the blood flow the corpuscle is transported in the brain where it blocks the flow to minor capillaries, leading to an ischemic stroke. The hypoperfusion cause is often related to a drop of blood pressure such as in case of cardiac arrest. The reduced amount of blood pressure does not allow the blood to reach all the territories of the brain leading to an ischemic condition. Mostly commonly, this happens in the watershed region, being the most distant from the Willis' circle. The arterial lumen obliteration is a reduction of the vessel's internal space, secondary to other pathologies like the external compression of the vessel, vasculopathy or vasculitis (49).

2.2.2. Local manifestations and effects of the iCVA

The iCVA is caused by the obstruction of a brain vessel by a solid body, not allowing the blood to reach all the districts (ischemia). The affected area of the cortex goes into hypoxia (lack of oxygen) leading to the cellular death in the core of the infarct zone (22; 24; 29; 38).

The physiological cerebral blood flow (CBF) in a healthy subject is between 38 and 55 ml/100g/min (CBFtot). When the CBF decreases to a value inferior than 20 ml/100g/min, the ischemic state is taking place but the neurons activity is still dependent on how intense is the ischemia:

- between 20 and 15 ml/100g/min: impairment of the neuron's electrical function
- < to 15 ml/100g/min: complete failure of the neuron's electrical system
- < to 5 ml/100g/min: extracellular K+ (potassium) release, generated by cellular death

The CBF is the most reduced in the ischemic core. The more we move away from the iCVA nucleus, the more the CBF tends, gradually, to get back into more physiological values. This translates into a zone called penumbra (20 to 40% of CBFtot), located all around ischemic core, which condition is still reversible through tissues revascularisation (9; 22; 24; 29).

In 1981, Astrup and Symon gave a first penumbra definition:
"the region of reduced CBF with absent spontaneous or induced electrical potentials that still maintained ionic homeostasis and transmembrane electrical potentials" (22)

Penumbra potential recovery is time-dependent. The earlier the treatment, the better results. The longer the neurons are exposed to penumbra, the higher risk of cellular death, leading to a greater function's impairment. For these reasons, the aim of medical and para-medical rehabilitation must be to revascularize the penumbra (22; 24).

In a physiologically functioning brain, the homeostasis is maintained through brain neurons, glial cells (micro and macro) and brain vasculature. The functional union of these structures is called Neurovascular Unit (NVU) (21; 24; 79). The NVU role is to modulate the blood-brain barrier (BBB) and the CBF. During an iCVA, the NVU assumes different functions. During the iCVA acute phase (until 6 hours after), the NVU promotes the BBB destruction via the proinflammatory effect of pericytes and through the matrix metalloproteinase (MMP) leading to the destruction of the BBB proteins, increasing its permeability and exposing to a cerebral oedema risk. Based on these findings, Wang et al. (2021) study highlight the importance of preserving BBB in the early stages of the iCVA (24; 44; 45; 79).

After the destruction of the NVU's cells, the debris of the destroyed ones negatively affects the surrounding healthy cells. In particular, glutamate, an important neurotransmitter also responsible of the neuronal growth, is conserved in high concentrations in the neurons. The neuron's destruction leads to a neurotoxic accumulation of glutamate. This process is called excitotoxicity (24; 44; 45; 79). Once released, glutamate affects the surrounding healthy cells, stimulating the NDMAR receptor (N-methyl-d-aspartate receptor), which secondarily induces the cell's death via a massive entrance of Ca^{2+} into the cell. The NDMAR stimulation, in a pathological situation, corresponds to the stimulation of the neural-death signalling (NDC) complex, which suppresses the neuron (44; 45). The final result is the massive neuron's death in the neighbouring tissues. On the other hand, *in vivo* studies proved that the astrocytes present in the ischemic area, result to be more resistant than the one in a healthy area, allowing a better tolerance to the neurotoxic environment (21; 79).
During the iCVA acute phase, a massive inflammatory reaction is triggered through the astrocytes which release high quantities of proinflammatory markers like MMPs, TNF, IL-1β, IL-6, IL-15 and NK cells. This reaction worsens the local ischemic state (79).

After the iCVA acute phase, the proinflammatory state starts to decrease, allowing the tissues repair through debris removal. An important protein for the neurovascular tissues repairs phase is ephrinB2 as well as VEGF (vascular endothelial growth factor) (21; 29; 79; 86). EphrinB2 main role is to fight towards the local tissues degeneration and to promote angiogenesis (21; 86). Elgebaly et al. (2020) study highlight that ephrin molecule

is the initial protein of a pathway called ephrin-Eph (erythropoietin-producing hepatocellular), being fundamental in in the post iCVA recovery phase. Eph class of receptors can be divided into two groups: Eph-A and -B. The first one is a superficial receptor whether the second is trans-membranal. The effects of the interaction between Ephrin and different subclasses of Eph is presented in Table 1 (21).

Ephrin–Eph interaction	Outcome
Neuroprotection strategy	
Ephrin-B2/EphB4 stimulation	Reduced edema, increased tight junction protein expression, and antiapoptotic neuroprotective effects
EphB2 antagonism	Reduction in infarct size, reduced edema and astrogliosis
	Reduced neovascularization indices such as vascular density, tortuosity, and branch density
	Neurocognitive function in diabetic rats improved using novel object recognition test
Ephrin-B2 antagonism	Reduced infarct size and neuroinflammation
EphA2 knockout/mutation	Preserved the integrity of tight junction proteins with NVU
	Improved neurological motor function tests
EphA4 knockout	Reduced neuronal death, glial cell scarring, axonal injury, and cell retractions
	Preserved tight junction protein Cx43
EphA3 blockade	Reduced glutamate toxicity
	Improved memory tested using Morris water maze
Ephrin-A5 antagonism	Improved late motor function recovery
Ephrin-B3 knockout	Enhanced neurogenesis

Table 1: Summary of outcomes in stroke with ephrin-Eph interactions according Elgebaly, 2020 (21)

High [VEGF] is also often detected in post-iCVA patients. The major role of this growth factor is to promote angiogenesis (29; 59; 65; 79).

VEGF gene's transcription is triggered by Hypoxia-Inducible factor 1 (HIF-1), who's concentration increase starts in hypoxic conditions. These values will return in the norms in hyperoxia conditions. After an average of 7 days [VEGF] reaches the peak and will return in the norms after about 28 days. The high concentration is mostly detected in the penumbra area (29; 59; 65).

For better understanding the iCVA mechanisms, scientists made some *in vivo* samples. The observation of these experiments firstly introduced the concept of evolving stroke damage, as previously mentioned, after the iCVA, the dead cells affect negatively

the healthy one. Furthermore, according Carmichael et al. (2005), the human iCVA is characterized by three features (9; 24; 44; 45; 79).

First, the dimension of the human iCVA is reduced (28-80 mm^3 / 4.5 to 14% of the ipsilateral hemisphere's mass) if compared with the animal one (in proportion). The advantages of a reduced volume stroke are a bigger marge of improvement and a lower chance to encounter a "malignant infarction", situation characterised by a rapid swelling of the tissues, arterial compression, widening of the infarct area and death (9; 85).

Second, *"recanalization of the arterial occlusion or reperfusion of the downstream territory"* (85) also simply called reperfusion. This happens thanks to two phenomena: the early clot lysis (15 to 18% of all the strokes) and the Leptomeningeal collaterals (LMCs) (9; 70). LMCs are a dormant group of vessels, which connect major arteries irrorating the skull. They are located along the surface of the brain. Their activation is triggered when the cerebral physiological vascularisation is impaired. Clinical observations show a better recanalization and a reduction of the infarct core size. Third, the damaging of a neural circuit corresponding to the impairment of a specific function. This type of functional damage appears to be way more specific in humans iCVA than in rodents (9; 70).

2.2.3. Systemic manifestations and effects of iCVA

2.2.3.1. Manifestations according affected artery

iCVA symptoms can be predicted according the affected artery (see "Encephalic Vascularization" chapter). According the occluded vessel, different syndromes are observed. In the next paragraphs, I will introduce the symptoms associated with the damages of the main vessels (ACA, MCA, PCA and ChA).

2.2.3.1.1. ACA syndromes

ACA occlusion is correlated with contralateral hemiparesis and hemianesthesia mostly affecting the contralateral LE rather than the UE. Another common signs of the ACA affection can be the abulia manifestation (lack of volunty), mutism or confusional state (the former is the dominant lobe is affected, the latter if non-dominant) (49).

Abdelrasoul et al. (2019) study introduces the azygos ACA infarction, a rare case of vascular damage which leads to the bilateral ischemia of frontal lobes (medial portion) coupled with the corpus callosum. The symptoms are not very well known given the rarity of this syndrome; generally, they are associated with the presence of primitive reflexes, hypophonia, rigidity and akinesia (common Parkinsonian symptoms) (1).

2.2.3.1.2. MCA syndromes

Common MCA artery occlusion leads to the following symptoms: contralateral hemianopia, hemianesthesia, hemiparesis and hemineglect syndrome. Other signs manifestations can be expressive and/or receptive aphasia (Broca's and/or Wernicke's area affection) if the dominant brain hemisphere is affected, otherwise when the same territories of the non-dominant hemisphere are touched, the patient can present impairments in the voice's rhythm and intonation (aprosodia). These symptoms can be verified in the expression or receptive ability (49).

More in detail, the MCA's superior branch affection (frontal lobe irroration) translates into movement and expression impairments. If the inferior branch is affected, hemianopsia and the ability to understand external language are affected.
Rarer, is the Gerstman's syndrome observed in case of dominant angular gyrus (inferior parietal lobe) lesion. The common symptoms are fingers agnosia, acalculia, agraphia and inability (or difficulties) to distinguish right and left. Despite the knowledge of these symptoms, it has been observed that they do not always manifests in on the same time (49; 69).
The infarct of the ChA (anterior) presents a phenotype similar to the MCA leading to marked hemianesthesia, hemianopia and hemiplegia (49).

2.2.3.1.3. PCA syndromes

PCA occlusions leading to iCVA are scarce (5 to 10% of all the ischemic strokes). Common signs of the iCVA provoked by the PCA obstruction are alexia (reading inability); if the dominant hemisphere is affected and contralateral homonymous hemianopsia (e.g. damage on left hemisphere corresponds to inability to see the right half from both eyes). Cortical blindness and Anton syndrome (negation of disturbs and confabulation) can be observed if both occipital lobes are affected (14; 49).

Other common symptomatology characteristics of the post-ischemic stroke patient are presented in the upcoming paragraphs.

2.2.3.2. Spasticity

From a motoric point of view, spasticity and increased muscle tone are between the most important pathological signs of iCVA (39; 58; 64).
Spasticity often takes place when one (or multiple) structure(s) controlling motricity are affected (see "Motoric Pathway").

