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1. Manganese exposure and neurotoxic effects in children.

Authors: Bjørklund, Geir; Chartrand, Max Stanley; Aaseth, Jan
Source: Environmental research; May 2017; vol. 155; p. 380-384
Publication Date: May 2017
Publication Type(s): Journal Article Review
PubMedID: 28282629
Database: Medline

Abstract: Manganese (Mn) is the fifth most abundant metal on earth. Although it is a well understood essential trace element, in excess, Mn is neurotoxic. Initial toxic symptoms associated with Mn are of psychiatric nature and are clinically defined as locura manganica. Neurological signs of Mn toxicity include dystonia, progressive bradykinesia, and disturbance of gait, slurring, and stuttering of speech with diminished volume. Studies indicate that children who ingested Mn in the drinking water (WMn) at or above a level of 0.241mg/L for a minimum of three years performed more poorly in school as measured by mastery of language, mathematics, and in their overall grade average. The Mn-exposed children also performed more poorly on a battery of neurobehavioral tests. It was also found a significant association between higher WMn and lower cognitive performance, verbal function, and full-scale intelligence quotient (IQ) scores. Young children appear to make up a vulnerable group in exposed populations. Toxicity of WMn is a problem particularly in areas of industrial waste or where Mn is leaching from the soil into public drinking water. Practical and cost-effective approaches are available to remove Mn from drinking water. It is crucial to protect developing brains against Mn toxicity.

2. Treatments for chronic inflammatory demyelinating polyradiculoneuropathy (CIDP): an overview of systematic reviews.

Authors: Oaklander, Anne Louise; Lunn, Michael Pt; Hughes, Richard Ac; van Schaik, Ivo N; Frost, Chris; Chalk, Colin H
Source: The Cochrane database of systematic reviews; Jan 2017; vol. 1; p. CD010369
Publication Date: Jan 2017
Publication Type(s): Research Support, Non-u.s. Gov't Journal Article Review
PubMedID: 28084646
Database: Medline
BACKGROUND Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a chronic progressive or relapsing and remitting disease that usually causes weakness and sensory loss. The symptoms are due to autoimmune inflammation of peripheral nerves. CIDP affects about 2 to 3 per 100,000 of the population. More than half of affected people cannot walk unaided when symptoms are at their worst. CIDP usually responds to treatments that reduce inflammation, but there is disagreement about which treatment is most effective.

OBJECTIVES To summarise the evidence from Cochrane systematic reviews (CSRs) and non-Cochrane systematic reviews of any treatment for CIDP and to compare the effects of treatments.

METHODS We considered all systematic reviews of randomised controlled trials (RCTs) of any treatment for any form of CIDP. We reported their primary outcomes, giving priority to change in disability after 12 months. Two overview authors independently identified published systematic reviews for inclusion and collected data. We reported the quality of evidence using GRADE criteria. Two other review authors independently checked review selection, data extraction and quality assessments.

On 31 October 2016, we searched the Cochrane Database of Systematic Reviews, the Database of Abstracts of Reviews of Effects (in the Cochrane Library), MEDLINE, Embase, and CINAHL Plus for systematic reviews of CIDP. We supplemented the RCTs in the existing CSRs by searching on the same date for RCTs of any treatment of CIDP (including treatment of fatigue or pain in CIDP), in the Cochrane Neuromuscular Specialised Register, CENTRAL, MEDLINE, Embase, and CINAHL Plus.

RESULTS Five CSRs met our inclusion criteria. We identified 23 randomised trials, of which 15 had been included in these CSRs. We were unable to compare treatments as originally planned, because outcomes and outcome intervals differed. Corticosteroids is uncertain whether daily oral prednisolone improves impairment compared to no treatment because the quality of the evidence was very low (1 trial, 28 participants). According to moderate-quality evidence (1 trial, 41 participants), six months’ treatment with high-dose monthly oral dexamethasone did not improve disability more than daily oral prednisolone. Observational studies tell us that prolonged use of corticosteroids sometimes causes serious side-effects. Plasma exchange according to moderate-quality evidence (2 trials, 59 participants), twice-weekly plasma exchange produced more short-term improvement in disability than sham exchange. In the largest observational study, 3.9% of plasma exchange procedures had complications. Intravenous immunoglobulin according to high-quality evidence (5 trials, 269 participants), intravenous immunoglobulin (IVIg) produced more short-term improvement than placebo.

