NLP TERM PROJECT 2019 REPORT

Team Name: Mame

Task: Biomedical Question answering

The results of the task are as follows:-

Type	Epoch	Loss	Accur	Accur
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			rain)	(test
)

Facto	1600	0.56	0.738	0.556
List	766	1.82	0.667	0.545

Our model was tested on a few questions and the working is shown below :-

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INFO:root:Calculating F1/EM for 10 examples in dev set... Refilling batches...
Refilling batches took 1.11 seconds
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CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 261

neurobiological basis of dyskinetic effects induced by antipsychotics: the contribution of animal models . tardive dyskinesia (td) is a movement disorder characterized by abnormal involuntary facial movements induced by chronic therapy with classical antipsychotic medications . currently , there is no satisfactory pharmacotherapy for td , which represents a major limitation to therapy with classical antipsychotics . in order to develop or optimize therapies for td , and to develop new apds with lower indices of motor side effects , the pathology underlying td must first be understood . the use of animal models has been used to further this objective . here , we review different preparations that have been used to model td and discuss the contribution of neuroimaging studies conducted in these models . studies in animal models have lead to several hypotheses of td pathology , although none has yet emerged as the ultimate underlying cause of this syndrome . we discuss alterations in functional indices , neuron and synapse morphology and changes in specific neurotransmitter systems that have been described in animal models of td , and outline how these findings have contributed to our understanding of antipsychotic-induced dyskinesias . we conclude that several

non-mutually exclusive theories of td are supported by animal studies , including increases in oxidative stress leading to structural and functional changes in specific neurotransmitter systems . elucidating the mechanisms underlying td neuropathology partly through the use of animal models will lead to the development of apds with superior side effect profiles or more effective therapies for td .

QUESTION: what is the cause of tardive dyskinesia ?

TRUE ANSWER: tardive dyskinesia (td) is a movement disorder characterized by abnormal involuntary facial movements induced by chronic therapy with classical antipsychotic medications .

PREDICTED ANSWER: tardive dyskinesia (td)

F1 SCORE ANSWER: 0.261

EM SCORE: False

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 188

novel anticoagulants for stroke prevention in atrial fibrillation : current clinical evidence and future developments . atrial fibrillation (af) is the most common cardiac rhythm disorder and a major risk factor for ischemic stroke . antithrombotic therapy using aspirin or vitamin kantagonists (vka) is currently prescribed for prevention for ischemic stroke in patients with af . a narrow therapeutic range and the need of regular monitoring of its anticoagulatory effect impair effectiveness and safety of vka , causing a need for alternative anticoagulant drugs . recently developed anticoagulants include direct thrombin antagonists such as dabigatran or factor xa inhibitors such as rivaroxaban , apixaban , betrixaban , and edoxaban . currently , data from a phase iii clinical trial are available for dabigatran only , which show the direct thrombin antagonist to be at least noninferior in efficacy to vka for the prevention of stroke and systemic embolism in patients with af . this review focuses on current advances in the development of directly acting oral anticoagulant drugs and their potential to replace the vka class of drugs in patients with af .

QUESTION: which clotting factor is inhibited by betrixaban ?

TRUE ANSWER: xa

PREDICTED ANSWER: xa

F1 SCORE ANSWER: 1.000

EM SCORE: True

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, underscores are unknown tokens). Length: 350 evaluation of the recognition of stroke in the emergency room (rosier) scale in chinese patients in hong kong . background and purpose : the objective of this study was to determine the performance of the recognition of stroke in the emergency room (rosier) scale in risk-stratifying chinese patients with suspected stroke in hong kong . methods : this was a prospective cohort study in an urban academic emergency department (ed) over a 7-month period . patients over 18 years of age with suspected stroke were recruited between june 2011 and december 2011 . rosier scale assessment was performed in the ed triage area . logistic regression analysis was used to estimate the impacts of diagnostic variables , including rosier scale , past history and ed characteristics . findings : 715 suspected stroke patients were recruited for assessment , of whom 371 (52 %) had acute cerebrovascular disease (302 ischaemic strokes , 24 transient ischaemic attacks (tia) , 45 intracerebral haemorrhages) , and 344 (48 %) had other illnesses i.e . stroke mimics . common stroke mimics were spinal neuropathy , dementia , labyrinthitis and sepsis . the suggested cut-off score of > 0 for the rosier scale for stroke diagnosis gave a sensitivity of 87 % (95 % ci 83-90), a specificity of 41 % (95 % ci 36-47), a positive predictive value of 62 % (95 % ci $_57\text{--}66_$) , and a negative predictive value of 75 % (95 % ci 68-81) , and the auc was 0.723 . the overall accuracy at cut off > 0 was 65 % i.e . (323+141) /715 . interpretation: the rosier scale was not as effective at differentiating acute stroke from stroke mimics in chinese patients in hong kong as it was in the original studies , primarily due to a much lower specificity . if the rosier scale is to be clinically useful in chinese suspected stroke patients , it requires further refinement .