Lance first defined spasticity in 1981 as follows: *"a motor disorder characterized by a velocity-dependent increase in muscle tone with exaggerated tendon reflex, resulting from hyper excitability of the stretch reflex."* (39; 80)
In 2005 the previous definition was updated in: *"a disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles"* (80)

Spasticity is not a common characteristic of iCVA-affected patients as only 20 to 40% of them exhibit spastic features (39; 43; 64). Clinically, it can be evaluated through the Ashworth Scale or the modified Ashworth Scale (60).

As it is known, spasticity generally affects the opposite side of the stroke-affected hemisphere (e.g. iCVA affecting the left brain hemisphere can lead to spasticity of the right side of the body). According Kuo and Hu (2018), the muscles which are more affected are mainly the upper extremity flexors (elbow, shoulder, wrist, fingers flexors, shoulder internal rotators and forearm pronators) and lower extremity extensors (plantar flexors, knee extensors, hip extensors and internal rotators) leading to the clinically known Wernicke Mann posture (43).

Spasticity invalidates the patient's activities of daily living (ADL) together with impairing the urinary continence, walking, sitting etc. resulting in a higher risk of falling and self-injury (28; 39; 58; 64; 80).

Clinically there is no official predictor for the spasticity onset, but different authors agree on asserting that an important sensorimotor impairment is correlated with the incidence of spasticity (39;58; 64).

Despite the spastic behaviour evidence, these characteristics do not manifest in an early stage. In the iCVA acute stage the patient usually exhibits a flaccid paralysis, which will later evolve in spasticity (Figure 4). The initial phase after CNS lesion is called "Shock-state". The transition from flaccid paresis to spasticity is probably triggered by an excessive compensative regeneration of the UMN terminations (80).

2.2.3.2.1. Spasticity pathophysiology

In the next paragraph, I will highlight the mechanisms leading to spastic manifestations.

A healthy CNS, presents balanced activity between excitatory and inhibitory MNs allowing fluid movements. After a central CNS lesion, this equilibrium is disbalanced. The specific manifestation of the lesion depends on the damaged location of the brain (80). Spasticity is connected with the lesion of the upper motoneuron (UMN) of the cortico-spinal tract (pyramidal tract) characterised by a higher speed-dependent response of the stretch reflex and leading to hyperreflexia, muscle jerks and clonus (28; 43; 51; 68; 80).

Spasticity can be explained following two different paths: the neural one and the muscle's local mechanical modifications (Figure 5) (43; 51; 68).

In the first one, the damaged UMN interferes in the exchange between brain and spinal cord. Therefore, the reflexes are completely enhanced (hyperreflexia). For better understanding this process, an analysis of the muscle spindles functioning is imposed. In the internal part of the muscle, we can observe some isolated groups of few muscular fibres called intrafusal fibres. They enclose the muscle spindles, which communicate to the CNS information about the dynamic muscle length and speed of elongation, through the Ia fibres. The fibres group II transmits only information about the static muscle length (at rest). When the muscle is stretched, the muscle spindles communicate the information through the Ia afferent fibres with a subsequent activation of the α-MN leading to the extrafusal muscle fibres contraction. In a physiological condition, the α-MN response is modulated by the central inhibitory control. In a spastic muscle, the inhibition does not take place resulting in an excessive reflex muscle contraction. Additionally, the diminution of the inhibitory influence on spinal interneurons (Ia and II), reduces the antagonists

protective response. The agonist's sensory neurons are then pushed to fire more information leading to a more intensive contraction (43; 68). A vicious circle.

For what concerns the muscle's mechanical modification, its onset is mostly observable in a chronic spastic condition and can be observed through active (tendon reflexes) and passive (passive stretch) testing. The chronic presence of spasticity reduces the length of the sarcomeres and increases the amount of connective tissue in the muscle leading to a higher intramuscular stiffness. This translates into a faster and stronger transmission of the tension to the muscle spindles (43; 51; 68).

In more detail, different elements are present in the interstitial space of a physiologically working muscle, between which the hyaluronic acid. His rheological properties are similar a non-Newtonian fluid, characterised by viscosity. Their reaction to an applied shear force is time-dependent.

The substance opposition to an applied shearing force is inversely proportional to the velocity of the velocity of the force, ergo the faster the force is applied, the more resistance the fluid will offer and vice versa (12; 68).

In the spastic muscle, concentration and viscosity of the hyaluronic acid are increased, leading to a grown accumulation of the latter in the interstitial space. An increased viscotic behaviour reduces the sliding between the intramuscular layers, justifying, from a mechanical point of view, the muscular increased stiffness and the spastic answer to the sudden stretch (43; 68).

Grade	Description
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the ROM when the affected part(s) is moved in flexion or in extension
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the reminder (less than half) of the ROM
2	More marked increase in muscle tone throughout most of the ROM, but affected part(s) easily moved
3	Considerable increase in muscle tone, passive movement is difficult
4	Affected part(s) rigid in flexion or extension

Table 2: Modified Ashworth Scale (by Bohannon and Smith, 1987) (91)

2.2.3.3. Somatosensory deficits

Somatosensory deficits are often part of the symptomatologic picture of a post-iCVA patient. They are present in 25 to 85% of the ischemic stroke-affected patients (40; 87).

Somatosensory perception is divided in exteroception (e.g. light touch, pain, temperature and superficial sensation) and proprioception (e.g. body position perception, stereognosis and discrimination) (40; 87).

The type and entity of somatosensory impairment is tightly correlated with the brain-affected area (40; 49). Kessner Simon et al. (2019) study underlines that the areas, which are mostly likeable to lead to perceptive impairments, are the following: thalamus, dorsal internal capsule, *corona radiata*, *pons* and cortical areas. According the same study, the two most affected areas are the superior thalamic radiation (superior component) and the secondary somatosensory cortex, located in the parietal lobe on the edge of the *sulcum lateralis* (40).

Unfortunately, a very low amount of information is present in the scientific research about the correlation between brain-damaged location and the somatosensory manifested symptoms (40). Damaged of primary/secondary sensory cortex and subcortical areas of the frontal or parietal lobe, are associated with a low RASP score (Rivermead Assessment of Somatosensory Performance) and with a somatosensory impairment (40).

2.2.3.4. Other systemic manifestations of iCVA

One of the other common manifestations of the iCVA, which is connected with the spastic behaviour is pain. Most patients presenting motoric and/or sensory impairments, present stroke-related pain (37; 68). According Stecco et al. (2014), this is connected with the automatic body compensatory strategies assumed for compensating the range of motion (RoM) reduction. These pathological tensions are transmitted through fascia, stimulating the pain receptors and giving the perception of musculoskeletal pain (68).

Common in post-iCVA patients is the affection of the facial nerve (VII cranial nerve). Often facial asymmetry, central and peripheral facial palsy can be mistaken during

the stroke acute phase. The more common cases where it is possible to observe central facial nerve palsy's symptomatology is when the MCA is affected as the affected territories are the corona radiata or the parietal cortex (84). Not enough studies have explored this domain, particularly considering that this type of lesion is mild and does not persist over time.

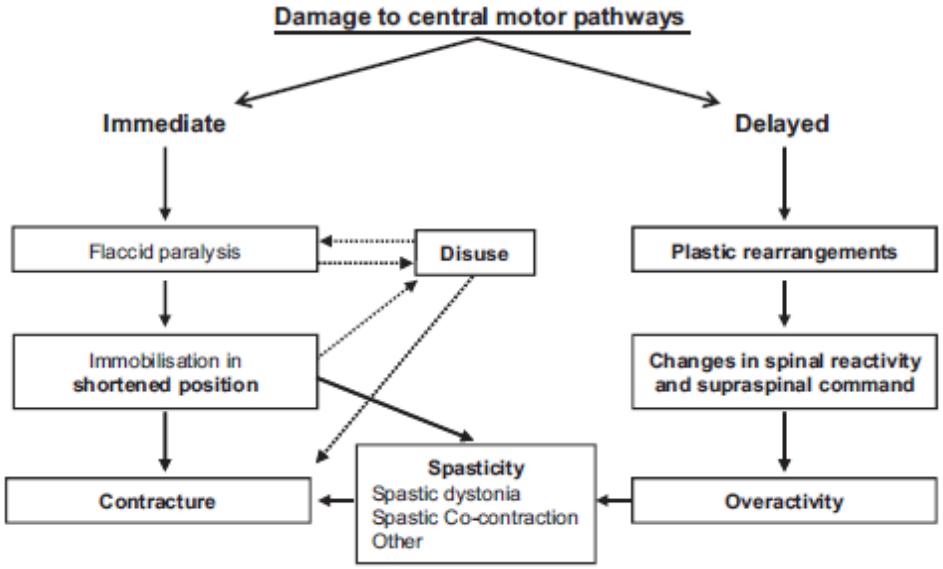

Figure 4: Evolution from flaccid paralysis to spasticity (80)

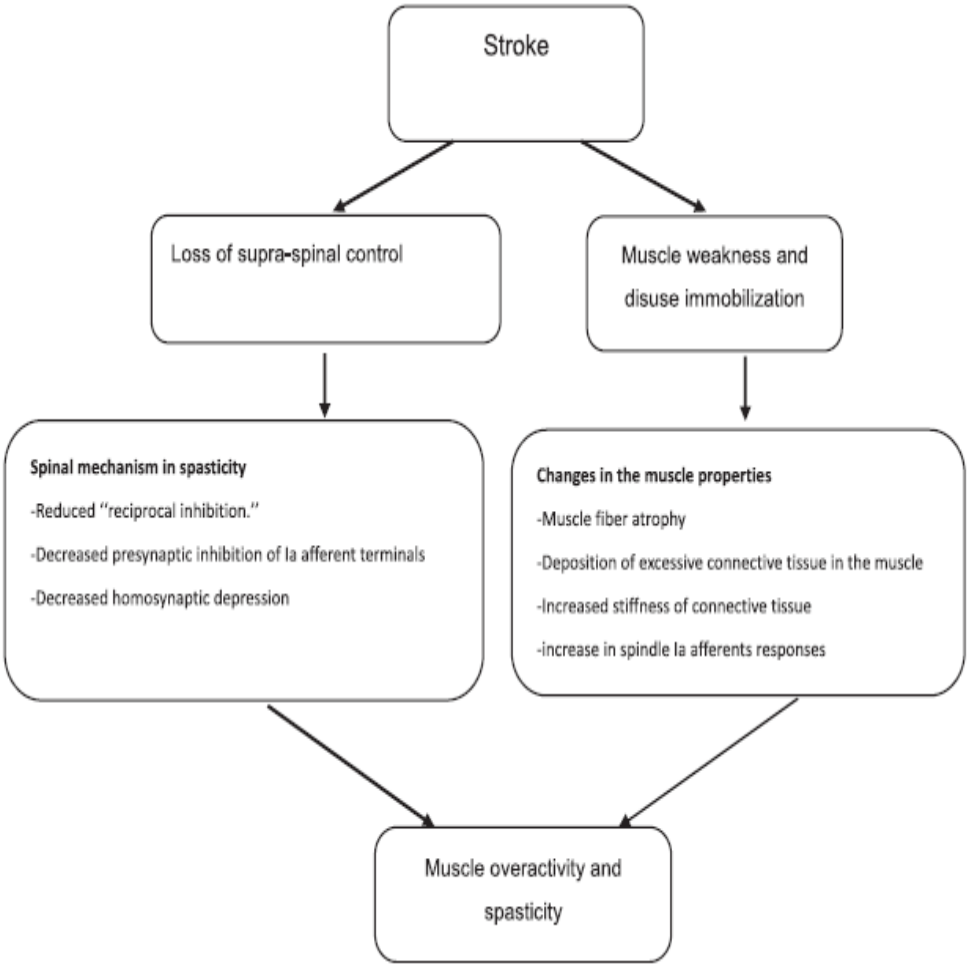

Figure 5: Mechanisms leading to spasticity (43)

2.3. Markers of pathologies predisposing to ischemic Cerebrovascular Accident

iCVA is multifactorial disease often being caused by subjective and non-subjective factors. The latter being almost non-modifiable (genetic). The former, instead, modifiable through a healthy life-style.