Adverse events were more common with IVIg than placebo (high-quality evidence), but serious adverse events were not (moderate-quality evidence, 3 trials, 315 participants). One trial with 19 participants provided moderate-quality evidence of little or no difference in short-term improvement of impairment with plasma exchange in comparison to IVIg. There was little or no difference in short-term improvement of disability with IVIg in comparison to oral prednisolone (moderate-quality evidence; 1 trial, 29 participants) or intravenous methylprednisolone (high-quality evidence; 1 trial, 45 participants). One unpublished randomised open trial with 35 participants found little or no difference in disability after three months of IVIg compared to oral prednisolone; this trial has not yet been included in a CSR. We know from observational studies that serious adverse events related to IVIg do occur. Other immunomodulatory treatments is uncertain whether the addition of azathioprine (2 mg/kg) to prednisone improved impairment in comparison to prednisone alone, as the quality of the evidence is very low (1 trial, 27 participants). Observational studies show that adverse effects of treatment in 10% of people. According to low-quality evidence (1 trial, 100 participants), compared to placebo, methotrexate 15 mg/kg did not allow more participants to reduce corticosteroid or IVIg doses by 20%. Serious adverse events were no more common with methotrexate than with placebo, but observational studies show that methotrexate can cause teratogenicity, abnormal liver function, and pulmonary fibrosis. According to moderate-quality evidence (2 trials, 77 participants), interferon beta-1a (IFN beta-1a) in comparison to placebo, did not allow more people to withdraw from IVIg. According to moderate-quality evidence, serious adverse events were no more common with IFN beta-1a than with placebo. We know of no other completed trials of immunosuppressant or immunomodulatory agents for CIDP. Other treatments We identified no trials of treatments for fatigue or pain in CIDP. Adverse effects Not all trials routinely collected adverse event data; when they did, the quality of evidence was variable. Adverse effects in the short, medium, and long term occur with all interventions. We are not able to make reliable comparisons of adverse events between the interventions included in CSRs.

AUTHORS’ CONCLUSIONS We cannot be certain based on available evidence whether daily oral prednisone improves impairment compared to no treatment. However, corticosteroids are commonly used, based on widespread availability, low cost, very low-quality evidence from observational studies, and clinical experience. The weakness of the evidence does not necessarily mean that corticosteroids are ineffective. High-dose monthly oral dexamethasone for six months is probably no more or less effective than daily oral prednisolone. Plasma exchange produces short-term improvement in impairment as determined by neurological examination, and probably produces short-term improvement in disability. IVIg produces more short-term improvement in disability than placebo and more adverse events, although serious side effects are probably no more common than with placebo. There is no clear difference in short-term improvement in impairment with IVIg when compared with intravenous methylprednisolone and probably no improvement when compared with either oral prednisolone or plasma exchange. According to observational studies, adverse events related to difficult venous access, use of citrate, and haemodynamic changes occur in 3% to 17% of plasma exchange procedures. It is uncertain whether azathioprine is of benefit as the quality of evidence is very low. Methotrexate may not be of benefit and IFN beta-1a is probably not of benefit. We need further research to identify predictors of response to different treatments and to compare their long-term benefits, safety and...
cost-effectiveness. There is a need for more randomised trials of immunosuppressive and immunomodulatory agents, routes of administration, and treatments for symptoms of CIDP.


Authors: Hillary, Frank G; Grafman, Jordan H
Source: Trends in cognitive sciences; May 2017; vol. 21 (no. 5); p. 385-401
Publication Date: May 2017
Publication Type(s): Journal Article Review
PubMedID: 28372878
Database: Medline
Abstract: A common finding in human functional brain-imaging studies is that damage to neural systems paradoxically results in enhanced functional connectivity between network regions, a phenomenon commonly referred to as 'hyperconnectivity'. Here, we describe the various ways that hyperconnectivity operates to benefit a neural network following injury while simultaneously negotiating the trade-off between metabolic cost and communication efficiency. Hyperconnectivity may be optimally expressed by increasing connections through the most central and metabolically efficient regions (i.e., hubs). While adaptive in the short term, we propose that chronic hyperconnectivity may leave network hubs vulnerable to secondary pathological processes over the life span due to chronically elevated metabolic stress. We conclude by offering novel, testable hypotheses for advancing our understanding of the role of hyperconnectivity in systems-level brain plasticity in neurological disorders.


