XIII.

WHAT IS THE CAUSE OF DEFECTIVE ORIENTATION OR EQUILIBRATION?

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The great demand for aviators and the large number of men being examined for service has led the writer to consider that it is an excellent opportunity for ophthalmologists and otologists to study deeply into the cause of defective orientation or equilibration.

Several months ago I wrote Dr. C. R. Holmes of Cincinnati, who was appointed to an aviation examining board, if he had the time, to investigate if there was any relation between defective eye muscle balance and defective equilibration from labyrinthine defects.

Over twenty years ago Dr. George T. Stevens, of New York, the pioneer and master of eye muscle defects, began a study of skulls in museums and colleges, and his researches showed that the bony orbits of some skulls were plumb with the horizon and some slanted, and some pitched too high or too low and declined at the same time. From this he drew the conclusion that this would make the eyes incline or pitch correspondingly. He used Helmholtz's haploscopic experiment and invented his clinoscope, which demonstrated that many people had more or less declination of the vertical meridians because the bony orbits were not plumb. He then devised his advancement operation of one or all four of the recti muscles of one or both eyes to successfully overcome this defect. Many people having declinations are subject to dizziness, afraid to go in crowds or high places, etc.

The connections of the eye muscle nuclei and labyrinthine nuclei in the brain are intimate, as we all know.

With the knowledge we have of the orbits being liable to stigmata of degeneration—i. e., inclinations or declinations—

down into the larynx, so that the upper end of the tube is under the finger holding up the epiglottis. The finger then holds the tube in position while the obturator is being removed.

If there is difficulty experienced in attempting to introduce the tube marked for the age of the patient, it is usually due to some edema or swelling in the cricoid ring, which is the narrowest point, or to a transient spasm of the glottis. In such cases a smaller tube may be introduced with slight force. Often our best judgment is put to a test as to when the tube should be removed. I am convinced that it should not be removed sooner than five days, in the majority of cases. If done in less time, before the pseudomembrane has disappeared, reintubation will most likely become necessary. The tube should remain longer if the pseudomembrane persists, and it may become necessary to reintubate a number of times if there are ulcerations about the cricoid cartilages, edema, granulations, abductor paralysis or cicatricial contractions.

The early administration of antitoxin has largely reduced the mortality in laryngeal diphtheria. I believe all cases of membranous laryngitis should be treated at first as diphtheritic and that antitoxin be given as soon as the stenosis is suspected or recognized, and the microscopic and cultural examinations be made afterward. If repeated findings are negative as regards Klebs-Loeffler bacilli, the antitoxin need not be continued; but, if still in doubt, it is best to continue it. If the intubating tube is carefully and gently introduced and the string removed, the presence of the tube is not going to disturb or annoy patients very much, and they are much more comfortable while the antitoxin and other medication are being administered.

The majority of cases intubated can be fed liquid food when in an upright position; but it is most always followed by some cough and, frequently, by complaint of some pain. If feeding in the upright position cannot be done, the Casselberry or Hillis position may be resorted to.