Recent studies indicate that metabolic syndrome, diabetes, obesity, increased BMI, cardiovascular diseases (CVD), hypertension and atherosclerosis could be factors predicting ischemic and haemorrhagic CVA (16; 23; 25; 46; 54; 76; 83). These pathologies' common characteristic is the provocation of an inflammatory state, triggered by pro-inflammatory cytokines (46; 71). Despite these results, the exact mechanisms linking the above-mentioned diseases with the CVA is still unclear (23; 34; 54).

The key point resides in white adipose tissue (composed by adipocytes) and in its humoral regulating role via the adipocytokines. Included in this group, the two most studied molecules are Leptin (LPT) and Adiponectin (APT). LPT and APT respectively intercede in the pro and anti-inflammatory state in pathologies like atherosclerosis (25; 34; 42; 46; 90).

2.3.1. Leptin

LPT protein is a class I cytokine encoded by *Ob* gene, widely present among vertebrates. From a structural point of view, LPT shows a similarity with interleukins (particularly IL-6), a group of pro and anti-inflammatory cytokines (25; 90). From a biological point of view, its function is to report the feeling of satiety and to trigger the energy expenditure (25). All over the body, six types of LPT receptors can be observed: Ob-Rb, Ob-Ra, Ob-Rc, Ob-Rd, Ob-Rf. The first one is the longest isoform and is present mostly in the hypothalamus. LPT main direct effect on the body is to generate platelets aggregation in the blood stream (25). High levels of LPT are observed in patients with CVD, diabetes and/or obesity showing a positive correlation with cardiovascular disease and stroke (34; 46).

2.3.2. Adiponectin

APT's molecular structure is similar to C1q and TNF-α. The former is an anti-inflammatory molecule diminishing cytokines production and the latter is a protein acting as cell signalling involved in the acute inflammatory phase reaction, phagocytosis stimulation and insulino-resistance modulation (13; 25; 72).

In physiological conditions, APT-blood concentration is 10^3 to 10^6, higher than LPT and other pro-inflammatory cytokines. Its functions are to regulate glucose concentration and lipidic metabolism. Furthermore, APT as anti-thrombotic, -atherogenic, -diabetic and -inflammatory properties (25; 34; 46). As a confirmation of its properties, *in vivo* studies proved that APT-deficient mice were more exposed to blood clotting (25).

On the other hand, APT's role is not clearly understood. In fact, numerous studies' results are in contradiction with what previously mentioned (34; 41; 46; 76).

Shen et al. observed that [APT] was higher in patient affected by iCVA rather than in healthy subjects (34). Larsen et al. (2018) express the potential correlation between high [APT] and death related to cardiovascular diseases, particularly in subjects with a reduced BMI or with chronical diseases (46).

2.3.3. LPT and APT involvement in CVA

Despite some contradictions on APT's role, many studies suggest the observation of [LPT] and [APT] as CVA predictors. In the scientific bibliography this relation is called L:A ratio (34; 41; 46).

Kim et al. (2012) furnishes a detailed explanation and interpretation of this ratio. According their study, the ratio would differ in function of the type CVA. L:A ratio would present a higher LPT level compared to APT's, if the stroke was originally caused by debris (atherosclerotic plaque rupture) belonging to a large artery. For what concerns a CVA originated from a capillary, no L:A ratio-stroke correlation was observed (41).

In conclusion, the topic involving the study of L:A ratio aiming to predict the possibility to be affected by CVA has still to be studied as well as the mechanism of action of LPT and APT has to be understood. Further studies are required.

2.4. Early medical treatment after iCVA

Different methods are proposed in the scientific literature as an early medical treatment in patients with acute iCVA. One of the mostly employed is the reperfusion therapy. This method aims to destroy the blood clot, which obstructs the blood flow, restoring the normal vascularization of the tissues, through two possible techniques: intravenous thrombolysis or mechanical thrombectomy. The former is performed within the 6 hours after the accident, with drugs which effect is to disaggregate the blood clot. The scientific reviews are still uncertain about until which this window f intervention could be extended for the observation of beneficial results. Commonly used drugs employed for this scope are tissue plasminogen activator, streptokinase or urokinase (7).

Despite what above-mentioned, this method presents some limitations. First, an increased risk of intracranial bleeding and ICA occlusion often leading to death, second,

reduced recanalization rate of the MCA, ICA and basilar artery and third, reduced effectiveness on big clots or distant located ones (7).

Mechanical thrombectomy, presents the advantage of being minimally invasive. The devices employed are differentiated according their mechanism of action: coil retrievers, stent, aspirators or mechanical destruction. Their immediate efficacy is clearly visible but long-term benefits aren't still so evident (7).

2.5. Physiotherapy after iCVA

A wide number of PT approaches can be employed in rehabilitation after iCVA. In the following pages, I will present modern and discussed methodologies.

2.5.1. Respiratory Physiotherapy

One of the most common impairments related to the iCVA is the muscles' property modification (e.g. paralysis, spasticity). This indirectly affects the connected structures. Commonly the thoracic cage mobility is limited, affecting the breathing pattern and opening to possible postural modifications (e.g. through diaphragm misfunctioning). Furthermore, post-iCVA patients present a higher risk of lung infection given the biomechanical problem at the level of the thoracic cage (3; 61).

Assumed the severe lack of strength which follows the ischemic stroke, the victims undergo an important reduction of the aerobic capacity, leading to decreased ADL performance, limiting even more the aerobic capacity as they are pushed to move less and less (5; 61).

For these reasons, respiratory physiotherapy assumes a primordial role in the rehabilitation process after ischemic stroke.

Arslan's et al. study (2021) proved that an inspiratory muscles training would improve the breathing capacity increasing the aerobic capacity, which translates into a greater capacity of achieving ADL. Furthermore, given that the main inspiratory muscle is the diaphragm, this would also improve the ability to maintain the posture and the balance (5).

Almasry's et al. study (2018) underlined the importance of chest muscles breathing physiotherapy particularly during the iCVA acute phase and in the early rehabilitative process, in order to prevent pneumonia and lungs infection. The techniques employed are mostly the deep breathing combined with manual techniques. This promotes the elimination of the fluid which accumulates in the lungs (expectoration). Combined with it also repositioning assumes a primordial role for the lung's complications prevention (3).

2.5.1. Motor recovery after iCVA

2.5.1.1. Mirror therapy

Mirror therapy is a commonly employed method in physiotherapy. Its aim is to improve the motor recovery in all the patients with motoric impairments of any sort.

This therapy was originally invented by Roger-Ramachandran for treating somatosensory disturbances (mostly pain and phantom pain) in amputated patients. During this method, the patient is able to see the healthy limb and its projection on the mirror, which hides the affected. Along the treatment, the patient is asked to provide specular movements with both extremities. Some studies suggest that its mechanism of action works by illuding the brain that both limbs work symmetrically trough the fake perception of the physiologically functioning limb and all the cerebral tasks related to it (movement and perception). From a neurological point of view the exact mechanism is still not well understood but it has been showed that it promotes inter-hemispherical communication, bilaterally activating cortical areas responsible for motricity, awareness and spatial attention (posterior cingulate and precuneal cortex) (26; 50; 73).

Different hypothesis has been done on the neurological functioning of Mirror therapy. The first one is based on the presence of mirror neuron, mainly in the temporal gyrus and in the frontal-temporal region. This neuron's function is to direct the movement of an extremity, imitating a similar externally observed action. The second one underlines the increasing activity of the areas responsible for spatial attention and self-awareness. The last hypothesis specifies that the incremented activity of the treated limb (after Mirror therapy) rely on the activation of the dormant ipsilateral motoric and sensory pathways (26).

In conclusion, combination of PT and Mirror therapy showed positive improvements in post-iCVA patients (acute, sub-acute and chronic stage) in peripheral articulations of the UE and LE. This is a beneficial factor knowing that most of the progressions are initially performed in proximal joints and later in distal ones (26; 50).
Other studies underline the Mirror therapy's beneficial effects on pain management and somatosensory impairments (26; 73).

2.5.1.2. Proprioceptive Neuromuscular Facilitation

PNF was invented during the first half of the 18th century by H. Kabat and M. Knott (neurologist and physiotherapist), for the treatment of poliomyelitis and cerebral palsy in young patients. This method stimulates (or inhibits) muscles via the proprioceptors' stimulation. The elongation of the chosen muscles initially triggers the facilitation mechanism. Other principle of this method are the verbal inputs, manual contact, resistance, movement timing, joint facilitation and irradiation. Knowing that for complex movements, different muscles cooperate for providing the motion, irradiation principle explains that it is possible to facilitate a weak muscle trough the stimulation of the muscles working on the same chain. The employ of this method stimulates also BDNF known as an important factor for neuroplasticity, vital for the CNS damage regeneration (10; 30; 31; 42).

Given PNF's efficacy, Guiu-Tula's et al. (2017) meta-analysis underlines the importance of including PNF method in the CVA-rehabilitation process. In accordance with these findings, Sharma et Kaur's study (2017) highlights the importance of the application of PNF method in the pelvis aiming to improve the trunk stability, gait stability, balance and abdominal muscles' activation (30; 31; 66).

2.5.2. Bobath concept

Also known as Neurodevelopmental Treatment, this individual approach suggests a problem-solving strategy aiming to improve the sensory-motor condition of neurologically affected patients. Originally developed during the late first half of the 18th century, incorporating three different theoretical basements: CNS plasticity, muscular tissues plasticity and sensorimotor learning. The key-point of Bobath concept is the treatment of the

function allowing us to achieve a specific task. From a neurophysiological point, this approach accords great importance on the afferent inputs allowing better musculoskeletal performance. The centers controlling these functions are located in the brainstem. The reason of a major interest on the afferent stimuli is based on the idea that our body behaves in function of the inputs received by the environment. The therapeutical effectiveness comparison between Bobath approach and other methods, is still under debate in the scientific community (18; 48).

