26_CONCORSO PUBBLICO, PER TITOLI ED ESAMI, PER LA COPERTURA A TEMPO DETERMINATO, DELLA DURATA DI CINQUE ANNI PER N. 1 POSTO DI RICERCATORE SANITARIO, CAT. D, LIVELLO D SUPER DA ASSEGNARE ALLA SC NEURORADIOLOGIA

PROVA I

1) Descrivere possibili applicazioni che i composti fluorurati possono avere come agenti di "bioimaging" multimodale

2) In Access cosa è una "Query"?

a. uno strumento idoneo all'interrogazione ed alla manipolazione dei dati
b. una tabella di visualizzazione di attributi di un elemento geografico
c. una tabella di visualizzazione di attributi di un elemento grafico

3) Leggere e tradurre

Amyotrophic lateral sclerosis

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Abstract | Amyotrophic lateral sclerosis (ALS), also known as motor neuron disease, is characterized by the degeneration of both upper and lower motor neurons, which leads to muscle weakness and eventual paralysis. Until recently, ALS was classified primarily within the neuromuscular domain, although new imaging and neuropathological data have indicated the involvement of the non-motor neuraxis in disease pathology. In most patients, the mechanisms underlying the development of ALS are poorly understood, although a subset of patients have familial disease and harbour mutations in genes that have various roles in neuronal function. Two possible disease-modifying therapies that can slow disease progression are available for ALS, but patient management is largely mediated by symptomatic therapies, such as the use of muscle relaxants for spasticity and speech therapy for dysarthria.

Amyotrophic lateral sclerosis (ALS) is a heterogeneous neurodegenerative disease that is characterized by the degeneration of both upper motor neurons (that is, neurons that project from the cortex to the brainstem and the spinal cord) and lower motor neurons (that is, neurons that project from the brainstem or spinal cord to muscle), leading to motor and extra-motor symptoms (FIG. 1). The initial presentation of ALS can vary between patients; some present with spinal-onset disease (that is, the onset of muscle weakness of the limbs), but other patients can present with bulbar-onset disease, which is characterized by dysarthria (difficulty with speech) and dysphagia (difficulty swallowing). In most patients, the cause of ALS is unknown, although some individuals have familial disease, which is associated with mutations in genes that have a wide range of functions, including roles in non-motor cells. In familial ALS, some of the implicated genes are incompletely penetrant, and with rare exceptions, genotype does not necessarily predict phenotype[1].

The classification of ALS can vary depending on the criteria used. The traditional definitions of ALS subgroups are based on the extent of involvement of upper and lower motor neurons, although other classification systems include different parameters, such as the site of onset (that is, bulbar-onset or spinal-onset disease), the level of certainty of diagnosis according to the revised El Escorial criteria and heritability (sporadic or familial disease)[1]. To date, none of these classification systems have incorporated the cognitive or behavioural symptoms, and within each classification system, a range of subphenotypes and clinical trajectories can be observed.

This Primer reviews the aspects of ALS that contribute to disease heterogeneity and looks to the future of new therapeutic trials that incorporate recent advances in our understanding. For new therapies, the challenge is to define mechanisms of disease that are amenable to drug targeting and to define patients who are likely to respond to these therapeutic agents.

PROVA NON SORTEGGIATA

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PROVA 2

1) Quali sono i composti fluorurati più utilizzati come sonde per 19F-MRI in studi pre-clinici e clinici e che caratteristiche hanno? Discutere i possibili vantaggi e svantaggi dei composti citati

2) In Excel cosa è una "funzione"?

a. un algoritmo di calcolo precostituito che ci permette di elaborare un calcolo complesso sui dati contenuti nelle celle
b. un comando che ci permette di stampare
c. un comando che ci permette di creare un grafico

3) Leggere e tradurre

Automatic Quality Assessment in Structural Brain Magnetic Resonance Imaging

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MRI has evolved into an important diagnostic technique in medical imaging. However, reliability of the derived diagnosis can be degraded by artifacts, which challenge both radiologists and automatic computer-aided diagnosis. This work proposes a fully-automatic method for measuring image quality of three-dimensional (3D) structural MRI. Quality measures are derived by analyzing the air background of magnitude images and are capable of detecting image degradation from several sources, including bulk motion, residual magnetization from incomplete spoiling, blurring, and ghosting. The method has been validated on 749 3D T_1-weighted 1.5T and 3T head scans acquired at 36 Alzheimer's Disease Neuroimaging Initiative (ADNI) study sites operating with various software and hardware combinations. Results are compared against qualitative grades assigned by the ADNI quality control center (taken as the reference standard). The derived quality indices are independent of the MRI system used and agree with the reference standard quality ratings with high sensitivity and specificity (>85%). The proposed procedures for quality assessment could be of great value for both research and routine clinical imaging. It could greatly improve workflow through its ability to rule out the need for a repeat scan while the patient is still in the magnet bore. Magn Reson Med 62:365–372, 2009. © 2009 Wiley-Liss, Inc.