Authors: Pichler, M; Hocker, S
Source: Handbook of clinical neurology; 2017; vol. 140 ; p. 131-151
Publication Date: 2017
Publication Type(s): Journal Article Review
PubMedID: 28187796
Database: Medline
Abstract: Status epilepticus is a neurologic and medical emergency manifested by prolonged seizure activity or multiple seizures without return to baseline. It is associated with substantial medical cost, morbidity, and mortality. There is a spectrum of severity dependent on the type of seizure, underlying pathology, comorbidities, and appropriate and timely medical management. This chapter discusses the evolving definitions of status epilepticus and multiple patient and clinical factors which influence outcome. The pathophysiology of status epilepticus is reviewed to provide a better understanding of the mechanisms which contribute to status epilepticus, as well as the potential long-term effects. The clinical presentations of different types of status epilepticus in adults are discussed, with emphasis on the hospital course and management of the most dangerous type, generalized convulsive status epilepticus. Strategies for the evaluation and management of status epilepticus are provided based on available evidence from clinical trials and recommendations from the Neurocritical Care Society and the European Federation of Neurological Societies.

5. The Pharmacologic and Clinical Effects of Illicit Synthetic Cannabinoids.

Authors: White, C Michael
Source: Journal of clinical pharmacology; Mar 2017; vol. 57 (no. 3); p. 297-304
Publication Date: Mar 2017
Publication Type(s): Journal Article Review
PubMedID: 27610597
Database: Medline
Abstract: This article presents information on illicitly used synthetic cannabinoids. Synthetic cannabinoids are structurally heterogeneous and commonly used drugs of abuse that act as full agonists of the cannabinoid type-1 receptor but have a variety of additional pharmacologic effects. There are numerous cases of patient harm and death in the United States, Europe, and Australia with many psychological, neurological, cardiovascular, pulmonary, and renal adverse events. Although most users prefer using cannabis, there are convenience, legal, and cost reasons driving the utilization of synthetic cannabinoids. Clinicians should be aware of pharmacologic and clinical similarities and differences between synthetic cannabinoid and cannabis use, the limited ability to detect synthetic cannabinoids in the urine or serum, and guidance to treat adverse events.


Authors: Liao, Xuhong; Vasilakos, Athanasios V; He, Yong
Source: Neuroscience and biobehavioral reviews; Jun 2017; vol. 77 ; p. 286-300
Publication Date: Jun 2017
Publication Type(s): Journal Article Review
PubMedID: 28389343
Database: Medline
9. Can adjunctive therapies augment the efficacy of endovascular thrombolysis? A potential role for activated protein C.

Abstract
Modelling the human brain as a complex network has provided a powerful mathematical framework to characterize the structural and functional architectures of the brain. In the past decade, the combination of non-invasive neuroimaging techniques and graph theoretical approaches enable us to map human structural and functional connectivity patterns (i.e., connectome) at the macroscopic level. One of the most influential findings is that human brain networks exhibit prominent small-world organization. Such a network architecture in the human brain facilitates efficient information segregation and integration at low wiring and energy costs, which presumably results from natural selection under the pressure of a cost-efficiency balance. Moreover, the small-world organization undergoes continuous changes during normal development and aging and exhibits dramatic alterations in neurological and psychiatric disorders. In this review, we survey recent advances regarding the small-world architecture in human brain networks and highlight the potential implications and applications in multidisciplinary fields, including cognitive neuroscience, medicine and engineering. Finally, we highlight several challenging issues and areas for future research in this rapidly growing field.
In the management of acute ischemic stroke, vessel recanalization correlates with functional status, mortality, cost, and other outcome measures. Thrombolysis with intravenous tissue plasminogen activator has many limitations that restrict its applicability, but recent advances in the development of mechanical thrombectomy devices as well as improved systems of stroke care have resulted in greater likelihood of vessel revascularization. Nonetheless, there remains substantial discrepancy between rates of recanalization and rates of favorable outcome. The poor neurological recovery among some stroke patients despite successful recanalization confirms the need for adjuvant pharmacological therapy for neuroprotection and/or neurorestoration. Prior clinical trials of such drugs may have failed due to the inability of the agent to access the ischemic tissue beyond the occluded artery. A protocol that couples revascularization with concurrent delivery of a neuroprotective drug offers the potential to enhance the benefit of thrombolysis. Analogs of activated protein C (APC) exert pleiotropic anti-inflammatory, anti-apoptotic, antithrombotic, cytoprotective, and neuroregenerative effects in ischemic stroke and thus appear to be promising candidates for this novel approach. A multicenter, prospective, double-blinded, dose-escalation Phase 2 randomized clinical trial has enrolled 110 patients to assess the safety, pharmacokinetics, and efficacy of human recombinant 3K3A-APC following endovascular thrombolysis.

