QUESTIONS – AUDIOVISUAL LECTURES

1. How does the skeletal and muscular face develop?
   a) Undifferentiated mesenchymal cells migrating from the neural crest? _____ _____
   b) Local cells in the face fail to develop? _____ _____
   c) Do all faces with lip and palate clefts have the full amount of bone tissue or are they deficient in bone tissue? _____ _____
   d) Does the extent of bone deficiency vary? _____ _____
   e) Is the cleft midface recessive at birth? _____ _____

2. How quickly does the face develop in utero?
   a) 0 to 5 weeks of pregnancy? _____ _____
   b) 5 to 8 weeks of pregnancy? _____ _____
   c) 9 to 12 weeks of pregnancy? _____ _____

3. Which racial type has the greater chance of clefting?
   a) African American? _____ _____
   b) Caucasian? _____ _____
   c) Asian? _____ _____

4. Do males and females usually have the same percentage of cleft types? _____ _____

5. Do all palates in a certain cleft palate type have the same degree of bone deficiency? _____ _____

6. Does the primary and secondary palate develop from the same source? _____ _____

7. a) Do the lateral palatal segments develop from a vertical position on each side of the tongue? _____ _____
   b) Does palatal segmental elevation depend on tongue position and its mandibular growth? _____ _____
   c) Can a micrognathic mandible be the cause of cleft palate? _____ _____
   d) Does the palatal segments elevate posteriorly -> anteriorly? _____ _____
   e) Does palatal fusion occur:
      1) At the same time? _____ _____
      2) Posteriorly -> anteriorly? _____ _____
      3) Anteriorly -> posteriorly? _____ _____

8. a) Do all palates within the same cleft type have the same proportionate amount of cleft size to mucosal covered palate medial to the alveolar ridge? _____ _____
   b) Less proportionate bone of various degrees? _____ _____
9. Can the premaxillary size vary?
   a) Due to number of teeth? YES NO
   b) Be always symmetrical in size? YES NO
   c) Not have any teeth? YES NO

10. PREMAXILLARY PROJECTION
   a) Vary in anterior projection degree from the lateral palatal segments? YES NO
   b) Vary due to differences in growth at the premaxillary vomerine suture (PVS)? YES NO
   c) Can united lip pressure retard PVS growth? YES NO
   d) Can a bodily retracted premaxilla occur from pressure by a presurgical orthopedic appliance (PSA)? YES NO
   e) PSA has no effect on the PVS and midfacial forward growth. It can always continue at a normal rate and degree? YES NO
   f) The ‘bodily’ retracted premaxilla may have a synostosis at the PVS? YES NO
   g) Can the retruded premaxilla still be advanced out of crossbite? YES NO
   h) There is no negative midfacial response at PVS if the premaxilla is ‘ventroflexed’? YES NO
   i) Lip adhesion causes’ bodily’ premaxillary retraction as well as a premaxillary ventroflexion? YES NO
   j) Elastic forces against the protruding premaxilla act the same way as lip adhesion? YES NO
   k) A bony bridge created by gingivoperiosteplasty after closing the lateral incisor space prevents premaxillary advancement and crossbite correction? YES NO

11. a) Upper and lower anterior arch congruency can only be attained when the lateral incisor space(s) are present? YES NO
    b) After premaxillary ventroflexion does synostosis of the PVS occur? YES NO
    c) After premaxillary ventroflexion only some inflammation at the PVS occurs but no synostosis?? YES NO
    d) After premaxillary ventroflexion secondary alveolar bone grafts (SABG) can replace missing bone at the lateral incisor space(s)? YES NO
    e) SABG utilizes:
        1) cortical bone alone? YES NO
        2) using medulary bone alone? YES NO
        3) using both types of bone together? YES NO
    f) Is premaxillary palatal segmental stabilization attained after SABG? YES NO
    g) Is palatal form stabilization required after SABG as well as after palatal expansion? YES NO
12. FACIAL GROWTH
   a) Does the facial growth pattern determine whether the premaxilla should be ventroflexed or bodily retracted or surgically set back? 
   b) What is to be done to the protruding premaxilla:
      1) ventroflexed? 
      2) bodily retracted? 

13. In CUCLP and CBCLP
   a) After lip adhesion does the anterior or buccal crossbite prevent palatal growth?
   b) Does anterior and buccal crossbite occur after NAM & GPP?
   c) Can this happen after NAM alone:
      1) Depending on the effect at the PVS in CBCLP?
      2) Some times in CUCLP?
   d) In non orthopedic cases does arch expansion in CBCLP & CUCLP require palatal expansion then retention?
   e) Does SABG prevent relapse in arch form?
   f) In CBCLP if the palatal segments are positioned behind the protruding premaxilla:
      1) Is the palate prevented from growing?
      2) Must the lateral palatal segments be immediately expanded and the premaxilla retruded?