Key words: magnetic resonance imaging; automatic quality assessment; image quality; artifact detection

MRI quality can be affected by a wide variety of artifacts. They can be broadly classified into two categories: those that are machine-specific and those that are related to the patient. Some of the machine-specific artifacts are not visually obvious, yet can potentially degrade images. This can cause inaccurate diagnosis or dramatically affect the efficiency of automated quantitative image analysis algorithms that are increasingly used in clinical practice and research. These techniques offer promise for improved clinical workflow, including clinical research studies, such as longitudinal monitoring of the evolution and the treatment of degenerative and inflammatory diseases (e.g., dementias, multiple sclerosis, Parkinson disease). In this context, recognizing artifacts becomes fundamental.

Recently, various investigators have proposed standardized quality assurance (QA) protocols and methodologies to test machine-related artifacts (1–3). These protocols are often based on specially designed phantoms to analyze image quality-related system parameters, such as: gradient linearity (4), geometric accuracy, high-contrast resolution, slice thickness/position accuracy, image intensity uniformity (5–7), percent signal ghosting, and low-contrast object detectability (8,9). These QA tests are of high interest to monitor scanner performance and retrospectively correct human images for drifts or discontinuities in gradient calibration (10,11).

Although QA tests are performed as standard procedure during tune-up and service of MR systems and are used in several clinical studies, very little has been reported about detecting and analyzing patient-related artifacts. The importance of such quality control might have been downplayed so far under the assumption that an experienced radiologist is able to "read-through" artifacts. Nevertheless, this issue has been investigated (12,13) and some

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PROVA 3

1) I composti fluorurati usati come traccianti per 19F-MRI sono spesso insolubili in acqua. Che tipo di formulazioni vengono utilizzate per la loro applicazione come:
   a) marcatori di cellule terapeutiche; b) marcatori di vettori di farmaci in vivo?

2) Il Plug & Play è una tecnologia che permette di:

   a. utilizzare una periferica semplicemente collegandola al computer
   b. attivare il risparmio energetico del computer
   c. aumentare il numero di dispositivi collegabili al computer

3) Leggere e tradurre

ABSTRACT

Background: Imaging studies have revealed cortical thinning and subcortical atrophy occurring in Parkinson's disease (PD); however, the topographical distribution and clinical associations related to advancing stages of PD remains unclear.

Objective: We aimed to investigate the topographical distribution of cortical and subcortical morphometric changes, and their clinical associations, related to increasing disease severity.

Methods: In this cross-sectional imaging study, T1-weighted structural magnetic resonance imaging data for 80 non-demented PD patients and 30 age-matched healthy controls were analysed using FreeSurfer software suite to derive morphometric changes using whole-brain vertex-wise analysis, and surface-based (cortical) and volume-based (subcortical) parcellation maps. PD patients were divided into three groups of mild ($n = 27$), moderate ($n = 27$), and severe ($n = 26$) PD based disease duration and Hoehn and Yahr and Unified Parkinson's Disease Rating Scale Part-III motor severity scores.

Results: Whole-brain vertex-wise analysis revealed cortical thinning in the orbitofrontal cortex in early PD ($P = .011$), and in the superior frontal ($P = .002$), caudal middle frontal gyrus ($P = .001$) and inferior parietal cortex ($P = .006$) in moderate PD. Severe PD patients showed additional cortical thinning in temporal and occipital cortices ($P < .005$). Subcortical volume loss was detected in the thalamus ($P = .012$) and hippocampus ($P = .032$) in moderate PD, which extended to the caudate ($P = .012$), putamen ($P = .042$) and amygdala ($P = .008$) in severe PD. Increasing disease duration and motor severity scores, correlated with cortical thinning in frontal, temporal, parietal and occipital cortices, and subcortical volumetric loss in the thalamus, caudate, putamen, amygdala and hippocampus. Lower global cognitive status, measured with MMSE, correlated with cortical thinning in temporal, parietal, frontal and cingulate cortices, and with volumetric loss in the hippocampus ($r = 0.31$; $P = .009$); suggesting subclinical pathogenic changes occur prior to the onset of cognitive impairment.

Conclusion: In conclusion, in more severe disease stages PD patients exhibit progressive cortical thinning and subcortical volume loss which could have relevance to the development of cognitive impairment.

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