**10. Clinical evoked potentials in neurology: a review of techniques and indications.**

**Authors** Lascano, Agustina M; Lalive, Patrice H; Hardmeier, Martin; Fuhr, Peter; Seeck, Margitta

**Source** Journal of neurology, neurosurgery, and psychiatry; Aug 2017; vol. 88 (no. 8); p. 688-696

**Publication Date** Aug 2017

**Publication Type(s)** Journal Article Review

**PubMedID** 28235778

**Database** Medline

**Abstract**

Evoked potentials (EPs) are a powerful and cost-effective tool for evaluating the integrity and function of the central nervous system. Although imaging techniques, such as MRI, have recently become increasingly important in the diagnosis of neurological diseases, over the past 30 years, many neurologists have continued to employ EPs in specific clinical applications. This review presents an overview of the recent evolution of 'classical' clinical applications of EPs in terms of early diagnosis and disease monitoring and is an extension of a previous review published in this journal in 2005 by Walsh and collaborators. We also provide an update on emerging EPs based on gustatory, olfactory and pain stimulation that may be used as clinically relevant markers of neurodegenerative disorders such as Parkinson’s disease, Alzheimer’s disease and cortical or peripheral impaired pain perception. EPs based on multichannel electroencephalography recordings, known as high-density EPs, help to better differentiate between healthy subjects and patients and, moreover, they provide valuable spatial information regarding the site of the lesion. EPs are reliable disease-progression biomarkers of several neurological diseases, such as multiple sclerosis and other demyelinating disorders. Overall, EPs are excellent neurophysiological tools that will expand standard clinical practice in modern neurology.

**11. Proposed recommendations for diagnosing and managing individuals with glutaric aciduria type I: second revision.**

**Authors** Boy, Nikolas; Mühlhausen, Chris; Maier, Esther M; Heringer, Jana; Assmann, Birgit; Burgard, Peter; Dixon, Marjorie; Fleissner, Sandra; Greenberg, Cheryl R; Harting, Inga; Hoffmann, Georg F; Karali, Daniela; Koeller, David M; Krawinkel, Michael B; Okun, Jürgen G; Opladen, Thomas; Posset, Roland; Sahm, Katja; Zschocke, Johannes; Kölker, Stefan; Additional individual contributors

**Source** Journal of inherited metabolic disease; Jan 2017; vol. 40 (no. 1); p. 75-101

**Publication Date** Jan 2017

**Publication Type(s)** Journal Article Review

**PubMedID** 27853989

**Database** Medline
Glutaric aciduria type I (GA-I; synonym, glutaric acidemia type I) is a rare inherited metabolic disease caused by deficiency of glutaryl-CoA dehydrogenase located in the catabolic pathways of L-lysine, L-hydroxylysine, and L-tryptophan. The enzymatic defect results in elevated concentrations of glutaric acid, 3-hydroxyglutaric acid, glutaconic acid, and glutaryl carnitine in body tissues, which can be reliably detected by gas chromatography/mass spectrometry (organic acids) and tandem mass spectrometry (acylcarnitines). Most untreated individuals with GA-I experience acute encephalopathic crises during the first 6 years of life that are triggered by infectious diseases, febrile reaction to vaccinations, and surgery. These crises result in striatal injury and consequent dystonic movement disorder; thus, significant mortality and morbidity results. In some patients, neurologic disease may also develop without clinically apparent crises at any age. Neonatal screening for GA-I is being used in a growing number of countries worldwide and is cost effective. Metabolic treatment, consisting of low lysine diet, carnitine supplementation, and intensified emergency treatment during catabolism, is effective treatment and improves neurologic outcome in those individuals diagnosed early; treatment after symptom onset, however, is less effective. Dietary treatment is relaxed after age 6 years and should be supervised by specialized metabolic centers. The major aim of this second revision of proposed recommendations is to re-evaluate the previous recommendations (Kölker et al. J Inherit Metab Dis 30:5-22, 2007b; J Inherit Metab Dis 34:677-694, 2011) and add new research findings, relevant clinical aspects, and the perspective of affected individuals.

