Introduction

Interactive image – guided neurosurgery for the resection of brain tumors was developed within the last 10 years at different neurosurgical centers around the world to improve the safety of the surgery and the functional outcome of the patient (**Chernove et al .,2005**).

In the late 80s the computer technology had developed to a stage that made it possible to use preoperative image data to tell the position of a tool in the brain. The concept of neuronavigation was born, and during the next decade a lot of effort was put into technical development and clinical evaluation of neuronavigation systems. Eventually, neuronavigation became a standard tool for planning and guidance of brain surgery **(Unsgård G, et al. 2002)**

The term neuronavigation is neologism used to describe the set of computer assisted technologies used by neurosurgeons to guide "navigate" within the confines of the skull during surgery (**Garslandt et al., 2002**).

Image-guided neuronavigation systems are very useful for intraoperative identification of the anatomic structures involved in the procedure. This is especially true in case of

distorted anatomy due to a particular growth pattern of the lesion which causes the classic landmarks being not easily identifiable. In such cases, neuronavigation can help to maintain the surgeon's orientation. **(de Divitiis E et al., 2007).**

Neuronavigation is useful in providing orientation to the surgeon with sufficient application accuracy. It facilitates a precise planning of the surgical vector to target small, subcortical lesions. **(Peter et al., 2006)**

Cranial openings should be exactly placed and not larger than necessary, but sufficiently large to not endanger the safety of the operation. Neuronavigation is routine in many practices, and intraoperative imaging may become so in the future (**Hernesniemi J et al., 2005**).

The clinical trials proved that the employed neuronavigation system is versatile, safe and that there are no adverse effects, complication or surgical mortality due to the devise. It enables the surgeon to plan smaller sized and better centered skin incisions and craniotomies and to approach the targeted lesions with less dissection of the intact brain tissue. Despite more radical removal of lesions the overall invasiveness of

the operation was decreased, and the surgeons feeling of safety could be improved (**Gumprecht et al., 2005**).

In general, neuronavigation can be used for all neurosurgical procedures. The additional time that is necessary for the navigation set up range is between 15 and 30 min and is justifiable. Sometimes, the navigation is used only at the beginning of surgery to perform a perfectly located small craniotomy and sometimes it is used throughout the entire procedure **(Gong J et al., 2007).**

Modern brain neuronavigation-using augmented reality navigation and realtime intraoperative MRI updated navigation-allows performing deep-seated operations more safely and accurately **(Kawamata T et al., 2002).**

Aim of the Work

The aim of this work is to demonstrate and evaluate using of neuronavigator in excision brain tumors.

Brain tumors

Brain tumors are one of the most common tumors in humans. They are the second most common form of malignancy in children and the sixth to eight most common form of malignancy in adults. Primary tumors of the brain and spine account for less than 2% of all malignancies but are responsible for 7% of the years of life lost from cancer prior to 70 years of age. In childhood, these figures are even more dramatic, with primary brain tumors accounting for 20% of malignant tumors diagnosed before 15 years of age. **(Black PM et al., 2008)**

The WHO classification of Brain tumors:

The World Health Organization established an international tumor classification system in 1957 in order to initiate a classification and grading system with worldwide acceptance and usage since without such a system and the clearly defind histopathological and clinical diagnostic criteria it was not possible to conduct epidemiological studies and clinical trials beyond institutional and national boundaries. **(Kleihues P. et al., 2002)**

The third edition was published 2000, the so-called WHO blue book not only described and graded all brain tumors in

precision, but also epidemiological, clinical, imaging and genetic information was presented in a structured fashion. **(Kleihues P. et al., 2002)**

Currently the fourth edition of the WHO classification of tumors of the central nervous system, published in 2007 lists new entities and histological variants. **(Louis DN et al., 2007)**

Table (1): The 2007 WHO classification of neuro-oncological entities **(Louis DN et al., 2007)**

