

# 國立中山大學 104 學年度碩士暨碩士專班招生考試試題

科目名稱：解剖學【醫科所碩士班選考】

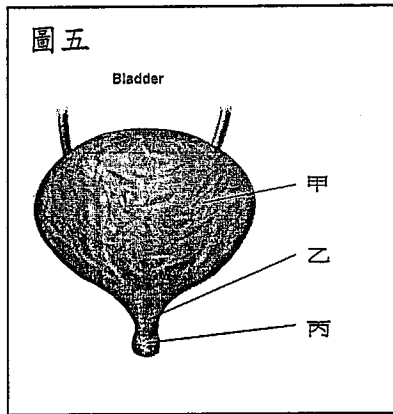
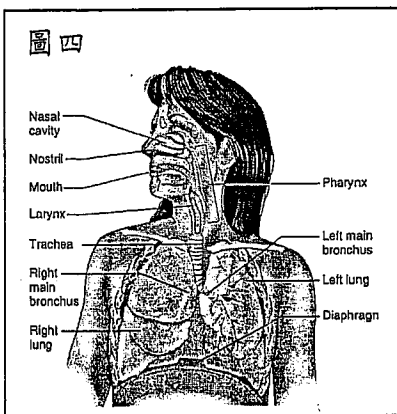
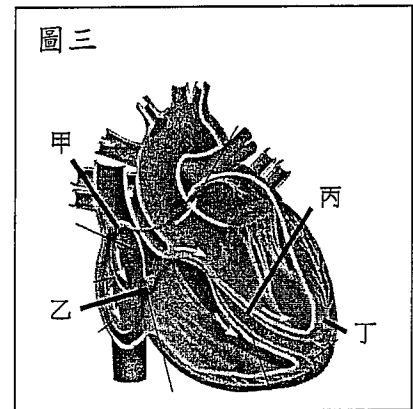
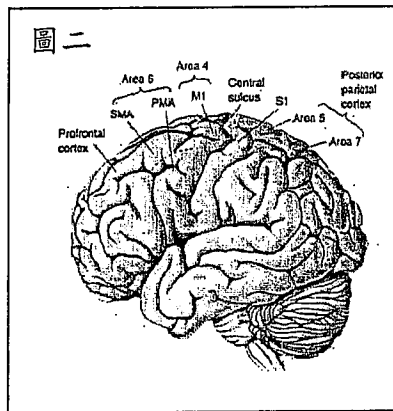
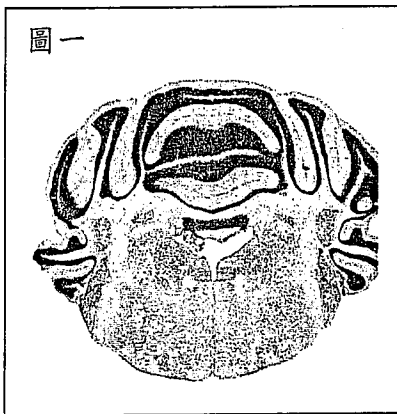
題號：428006

※本科目依簡章規定「不可以」使用計算機(混合題)

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一、單選題(每題 4 分，共 20 分)

1. 圖一為甲苯酚紫(cresyl violet)染色的大鼠腦切片，請問此切片屬於何種方位切片？  
(A) 水平 (B) 矢狀 (C) 冠狀切面
2. 圖二為人類大腦皮質的分區。請問哪個分區的活化可直接驅動四肢的運動功能？  
(A) Area 4 (B) Area 5 (C) Area 6 (D) Area 7
3. 圖三為人類心臟的剖面圖。請問標示的哪個區域具有自發性放電的特性？  
(A) 甲、乙 (B) 乙、丙 (C) 丙、丁 (D) 甲、丁
4. 圖四為人體呼吸系統的架構圖，請問哪個構造的活化或收縮會造成吸氣活動？  
(A) Trachea (B) Diaphragm (C) Larynx (D) Pharynx
5. 圖五為人類膀胱的示意圖，請問哪個部位的肌肉可受到意識的控制？  
(A) 甲 (B) 乙 (C) 丙



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## 二、問答題(共 50 分)

1. 請描繪一個典型的神經細胞並標示及說明以下構造(樹突、軸突、髓鞘、突觸)的功能。(15 分)
2. 請描繪一個腎元的構造並說明尿液形成的機制。(15 分)
3. 請描繪與標示大靜脈的結構並說明靜脈回心血受到哪些因素的調控。(10%)
4. 呼吸系統中有所謂的死腔(dead space)，請解釋生理(physiologic dead space)與解剖(anatomic dead space)死腔的定義。(10 分)

## 三、閱讀與實驗設計(共 30 分)

1. 請閱讀下方文章內容並寫出中文摘要。(20%)
2. 請設計實驗驗證此篇論文所使用的神經追蹤劑的確是"activity-dependent"。(10%)

文章來源 *Exp Neurol.* 261:440-50, 2014

### **Injection of WGA-Alexa 488 into the ipsilateral hemidiaphragm of acutely and chronically C2 hemisectioned rats reveals activity-dependent synaptic plasticity in the respiratory motor pathways.**

WGA-Alexa 488 is a fluorescent neuronal tracer that demonstrates transsynaptic transport in the central nervous system. The transsynaptic transport occurs over physiologically active synaptic connections rather than less active or silent connections. Immediately following C2 spinal cord hemisection (C2Hx), when WGA-Alexa 488 is injected into the ipsilateral hemidiaphragm, the tracer diffuses across the midline of the diaphragm and retrogradely labels the phrenic nuclei (PN) bilaterally in the spinal cord. Subsequently, the tracer is transsynaptically transported bilaterally to the rostral Ventral Respiratory Groups (rVRGs) in the medulla over physiologically active connections. No other neurons are labeled in the acute C2Hx model at the level of the phrenic nuclei or in the medulla. However, with a recovery period of at least 7 weeks (chronic C2Hx), the pattern of WGA-Alexa 488 labeling is notably changed. In addition to the bilateral PN and rVRG labeling, the chronic C2Hx model reveals fluorescence in the ipsilateral ventral and dorsal spinocerebellar tracts, and the ipsilateral reticulospinal tract. Furthermore, interneurons are labeled bilaterally in laminae VII and VIII of the spinal cord as well as neurons in the motor nuclei bilaterally of the intercostal and forelimb muscles. Moreover, in the chronic C2Hx model, there is bilateral labeling of additional medullary centers including raphe, hypoglossal, spinal trigeminal, parvocellular reticular, gigantocellular reticular, and intermediate reticular nuclei. The selective WGA-Alexa 488 labeling of additional locations in the chronic C2Hx model is presumably due to a hyperactive state of the synaptic pathways and nuclei previously shown to connect with the respiratory centers in a non-injured model. The present study suggests that hyperactivity not only occurs in neuronal centers and pathways caudal to spinal cord injury, but in supraspinal centers as well. The significance of such injury-induced plasticity is that hyperactivity may be a mechanism to re-establish lost function by compensatory routes which were initially physiologically inactive.