aneurysm and what does not, I think it safe to assume that such a large sized saccular dilatation of an artery as was found in the case presented here is really an aneurysm. It represents the only pulmonary aneursym in the records of the Office of the Chief Medical Examiner of the City of New York, since Jan. 1, 1918. During the six years from 1918 to 1924, about 65,000 cases with almost 10,000 necropsies have been handled. This is proof positive that pulmonary aneurysm is rare.

Nevertheless, I found more cases of pulmonary aneurysm than I expected reported in the literature. Henschen,[2] for example, in 1906, after carefully sifting the literature, compiled forty-two such cases. Posselt,[3] in 1909, added nine more, and gave a bibliography of seventy-six authors on the subject. In addition, a few doubtful cases have been reported. Nearly all the aneurysms were necropsy findings.

Since 1909, a cursory review of the literature shows cases by Ploeger,[4] Warthin,[5] Foster,[6] Stoddard[7] and Pissot.[8] Zak[9] describes a pulmonary aneurysm and patent ductus arteriosus, but this was diagnosed only by roentgen-ray examination, and the diagnosis was not corroborated by necropsy. A number of pathologists also have found aneurysm of the ductus arteriosus associated with more or less dilatation of the plumonary artery.

As far as the etiology of pulmonary aneurysm is concerned, the most important factor seems to be a patent arterial duct, and a large number of the cases reported by Henschen, Posselt and others showed such a condition, although in no instance was the duct as large as that of the one I report here. Henschen also regards syphilis as an important causative agent, but Posselt does not agree with him. Although age does not seem to exert much influence (the ages in the various cases varied all the way from 5 months to 80 years), pulmonary aneurysm was nevertheless most frequently found in patients between 35 and 45 years of age. Unlike aortic aneurysm, pulmonary aneurysm is more frequent in women than in men, just as a patent arterial duct is oftener found in female than in male infants. The cause of the patency of the ductus arteriosus must be sought in the same mechanism that keeps the foramen ovale open and, as such, is intimately connected with the local changes in blood pressure due to the respiratory efforts of the new-born. Pulmonary atelectasis, therefore, undoubtedly favors a patency of the arterial duct.

Contrary to what one would expect, sudden death is not common with pulmonary aneurysm, and only two cases have been reported in which the patients died from a rupture of the aneurysm. In one of these, hemorrhage took place into the pleural cavity; in another, reported by Durno and Langdon Brown,[10] the hemorrhage was into the pericardium.

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SPEECH DEFECTS

CLASSIFICATION AND TREATMENT AT THE UNIVERSITY OF CALIFORNIA HOSPITAL

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The study of the treatment of speech defects and disturbances has been left, for many years, to a few specialists or teachers with a limited knowledge of the causes of these disturbances. Not until recent years have physicians realized the need of a speech clinic to which such cases could be referred. After eight years of experience in conducting the speech defect clinic of the University of California medical department, I feel that a general discussion of this subject might be of interest.

In 1915, our clinic was opened under the auspices of the pediatric department. Since then the other departments also have referred to the clinic all cases needing speech improvement. Every kind of a speech defect, from the simplest type—that of poor enunciation, in cases sponsored by interested teachers—to the most difficult, i. e., the nervous type, has crossed the threshold of the speech clinic at the University of California Hospital.

For convenience in classifying, the different speech defects may be thus arranged:

1. The nervous speech disorders, under which come stammering, stuttering, cluttering, and nervous hesitation.
2. Retarded speech in both normal and subnormal children.
3. Infantile substitutions not caused by mouth malformations.
4. Substitution of sounds or imperfect speech caused by malformations of the speech organs.
5. Voice defects, not organic.
6. Voice defects after certain operations, such as cleft palate or adenoid.
7. Imperfect speech through partial deafness.
8. Aphasia, sensory or motor.
9. Foreign substitution of speech sounds, caused by foreign environment.
10. Sluggish enunciation due to poor muscular coordination, which may possibly have been caused by certain diseases of childhood.