TUMOURS OF NEUROEPITHELIAL TISSUE	
Astrocytic tumours	
Pilocytic astrocytoma	9421/1[1]
Pilomyxoid astrocytoma	*9425/3**
Subependymal giant cell astrocytoma	9384/1
Pleomorphic xanthoastrocytoma	9424/3
Diffuse astrocytoma	9400/3
Fibrillary astrocytoma	9420/3
Gemistocytic astrocytoma	9411/3
Protoplasmic astrocytoma	9410/3
Anaplastic astrocytoma	9401/3
Glioblastoma	9440/3
Giant cell glioblastoma	9441/3
Gliosarcoma	9442/3
Gliomatosis cerebri	9381/3
Oligodendroglial tumours	
Oligodendroglioma	9450/3
Anaplastic oligodendroglioma	9451/3
Oligoastrocytic tumours	
Oligoastrocytoma	9382/3
Anaplastic oligoastrocytoma	9382/3
Ependymal tumours	
Subependymoma	9383/1
Myxopapillary ependymoma	9394/1
Ependymoma	9391/3
Cellular	9391/3
Papillary	9393/3
Clear cell	9391/3
Tanycytic	9391/3
Anaplastic ependymoma	9392/3
Choroid plexus tumours	
Choroid plexus papilloma	9390/0
Atypical choroid plexus papilloma	*9390/1**
Choroid plexus carcinoma	9390/3
Other neuroepithelial tumours	
Astroblastoma	9430/3
Chordoid glioma of the third ventricle	9444/1
Angiocentric glioma	*9431/1**
Neuronal and mixed neuronal-glial tumours	
Dysplastic gangliocytoma of cerebellum (Lhermitte-Duclos)	9493/0
Desmoplastic infantile astrocytoma/ ganglioglioma	9412/1
Dysembryoplastic neuroepithelial tumour	9413/0
Gangliocytoma	9492/0
Ganglioglioma	9505/1
Anaplastic ganglioglioma	9505/3
Central neurocytoma	9506/1
Extraventricular neurocytoma	*9506/1**
Cerebellar liponeurocytoma	*9506/1**
Papillary glioneuronal tumour	*9509/1**
Rosette-forming glioneuronal tumour of the fourth ventricle	*9509/1**
Paraganglioma	8680/1
Tumours of the pineal region	
Pineocytoma	9361/1
Pineal parenchymal tumour of intermediate differentiation	9362/3
Pineoblastoma	9362/3
Papillary tumour of the pineal region	*9395/3**
Embryonal tumours	
Medulloblastoma	9470/3
Desmoplastic/nodular medulloblastoma	9471/3
Medulloblastoma with extensive nodularity	*9471/3**
Anaplastic medulloblastoma	*9474/3**
Large cell medulloblastoma	9474/3
CNS primitive neuroectodermal tumour	9473/3
CNS Neuroblastoma	9500/3
CNS Ganglioneuroblastoma	9490/3
Medulloepithelioma	9501/3
Ependymoblastoma	9392/3
Atypical teratoid / rhabdoid tumour	9508/3
TUMOURS OF CRANIAL AND PARASPINAL NERVES	
Schwannoma (neurilemoma, neurinoma)	9560/0
Cellular	9560/0
Plexiform	9560/0
Melanotic	9560/0
Neurofibroma	9540/0
Plexiform	9550/0

[1] Morphology code of the International Classification of Diseases for Oncology (ICD-O) {614A} and the Systematized Nomenclature of Medicine (http://snomed.org). Behaviour is coded /0 for benign tumours, /3 for malignant tumours and /1 for borderline or uncertain behaviour.

* The italicised numbers are provisional codes proposed for the 4th edition of ICD-O. While they are expected to be incorporated into the next ICD-O edition, they currently remain subject to change.

Perineurioma	
Perineurioma, NOS	9571/0
Malignant perineurioma	9571/3
Malignant peripheral nerve sheath tumour (MPNST)	
Epithelioid MPNST	9540/3
MPNST with mesenchymal differentiation	9540/3
Melanotic MPNST	9540/3
MPNST with glandular differentiation	9540/3

TUMOURS OF THE MENINGES

Tumours of meningothelial cells	
Meningioma	9530/0
Meningothelial	9531/0
Fibrous (fibroblastic)	9532/0
Transitional (mixed)	9537/0
Psammomatous	9533/0
Angiomatous	9534/0
Microcystic	9530/0
Secretory	9530/0
Lymphoplasmacyte-rich	9530/0
Metaplastic	9530/0
Chordoid	9538/1
Clear cell	9538/1
Atypical	9539/1
Papillary	9538/3
Rhabdoid	9538/3
Anaplastic (malignant)	9530/3
Mesenchymal tumours	
Lipoma	8850/0
Angiolipoma	8861/0
Hibernoma	8880/0
Liposarcoma	8850/3
Solitary fibrous tumour	8815/0
Fibrosarcoma	8810/3
Malignant fibrous histiocytoma	8830/3
Leiomyoma	8890/0
Leiomyosarcoma	8890/3
Rhabdomyoma	8900/0
Rhabdomyosarcoma	8900/3
Chondroma	9220/0
Chondrosarcoma	9220/3
Osteoma	9180/0
Osteosarcoma	9180/3
Osteochondroma	9210/0
Haemangioma	9120/0
Epithelioid haemangioendothelioma	9133/1
Haemangiopericytoma	9150/1
Anaplastic haemangiopericytoma	9150/3
Angiosarcoma	9120/3
Kaposi sarcoma	9140/3
Ewing sarcoma - PNET	9364/3
Primary melanocytic lesions	
Diffuse melanocytosis	8728/0
Melanocytoma	8728/1
Malignant melanoma	8720/3
Meningeal melanomatosis	8728/3
Other neoplasms related to the meninges	
Haemangioblastoma	9161/1