Diaz-Arribas' et al. review (2020) compared the results of Bobath approach with other methods (PNF, traditional functional training, constraint-induced movement, motor relearning program, problem-oriented willed-movement and robotic-assisted therapy), over 15 studies driven on post-iCVA patients. The results highlighted a lack of statistical difference in terms of efficacy in ADL, LE control, general mobility, balance, posture and UE dexterity or control (18).

2.5.3. Spasticity management

2.5.3.1. Muscle stretching

Assumed that one of the most common complications post-iCVA is the muscles' spasticity often combined with muscular contractures and RoM reduction, one of the commonly presented strategies is static stretching. Furthermore, stretching effectiveness, intended as a general domain (applied in sport, recovery, prevention, rehabilitation, etc.), is a widely discussed thematic given the inconsistency of the results (63).

On this subject, Salazar's et al. (2019) meta-analysis investigated the effect of passive stretching in combination with physiotherapy on spastic patients. Their results evidenced a low efficacy-magnitude of passive stretching through orthotic devices (or manually performed). Despite this fact, this treatment still presented benefits if compared with no therapy. In addition, the combination of physiotherapy and passive stretching showed no difference in terms of effectiveness, if compared with general physiotherapy (63).

On the other hand, Ganvir's et al. (2020) review affirm the positive effectiveness of stretching over spasticity (30 min, 4 times a day). Furthermore, they investigated a method for enhancing the stretching effects. They reduced the pathological muscular contraction reflex during elongation with ice (cold) application (see "Cryotherapy" chapter).

Despite highlighting the debate on stretching effectiveness on spastic patients, Ganvir et al. agreed on asserting that deep cryotherapy application is beneficial for muscle relaxation. Thus, stretching following cold application leads to spasticity diminution and improvement of functionality (27).

2.5.3.1. Cryotherapy

Between non-pharmacological treatments, I chose to introduce cryotherapy because is cheap, accessible and can be employed in every day's clinical practice. It is still unclear what is the mechanisms on which spasticity treatment through cryotherapy is based on.

Garcia et al. (2019) study proved that a 20-min local ice-pack application on plantar flexors, reduced spasticity for a short-medium period allowing a better PT session performance, without modifying the proprioception perception. As mentioned in the study, it is important to underline that the observed spasticity doesn't reflects the spastic behaviour observed during ADL activities (e.g. walking) (28).

Alcantara's et al. (2019) study agrees on the cryotherapy beneficial effects on hypertonia. This study adds that ice-packs application leads to no effects on isometric, eccentric and concentric ankle muscles' strength and that this therapy doesn't affect the gait cycle at any level (2).

Naro's et al. (2017) review, other than confirming what above-mentioned, suggests an explanation justifying the transitory beneficial effects of this method. The muscular relaxation appears to be enhanced by the Ia fibres' reduction meaning a reduced muscle spindle's stimulation and a subsequent lower α-MN response (52).

2.5.3.2. Deep Dry Needling

Despite DN is not a common (or allowed) treatment for PTs, a wide number of studies show favourable results in spasticity treatment in combination with conventional rehabilitation (4; 78; 88).

Anasari et al. (2015) showed that DN's beneficial effects could be observed, on the UE, already after one session (treating pronator teres, flexor carpi radialis and flexor carpi ulnaris) (4).

Zaldivar et al. (2020) compared the results of a conventional rehabilitation with the combination of DN + rehabilitative program on the UE, over a period of 8 weeks. In this period, the second group received 6 DN sessions. Despite the lack of knowledge concerning the underlying mechanisms, remarkable benefits were observed in the reduction of the spastic phenomenon, thus this did not affect pain perception nor functionality (88).

Valencia-Chulian et al. (2020) other than highlighting the beneficial effects of DN spasticity, showed improvements also on perceived musculoskeletal pain and PRoM. Another important factor mentioned in this article, is the low chance (~20% of patients) of undesired effects like bleeding, blood pressure reduction, bruising and short-lasting pain (78).

2.6. New frontiers for iCVA rehabilitation

2.6.1. Gait Rehabilitation: Robotic or Conventional?

Walking impairments are common in post-iCVA patients. Walking rehabilitation assumes a primordial role in restoring the patient's autonomy.

Dierick et al. (2017) study presents the comparison between the gait robotic assisted (GRAr) with Lokomat and conventional rehabilitation (e.g. Bobath approach). Through their study, they highlighted that both methods are similarly beneficial from a functional level, underling the lack of difference between GRAr and conventional rehabilitation in both iCVA and hCVA. Park's et al. (2018) study also underlines that the combination between GRA and conventional rehabilitation could be a supplementary tool for improving gait and postural rehabilitation (19; 55; 56).

In conclusion, if compared GRA and conventional rehabilitation have the same beneficial effect. The best solution would be their combination.

2.6.2. Virtual reality

"use of interactive simulations created with computer hardware and software to present users with opportunities to engage in environments that appear and feel similar to real-world objects and events" (47)

Virtual Reality is recent full-immersive technology that initially found its employ as a formation-practice system (e.g. surgeons and pilots), then as a recreative tool and only lately started to be introduced into the world of rehabilitation. VR can be globally divided into immersive and non-immersive; the main difference is that the former transmits the perception of being in another world whether the latter still allows the perception of being in a "secondary" dimension. In terms of clinical efficacy, the comparison between immersive and non-immersive VR is still subject of debate (15; 47; 53; 57).

Immersive VR works through the projection of digital images in two concave screens located in a head-mounted "helmet" and placed in front of the eyes, offering a 360° stereoscopic vision and allowing the interaction with the virtual environment

through specific controllers. Stereopsis is the physiological ability to perceive the three-dimensionality of the environment. Two (almost) identical images of the surroundings are projected in each eye. The mechanisms, which lead to the perception of one single image, are two: motor fusion and sensory fusion. The former is the ability of the eyes to have the same alignment. The latter, is the adjusted fusion of the two images ultimately leading to the seen figure (6; 53; 81).

Concerning the clinical effects, preliminary *in vivo* testing showed improvements in problem-solving tasks; VR beneficial results in humans include an increased neuroplasticity effect leading to a general improvement of the neuromuscular response, particularly through the activation of the primary motor region. More specifically improvement of balance, posture, motor functions (manipulation and gait), cognitive functions were observed, leading to an improvement of the ADL and a diminution of iCVA-related pain and falls (6; 15; 47; 53).

Palma's et al. study (2017) partially disagrees with what above-mentioned. Their results are in accordance with the improvement of structure and function of UE, LE and mental functions but, on the other hand, they showed no VR's statistical impact on patient's activity and participation. These two aspects play a fundamental psycho-somatic role in the psychological involvement of the patient in the rehabilitation process, affecting the final degree improvements (53). Warland et al. (2019) highlight that the risks of adverse effects are minor and in case their intensity is relatively low (82).

In conclusion, VR in combination with PT could be a new frontier for a more effective rehabilitation process for the patients post-iCVA (15; 53).

3. Special part (Case study)

3.1. Methodology

My clinical practice took place at Rehabilitační nemocnice Beroun starting from the 18th January until the 12th February 2021. The total amount of hours spent for my practice is 127. During my placement, I was supervised by Bc. Aleš Nesvadba and my Academic supervisor was PhDr. Jitka Malá, Ph.D.

I spent 9 sessions with the patient designed as case-study, starting from the 18th January until the 2nd February 2021, counting the initial and conclusive examinations. During the whole placement, the patient was conscient and aware of the goals of the employed procedures. All the employed techniques and examination methods, have been learned during my studies in FTVS at Charles University (Fakulta tělesné výchovy a sportu, Univerzity Karlovy), Prague.

This thesis was approved by the ethics Committee of the above-mentioned faculty.

3.2. Anamnesis

Examined Subject: Z.J. **Year of birth:** 1970

Diagnosis: I63.9 - Right hemisphere ischemic stroke

3.2.1. Status praesens

3.2.1.1. Objective

Height (cm): 174

Weight (kg): 100

BMI: 33

Blood pressure, Heart rate, Body temperature: 124/85 mmHg; 55 bpm; 36,5°

Dominant side: right

Glasses: none

Communication ability: normal

3.2.1.2. Subjective

3.2.1.2.1. Chief complaint

The patient's major complaints are related to inability to walk and to bear the weight on the left leg, the inability to move the left arm (mainly shoulder) and reduced functionality of the left hand and elbow. The patient presents signs of central lesion of the VII cranial nerve on the left side of the face. The patient doesn't report any sort of pain in any part of the body.

3.2.1.2.2. Personal anamnesis

The patient is oriented, conscious and cooperative. Normal development.

3.2.1.2.3. Family anamnesis

Nothing correlated to report.

3.2.1.2.4. Injury anamnesis

The patient was hospitalized (17-01-2021) after an ischemic stroke caused by the obstruction of the right internal carotid. The lesion happened the 1st January 2021 and was treated with Intravenous thrombolysis in UVN Střešovice.

3.2.1.2.5. Past medical and surgical history

Diabetes mellitus type II

Dyslipidemia

GERD

3.2.1.2.6. Medical anamnesis

Clopidogrel actavis 75mg: 1/d

Torvacard neo 40mg: 1/d

Glimepirid Sandoz 3mg: 1/d

Stadamet 1000mg: 2/d

Miraklide 10mg: 1/d

Pantoprazol 40mg: 1/d

Lactulosa 3/d

Zaldiar 37.5/325mg 3/d

3.2.1.2.7. Allergy anamnesis

None

3.2.1.2.8. Abuses

Alcohol consumer, smoker (30/d)

3.2.1.2.9. Diet

Fat and sugar-rich diet

3.2.1.2.10. Functional anamnesis

The patient is unable to walk, climb stairs and use for any reasons the left upper extremity. No sphincters problems are reported. The Barthel scale index is 70/100.

3.2.1.2.11. Social anamnesis

The patient is married without children.

3.2.1.2.12. Occupational anamnesis

Worker in steel industry. The past occupation is described as moderately active and performed most of the time standing or on complex positions (kneeling etc.). The displacements in the industry were performed walking.

3.2.1.2.13. Sport, Physical activity

Garden work

3.3. Neurological examination

3.3.1. Cranial nerves

VII nerve central paresis affecting the left inferior quarter of the face. The other nerves did not show any pathological signs.

3.3.2. Upper limbs

- Deep tendon reflexes testing: global areflexia of the left body hemisphere. The tested deep tendon reflexes are the following: Biceps brachii (C5-6), Brachioradialis (C5-6) and Triceps brachii (C7-8)
- Paretic signs: unable to be properly performed. The tested paretic signs are the following: Mingazzini, Barré, Hanzal and Du Four.
- Spastic-irritative signs: negative. The tested spastic-irritative signs are the following: Hoffman, Juster and Tromner.
- Cerebellar examination: negative to taxes test, adyadocokinesa, intentional tremor.