NERVOUS SPEECH DISORDERS

Of the nervous speech disorders, stammering is a spasmodic action of the speech muscles which may be manifested visibly or audibly in degrees ranging from the slightest, almost imperceptible, to the most violent contortion. Stuttering, as understood in this country, is a rapid repetition of the initial sound of words. Cluttering is a rapid, choppy utterance because of which certain parts of words are slurred over or lost. Nervous hesitation in its very title is self-defined.

These four defects, after years of struggle and fruitless experimentation, often to the detriment of the patient's health or life, have at last been properly classified as to origin. German articles as recent as prewar days still confine stammering to the purely mechanical speech defect group. In fact, as late as the summer of 1923, I was amazed on visiting the speech clinics of Vienna, Paris and Berlin, to find them still clinging to the mechanical aspect, advocating physical speech drills, imposing torture through the use of numerous instruments for tests of breathing, phonation and muscle stimulation—attempting everything, in fact, but the

most direct, and from my point of view, the only method: that of reaching the seat of the trouble through psychologic reeducation. For I am convinced, after years of history-taking and of progressive familiarity with the inner lives of hundreds of speech defectives, children and adults, that the origin of their nervous disturbances is a psychic one—perhaps a sudden shock, perhaps continued emotional disturbance.

The treatment, granting this assumption, follows naturally. It consists in teaching the conscious control of the speech mechanism, and, at the same time, in associating new positive ideas of control, poise and confidence. The patient's obsession that his speech organs will refuse to work, his horror of being the object of ridicule, and his dread of being thought inferior—these are the three predominant fears that must be destroyed. Earnest application on the part of the patient, and intelligence and understanding on the teacher's side, will bring about results in a few months.

RETARDED SPEECH IN BOTH NORMAL AND SUBNORMAL CHILDREN

The end of the third year has been set by the Germans as the extreme limit for the attainment of complete speech control. This varies, however, with the individual child. Our records at the university clinic show normal speech development as early as at 2 and as late as at 9 years of age.

The normal child learns speech mostly through imitation, the child's accuracy depending on its sense of perception and its ear for the delicate shades of sound. When the desire for imitation, which is a primal instinct, is lacking, there exists a type of retarded speech defect caller "Hörstummheit" in German—dumbness without deafness. Children suffering with this defect must be very tactfully handled. Efforts to stimulate their imitative powers are usually of no avail, and it is only through patient, indirect teaching, the use of pictures, etc., that results are obtained.

Another type of retardation in speech development when the child is normal may be caused by inattention. This, bringing in its wake lack of concentration, and implying lack of, or at least dulness of perception, lays an excellent foundation for retarded speech. Treatment consists in first securing the attention of the child. Thereupon, auditory stimulation is fostered, the child actually being taught the elements of the spoken language, first sounds and then words. Later, he is taught language structure.

Cases of retarded speech in normal children are also occasionally found when there are unnaturally silent home conditions; there are instances in which on investigation, it has been found that the parents rarely speak to each other, or that the child has no companions.

Subnormal children must be judged from a different point of view. With them it is a question of mentality and inability to comprehend. The treatment in these cases is a slow building up of word associations by means of pictures, toys, and the like. This succeeds according to the degree of intelligence involved.

INFANTILE SUBSTITUTIONS NOT CAUSED BY MOUTH MALFORMATIONS

To the class of infantile substitutions not caused by mouth malformations also belong children who lack perception. Mistakes in speech made in early childhood are carried on into youth and adult life, merely because the patients themselves have never realized their mistakes, or have never had their attention called to them by others. Various forms of lisping come under this head. Mere placing of the tongue in the right position by means of a wooden spatula, or with the use of a mirror, followed by drill, will correct this defect.