LYMPHOMAS AND HAEMATOPOIETIC NEOPLASMS

Malignant lymphomas	9590/3
Plasmacytoma	9731/3
Granulocytic sarcoma	9930/3

GERM CELL TUMOURS

Germinoma	9064/3
Embryonal carcinoma	9070/3
Yolk sac tumour	9071/3
Choriocarcinoma	9100/3
Teratoma	9080/1
Mature	9080/0
Immature	9080/3
Teratoma with malignant transformation	9084/3
Mixed germ cell tumour	9085/3

TUMOURS OF THE SELLAR REGION

Craniopharyngioma	9350/1
Adamantinomatous	9351/1
Papillary	9352/1
Granular cell tumour	9582/0
Pituicytoma	*9432/1**
Spindle cell oncocytoma of the adenohypophysis	*8291/0**

METASTATIC TUMOURS

Management of brain tumors

The role of surgery in the treatment of intrinsic cerebral tumors is based mainly on the ability to maximize safe tumor removal and alleviate focal neurological deficits secondary to mass effect and increased intracranial pressure. Understanding the localization of function in the human cerebrum is important in the surgical decision making process. Both the cortical and subcortical pathways within and adjacent to intrinsic cerebral tumors can be identified during tumor removal to prevent permanent morbidity **(Berger MS et al., 2007)**

Advances in both surgical techniques and the surgical armamentarium have made possible access to and complete removal of the tumor mass in a great many patients. Many benign tumors are thereby cured, and many patients experience a reduced incidence of complications and an improved quality of life. A good example of what can be achieved with modern techniques and approaches is seen with tumors located at the base of the skull. **(Brem H et al., 2011)**

Radical removal of even malignant tumors can result in prolongation of survival with a relatively low complication

rate. This has been seen after the resection of low- and high-grade gliomas, as well as metastatic brain tumors. Such resections have been aided by imaging guidance, ultrasound localization, intraoperative magnetic resonance imaging and computed tomography, cortical mapping, and awake procedures whenever necessary, with the ultimate goal being maximal removal of the mass with preservation of zones of function that might lie adjacent to its borders. **(Brem H et al., 2011)**

In addition to surgery, improved localization of the tumor mass through modern imaging techniques has also resulted in advances in radiation therapy. Conformal radiation therapy delivered as a single dose (also called *radiosurgery*) or in multiple doses, such as with stereotactic radiotherapy or intensity-modulated radiation therapy, is dependent on accurate localization of the tumor mass in relation to its boundaries. These techniques have been capable of delivering higher doses of radiation to the tumor mass with better dose distribution within and immediately surrounding the mass and steeper dose gradients into adjacent structures. **(Brem H et al., 2011)**

The effectiveness of chemotherapy for brain tumors has historically been limited because of resistance of the tumors to available drugs and reduced delivery of the drugs through the blood-brain barrier. Despite these drawbacks, several tumor types can respond well to chemotherapy drugs, including lymphomas, germinomas, anaplastic astrocytomas, oligodendrogliomas, and glioblastomas. **(Brem H et al., 2011)**

The *principles* that guide the neurosurgeon in the treatment of patients harboring a brain tumor are as follows:

1. Acquisition of a representative tumor biopsy for the purpose of histological diagnosis
2. Reduction of the tumor mass to the maximal extent consistent with preservation of neurologic function
3. Appropriate use of adjuvant therapy based on clinical and laboratory observations.

In the following sections the major groups of intracranial tumors which are frequently seen in practice will be discussed focusing on classification, epidemiologic variables (incidence, age distribution), main histopathological characteristics, features on radiological images, clinical presentation, treatment possibilities and patients' prognosis.

Tumor entities

Major tumor entities are neuroepithelial (glial) tumors, meningiomas and metastasis. Primary brain tumors that arise from glial cells (gliomas) are classified according to their appearance under the microscope into four grades (I, II, III, and IV) and the treatment and prognosis depend on the tumor grade. **(Louis DN et al., 2007)**

For clinical considerations grade I or II gliomas are termed low grade gliomas, and grade III or IV gliomas are termed high grade gliomas.