QUESTION: rosier scale is used for which disorder ?

TRUE ANSWER: stroke

PREDICTED ANSWER: spinal neuropathy

F1 SCORE ANSWER: 0.000

EM SCORE: False

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 386

post-surgical outcome for epilepsy associated with type i focal cortical dysplasia subtypes . focal cortical dysplasias are a well-recognized cause of medically intractable seizures . the clinical relevance of certain subgroups of the international league against epilepsy (ilae) classification scheme remains to be determined . the aim of the present work is to assess the effect of the focal cortical dysplasia type ib and ic histologic subtypes on surgical outcome with respect to seizure frequency . this study also provides an opportunity to compare the predictive value of the ilae and palmini et al classification schemes with regard to the type i focal cortical dysplasias . we retrospectively reviewed 91 focal cortical dysplasia patients (55 % female ; median age : 19 years (interquartile range 8-34); median seizure duration: 108 months (interquartile range 36-204)) with chronic epilepsy who underwent surgery . we compared the pathological subtypes , evaluating the patients ' post-surgical outcome with respect to seizure frequency according to the engel 's classification and the ilae outcome classification . both the ilae classification scheme and palmini et al classification scheme were utilized to classify the histologic subtype . using χ (2) and fisher 's exact tests , we compared the post-surgical outcomes among these groups . of the 91 patients , there were 50 patients with ilae focal cortical dysplasia type ib , 41 with ilae focal cortical dysplasia type ic , 63 with palmini et al focal cortical dysplasia type ia , and 28 with palmini et al focal cortical dysplasia type ib . after surgery , 44 patients ($48\ \%$) were seizure-free . crude analysis revealed no significant difference between patients with subtypes of ilae focal cortical dysplasia type i or palmini et al focal cortical dysplasia type i concerning postoperative outcome according to the engel and ilae scoring systems on seizure frequency . our findings revealed no significant difference concerning surgical outcome with respect to seizure frequency for the histologic subtypes of ilae focal cortical dysplasia type i (ib vs ic) or palmini et al focal cortical dysplasia type i (ia vs ib) . in isolation , the histologic subtype of focal cortical dysplasia type i does not appear predictive of postoperative outcome .

QUESTION: which disorder is rated by palmini classification ?

TRUE ANSWER: focal cortical dysplasia

PREDICTED ANSWER: dysplasia type ib

F1 SCORE ANSWER: 0.333

EM SCORE: False

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 200

novel exon skipping mutation in the fibrillin-1 gene : two 'hot spots ' for the neonatal marfan syndrome . the marfan syndrome is an autosomal dominant heritable disorder of connective tissue that involves principally the skeletal , ocular , and cardiovascular systems . the most severe end of the phenotypic spectrum , the neonatal marfan syndrome (nmfs) , is characterized by pronounced atrioventricular valve dysfunction , and death often occurs within the first year of life due to congestive heart failure . mutations in the gene coding for fibrillin-1 , fbn1 , are known to cause marfan syndrome , and have been identified in almost all exons of fbn1 . here , we describe a novel mutation affecting the invariant + 1 position of the splice donor site in intron 31 , associated with skipping of exon 31 , in a patient with nmfs . published reports of nmfs are reviewed and a strict definition for nmfs is suggested . if this definition is used , all nmfs mutations reported to date lie in one of two hot spots , comprising mainly missense mutations in $_fbn1_$ exons 24-27 and mutations causing skipping of exon 31 or 32 .

QUESTION: which gene mutations cause the marfan syndrome ?