SUBSTITUTION OF SOUNDS OR IMPERFECT SPEECH CAUSED BY MALFORMATIONS OF THE SPEECH ORGANS

The substitution of sounds or imperfect speech caused by malformations of the speech organs is a defect of a purely mechanical nature. In this group are harelip, feeble or very short upper lip, overshot jaw, narrow palate, cleft palate, sluggish palate, nasal obstructions, tongue-tie, and very irregular teeth. The treatment in most of these cases is surgical, followed by speech reeducation.

VOICE DEFECTS—NOT ORGANIC

The voice reflects every emotional state. High-pitched voices are caused by various psychologic disturbances, such as excitement, tenseness, anger or irritability. Timidity, on the other hand, expresses itself by low, weak tones. Nervous haste brings forth rapid, incoherent speech. Sarcasm is expressed by sharpness, nervousness by choppiness, and hatred by hardness. Coarseness and loudness are caused by a lack of breeding. Many of the nasal tones, as characterized by speech in certain parts of the country, are purely imitative.

Since this defect partakes of a dual nature, psychologic as well as physiologic, it must be approached from both standpoints. An agreeable speaking voice, either the teacher's or that of a mechanical substitute such as is furnished by a phonograph, is used to create a standard for imitation. Psychologic reeducation includes not only the pointing out of the basic trouble, whether it be irritability, timidity or what not, but also the supervision of the cure.

VOICE DEFECTS AFTER CERTAIN OPERATIONS, SUCH AS CLEFT PALATE OR ADENOIDS

Voice defects after operations are of common occurrence. No matter how technically perfect the operation and its results, speech itself must be retrained. The old speech habits due to, say, a cleft palate, are commonly clung to even when the defect has been righted. For this reason, it is very important that the patient be taught to raise and lower the palate and to redirect sound, in order to develop resonance. The formation of the explosive consonants, p, b, t, d, k and g, must actually be taught for the first time, since the very nature of the patient's affliction precluded the forming of these consonants.

Incidentally, it has been my experience that in cleft palate, better speech is obtainable in patients who have not been operated on than in those who have; that is, when the patients who have not been operated on have been supplied with a dental contrivance that is a combination of the hard and the soft palate, fitted in like a plate. The muscles in such cases are free to adjust themselves to the new apparatus, and the results from a speech standpoint are much more normal. A cleft palate operation in itself may be a perfect success, but the results, as far as the flexibility of the palate is concerned, do not compare with the dental method of procedure.

An unusually interesting example under this heading is the case of voice development after laryngectomy, which I recently witnessed in Vienna. A woman, aged 40, whose larynx had been removed the previous year,

was seen at her work, that of a superintendent of a hospital, telephoning and giving instructions. Her voice, while loud, was monotonous, carrying no inflections. It sounded hoarse. She had been taught to speak by developing an unusual function of the stomach. Instruction had been started by the giving of soda-water, which had started gas. From this belching, vowel sounds and words had been developed until a continuous sound resulted, and the patient had learned to use inhalations and exhalations at will, without recourse to the original stimulation.

IMPERFECT SPEECH THROUGH PARTIAL DEAFNESS

The first test applied to speech defectives, barring nervous speech defectives, is that of hearing. The discovery of partial deafness easily accounts for imperfect speech, and the patient is promptly referred to the proper specialist. If treatment on his part is of no avail, the residual hearing is developed, and lip-reading taught in the defective speech department.

APHASIA, SENSORY OR MOTOR

Complete sensory aphasia is hopeless from the point of view of treatment. Partial sensory aphasia is amenable to training, according to some authors, in patients up to the age of 12. Under this heading may be mentioned certain types of poor spellers and defective readers. Defects in visual perception, which are the cause of poor spelling, for instance, are approached by means of unusual size or color of type or lettering, thus emphasizing the troublesome letters.

Complete motor aphasia, since memory for the required speech action is gone, is also hopeless. Some patients with partial motor aphasia can be improved by exercises in muscular coordination and conscious word development.

