I. 簡答題，每題 5 分共 30 分

1. 請例舉一生理參數(如血壓)的調節，來敘明何謂恆定控制(homeostatic control)？
2. 請說明主動運送(active transport)與促進性擴散(facilitated diffusion)間的異同？
3. 請就學理上推論，以外科手術方式破壞脊髓(spinal cord)哪一部份，能產生止痛的效果？
4. 請描述副甲狀腺激素(parathyroid hormone)如何調節血漿中鈣離子的濃度？
5. 承上題，若副甲狀腺激素分泌不足，則其對心肌與骨骼肌細胞收縮的影響是否有不同？
6. 某人心跳速率為每分 40 次，心電圖顯示正常 QRS 波但無 P 波，請解釋為什麼？

II. 藥物X能立即且長期阻斷 Na^+/K^+-ATPase pump 的功能，某生以X藥物處理培養中的神經細胞，並觀察神經細胞的生理功能變化。請回答以下問題：

(1)何謂 Na^+/K^+-ATPase，以及為何其俱有電生性(electrogenic)的特徵? (3 分)

(2)假設不考慮其電生性特徵，以X藥物阻斷 Na^+/K^+-ATPase 功能後，對神經細胞的膜電位 (membrane potential)，其瞬間與長期的影響各為何? (6 分)

(3)若神經細胞是以二級主動運送方式吸收葡萄糖，則以X藥物阻斷 Na^+/K^+-ATPase 功能後，對神經細胞的葡萄糖吸收，其瞬間與長期的影響各為何? (6 分)

III. 請描述上皮組織(epithelium)的形態、構造與功能特徵為何？ (7 分)；試再以腸道為例，這些上皮組織的形態、構造特徵如何幫助動物對食物的消化、吸收以及保護其他組織不被食物侵噬? (8 分)

IV. 動物面臨缺水的情形，會排出高濃度的尿液來減緩水分喪失。請以含下面的專有名詞(可不考慮其出現次序)寫一短文來解釋此一過程(20 分)。

interstitial osmolarity、countercurrent multiplier system、hypothalamus、collecting duct、Loop of Henle、active transport、ascending limb、vasopressin、Renal Cortex-Medulla、negative feedback。

V. 請詳閱下一頁所附英文短文後，回答以下問題：

(1) 由文中推知 tau 蛋白質在神經細胞所扮演的功能角色是什麼？而它在 Alzheimer's disease 中又產生哪些異常而可能造成疾病？(10 分)

(2) 由文中推知退化性神經病變，如 Alzheimer's diseases 與 Parkinson's disease 的發生，在細胞學上其共同特徵是什麼？(10 分)

見背面

短文開始：

Tau is a microtubule-binding protein normally present in nerve cells. In Alzheimer disease abnormal aggregates of tau are visible in the light microscope in neurons and glia as well as in the extracellular space. Highly phosphorylated tau molecules arranged in long, thin polymers wind around one another to form paired helical filaments. Bundles of the polymers, known as *neurofibrillary tangles,* accumulate in neuronal cell bodies, dendrites, and axons.

In normal neurons tau is either bound to microtubules or free in the cytosol. In the tangles it is not bound to microtubules but is highly insoluble. The tangles form at least in part because tau is not proteolytically degraded. The accumulations disturb the polymerization of tubulin and therefore interfere with axonal transport. Consequently, the shape of the neuron is not maintained.

Tau accumulations are also found in neurons of patients with progressive supranuclear palsy, a movement disorder, and in patients with frontotemporal dementias, a group of neurodegenerative disorders that affect the frontal and temporal lobes. The familial forms of fronto temporal dementias are caused by mutations in the *tau* gene.

The peptide β-amyloid also accumulates in the extracellular space in Alzheimer disease. It is a small proteolytic product of a much larger integral membrane protein, amyloid precursor protein, which is normally processed by several proteolytic enzymes associated with intracellular membranes. The proteolytic pathway that generates β-amyloid requires the enzyme β-secretase.

For unknown reasons, in Alzheimer disease abnormal amounts of the amyloid precursor are processed by β-secretase. Some patients with early-onset familial Alzheimer disease either have mutations in the amyloid precursor gene or in the genes coding for the membrane proteins presenilin 1 and 2, which are closely associated with β-secretase activity.

In Parkinson disease abnormal aggregates of α-synuclein accumulate in cell bodies of neurons. Like tau, α-synuclein is a normal soluble constituent of the cell. But in Parkinson disease it becomes insoluble, forming spherical inclusions called *Lewy bodies*.

Do these abnormal protein accumulations affect the physiology of the neurons and glia? On the one hand, the accumulations may form in response to altered post-translational processing of the proteins and serve to isolate the abnormal proteins, permitting normal cell activities. On the other hand, the accumulations may disrupt cellular activities such as membrane trafficking and axonal and dendritic transport. In addition, the altered proteins themselves, aside from the aggregations, may have deleterious effects. With β-amyloid there is evidence that the peptide itself is toxic.

短文結束

試題隨卷繳回