12. Riboflavin Has Neuroprotective Potential: Focus on Parkinson’s Disease and Migraine.

Authors Marashly, Eyad T; Bohlega, Saeed A
Source Frontiers in neurology; 2017; vol. 8; p. 333
Publication Date 2017
Publication Type(s) Journal Article Review
PubMedID 28775706
Database Medline

Abstract With the huge negative impact of neurological disorders on patient’s life and society resources, the discovery of neuroprotective agents is critical and cost-effective. Neuroprotective agents can prevent and/or modify the course of neurological disorders. Despite being underestimated, riboflavin offers neuroprotective mechanisms. Significant pathogenesis-related mechanisms are shared by, but not restricted to, Parkinson’s disease (PD) and migraine headache. Those pathogenesis-related mechanisms can be tackled through riboflavin proposed neuroprotective mechanisms. In fact, it has been found that riboflavin ameliorates oxidative stress, mitochondrial dysfunction, neuroinflammation, and glutamate excitotoxicity; all of which take part in the pathogenesis of PD, migraine headache, and other neurological disorders. In addition, riboflavin-dependent enzymes have essential roles in pyridoxine activation, tryptophan-kynurenine pathway, and homocysteine metabolism. Indeed, pyridoxal phosphate, the active form of pyridoxine, has been found to have independent neuroprotective potential. Also, the produced kynurenines influence glutamate receptors and its consequent excitotoxicity. In addition, methylenetetrahydrofolate reductase requires riboflavin to ensure normal folate cycle influencing the methylation cycle and consequently homocysteine levels which have its own negative neurovascular consequences if accumulated. In conclusion, riboflavin is a potential neuroprotective agent affecting a wide range of neurological disorders exemplified by PD, a disorder of neurodegeneration, and migraine headache, a disorder of pain. In this article, we will emphasize the role of riboflavin in neuroprotection elaborating on its proposed neuroprotective mechanisms in opposite to the pathogenesis-related mechanisms involved in two common neurological disorders, PD and migraine headache, as well as, we encourage the clinical evaluation of riboflavin in PD and migraine headache patients in the future.


Authors Sarfo, Fred S; Adamu, Sheila; Awuah, Dominic; Ovbiagele, Bruce
Source Journal of the neurological sciences; Sep 2017; vol. 380; p. 196-199
Publication Date Sep 2017
Publication Type(s) Journal Article Review
PubMedID 28870567
Database Medline
BACKGROUND The rapid advancement in telecommunications on the African continent has opened up avenues for improving medical care to underserved populations. Although the greatest burden of neurological disorders is borne by Low-and-Middle Income Countries (LMICs) including sub-Saharan Africa (SSA), there is a profound paucity of neurologists to serve the population. Telemedicine presents a promising avenue for effective mobilization and utilization of the few neurologists in Africa.

OBJECTIVE To systematically review the published literature on the use of telemedicine for improved care and outcomes for patients with neurological disorders in SSA.


RESULTS This search yielded 6 abstracts. By consensus between two investigators, 1 publication met the criteria for inclusion and further review. The one study identified utilized telemedicine for the purpose of improving education/knowledge of 16 doctors and 17 allied health professionals in Parkinson's disease (PD) in Cameroon. The study noted feasibility and satisfaction of participants with telemedicine as well as improved knowledge base of participants after the educational course but noted access to healthcare by patients did not change. No studies have evaluated the use of telemedicine for care of patients with neurological disorders.

CONCLUSION The indication is that teleneurology may be feasible in SSA and studies are needed to assess feasibility, acceptability, efficacy, cost-effectiveness of this promising discipline of neurology in these resource-limited settings. We propose the setting up of trans-continental, inter-regional, intra-regional, and national networks of neurologists to utilize teleneurology platforms to improve the reach of neurology care in SSA.


Authors Georgieff, Michael K
Source The American journal of clinical nutrition; Oct 2017
Publication Date Oct 2017
Publication Type(s) Journal Article Review
PubMedID 29070550
Database Medline
Abstract Iron deficiency (ID) before the age of 3 y can lead to long-term neurological deficits despite prompt diagnosis of ID anemia (IDA) by screening of hemoglobin concentrations followed by iron treatment. Furthermore, pre- or nonanemic ID alters neurobehavioral function and is 3 times more common than IDA in toddlers. Given the global prevalence of ID and the enormous societal cost of developmental disabilities across the life span, better methods are needed to detect the risk of inadequate concentrations of iron for brain development (i.e., brain tissue ID) before dysfunction occurs and to monitor its amelioration after diagnosis and treatment. The current screening and treatment strategy for IDA fails to achieve this goal for 3 reasons. First, anemia is the final state in iron depletion. Thus, the developing brain is already iron deficient when IDA is diagnosed owing to the prioritization of available iron to red blood cells over all other tissues during negative iron balance in development. Second, brain ID, independently of IDA, is responsible for long-term neurological deficits. Thus, starting iron treatment after the onset of IDA is less effective than prevention. Multiple studies in humans and animal models show that post hoc treatment strategies do not reliably prevent ID-induced neurological deficits. Third, most currently used indexes of ID are population statistical cutoffs for either hematologic or iron status but are not bioindicators of brain ID and brain dysfunction in children. Furthermore, their relation to brain iron status is not known. To protect the developing brain, there is a need to generate serum measures that index brain dysfunction in the preanemic stage of ID, assess the ability of standard iron indicators to detect ID-induced brain dysfunction, and evaluate the efficacy of early iron treatment in preventing ID-induced brain dysfunction.

