Kitty M., aged 11 years, an American schoolgirl, first came to my clinic January 17, 1902.

Family History.—Mother living and well; father died of pneumonia; she has four brothers and sisters in good health; both maternal grandparents lived to old age; her paternal grandmother developed some form of paralysis after childbirth. Her paternal grandfather was paralyzed late in life, and had some disturbance of speech which passed away before death.

Previous History.—Birth and early history negative except for the usual diseases of infancy. She had pneumonia when six years of age; diphtheria three years ago and again last April (1901) from which she apparently made a good recovery. For the past two or three years the mother has noticed more or less impairment of hearing.

Present Illness.—Early in September the mother detected slight difficulty in swallowing and some defect in the child's speech; it seemed as if there were something in the mouth. About the middle of September she was sent home from school with instructions to have her eyes tested. This was done, and she was given glasses for astigmatism.

Drooling and speech defects led to a consultation with a throat specialist, who advised the removal of post-nasal adenoids. The operation was performed about the first of October. She had occasional fits of obstinacy and cried frequently, i.e., she was emotional but showed no evidence of impaired mentality. Speech and deglutition became increasingly difficult so that the hand was occasionally employed to retain food in the mouth.

Status Praesens.—A girl of average size for her age, but somewhat pale and poorly nourished. The expressionless face, fallen lip and drooling mouth first arrest attention. She answers questions indistinctly and laboriously; the speech defect shows inability to pronounce labials and denti-linguals; she cannot

*This case was shown before The Chicago Pediatric Society in February: also before The Chicago Academy of Medicine in March, last.
wrinkle the forehead, elevate the eyebrows, close the eyes completely, purse the lips, or smile; the tongue cannot be protruded beyond the teeth or raised to the roof of the mouth; it is thickly coated, plainly shriveled and lies passively in the floor of the mouth with occasional flickering tremors. The pharynx presents no gross lesions, but the uvula and palate show some anesthesia; the voice is monotonous and of nasal quality; the mouth is full of saliva which pours over the pendulous lip; the child masticates food slowly and swallows with marked difficulty, at times choking and coughing. Fluids do not regurgitate through the nose, nor is there any history of this symptom.

The respiration is shallow and under excitement or exertion becomes slightly snoring; she also snores while asleep.
The examination of the ocular fundus and ear gave negative results.† Pupillary reflex normal. The range of ocular movements is somewhat restricted toward the right. Slight horizontal nystagmus is at times noticeable. She cannot close the eyes completely, prolonged effort causing parallel deflection upward and to the left. Eyes are constantly suffused, and there is conjunctival anesthesia. The general muscular system shows but little wasting. Patellar reflexes normal and co-ordination good. Electrical response to both galvanism and faradism normal, with the exception of the facial muscles, which show reaction of degeneration—

- A. O. C. = 2.5 M. A.
- C. C. C. = 4.5 M. A.
- A. C. C. = 4.5 to 5 M. A.

There is no response to faradism (the strongest current) in the facial muscles. (Kindness of G. W. Hall, M.D.) No anesthetic areas, either thermal or tactile, were found except on palate and conjunctiva. No tremors were present, except occasional fibrillary twitchings of tongue and facial muscles.

Taste as far as observed not affected.

Chest examination negative with exception of systolic murmur; abdomen negative; urine negative.

The child has been under our observation for seven weeks and during that time the lowest and highest temperatures have been 97.4° and 100.4° F.; pulse 75 to 130 and easily disturbed; respiration 10 to 26.

She has lost 5 pounds in weight, with perceptible diminution of muscular vigor. During the last week the gait has shown some uncertainty, and there is increasing tendency to sleep during the day.

All the plegic symptoms have steadily intensified since the first observation.

Bulbar paralysis is certainly so rare a disease among children that no apology is needed for bringing it to your attention. As you will recall, this disease was described for the first time in 1860 by Duchenne, and it was not until ten years later that the real seat of the lesion was recognized by Charcot and Leyden.

The fact that the symptom-complex of glosso-labio-laryngeal paralysis may accompany lesions other than the degenerative process in the nuclei of medulla, would excuse a brief examination by Prof. Hotz.
consideration of the classification given by Collins in *Twentieth Century Practice*.

This author presents these paralyses with reference to their etiology under eight heads. With your permission I will give them *seriatim* in comparison with the clinical findings of the case before us.

**Collins' Classification.**

1. Progressive labio-glosso-laryngeal paralysis.
2. Primary vascular lesions; (a) acute inflammatory; (b) bulbar hemorrhage; (c) thrombosis and embolism.
3. Bulbar neuritis (associated neuritis of bulbar nerves).
4. Infantile bulbar paralysis; (a) familiar; (b) hereditary.
5. Secondary degenerative lesions of the bulb; (a) in amyotrophic lateral sclerosis; (b) in syringomyelia; (c) in tabes and multiple sclerosis.
7. Tumors of medulla oblongata, including gummata.
8. Bulbar paralysis without anatomical foundation (asthenic bulbar paralysis).