3.3.3. Lower limbs examination

- Deep reflexes: global areflexia of the left body hemisphere. The tested deep tendon reflexes are the following: Patellar (L3-4) and Achilles' tendon (S1-S2).
- Paretic signs: negative paretic signs. The tested paretic signs are the following: Barré, Mingazzini.
- Spastic-irritative signs: negative. The tested spastic-irritative signs are the following: Babinski, Chaddock, Oppenheim and Rossolimo.
- Cerebellar examination: negative to taxes test.
- Clonus: absent

3.3.4. Lasègue / Reverse Lasègue

Negative

3.3.5. Sensation

The patient results to be normosensitive to deep and superficial sensation in the whole affected side, compared to contralateral side.

3.4. Musculoskeletal examination

3.4.1. Plumb-line

The plumb-line evaluation could not be performed given the patient's inability to stand without support.

3.4.2. Pelvis Palpation

The pelvis palpation could not be performed given the patient's inability to stand without support.

3.4.3. Breathing stereotype (calm breathing)

Sitting: No motion nor muscular activity in the abdominal department. Marked activity of the auxiliary (scaleni and sternocleidomastoid) muscles during inhalation leading to cranial shift of the thoracic cage.

- Supine: Slight abdominal department activation. Marked activity of the auxiliary muscles during inhalation leading to cranial shift of the thoracic cage.

3.4.4. Specific posture testing

- Two scales standing: Can't be tested
- Romberg test:
    I. Instable
- Trendelenburg sign: Can't be tested
- Véle's test: Can't be tested

3.4.5. Modification of standing

- Toes standing: Can't be tested
- Heels standing: Can't be tested
- One-leg standing: Can't be tested

3.4.6. Dynamic spine examination (performed in sitting position)

- Entire spine anteflexion

All the motion takes starting from L2 until C6. Cervical and lumbar tract are straight and in erect position they stand in hyperlordosis.

- Entire spine retroflexion

The motion takes places in L1 to L3 segment. The thoracic spine is blocked into flexion.

- Entire spine right side-bending

Movement taking place between T8 to L1 and in C-T junction. Reduced mobility of the rest of the spine.

- Entire spine left side-bending

Movement taking place in T-L junction. The rest of the spine presents general stiffness.

3.4.7. Gait analysis

The gait analysis was performed while walking (assisted) on the high walker (support under the elbows). The patient is unable to support his body-weight (even partially) on his left lower extremity leading to right side-bending of the whole body. The trunk position is in flexion highlighting a dominance of the thoracic kyphosis. For compensating the thoracic kyphosis and the trunk forward flexion, the cervical spine intercedes into hyperlordosis. The stride length is extremely reduced and asymmetrical (left-step longer). During the gait, the left foot shows “drop-foot sign” and the left hip is circumducted permitting a slight advancement of the left lower extremity. The gaze is ground-oriented, unless verbally corrected. The gait modification could not be tested.

3.4.8. Length test

The grades for the length test (Janda and Kendall methodology) are reported in Table 3.

Length test	L	R
Hamstring (Janda)	1	2
Hip flexors (bi and mono-articular)	moderate	marked
Piriformis	1	1
Triceps surae	slight (soleus)	moderate (soleus)
Pectoralis minor (Kendall)	moderate	moderate
Pectoralis major (90/125° - Kendall)	nn	nn
Upper trapezium	moderate	marked
Levator scapulae	moderate	marked
Paravertebrals	2	

Table 3: Length test grading

3.4.9. Strength test (Janda's scale grading)

In Table 4 and Table 5 are reported the strength test grading for UEE, LEE and facial muscles (Kendall's methodology).

Strength test		**L**	**R**
Ankle	Plantar flexors	1	4+
	Flexors digitorum l/b	1	5
	Flexor hallucis l/b	0	5
	Plantar dorsiflexors	1	4+
	Extensor digitorum l/b	2-	5
	Extensor hallucis l/b	0	4
Knee	Extensors	1	4
	Flexors	2	4+
Hip	Flexors	2	4+
	Abductors	1	4
	Extensors	1	4-
	Adductors	1	5
Facial	Buccinator	4	5
	Orbicularis orii	4	5
	Zygomaticus	3	5
	Orbicularis oculi	5	5
	Risorius	4	5
	Mentalis	4	5
	Frontalis	5	5

Table 4: Strength test grading LEE and facial muscles

Strength test		L	R
Wrist/Hand.	Flexor carpi rad/uln	3-	4+
	Flexor digitorum s/p	3	5
	Flexor pollicis s/p	2-	4
	Extensor carpi rad./uln.	2-	4
	Extensor digitorum com.	1	4
	Extensor pollicis l/b	1	4
Elbow	Biceps brachii	3-	5
	Triceps brachii	3-	5
	Pronator Teres	2	5
	Pronator Quadratus	2	4+
	Supinator	1	4+
Shoulder	Deltoid (medius)	2	5
	Pectoralis major (Kendall) (90/135°)	2	5
	Pectoralis minor (Kendall)	3	5
	Latissimus dorsi	3-	5
	Teres Minor	1	4-
	Teres Major	1	5
	Supraspinatus	1	4-
	External rotators	1	4+
	Internal Rotators	2	5
	Rhomboids	1	3+
	Upper trapezium	3	5

Table 5: Strength test grading UEE

3.4.10. RoM Measurements

In Table 6 and Table 7 are reported the active and passive range of motion values expressed in degrees (ARoM and PRoM, according Janda's methology).

Left side		AROM			PROM		
Shoulder	S:	10	0	40	30	0	170
	F:	30	0	0	80	0	0
	T:	15	0	10	130	0	110
	R:	20	0	50	60	0	55
Elbow	S:	0	0	80	0	0	140
	R:	60	0	30	90	0	65
Wrist	S:	20	10	50	60	0	80
	F:	10	0	0	15	0	25
Hip	S:	0	5	100	20	0	130
	F:	20	0	25	30	0	25
	R:	20	0	5	55	0	10
Knee	S:	0	0	80	5	0	110
Ankle	S:	0	0	25	10	0	40
	R:	5	0	5	15	0	40

Table 6: Left side RoM measurements

Right side		AROM			PROM		
Shoulder	S:	30	0	155	40	0	155
	F:	80	0	0	90	0	0
	T:	20	0	115	35	0	120
	R:	40	0	70	50	0	70
Elbow	S:	0	0	140	0	0	150
	R:	90	0	60	90	0	70
Wrist	S:	55	0	75	60	0	75
	F:	10	0	20	20	0	20
Hip	S:	5	5	110	5	0	125
	F:	30	0	30	35	0	30
	R:	45	0	5	50	0	5
Knee	S:	0	5	120	0	5	130
Ankle	S:	15	0	40	20	0	40
	R:	10	0	40	15	0	35

Table 7: Right side RoM measurements

3.4.11. Muscle tone palpation

In Table 8 are reported the values following the Muscle tone palpation. The results are reported according the following scale:

0	Atonic
1	Hypotonic
2	Normotonic
3	Slightly hypertonic
4	Moderately hypertonic
5	Markedly hypertonic

	L	R
Triceps Surae	1	3
Tibialis anterior	1	2
Rectus femoris	3	3
Psoas (inguinal palpation)	3	4
Quadratus lumborum	1	3
Lumbar paravertebral	2	4
Thoracic paravertebral	4	4
Cervical paravertebral	2	5
Suboccipital muscles	3	5
Upper trapezium	3	5
Pectoralis minor	1	5
Subscapularis	1	3
Biceps brachii	1	2
Triceps brachii	1	2
Wrist and fingers flexors	3	4
Wrist and fingers extensors	3	3

Table 8: Muscle tone palpation results

3.4.12. Joint play examination

In Table 9 are presented the values after the joint play examination ("R": restricted; "NR": non-restricted) (according Lewit's techniques).

		L	R
Lisfranc's joint		NR	NR
Chopard's joint		NR	NR
Sub-Talar joint		R	R
Talocrural joint		R	R
Patellar mobility		NR	NR
Tibio-fibular joint		NR	NR
Gleno-humeral joint		NR	NR
Acromio-clavicular joint		NR	NR
Sterno-clavicular joint		NR	R
Elbow joint		NR	NR
Carpo-metacarpal joint		NR	NR
Carpo-carpal joint		NR	NR
Sacro-Iliac joint		NR	R
Lumbar spine	Side-bending	R: L2 to L5 (left side-bending)	
	Anteflexion	R: L3 to L5	
	Retroflexion	R: L2/L3 segment	
Thoracic spine	Side-bending	R: Global (left side-bending)	
	Anteflexion	N R	
	Retroflexion	R: T1 to T5 segments	
First rib		R	R
Ribs		R: 2nd to 5th (inhalation, exhalation)	R: 4th and 5th (exhalation)
Cervical spine		Can't be tested	

Table 9: Joint play examination results

3.5. Conclusion of the Initial Examination

The patient reports the typical condition of the "Spinal-shock" stage. This variable period is the one interposing between the nervous system central lesion and the instauration of the typical spastic characteristics. The "Spinal shock" stage duration goes from few days to months. During this stage, on the affected side, we observe muscle flaccidity, areflexia, strength loss, reduction of joint stability, decreased ARoM and increased PRoM. The patient's Barthel Index scale was 70/100.

3.5.1. Therapeutic goals

- Restore functionality of lower and upper extremity in equal measure
- Reduce the effects of spasticity onset
- Allow ADL performance
- Restore symmetrical activity of facial muscles

3.5.2. Therapeutic plan

During the hospitalisation period (18/01/2021 to 03/02/2021), the patient accessed these different treatments:

- Upper extremity conditioning (motomed): 1/day for 15min, subjectively-applied resistance.
- Lower extremity conditioning (motomed): 1/day for 15min, subjectively-applied resistance.
- Antispastic electrostimulation: UE even days, LE odd days; 1/day
  Applied for proximal (e.g. knee extensors, knee flexors) and peripheral (e.g. plantar flexors, plantar dorsiflexors) muscles.

Duration: 10 min per muscular compartment (20 min in total every session)
Frequency (Hz): 1.00
Amplitude (mA): 25 (forearm), 44 (arm), 32 (lower leg), 57 (thigh)
Duration (ms): 0.20 (electrode 1), 0.50 (electrode 2)

- Reflex massage: 2/week
- Individual ergotherapy: 1/day for 45 to 60 min. This treatment is mostly focused on the rehabilitation of the upper extremity

4. Daily examination

In the following chapter, I am reporting the Daily examination content from the 19th January until the 2nd February 2021. The 29th February session is missing as the patient was unable to participate.