15. Prevention of epilepsy: Should we be avoiding clinical trials?

Authors Klein, Pavel; Tyrlikova, Ivana
Source Epilepsy & behavior : E&B; Jul 2017; vol. 72 ; p. 188-194
Publication Date Jul 2017
Publication Type(s) Journal Article Review
PubMedID 28647441
Database Medline
Abstract
Epilepsy prevention is one of the great unmet needs in epilepsy. Approximately 15% of all epilepsy is caused by an acute acquired CNS insult such as traumatic brain injury (TBI), stroke or encephalitis. There is a latent period between the insult and epilepsy onset that presents an opportunity to intervene with preventive treatment that is unique in neurology. Yet no phase 3 epilepsy prevention studies, and only 2 phase 2 studies have been initiated in the last 16 years. Current prevailing opinion is that the research community is not ready for clinical preventive epilepsy studies, and that animal models should first be refined and biomarkers of epileptogenesis and of epilepsy discovered before clinical studies are embarked upon. We review data to suggest that there is basis to do epilepsy prevention studies now with the current knowledge and available drugs, and that those studies are feasible with currently available tools. We suggest that a different approach is needed from the past in order to maximize chances of success, minimize the cost, and set up platform for future preventive treatment development. That approach should include close coordination of preclinical and clinical development programs in a combined PTE prevention strategy, consideration of polytherapy, and simultaneous, combined clinical development of preventive treatment and of biomarker discovery. We argue that the currently favored approach of eschewing clinical studies until biomarkers are available will delay the discovery of epilepsy prevention treatment by at least 10 years and significantly increase the cost of such discovery.

Authors
Barone, Daniel A; Chokroverty, Sudansu
Source
Sleep medicine clinics; Mar 2017; vol. 12 (no. 1); p. 73-85
Publication Date
Mar 2017
Publication Type(s)
Journal Article Review
PubMedID
28159099
Database
Medline
Abstract
Sleep disorders and neurologic illness are common and burdensome in their own right; when combined, they can have tremendous negative impact at an individual level as well as societally. The socioeconomic burden of sleep disorders and neurologic illness can be identified, but the real cost of these conditions lies far beyond the financial realm. There is an urgent need for comprehensive care and support systems to help with the burden of disease. Further research in improving patient outcomes in those who suffer with these conditions will help patients and their families, and society in general.

17. An update on anesthetics and impact on the brain.
Authors
Fodale, Vincenzo; Tripodi, Vincenzo F; Penna, Olivia; Famà, Fausto; Squadrito, Francesco; Mondello, Epifanio; David, Antonio
Source
Expert opinion on drug safety; Sep 2017; vol. 16 (no. 9); p. 997-1008
Publication Date
Sep 2017
Publication Type(s)
Journal Article Review
PubMedID
28697315
Database
Medline
Abstract
INTRODUCTION While anesthetics are indispensable clinical tools and generally considered safe and effective, a growing concern over the potential neurotoxicity of anesthesia or specific anesthetic agents has called into question the safety of general anesthetics, especially when administered at extremes of age. Areas covered: This article reviews and updates research findings on the safety of anesthesia and anesthetics in terms of long-term neurotoxicity, with particular focus on postoperative cognitive dysfunctions, Alzheimer’s disease and dementias, developing brain, post-operative depression and autism spectrum disorder. Expert opinion: Exposure to general anesthetics is potentially harmful to the human brain, and the consequent long-term cognitive deficits should be classified as an iatrogenic pathology, and considered a public health problem. The fact that in laboratory and clinical research only certain anesthetic agents and techniques, but not others, appear to be involved, raises the problem on what is the safest and the least safe anesthetic to maximize anesthesia efficiency, avoid occurrence of adverse events, and ensure patient safety. New trends in research are moving toward the theory that neuroinflammation could be the hallmark of, or could have a pivotal role in, several neurological disorders.