TRUE ANSWER: fbn1

PREDICTED ANSWER: fbn1

F1 SCORE ANSWER: 1.000

EM SCORE: True

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 261

retrovirus-mediated gene transfer and galactocerebrosidase uptake into twitcher glial cells results in appropriate localization and phenotype correction . galactocerebrosidase (galc) is deficient in all tissues from human patients and animal models with globoid cell leukodystrophy (gld) or krabbe disease . the deficiency results in decreased lysosomal catabolism of certain galactolipids including galactosylceramide and psychosine that are synthesized maximally during myelination . according to current theories , the accumulation of psychosine in humans and animals with gld induces oligodendrocyte degeneration and myelination ceases . transduction of oligodendrocytes from twitcher mice with a retroviral vector containing the galc cdna can correct the enzyme deficiency in these cells . our data show that twitcher astrocytes and oligodendrocytes can internalize exogenous galc , as well as donate the enzyme to the mutant glial cells . antibodies against human galc localized the galc antigen in retrovirally transduced cells and cells receiving enzyme via cell to cell secretion and uptake to the lysosomal fraction . in fact immunocytochemical studies in transduced oligodendrocytes revealed that the $_{galc}__{colocalizes}_$ in vesicles lysosomal-associated membrane protein-2 (lamp2) (+) . moreover , labeling cells with anti-galc and a marker for oligodendrocytes demonstrated that , upon differentiation , transduced , twitcher oligodendrocytes attained the normal branched process configuration , while untransduced cells show only abnormal morphology . phenotype correction in mutant oligodendrocytes has also been observed after enzyme transfer . these studies indicate that galc activity supplied to cultured oligodendrocytes from twitcher mice by different methods can correct the pathological phenotype of these cells .

QUESTION: which enzyme is deficient in krabbe disease ?

TRUE ANSWER: galactocerebrosidase

PREDICTED ANSWER: galactocerebrosidase

F1 SCORE ANSWER: 1.000

EM SCORE: True

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 207

betrixaban (prt054021) : pharmacology , dose selection and clinical studies . the recently introduced oral anticoagulants , dabigatran , rivaroxaban and apixaban , were shown , in randomized controlled trials , to be at least as effective and safe as monitored warfarin therapy for the treatment of venous thromboembolism and stroke prevention in atrial fibrillation . these new oral anticoagulants have predictable pharmacology , less variability in anticoagulant effect and fewer drug and food interactions than warfarin , allowing unmonitored and fixed dosing , which renders their use appealing . the remaining limitations of currently available new oral anticoagulants include their dependence on renal and hepatic clearance , and the lack of an antidote , which is problematic in bleeding patients and those requiring urgent surgery . betrixaban is a new direct factor xa inhibitor with distinct pharmacological characteristics , including a long half-life , minimal renal clearance and minimal hepatic metabolism . betrixaban was tested in phase ii studies in orthopedic thromboprophylaxis (expert) and atrial fibrillation (explore-xa) , and is being evaluated in a phase iii trial of extended thromboprophylaxis in medical patients (apex). this article details the pharmacology , preclinical and clinical development of betrixaban .

QUESTION: which clotting factor is inhibited by betrixaban ?

TRUE ANSWER: xa

PREDICTED ANSWER: xa

F1 SCORE ANSWER: 1.000

EM SCORE: True

CONTEXT: (green text is true answer, magenta background is predicted start, red background is predicted end, _underscores_ are unknown tokens). Length: 334

evaluation of the oral direct factor xa inhibitor - _betrixaban_ . introduction : for over 60 years vitamin k antagonists have been the mainstay of oral therapy for treatment and prevention of venous and arterial thromboembolic disease . the emergence of two new classes of orally administered anticoagulants , direct thrombin and factor xa inhibitors have drastically changed the landscape in the management of these disease states . betrixaban , an orally administered direct factor

xa inhibitor , is entering a phase iii trial and undergoing investigation for similar indications as apixaban , dabigatran and rivaroxaban . areascovered: the chemical development of betrixaban , pharmacokinetic differences between betrixaban and currently available novel anticoagulants and future considerations for clinical use . expert opinion : betrixaban , the fifth novel oral anticoagulant in line for the food and drug administration (fda) approval, possesses some unique pharmacokinetic characteristics in comparison with the currently available novel anticoagulants , including limited renal excretion , minimal metabolism through the cytochrome p450 system and a long half-life . this pharmacokinetic profile may allow greater flexibility for use in patients with poor renal function , offer the convenience of once daily dosing , and exhibit less drug interactions . betrixaban is currently being evaluated for prophylaxis against venous thromboembolic disease (vted) and the prevention of stroke and systemic embolism associated with nonvalvular atrial fibrillation , its role in the management of acute vted and acute coronary syndromes is yet to be defined based on clinical data and evaluation . of interest , a factor xa decoy , prt4445 , is currently under evaluation in conjunction with betrixaban , and may be a universal reversal agent for all anticoagulants with anti-xa activity . currently , there are no specific reversal agents for the novel anticoagulants . the availability of an effective reversal agent would be very attractive for the management of associated bleeding , bleeding due to trauma , or the need for emergent surgery .

QUESTION: which clotting factor is inhibited by betrixaban ?

TRUE ANSWER: xa

PREDICTED ANSWER: xa

F1 SCORE ANSWER: 1.000

EM SCORE: True