1. 請描述矯正牙齒移動過程中，

a. 目前已知的由機械力之給予後所發生的細胞、組織的反應，以致牙齒能移動至理想位置。

b. 假設有某蛋白分子爲過程必需的，試述如何以實驗直接證明此假設，或其他間接實驗可證實其重要性？

c. 若某蛋白分子功能爲加速牙齒移動，試述有何實驗能直接證明此蛋白分子功能，或其他間接實驗可證明其加速牙齒移動功能？

35%

2. 試述自古至今的矯正器種類，與其優缺點。20%

3. "Mini-implants used in orthodontic anchorage usually do not require osteointegration for stability." 這句話的根據爲何？迷你植體的穩定度受何影響？這些影響因子是如何找出、或要如何測試? 15%

4. Treacher Collins syndrome, which is characterized by hypoplasia of the facial bones, as well as cleft palate and ear abnormalities, is a rare autosomal dominant disorder affecting 1 in 50,000 people. It is caused by haploinsufficiency of the gene TCOF1, encoding the nucleolar phosphoprotein Treacle, which is involved in ribosome biogenesis. Tcof1 is expressed widely at low levels during early development and show peak expression in the premigratory crest in the branchial arches. The expression patter of Tcof1, the phenotype of Treacher Collins syndrome, and other findings all show that this syndrome is caused by abnormalities in the development of the neural crest. A mouse model of Treacher Collins syndrome ($Tcof1^{+/-}$ mice) has increased apoptosis, as well as reduced cell proliferation in the neuroepithelium, preceding the facial bone hypoplasia. Haploinsufficiency of Treacle causes a reduction in ribosome biogenesis within the neuroepithelium of $Tcof1^{+/-}$ mice. Because reduced ribosome biogenesis causes decreased cell proliferation, it was assumed that the impact of Treacle on ribosome biogenesis caused a decrease in cell proliferation, thus explaining the apoptosis and hypoplasia that characterizes Treacher Collins syndrome.

Treacher Collins syndrome 病人的矯正治療爲何？得到上述之研究新知，對於治療 Treacher Collins syndrome 的病人上，有何助益？30%

試題隨卷繳回