Authors
Konakondla, Sanjay; Fong, Reginald; Schirmer, Clemens M
Source
Advances in medical education and practice; 2017; vol. 8 ; p. 465-473
Publication Date
2017
Publication Type(s)
Journal Article Review
PubMedID
28765716
Database
Medline
Available at Advances in Medical Education and Practice from Europe PubMed Central - Open Access
The current simulation technology used for neurosurgical training leaves much to be desired. Significant efforts are thoroughly exhausted in hopes of developing simulations that translate to give learners the “real-life” feel. Though a respectable goal, this may not be necessary as the application for simulation in neurosurgical training may be most useful in early learners. The ultimate uniformly agreeable endpoint of improved outcome and patient safety drives these investments. We explore the development, availability, educational taskforces, cost burdens and the simulation advancements in neurosurgical training. The technologies can be directed at achieving early resident milestones placed by the Accreditation Council for Graduate Medical Education. We discuss various aspects of neurosurgery disciplines with specific technologic advances of simulation software. An overview of the scholarly landscape of the recent publications in the realm of medical simulation and virtual reality pertaining to neurologic surgery is provided. We analyze concurrent concept overlap between PubMed headings and provide a graphical overview of the associations between these terms.

19. Recent advances in understanding the roles of whole genome duplications in evolution.

Authors: MacKintosh, Carol; Ferrier, David E K
Source: F1000Research; 2017; vol. 6; p. 1623
Publication Date: 2017
Publication Type(s): Journal Article Review
PubMedID: 28928963
Database: Medline
Available at: F1000Research from Europe PubMed Central - Open Access

Abstract: Ancient whole-genome duplications (WGDs)- paleopolyploidy events-are key to solving Darwin’s ‘abominable mystery’ of how flowering plants evolved and radiated into a rich variety of species. The vertebrates also emerged from their invertebrate ancestors via two WGDs, and genomes of diverse gymnosperm trees, unicellular eukaryotes, invertebrates, fishes, amphibians and even a rodent carry evidence of lineage-specific WGDs. Modern polyploidy is common in eukaryotes, and it can be induced, enabling mechanisms and short-term cost-benefit assessments of polyploidy to be studied experimentally. However, the ancient WGDs can be reconstructed only by comparative genomics: these studies are difficult because the DNA duplicates have been through tens or hundreds of millions of years of gene losses, mutations, and chromosomal rearrangements that culminate in resolution of the polyploid genomes back into diploid ones (rediploidisation). Intriguing asymmetries in patterns of post-WGD gene loss and retention between duplicated sets of chromosomes have been discovered recently, and elaborations of signal transduction systems are lasting legacies from several WGDs. The data imply that simpler signalling pathways in the pre-WGD ancestors were converted via WGDs into multi-stranded parallelised networks. Genetic and biochemical studies in plants, yeasts and vertebrates suggest a paradigm in which different combinations of sister paralogues in the post-WGD regulatory networks are co-regulated under different conditions. In principle, such networks can respond to a wide array of environmental, sensory and hormonal stimuli and integrate them to generate phenotypic variety in cell types and behaviours. Patterns are also being discerned in how the post-WGD signalling networks are reconfigured in human cancers and neurological conditions. It is fascinating to unpick how ancient genomic events impact on complexity, variety and disease in modern life.

20. The Medical and Economic Burden of Narcolepsy: Implications for Managed Care.

Authors: Thorpy, Michael J; Hiller, George
Source: American health & drug benefits; Jul 2017; vol. 10 (no. 5); p. 233-241
Publication Date: Jul 2017
Publication Type(s): Journal Article Review
PubMedID: 28975007
Database: Medline
BACKGROUND The neurologic disorder narcolepsy results from dysregulation of the sleep-wake cycle and is primarily characterized by chronic, severely excessive daytime sleepiness and cataplexy, an emotionally induced muscle weakness. The prevalence of narcolepsy is approximately 0.05%, and onset generally occurs during the first 2 decades of life. Narcolepsy is believed to be an autoimmune disorder with destruction of hypocretin-producing neurons in the lateral hypothalamus.

OBJECTIVES To provide an enhanced understanding of narcolepsy and establish the need for early diagnosis and rapid initiation of effective treatment for patients with narcolepsy.