19/01/2021

Subjective

Pain grading: No pain described

Feeling: Major complaint associated with walking inability, instability and inability to accomplish daily tasks with the upper extremity.
In the present session he reports upper body tiredness, following ergotherapy.

Medication None

Objective

Inspection:
- Generalized hypotonus of the left body hemisphere
- Reduced ability to activate left body hemisphere muscles
- Increased muscle activation latency on affected side

Assessment

Patient's main problem consists in muscle activation. If solicited, he is able to activate the involved muscles except for more distal and spastic-antagonist muscles.
Blockage of sub-talar and talocrural joint on the left side.

Plan

Main objective:
-Anti-spastic muscles strengthening and improving/maintaining general mobility in anti-spastic movements. Main focus on activation of left ankle dorsiflexors, knee flexors and hip general mobility (abduction++)
- Stretching of plantar flexors, hip flexors (mono and bi-articular) and hip adductors
- Blocked joints mobilisation

Secondary objective:
- Gait pattern correction (assisted): improvement of weight bearing and symmetrical stride length

20/01/2021

Subjective

Pain grading: No pain described except for slight pain (2/10) on inferior part of left patella when walking.

Feeling: Major complaint associated with walking inability, instability and inability to accomplish daily tasks with the upper extremity.
In the present session he reports to be tired in the upper body after the ergotherapy session.

Medication: None

Objective

Inspection:
- Generalized hypotonus of the left body hemisphere
- Reduced ability to activate left body hemisphere muscles
- Increased muscle activation latency on affected side
- No sensory impairment
- Left sub-talar and talocrural joint blockage

Assessment

Patient's main problem consists in muscle activation. If solicited, he is able to activate the involved muscles except for more distal and spastic-antagonist muscles.
Blockage of sub-talar and talocrural joint on the left side.
Improved contraction ability of left plantar dorsiflexors hallucis and toes extensors.

Plan

Main objective:
-Anti-spastic muscles strengthening and improving/maintaining general mobility in anti-spastic movements. Main focus on activation of left ankle dorsiflexors, knee flexors and hip general mobility (abduction++)
- Stretching of plantar flexors, hip flexors (mono and bi-articular) and hip adductors
- Blocked joints mobilisation

Secondary objective:
- Gait pattern correction (assisted): improvement of weight bearing and symmetrical stride length.

21/01/2021

Subjective

Pain grading: No pain described

Feeling: The patient describes less tiredness after ergotherapy and electrostimulation session

Medication: None

Objective

Inspection: - Physiological movement in left talocrural and sub-talar joints
- Improved ability and confidence during wheelchair's transfers
- Prolongation of time spent standing (still under 1 minute)

Assessment

Improved muscles activation ability and reduced latency in muscular activation. The patient still shows greater weakness signs in the upper extremity compared to the lower extremity of the affected side. The weakness is observed particularly in the antispastic muscles.

Plan

Main objective: -Anti-spastic muscles strengthening and improving/maintaining general mobility in anti-spastic movements. Main focus on activation of left ankle dorsiflexors, knee flexors and hip general mobility (abduction++)
- Stretching of plantar flexors, hip flexors (mono and bi-articular) and hip adductors
- Recover symmetrical functionality of the facial muscles: Facial muscles' motor control training.

Secondary objective: - Gait pattern correction (assisted with right-side stick): improvement of weight bearing and symmetrical stride length. Improving the covered distance without rest (50m).
- Assisted stairs climbing: approaching stairs climbing with pathology-related modifications.

22/01/2021

Subjective

Pain grading: No pain described

Feeling: The patient describes tiredness after ergotherapy session

Medication: None

Objective

Inspection:
- General lower extremity strength improvement
- Autonomy improvement in displacements and bed transitions
- More visible reflexes (paretic and spastic-irritative)
- Still marked weakness of the upper extremity compared to lower

Assessment

The patient observes the first benefits of the therapy in terms of autonomy and ability of performing ADL. During walking (assisted with high-walker), the patient performs hip circumduction on the affected side and holds a narrow base of support. The steps are uncertain and they might be one in front of the other (dangerously).

Plan

Main objective:
- Improve voluntary muscles activity in the upper extremity: PNF 1st and 2nd diagonal (with important assistance)
- Recover symmetrical functionality of the facial muscles: Facial muscles' motor control training.
- Removal of wrong walking pattern (knee extension stiffness and hip circumduction): functional training for proper hip and knee flexion/extension motion.

Secondary objective:
- Gait pattern correction (assisted with right-side stick): improvement of weight bearing and symmetrical stride length.
- Assisted stairs climbing: correcting wrong patterns

25/01/2021

Subjective

Pain grading: No pain described

Feeling: Patient is satisfied of the progressions made. Satisfied of being able to walk again. Still problems in upper extremity functionality.

Medication None

Objective

Inspection: - General strength improvement notoriously in the lower extremity
- Autonomy improvement in general transitions (stick-assisted walking)
- More visible reflexes (paretic and spastic-irritative)
- Still marked weakness of the upper extremity compared to lower

Assessment

The patient lower extremity strength and autonomy is increased. In particular the patient is able to activate easily the anti-spastic muscles (e.g. tibialis anterior 3- / 5). Still marked weakness is detected on the upper extremity. Paretic facial muscles show signs of improvement being symmetrical in terms of activity.

Plan

Main objective: - Improve voluntary muscles activity in the upper extremity: PNF 1st and 2nd diagonal (with important assistance)
- Recover symmetrical functionality of the facial muscles: Facial muscles' motor control training.
- Removal of wrong walking pattern (knee extension stiffness and hip circumduction): functional training for proper hip and knee flexion/extension motion
- Hip flexors relaxation (mono and bi-articular): PIR method
- Plantar flexors relaxation (mono and bi-articular): PIR method

Secondary objective: - Gait pattern correction (assisted with right-side stick): improvement of weight bearing and symmetrical stride length.

26/01/2021

Subjective

Pain grading: No pain described

Feeling: Tiredness particularly related to the upper extremities

Medication: None

Objective

Inspection: - Upper extremity general weakness
- Stable symmetry of facial muscles activity
- Ribs (1st to 6th) blockage in both anterior and posterior aspect (inhalation and exhalation)
- SI joint bilateral blockage

Assessment

Despite the Ergotherapy intensive training, the upper extremity doesn't show marked progression of proximal joints (shoulder). The wrist and fingers extensors neither show progression signs. The lower extremity improved ARoM and mobility in anti-spastic patterns.

Plan

Main objective: - All examined blocked joint mobilisation:
- Scapular mobility: PNF diagonals (all 4)
- Scapulo-thoracic passive mobilisation
- Active-Assistive joint mobilisation (sitting and supine: Flexion, Extension, Abduction, Horizontal abduction and adduction)

Secondary objective: - Gait pattern correction (assisted with right-side stick): improvement of weight bearing and symmetrical stride length.
- Assisted stairs climbing: correcting wrong patterns.

27/01/2021

Subjective

Pain grading: No pain described

Feeling: Satisfied by the improvements in walking. Able to move without wheelchair. Walking with stick support on the right hand.

Medication: None

Objective

Inspection: - Improvement of voluntary contraction of left upper extremity muscles
- Free bilateral SI joint
- Blocked left sub-talar and talocrural joints

Assessment

Patient's main problem consists in muscle activation. If solicited, he is able to activate the proximal joints involved muscles (hip and shoulder joint). More distal muscles are more sensitive to voluntary contraction but still weak (particularly anti-spastic ones). Blockage of sub-talar and talocrural joint on the left side.

Plan

Main objective: - Pelvis mobilisation: PNF pelvic diagonals
- Crossed-chain muscles activation: 4 ½ months position
- Blocked joints mobilisation
- Hip adductors PIR
- Hip external rotators PIR

Secondary objective: - Gait pattern correction (assisted): improvement of weight bearing and symmetrical stride length.

28/01/2021

Subjective

Pain grading: No pain described

Feeling: In the present session the patient reports marked general tiredness

Medication: None

Objective

Inspection:
- Lower extremity spastic position visible onset
- Constant autonomy improvement
- Marked spastic reflex, paretic (notoriously in the lower extremity) and spastic-irritative signs
- Global progression of ARoM in the upper extremity (shoulder and elbow joint)
- Left carpo-metacarpal joint blockage

Assessment

The patient lower extremity strength and autonomy is increased. In particular the patient is able to activate easily the anti-spastic muscles (e.g. tibialis anterior). Still moderate weakness is detected on the upper extremity. Paretic facial muscles show signs of improvement being symmetrical in terms of activity.

Plan

Main objective:
- Restore physiological ARoM in scapula mobility: PNF mobilisation in both diagonals
- Restore physiological ARoM in shoulder girdle: AARoM (stick) in Flexion, Horizontal Abduction/Adduction
- Contracture's prevention: finger flexors passive stretching
- Wrist, elbow, shoulder and scapula-thoracic mobilisation

Secondary objective:
- Gait pattern correction (assisted with right-side crutch): improvement of weight bearing and symmetrical stride length.
- Assisted stairs climbing: correcting wrong patterns (hip circumduction, reduced knee flexion and drop-foot)

01/02/2021

Subjective

Pain grading: No pain described

Feeling: The patient describes an intense sense of tiredness related to the ADL (forcing him-self to do them with impaired side)

Medication: None

Objective

Inspection:
- Left SI joint blockage
- Marked weakness of left UE extensors (mainly fingers and wrist) (1 and 3 out of 5)
- Plantar flexors spasticity and shortness
- Hip flexors (mono and bi-articular) shortness

Assessment

The global patient's condition is improved but is conditioned by the tiredness.

The lower extremity shows the typical Wernicke-Mann positioning and functionality. Particularly when walking, the leg presents stiffness (straightening), foot in plantar flexion and the hip circumducted.

Plan

Main objective:
- SI joint mobilisation
- Elbow and shoulder ARoM improvement: A-ARoM into flexion, horizontal abduction, horizontal adduction, extension, abduction
- Pelvic mobilisation: PNF diagonals (1st and 2nd)
- Symmetrical facial muscles activity: Facial muscles' motor control training.
- Hip flexors and plantar flexors stretching (mono and bi-articular)

Secondary objective:
- Gait pattern correction: reduction of stiff-leg phenomenon and compensative hip circumduction
- Assisted stairs climbing: stimulating knee flexion and ankle dorsiflexion while climbing stairs.

5. Kinesiological Examination at Discharge

The present Kinesiological examination took place on the 02/02/2021. It reports exclusively the differences between the status praesens and the initial examination. The patient spent a period of 12 days of therapy (16 of hospitalisation) in the Rehabilitation centre.

Ashworth scale at discharge: 1+

5.1. Neurological examination

5.1.1. Cranial Nerves

The Facial nerve strength evaluation is reported in the paragraph “Strength test at Discharge”.