From his second class, due to vascular disturbances, this case may be excluded by its gradual onset and absence of acute symptoms, as fever, disturbance of pulse-rate, vomiting, etc.

For the same reason (gradual onset) we may rule out class three, viz., associated neuritis of bulbar nerves, which condition not only causes a rapid development of symptoms, but is rarely reported as furnishing all the symptom-complex of true bulbar paralysis.

The fourth class (familiar or hereditary type) we may possibly exclude for want of corroborative history, her brothers and sisters being free from any suggestion of similar trouble.

There is no evidence of any involvement of the cord, such as spasticity, increased muscular irritability or atrophy.

Lack of sensory disturbances, as analgesia, thermic anesthesia and also lack of muscular atrophies may exclude syringomyelia.

The persistence of the patellar reflex, absence of crises and the Argyll-Robertson pupil may allow us to dismiss tabes from consideration. And although we have a slight nystagmus the intention tremor, scanning speech, rigidity and exaggerated knee-jerks of multiple sclerosis are wanting. If, as it has been
claimed, an isolated area of a developing multiple sclerosis have its seat primarily in the medulla and produce the symptoms seen in this patient, may we not claim that it is time (seven months) for symptoms of other sclerosed areas to manifest themselves?

In pseudobulbar (cerebral) paralysis we should expect a sudden onset with possible subsequent improvement until there is a second attack, possibly hemiplegia; the atrophy should be that of inanition and the muscles should not show the reaction of degeneration. Especially is atrophy of the tongue wanting. Nor is pseudo-bulbar paralysis long free from other evidences of cerebral involvement, as mental impairment and emotional disturbances.

From tumor of the medulla it is possible that enough time has not yet elapsed for the development of sufficient symptoms to make positive differentiation. Indeed, we have already involvement of the upper facial muscles and slight involvement of the ocular muscles.

However, the negative condition of the fundus, absence of sensory disturbances, cephalalgia etc., might lead us to exclude a tumor of any considerable size.

The entire absence of any other signs of syphilis renders the diagnosis of gummata improbable.

Asthenic bulbar paralysis, without anatomic foundation, is not recorded as showing true muscular atrophy with the reaction of degeneration. Nor is its course steadily progressive; on the contrary, almost complete cessation of symptoms follows intervals of rest.

The extensive involvement of the facial nerve seen in this case, though not of itself a part of the symptom-complex of true bulbar paralysis, still occurs with sufficient frequency not to jeopardize our diagnosis, providing the pathognomonic symptoms be present.

As to the ear involvement, the history of partial deafness for the past three years would indicate merely a coincidental condition.

It seems to the writer that from the symptoms present, the course of the disease and the onset, he is justified in classing this case as degenerative bulbar paralysis. The extreme rareness of this affection in a female child lends exceptional interest to this case.

1485 Jackson Boulevard.
Dr. Charles S. Bacon read a paper entitled "The Importance of Rickets in Girls from an Obstetrical Standpoint."

Rickets is a rather common disease of infancy. Its most serious results, so far as girls are concerned, do not manifest themselves until the child-bearing period. It is estimated that from 3 to 7 per cent. of all women have contracted pelves due to rickets. The importance of a pelvic contraction of 1 to 2 cm. is considerable. Concerning the etiology of the disease, the effects of unsanitary surroundings, improper food and gastrointestinal disease are acknowledged but the essence of the trouble is not yet determined. Among the food deficiencies, the lack of fat is undoubtedly the most important.

The most common pelvic deformity is the simple flat pelvis. This is caused by the weight of the trunk when the child is in the sitting or standing position. The soft bodies of the sacral vertebrae are crowded forward between the wings of the sacrum thus contracting the antero-posterior diameter. The strong sacro-iliac ligaments pull backwards the posterior margins of the ilia, and because of the anterior fixation of these bones at the symphysis the transverse diameter of the pelvis is increased.

The indications for the treatment of rickets in its acute stage are to control the disease process as soon as possible and to prevent the pelvic deformity. The disease process is corrected by dietetic and hygienic management. Gastrointestinal infection is overcome and the child placed in as good sanitary surroundings as possible. Fat, which is often lacking in the diet, is given, often in the form of cod-liver oil.

The question of the prevention of pelvic deformity is new. Its importance has been overlooked by orthopedists. It is very difficult to keep the child in the horizontal position. If it were possible to devise apparatus which would take the trunk pressure from the sacrum it would be a valuable device.