1.1. Low grade gliomas:

Low grade gliomas (LGGs) accounting for about 15% of all brain tumors in adults. The subset of tumors classified as LGGs is a heterogeneous group of tumors with astrocytic, oligodendroglial, ependymal, neuroepithelial tumors gang1iogllomas, gangliocytomas, Juvenile pilocytic astrocytoma, pleomorphic xanthoastrocytomas, dysembryoplastic or mixed cellular histology. In adults, the term LGGs typically refers to the diffuse, infiltrating variety of tumors classified as WHO grade II lesions—specifically, low-grade astrocytomas, oligodendrogliomas, or mixed oligoastrocytomas. **(Bondy ML et al., 2008)**

The majority of patients who are diagnosed with low-grade gliomas usually come to medical attention because of sudden occurrence of seizures. These patients are generally intact at the gross neurologic examination, but they frequently present more subtle symptoms affecting complex neurologic functions (memory, language, character, visuospatial orientation, etc.) that require a specific testing by a neuropsychologist **(Bello L et al., 2010)**

Those patients who present with frank neurologic deficits (e.g., hemiparesis, ataxia, aphasia) are usually candidates for surgery because their symptoms are related to direct mass effect of the tumor on the cortex or on the subcortical white matter tracts. This category of patients carries higher surgical risks in terms of morbidity and mortality than that of neurologically intact patients. **(Bello L et al., 2010)**

MRI is now the preferred study for brain tumors. LGGs are characteristically homogeneously isointense to hypointense on T1-weighted images and hyperintense on T2-weighted images. Contrast enhancement is uncommon, but more often is seen in oligodendrogliomas (25-50%). Calcifications are apparent in 20%. (**Cha S et al., 2005)**

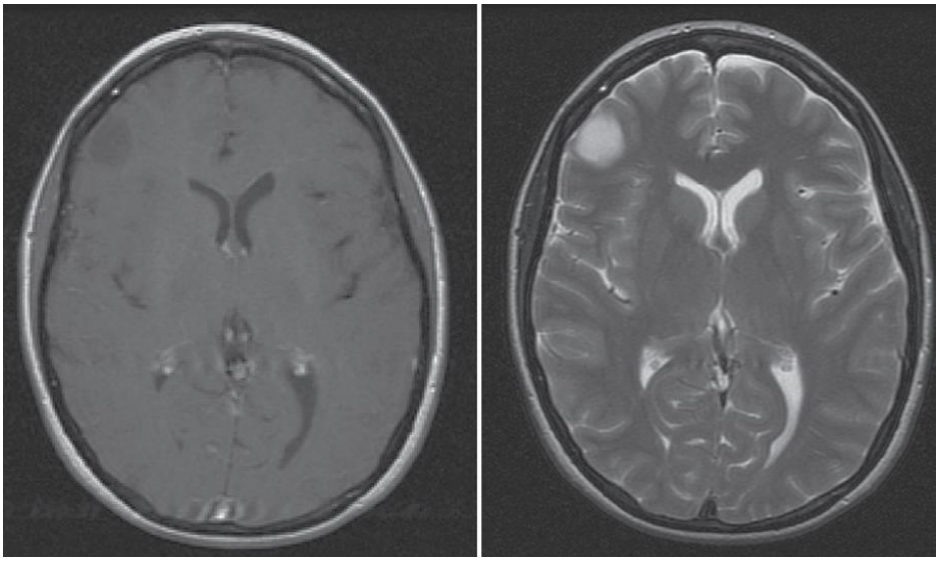

Figure (1): T1-weighted, axial (right) and T2-weighted, axial (left) magnetic resonance imaging (with gadolinium contrast) of a non–contrast enhancing, low-grade astrocytoma in a 41-year-old female patient. **(Cha S et al., 2005)**

The role of surgery for gliomas, low-grade as well as high-grade, has been questioned for a long time, but evidence has now been produced as a collateral result from the phase III trial with fluorescence-guided resection of newly diagnosed glioblastoma ,showing that the extent of resection correlates with survival **(Stummer W et al., 2008)**

1.2. High grade gliomas:

Malignant astrocytomas, which include anaplastic astrocytoma (WHO grade III), glioblastoma multiforme (GBM, WHO grade IV), and gliosarcoma, are the most

common malignant primary central nervous system (CNS) tumors in adults **(Bondy ML et al., 2008)**

Glioblastoma multiforme (GBM) may arise through two distinct pathways of neoplastic progression. Tumors that progress from lower grade (II or III) astrocytic tumors, termed *secondary* or *type 1 GBMs*, typically display both well-differentiated and poorly differentiated foci. Secondary GBMs develop in younger patients (fifth to sixth decade), with time to progression from lower-grade lesions ranging from months to decades. In contrast, primary type 2 GBMs develop in older individuals (sixth to seventh decade), have short clinical histories (less than 3 months), and arise de novo without any evidence of a lower-grade precursor **(Ohgaki H et al., 2007)**

High-grade glioma normally appears as an irregular hypointense lesion on T1-weighted MRI with various degrees of contrast enhancement and edema. The presence of ring-like enhancement surrounding irregularly shaped areas of presumed necrosis suggests glioblastoma. Magnetic resonance spectroscopy may be used to help differentiate tumors from stroke, old trauma, radionecrosis, infection, and multiple sclerosis **(Talos IF et al., 2007)**