5.1.2. Upper limbs

- Deep tendon reflexes testing: global mild hyperreflexia of the left body hemisphere.

  The tested deep tendon reflexes are the following: Biceps brachii (C5-6), Brachioradialis (C5-6) and Triceps brachii (C7-8)
- Paretic signs: positive. The tested paretic signs are the following: Mingazzini, Barré, Hanzal and Du Four.
- Spastic-irritative signs: positive. The tested spastic-irritative signs are the following: Hoffman, Juster and Tromner.

5.1.3. Lower limbs examination

- Deep reflexes: global mild hyperreflexia of the left body hemisphere. The tested deep tendon reflexes are the following: Patellar (L3-4) and Achilles’ tendon (S1-S2).
- Paretic signs: positive. The tested paretic signs are the following: Barré, Mingazzini.
- Spastic-irritative signs: positive. The tested spastic-irritative signs are the following: Babinski, Chaddock, Oppenheim and Rossolimo.
- Clonus: present

5.2. Musculoskeletal examination at Discharge

5.2.1. Specific posture testing

- Two scales standing: Can't be tested
- Romberg test:
    I. Negative
    II. Negative
    III. Negative

- Modification of standing
    I. Tip-toes standing: able to be performed bilaterally but the weight loading mostly takes place on the right side (shift of the trunk to the right and support of the crutch).
    II. Heels standing: able to be performed only on non-affected side. On affected side, it is possible to observe the extensor's activation and the toes extension but the ankle motion is not performed.

5.2.2. Gait analysis at Discharge

The gait analysis was performed while walking (assisted with stick on the right side). The patient shows the typical spastic signs (Wernicke-Mann's posture) on the left-body hemisphere. The left foot is in complete plantar flexion and inversion. The left knee is held straight while the hip is in internal rotation and constant slight flexion. During the gait, the patient circumducts the hip allowing the forward advancement of the whole lower extremity. The stride length is asymmetrical (right-step longer) and on a narrow base of support. The whole-body axis leans on the right side providing better stability (quadruped walking stick). As a consequence, the right shoulder as well as the scapula is higher than contralateral, and the head is slightly bent to the left. The left UE is straight but the fingers are in constant flexion. The left scapula is more abducted and presents the characteristics of the scapula alata.

5.2.3. Length test at Discharge

The grades for the length test (Janda's and Kendall's methodology) are reported in Table 10.

Length test	L	R
Hamstring (Janda)	1	2
Hip flexors (bi and mono-articular)	moderate	moderate
Piriformis	2	1
Triceps surae	moderate (soleus)	moderate (soleus)
Pectoralis minor (Kendall)	slight	moderate
Pectoralis major (90/125° - Kendall)	nn	nn
Upper trapezium	moderate	marked
Levator scapulae	moderate	marked
Paravertebrals	2	

Table 10: Length test grading at Discharge

5.2.4. Strength test at Discharge (Janda's scale grading)

In Table 11 and Table 12 are reported the strength test grading for UEE, LEE and facial muscles at Discharge (Kendall's methodology).

	Strength test	L	R
Ankle	Plantar flexors	4	4+
	Flexors digitorum l/b	4	5
	Flexor hallucis l/b	4	5
	Plantar dorsiflexors	2	4+
	Extensor digitorum l/b	3+	5
	Extensor hallucis l/b	3	4
Knee	Extensors	4-	5
	Flexors	4	4+
Hip	Flexors	4	5
	Abductors	3	4
	Extensors	3	4-
	Adductors	4+	5
Facial	Buccinator	5	5
	Orbicularis orii	4	5
	Zygomaticus	4	5
	Orbicularis oculi	5	5
	Risorius	4	5
	Mentalis	4	5
	Frontalis	5	5

Table 11: Strength test grading LEE and facial muscles at Discharge

Strength test		L	R
Wrist	Flexor carpi rad/uln	3-	4+
	Flexor digitorum s/p	3+	5
	Flexor pollicis s/p	3-	4
	Extensor carpi rad/uln	2	4
	Extensor digitorum com	2	4
	Extensor pollicis l/b	2	4
Elbow	Biceps brachii	3	5
	Triceps brachii	3-	5
	Pronator Teres	3	5
	Pronator Quadratus	2+	4+
	Supinator	2	4+
Shoulder	Deltoid (medius)	3+	5
	Pectoralis major (Kendall) (90/135°)	4-	5
	Pectoralis minor (Kendall)	4	5
	Latissimus dorsi	3	5
	Teres Minor	3-	4-
	Teres Major	3-	5
	Supraspinatus	2	4-
	External rotators	2	4+
	Internal Rotators	3	5
	Rhomboids	2+	3+
	Upper trapezium	4-	5

Table 12: Strength test grading UEE at Discharge

5.2.5. RoM Measurements at Discharge

In Table 13 and Table 14 are reported the active and passive range of motion values expressed in degrees at Discharge (ARoM and PRoM, according Janda's methology).

Left side		AROM			PROM		
Shoulder	S:	15	0	40	30	0	170
	F:	110	0	0	120	0	0
	T:	95	0	90	130	0	110
	R:	40	0	50	60	0	55
Elbow	S:	0	0	120	0	0	140
	R:	65	0	45	90	0	65
Wrist	S:	20	10	60	60	0	80
	F:	10	0	0	15	0	25
Hip	S:	5	0	100	20	0	110
	F:	20	0	25	30	0	25
	R:	30	0	10	55	0	10
Knee	S:	0	0	110	5	0	120
Ankle	S:	5	0	35	10	0	40
	R:	5	0	10	15	0	40

Table 13: Left side RoM measurements at Discharge

Right side		AROM			PROM		
Shoulder	S:	30	0	155	40	0	155
	F:	80	0	0	90	0	0
	T:	20	0	115	35	0	120
	R:	40	0	70	50	0	70
Elbow	S:	0	0	140	0	0	150
	R:	90	0	60	90	0	70
Wrist	S:	55	0	75	60	0	75
	F:	10	0	20	20	0	20
Hip	S:	5	5	110	5	0	125
	F:	30	0	30	35	0	30
	R:	45	0	5	50	0	5
Knee	S:	0	5	120	0	5	130
Ankle	S:	15	0	40	20	0	40
	R:	10	0	40	15	0	35

Table 14: Right side RoM measurements at Discharge

5.2.6. Muscle tone palpation at Discharge

In Table 15 are reported the values following the Muscle tone palpation at Discharge. The results are reported according the following scale:

0	Atonic
1	Hypotonic
2	Normotonic
3	Slightly hypertonic
4	Moderately hypertonic
5	Markedly hypertonic

	L	R
Triceps Surae	4	3
Tibialis anterior	1	2
Rectus femoris	3	3
Psoas (inguinal palpation)	4	4
Quadratus lumborum	2	3
Lumbar paravertebral	3	4
Thoracic paravertebral	5	4
Cervical paravertebral	4	5
Suboccipital muscles	4	4
Upper trapezium	5	5
Pectoralis minor	5	5
Subscapularis	3	3
Biceps brachii	1	2
Triceps brachii	1	2
Wrist and fingers flexors	4	3
Wrist and fingers extensors	1	3

Table 15: Muscle tone palpation results at Discharge

5.2.7. Joint play examination at Discharge

In Table 16 are presented the values following the joint play examination at discharge ("R": restricted; "NR": non-restricted) (according Lewit's techniques).

		L	R
Lisfranc's joint		NR	NR
Chopard's joint		NR	NR
Sub-Talar joint		NR	NR
Talocrural joint		R	NR
Patellar mobility		R: lateral	NR
Tibio-fibular joint		NR	NR
Gleno-humeral joint		NR	NR
Acromio-clavicular joint		NR	NR
Sterno-clavicular joint		NR	R
Elbow joint		NR	NR
Carpo-metacarpal joint		NR	NR
Carpo-carpal joint		NR	NR
Sacro-Iliac joint		NR	R
Lumbar spine	Side-bending	R: L2 to L5 (left side-bending)	
	Anteflexion	R: L3 to L5	
	Retroflexion	R: L2/L3 segment	
Thoracic spine	Side-bending	R: Global (left side-bending)	
	Anteflexion	NR	
	Retroflexion	R: T1 to T5 segments	
First rib		R	NR
Ribs		R: 2nd to 3rd (inhalation; exhalation)	NR
Cervical spine		Can't be tested	

Table 16: Joint play examination results at Discharge

5.3. Kinesiological Examination Conclusion at Discharge

After a period of 12 days of therapy (16 of hospitalisation), the patient is discharged with part of the typical signs of "Wernicke-Mann's" posture. The characteristic spasticity is mainly observable in the left LE (see Gait analysis). The global body alignment has tendency to lean on the right side, looking for a better body support through the stick held on the right hand.

Concerning the facial muscles activity, the symmetry is not complete but allows the patient to better express himself (see "Strength Test at Discharge").

The left UE is aligned along the body but the strength profile is still lacking, particularly in what concerns the extensors posterior chain but, from a global point of view, the strength improved. The ARoM of the affected extremity has also improved parallelly with the strengthening. The ability of the patient to perform ADL has improved as shows the Barthel index scale result (80/100).

Unfortunately, correlated with the reduced treatment period, the results are positive but limited.

5.4. Initial and final Kinesiological Examination comparison

Comparing the two Kinesiological Examination, the patient showed a marked improvement in terms of general strength and muscle control. Parallelly, the ARoM of the affected side improved.

It is important to notice the positive evolution of the patient's autonomy. Indeed, in the initial examination, the patient was unable to bear his own weight, stand and walk. At Discharge instead, he was able to walk and climb stairs. From a broader point of view, his ability to perform ADL improved as well, as the Barthel Scale index's evolution confirms (from 70 to 80/100).

On the other hand, despite the efforts made for reducing its onset, spasticity started to manifest in the end of the treatment period (at Discharge, Ashworth scale: 1+). Connected to it, typical spastic posture and gait are observed.

In the next tables (Table 17, Table 18, Table 19), I compare the differences between before and after the treatment period, concerning the left side of the body. The reported values are only the one which reported a relevant modification of the value.