14. PRESURGICAL ORTHOPEDICS
   a) The Latham –Millard presurgical orthopedics with periosteoplasty followed by lip adhesion (POPLA)
      1) Causes severe midfacial growth disturbance?
      2) Causes no facial growth problems?
      3) Are there any longitudinal studies to support use of the procedure?
   b) Nasoalveolar Molding + Gingivoperiosteoplasty
      1) Is it a modification of POPLA and can also cause midfacial growth problems by causing synostosis of the PVS?
      2) Causes negative facial/occlusal development in all cases?
      3) Are there any longitudinal objective records to support its use?
      4) NAM alone in CUCLP cases can be satisfactorily used but only in some CBCLP cases when it is limited to premaxillary ventroflexion?

15. PROTRACTION FACIAL MASK
   a) Can be successfully used in the:
      1) Deciduous dentition to correct maxillary recessiveness in some cases?
      2) Mixed dentition to correct maxillary recessiveness or anterior crossbite?
      3) Adult dentition to correct maxillary recessiveness or anterior crossbite in some cases?
b) Protraction forces have to emanate from
   1) The maxillary cuspid area in all cases?
   2) From the molar area?
   3) From both the cuspid and molar area in very few cases?

c) Limited to correct anterior crossbites in most cases?

d) Can correct Class 3 in only some cases?

16. NASOPHARYNGEAL SPACE

a) VPI is mainly caused by the late closure of the palatal cleft?
   1) After 6 months?
   2) After 12 months?
   3) After 2 years in most cases?

b) VPI only involves inadequate velar size?

c) Spinal form is an important factor to be considered?

d) The position and size of the tubercle at the first cervical vertebrae is not a
   factor in causing VPI?

e) The normal size and position of the palate within the face is important?

f) Can the scarred hard palate fail to descend to a proper velar position and
   influence velar position and function?

g) Can one or both lateral pharyngeal muscles be a factor in VPI?

h) Where to attach the velar flap to the retropharyngeal wall?

i) Does the flap’s width determine success or failure of VPI surgery?

j) Can the angle of the cranial base be a factor in causing either
   hypernasality or hyponasality?

17. PALATAL GROWTH

a) The velocity of palatal growth is always limited to 6 to 12 months of age?

b) To 18 to 24 months in well treated and normal cases?

c) Do all cleft types have the same degree of palatal bone deficiency?

d) Is cleft size the same after molding is complete and crossbite corrected?

e) Can some surgical procedures reduce palatal growth?

f) Can some presurgical prosthetic procedures stimulate palatal growth and
   reduce the cleft space?

g) Can palatal surgery performed at 18 to 24 months lead to speech that
   requires speech therapy in most cases but leads to good facial growth?

h) Can palatal surgery at 36 to 48 months as reported by European speech
   therapists lead to good speech with minimum therapeutic needs?

i) Can palatal closure be delayed two years to obtain good facial/palatal
   growth as well as speech?

j) Be delayed to 3 years to obtain good growth as suggested by Goteborg?
18. What are the three main factors to be considered in Treatment Planning:
   a) 1) Facial growth pattern?  
       2) Ratio of cleft size to available palatal soft tissue area?  
       3) Avoiding palatal scarring?  
   b) Can good facial aesthetics be achieved in all cases without the use of presurgical orthopedics?

19. FACIAL GROWTH
   a) Will good lip/nose aesthetics created at 6-12 months of age using presurgical orthopedics always be present at 6 to 12 years of age and later?  
   b) Are different facial growth patterns a factor in treatment decision making?  
   c) Are there different degrees of palatal bone deficiencies?  
   d) Can the degree of premaxillary protrusion vary in different facial growth patterns and even within the same growth pattern?  
   e) Can facial aesthetics improve with facial growth alone, that is, without the need for presurgical orthopedics?  
   f) Is it better to have good facial aesthetics when the child starts school rather than soon after birth?  
   g) Is the surgeon treating the parents rather than the child by aiming for good facial aesthetics soon after birth?  
   h) Is staged facial/palatal surgery including secondary alveolar bone grafting, more physiological than completing all surgery by 2 years of age?  
   i) Does Differential Diagnosis in Treatment Planning mean that all children within the same cleft type may need different treatment plans according to the facial growth pattern and degree of palatal bone deficiency?  
   j) A child with Class 1, 2 or 3 may have different palatal cleft sizes and therefore cannot have all cleft palate surgery completed by 2 years of age?

20. 
   a) Are all goals of good facial aesthetics, speech, dental occlusion and psycho-social development attainable?  
   b) Should a priority of one goal over another be acceptable?  
   c) Does this mean that some cleft problems may require different degrees of speech therapy?  
   d) If additional speech therapy at a later age is acceptable does this mean a trade-off of one goal over the other goals is recommended, that is, early palatal surgery?