DISCUSSION Narcolepsy reduces daily functioning and is associated with a substantial medical and economic burden, with many patients being on full disability. The annual direct medical costs are approximately 2-fold higher in patients with narcolepsy than in matched controls without this condition ($11,702 vs $5261, respectively; P < .0001). Further contributing to the overall burden is a lack of recognition of the signs and symptoms of narcolepsy and an absence of easily measurable biomarkers, resulting in a diagnostic delay that often exceeds 10 years and may be associated with misdiagnosis and inappropriate resource utilization. Because narcolepsy generally has an onset in childhood or in adolescence, is often misdiagnosed, has no known cure, and requires lifelong treatment, it is an important disease from a managed care perspective. Clinical features, as well as objective testing, should be used to ensure the timely diagnosis and treatment of patients with narcolepsy.

CONCLUSION Policies for the diagnosis and treatment of narcolepsy should be based on the current treatment guidelines, but they should also encourage shared decisions between clinicians and patients to allow for individualized diagnostic and treatment choices, as suggested in best practice recommendations.

21. The Interplay of Axonal Energy Homeostasis and Mitochondrial Trafficking and Anchoring.
Authors Sheng, Zu-Hang
Source Trends in cell biology; Jun 2017; vol. 27 (no. 6); p. 403-416
Publication Date Jun 2017
Publication Type(s) Journal Article Review
PubMedID 28228333
Database Medline
Abstract Mitochondria are key cellular power plants essential for neuronal growth, survival, function, and regeneration after injury. Given their unique morphological features, neurons face exceptional challenges in maintaining energy homeostasis at distal synapses and growth cones where energy is in high demand. Efficient regulation of mitochondrial trafficking and anchoring is critical for neurons to meet altered energy requirements. Mitochondrial dysfunction and impaired transport have been implicated in several major neurological disorders. Thus, research into energy-mediated regulation of mitochondrial recruitment and redistribution is an important emerging frontier. In this review, I discuss new insights into the mechanisms regulating mitochondrial trafficking and anchoring, and provide an updated overview of how mitochondrial motility maintains energy homeostasis in axons, thus contributing to neuronal growth, regeneration, and synaptic function.

Authors Yao, Xiyang; Ma, Junwei; Li, Haiying; Shen, Haitao; Lu, Xiaojun; Chen, Gang
Source The Journal of international medical research; Feb 2017; vol. 45 (no. 1); p. 11-21
Publication Date Feb 2017
Publication Type(s) Meta-analysis Journal Article Review
PubMedID 28222628
Database Medline
Abstract Background We evaluated the safety and efficiency of flow diverters (FDs) in treating small intracranial aneurysms (IAs). Materials and Methods We reviewed the literature published in PubMed and EMBASE. R for Project software was used to calculate the complete aneurysm occlusion rates, procedure-related neurologic mortality, procedure-related neurologic morbidity and procedure-related permanent morbidity. Results Ten observational studies were included in this analysis. The complete aneurysm occlusion rate was 84.23% (80.34%-87.76%), the procedure-related neurologic mortality was 0.87% (0.29%-1.74%), the procedure-related neurologic morbidity rate was 5.22% (3.62%-7.1%), the intracerebral haemorrhage rate was 1.42% (0.64%-2.49%), the ischemic rate was 2.35% (1.31%-3.68%), the subarachnoid haemorrhage rate was 0.03% (0%-0.32%) and the procedure-related permanent morbidity was 2.41% (0.81%-4.83%). Conclusions Treatment of small IAs with FDs may be correlated with high complete occlusion rates and low complication rates. Future long-term follow-up randomized trials will determine the optimal treatment for small IAs.

Authors Sweis, Rochelle; Biller, José
Source Current neurology and neuroscience reports; Feb 2017; vol. 17 (no. 2); p. 8
Publication Date Feb 2017
Publication Type(s) Journal Article Review
PubMedID 28188542
Database Medline
PURPOSE OF REVIEW: To review the acute and chronic systemic complications of spinal cord injury and discuss treatment recommendations.

RECENT FINDINGS: The psychological, social, economic, and permanent neurologic effects associated with spinal cord injury (SCI) have universally persisted over time. Treating acute complications and preventing secondary injury can influence outcome, highlighting the importance of proper management of this patient population. Spinal cord injury (SCI) is due to traumatic or non-traumatic causes. Outcome depends on the level of injury and degree of sensorimotor deficits. After the primary injury occurs, it is crucial to detect and treat secondary mechanisms of injury. Correct method of intubation, preventing avoidable complications, and treating cardiovascular, pulmonary, renal, and infectious systemic complications are crucial as they all impact morbidity and mortality in SCI patients.
## Search Strategy

### Strategy 308026

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