Strength		Initial Exam.	Final Exam.
Ankle	Plantar flexors	1	4
	Plantar dorsiflexors	1	2
Knee	Knee extensors	1	4-
	Knee flexors	2	4
Hip	Flexors	2	4
	Abductors	1	3
	Extensors	1	3
	Adductors	1	4+
Wrist/Hand	Flexor digitorum s/p	3	3+
	Flexors pollicis s/p	2-	3-
	Extensor digitorum com	1	2
	Extensor pollicis l/b	1	2
	Deltoid (medius)	2	3+
	Pectoralis major (Kendall) (90/135°)	2	4-
	Pectoralis minor (Kendall)	3	4
	Teres minor	1	3-
	Teres major	1	3-
	Supraspinatus	1	2
	External rotators	1	2
	Internal rotators	2	3
	Rhomboids	1	2+
	Upper trapezium	3	4-

Table 17: Strength comparison (left-side)

ARoM		Initial Exam.			Final Exam.		
Shoulder	F:	30	0	0	110	0	0
	T:	15	0	10	95	0	90
	R:	20	0	50	40	0	50
Elbow	S:	0	0	80	0	0	120
	R:	60	0	30	65	0	45
Hip	S:	0	5	100	5	0	100
	R:	20	0	5	30	0	10
Knee	S:	0	0	80	0	0	110
Ankle	S:	0	0	25	5	0	35

Table 18: ARoM comparison (left-side)

The results for the muscle tone palpation are reported according the following scale:

0	Atonic
1	Hypotonic
2	Normotonic
3	Slightly hypertonic
4	Moderately hypertonic
5	Markedly hypertonic

Muscle tone palpation	Initial Exam.	Final Exam.
Triceps surae	1	4
Psoas (inguinal palpation)	3	4
Quadratus lumborum	1	2
Lumbar paravertebral	2	3
Thoracic paravertebral	4	5
Cervical paravertebral	2	4
Suboccipital muscles	3	4
Upper trapezium	3	5
Pectoralis minor	1	5
Subscapularis	1	3
Wrist and fingers flexors	3	4
Wrist and fingers extensors	3	1

Table 19: Muscle tone comparison (left-side)

5.4.1. Suspected prognosis of the patient's progression

After the Discharge, the patient might observe a gradual evolution of the spasticity characteristics which could also be directly associated with muscular pain. The newly assumed posture might also bring to other musculoskeletal problems which are the result of the body compensatory strategy.

From the motoric point of view, it is important to force himself to use the affected limbs in the daily activities. IN order to better accomplish daily tasks, it is very important to strengthen the weak muscles stretch and relax the spastic one.

Ideally, the patient should pursue the physiotherapeutic treatments privately or in a structure. An idea of self-treatment which the patient should perform daily is reported underneath.

5.4.2. Self-treatment

As a self-treatment, the patient was instructed, for what concerns the affected LE, to regularly perform movements in full RoM and slowly, allowing a better control. Concerning the affected UE, AARoM exercises are highly recommended (e.g. are reported following)

UE:

- AARoM exercises with stick (held with both hands): movement in shoulder and elbow flexion/extension, shoulder horizontal abduction/adduction
- Finger and hand flexors stretching/relaxation through Passive stretching and PIR method
- Wrist/Fingers extensors strengthening through AARoM with contralateral hand support

LE:

- Hip flexors relaxation/stretching through PIR method and passive stretching
- Plantar flexors stretching/relaxation through Passive stretching and PIR method
- Foot/Toes extensors strengthening through AARoM (motion is accompanied with hand support)
- Hip adductors relaxation through PIR method
- Hip abductors strengthening in side lying position or in supine

The Self-treatment exercises should be performed as often as possible with the limit of quality/tiredness.

6. Personal Conclusion

The realisation of the theoretical part of this thesis taught me a lot about the stroke somatic manifestations and their background mechanisms.

The placement permitted to experience a practical application of all the acquired knowledge during the bachelor. Furthermore, I understood that everything I learned can be shaded. This worth also for the treatment approach; particularly through the guide of my thesis supervisors I learned that every therapeutic goal can be achieved in a wide multitude of ways. Which treatment is the best, depends on the patient and on its psychological condition. For the therapist, two factors appear essential: curiosity and passion.

An aspect which is totally new when performing the first placements is the human dimension; from an academic point of view, we have tendency to forget about empathy and the relevance this one has in the patient's eyes.

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8. Approval by Ethic Committee

CHARLES UNIVERSITY
FACULTY OF PHYSICAL EDUCATION AND SPORT
José Martího 31, 162 52 Prague 6-Veleslavín

Application for Approval by UK FTVS Ethics Committee

of a research project, thesis, dissertation or seminar work involving human subjects

The title of a project: Physiotherapy Case Study: treatment of patient after Ischemic Cerebral Vascular Accident

Project form: research work – bachelor

Period of realization of the project: January 2021, February 2021

Applicant: Giammaria Cattozzo, UK FTVS Physiotherapy department

Main researcher: Giammaria Cattozzo, UK FTVS Physiotherapy department

Workplace: Rehabilitační nemocnice Beroun

Co-researcher(s):

Supervisor: PhDr. Mala Jitka

Financial support:

Project description: The aim of this longitudinal study is to propose an overview on the theoretical contents concerning the Ischemic Cerebral Vascular Accident. The present study will constitute my Bachelor Thesis. It is divided into a first theoretical part followed by a Study Case reporting the complete initial and final assessment procedure, followed by a daily therapy description of the patient. The data collection will be subjectively performed by the thesis author. The participant will follow a rehabilitation program specific to his pathology.

Characteristics of participants in the research: The involved participant is one. The selection criteria are related to his pathology (Ischemic Cerebral Vascular Accident). The patient selection will be performed according the hospitalisation duration (at least 8 physiotherapeutic session have to be completed). Contraindications include potential deadly complications of the patient or inability to satisfy the above-mentioned criteria.

Ensuring safety within the research: All the practical component will be performed under the supervision of an experienced and qualified supervisor in a safe environment. The only employed methods will be non-invasive: manual contact, electrotherapy and hydrotherapy. No invasive methods is performed. Risks of therapy and methods will not be higher than the commonly anticipated risks for this type of therapy.

Ethical aspects of the research: The collected data will be anonymized within one week after the end of working with the patient. I understand that anonymization means that the text does not use any item of information or combination of items that could lead to the identification of a person. I will be careful not to enable recognition of a person in the text of the thesis, especially within the anamnesis. After the text has been anonymized, any personal data still kept elsewhere will be deleted.
Photographs of the participant will be anonymized within one week after being taken by blurring the face, parts of the body or any characteristics that could lead to identification of the person. After anonymization any non-anonymized photographs will be deleted.
All collected data will be safely stored on a PC safeguarded by a keyword in a locked room, any data in paper form will be kept safely under lock and key in a locked room. The data will be processed, safely retained and published in an anonymous way in the bachelor thesis.
I shall ensure to the maximum extent possible that the research data will not be misused.
Informed Consent: attached

It is the duty of **all participants of the research team** to protect life, health, dignity, integrity, the right to self-determination, privacy and protection of the personal data of all research subjects, and to undertake all possible precautions. Responsibility for the protection of all research subjects lies on the researcher(s) and not on the research subjects themselves, even if they gave their consent to participation in the research. All participants of the research team must take into consideration ethical, legal and regulative norms and standards of research involving human subjects applicable not only in the Czech Republic but also internationally.

CHARLES UNIVERSITY
FACULTY OF PHYSICAL EDUCATION AND SPORT
José Martího 31, 162 52 Prague 6-Veleslavín

I confirm that this project description corresponds to the plan of the project and, in case of any change, especially of the methods used in the project, I will inform the UK FTVS Ethics Committee, which may require a re-submission of the application form.

In Prague, 25/01/2021 Applicant's signature:

Approval of UK FTVS Ethics Committee

The Committee: Chair: doc. PhDr. Irena Parry Martínková, Ph.D.
Members: prof. PhDr. Pavel Slepička, DrSc.
prof. MUDr. Jan Heller, CSc.
PhDr. Pavel Hráský, Ph.D.
Mgr. Eva Prokešová, Ph.D.
Mgr. Tomáš Ruda, Ph.D.
MUDr. Simona Majorová

The research project was approved by UK FTVS Ethics Committee under the registration number: 072/2021

Date of approval: 26.1.2021

UK FTVS Ethics Committee reviewed the submitted research project and **found no contradictions** with valid principles, regulations and international guidelines for carrying out research involving human subjects.

The applicant has met the necessary requirements for receiving approval of UK FTVS Ethics Committee.

Stamp of UK FTVS

UNIVERZITA KARLOVA
Fakulta tělesné výchovy a sportu
José Martího 31, 162 52, Praha 6
– 20 –

Signature of the Chair of
UK FTVS Ethics Committee

8.1. Informed consent

UNIVERZITA KARLOVA
FAKULTA TĚLESNÉ VÝCHOVY A SPORTU
José Martího 31, 162 52 Praha 6-Veleslavín

INFORMOVANÝ SOUHLAS

Vážená paní, vážený pane,

v souladu se Všeobecnou deklarací lidských práv, nařízením Evropské Unie č. 2016/679 a zákonem č. 110/2019 Sb. – o zpracování osobních údajů, Helsinskou deklarací, přijatou 18. Světovým zdravotnickým shromážděním v roce 1964 ve znění pozdějších změn (Fortaleza, Brazílie, 2013) a dalšími obecně závaznými právními předpisy Vás žádám o souhlas s prezentováním a uveřejněním výsledků vyšetření a průběhu terapie prováděné v rámci praxe na[1] ……., kde Vás příslušně kvalifikovaná osoba seznámila s Vaším vyšetřením a následnou terapií. Výsledky Vašeho vyšetření a průběh Vaší terapie bude publikován v rámci bakalářské práce na UK FTVS, s názvem[2] ……

Cílem této bakalářské práce je ……

Získané údaje, fotodokumentace, průběh a výsledky terapie budou uveřejněny v bakalářské práci v anonymizované podobě. Osobní data nebudou uvedena a budou uchována v anonymní podobě. V maximální možné míře zabezpečím, aby získaná data nebyla zneužita.

Jméno a příjmení řešitele ……. Podpis:……

Jméno a příjmení osoby, která provedla poučení[3]…… Podpis:……

Prohlašuji a svým níže uvedeným vlastnoručním podpisem potvrzuji, že dobrovolně souhlasím s prezentováním a uveřejněním výsledků vyšetření a průběhu terapie ve výše uvedené bakalářské práci, a že mi osoba, která provedla poučení, osobně vše podrobně vysvětlila, a že jsem měl(a) možnost si řádně a v dostatečném čase zvážit všechny relevantní informace, zeptat se na vše podstatné a že jsem dostal(a) jasné a srozumitelné odpovědi na své dotazy. Byl(a) jsem poučen(a) o právu odmítnout prezentování a uveřejnění výsledků vyšetření a průběhu terapie v bakalářské práci nebo svůj souhlas kdykoli odvolat bez represí, a to písemně zasláním Etické komisi UK FTVS, která bude následně informovat řešitele.

Místo, datum ……

Jméno a příjmení pacienta …… Podpis pacienta: ……

Jméno a příjmení zákonného zástupce[4] ……..

Vztah zákonného zástupce k pacientovi …… Podpis: ……

1 *Uveďte pracoviště.*
2 *Uveďte název práce, nebo alespoň název předběžný.*
3 *Je-li řešitel s pacientem v závislém postavení, poučení provádí jiná příslušně kvalifikovaná osoba.*
4 *Uveďte pouze v případě, má-li pacient omezenou způsobilost k právním úkonům (např. je-li nezletilý).*