FOREIGN SUBSTITUTION OF SPEECH SOUNDS CAUSED BY FOREIGN ENVIRONMENT

Each language has its definite tone inflection and its peculiar mouth action. In learning a new language, the student often carries over the habits of his own language, or substitutes the nearest sound if in doubt of the new sound formation. German and French, for instance, require greater action of the lips; English of the tongue and jaw; the Oriental languages call for practically no jaw action. The treatment is direction of attention to the existing facts, ear training, and correct tongue and lip positions.

SLUGGISH ENUNCIATION, DUE TO POOR MUSCULAR COORDINATION

Poor muscular coordination may be either physiologic or mental in origin. Physiologic sluggishness may be caused by an inertness of the speech muscles, often accompanied by mental inertness, or result from diseases of childhood, such as diphtheria, infantile paralysis or hemiplegia. The method of approach in such cases is a gymnastic one. Carefully organized and directed mouth gymnastics, followed by speech sounds and words, are repeated until greater flexibility and precision of movement are obtained.

CONCLUSIONS

Certain truths stand out preeminently. Again and again, I have found physicians who have advised anxious parents of children coming under one of the foregoing categories to wait, that they would "outgrow it." Nothing is more harmful than this waiting. The older the child grows, the more established and the firmer rooted does the speech defect habit become. If it is of a mechanical nature, early surgical intervention is advisable—nothing is gained by waiting. Only when the cases are light ones, caused by imitation, or when the increasing mental development of the child enables him to see and to correct his own mistake, are the defects "outgrown."

Waiting has another harmful angle. Any one of the aforementioned nervous speech disorders, if allowed to continue, has a destructive effect on the patient's morale. He becomes a warped, self-conscious and negative individual, full of fears. At times these fears are repressed, causing neuroses. All of these nervous speech disorders are curable if attacked in the right way by the right instructor. Their origin is psychologic, and psychologic reeducation is their solution.

EFFECT OF EXPOSURE TO LOW TEMPERATURES ON DIPHTHERIA TOXIN-ANTITOXIN MIXTURE*

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Early in the last week of January of the present year, an experience due to a peculiar set of unique and fortuitous circumstances occurred which could not have been anticipated by the application of any knowledge then existing. In two towns in Massachusetts, two different lots of diphtheria toxin-antitoxin mixture, after having suffered prolonged exposures to unusually low temperatures, were injected into a total of fifty-four children for the purpose of immunizing them against diphtheria, and produced severe reactions in forty-two of them. Both the local and the systemic reactions were typical of diphtheria intoxication.

All the facts strongly suggested the hypothesis that the exposure to temperatures below freezing had caused the antitoxin to dissociate from the toxin, leaving free toxin present in the mixture. In order to determine the validity of this hypothesis, the experiments here reported were carried out.

I. PREPARATION OF THE TOXIN-ANTITOXIN MIXTURE

The mixtures were made according to the recommendations issued by the United States Hygienic Laboratory. The details of the preparation of one of the mixtures (100 T) were as follows: The toxin made from *B. coli* fermented veal infusion broth containing 2 per cent. proteose peptone had been aged for nineteen and one half months. It originally had a minimal lethal dose of ± 0.0025 c.c., and at the time it was used in making the mixture its L + dose was 0.19 c.c. To 1,475 c.c. of this toxin was added 5.48 c.c. of concentrated antitoxic globulin, twenty-two and one half months old, and containing approximately 1,250 antitoxin units per cubic centimeter. The antitoxin was diluted just before its addition to the toxin with 5,900 c.c. of sterile 0.85 per cent. sodium chlorid solution containing 0.4 per cent. phenol (carbolic acid). The total volume of the mixture was 7,400 c.c., and each cubic centimeter contained approximately 1.05 L + doses of toxin. The mixture was made, Nov. 11, 1923, filtered

* From the Massachusetts Department of Public Health, Antitoxin and Vaccine Laboratory.