

creased, and the bundles appeared compressed, but Weigert's stain indicated no localized degeneration. The intervaginal space was not at all well marked.

Amaurotic family idiocy was considered in three papers published¹ a few years ago. The discussion of these papers was general and exhaustive, and the consensus of opinion then was that the disease was one of degeneration of the ganglion cells of the entire nervous system, and that the changes in the retina and optic nerve were simply the ocular manifestations of a general disease.

It was unfortunately impossible to obtain an autopsy in the case just reported. It seems to me that the arrested development theory of Sachs, the degeneration theory of Kingdon and Russell, and the toxin theory of Hirsch fit together very well. There is every reason to believe that a child can be born normal and for a short time give no indication that it will not continue in this condition. We have all seen this over and over again, in fact, it is the rule. A child does not cease to develop when it is able to breathe, and the central nervous system is the most backward of all. If the central ner-

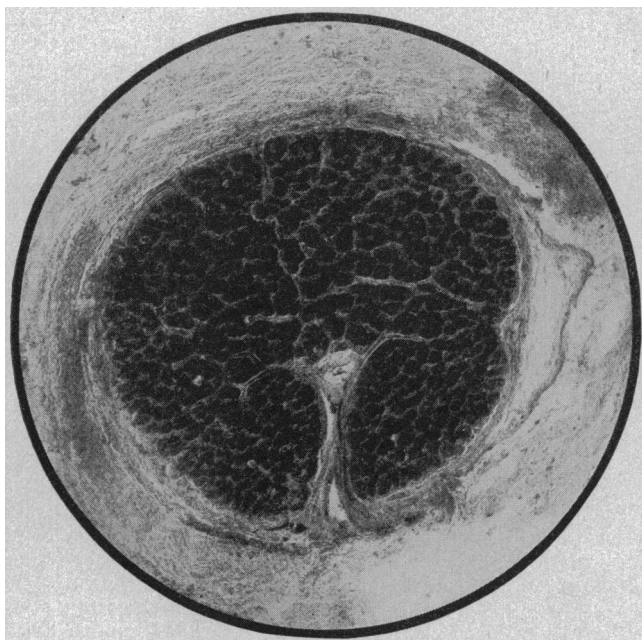


Fig. 2.—Transverse section of optic nerve at point of entrance of vessels. Photomicrograph by Dixon.

vous system fails to develop properly the finer degenerations must follow, and it is only a step further to the development of toxins due to errors in metabolism.

A word as to fixing and postmortem changes. I am of the opinion that the best solution for fixing the retina is Zenker's fluid, that Müller's or Orth's is next, and that formalin alone is the poorest of all. It depends altogether on the individual specimen whether or not the retina be detached by either of the fluids. As a matter of experience, formalin has appeared to be responsible for more detachments than the other fluids. I think we would encounter little trouble in this particular direction if it were possible to discard the use of alcohol. As to postmortem changes, Parsons says:²

The finer postmortem changes have been studied by Birch-Hirschfeld by Nissl's method, using thionin as the stain. The results obtained by this extremely delicate method must be accepted with caution, as they are only strictly comparable

when the minutest details of technic are kept constant. Post-mortem changes commence in the ganglion cells two hours after death. The perinuclear space becomes visible, and the Nissl granules become blurred at the edges, the smallest breaking up. In three and a half hours the nucleus shows vacuoles and commences to shrink, larger and more numerous vacuoles appear in the periphery of the cytoplasm. In five hours the Nissl granules have broken up and formed a dusty, diffuse stain, while the other changes have progressed. In seven hours the cells have been transformed into a mere granular mass.

Practically the same is said of the bipolars. In the same volume, p. 544, he says: "The chromatin diminishes with the exposure to light, and after prolonged exposure to bright light the Nissl bodies break up, vacuoles appear and the cells shrink."

Now, if these statements are correct we can not be too careful in drawing conclusions from the apparent finer details which are brought out by delicate cytologic methods, and it requires no great stretch of the imagination to assume that much of our painstaking labor may be rendered useless by minute changes occurring soon after circulation has ceased.

In conclusion, attention is called to the fact that the eyes in this case were removed and placed in the fixing solutions two and one-half hours after death. The only changes that have been detected are the swelling of the multipolar ganglion cells, the displacement of their nuclei, the retraction of the cell reticulum, the occasional disappearance of ganglion cells, and the general disappearance of Nissl's granules; the appearance of dark granules by Weigert's stain in all the ganglion cells, the peculiar formation of the macula and fovea, the so-called "spacing out" of the external reticular layer near the macula, and beginning simple atrophy of the optic nerve.

I am indebted to Mr. E. B. Burchell for valuable assistance in the preparation of this case.

THYMIC TRACHEOSTENOSIS, TRACHEOSCOPY, THYMECTOMY, CURE.

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Much of the literature of the diseases of the thymus gland is devoted to theoretical arguments as to the cause of "thymus death" (thymustod). A child dies suddenly with more or less stridor and cyanosis, and nothing pathologic but an enlarged gland is found at autopsy. The gland, however, does not seem to press on the trachea. The authors then speculate on theories as to pressure on the pneumogastric, pressure on the pulmonary artery, "hyperthymization of the blood," irritation of the large vessels and nerves by the friction of the to-and-fro movements of respiration, paralysis of the vasoconstrictors, status lymphaticus, etc. Theoretic arguments and anatomic demonstrations are brought forward to show the softness of the gland and the relative incompressibility of the trachea.

It has been my privilege, with the aid of the bronchoscope, to demonstrate beyond all doubt on the living patient the purely mechanical nature of "thymic asthma" in one instance. This, of course, does not prove that every case has this same pathologic mechanism, but it does prove the occurrence of that which many, including Friedleben, von Kundrat, d'Escherich and Paltauf, have denied, namely, that a hypertrophic thymus can

1. Peterson (Frederick), Hirsch (William), and Holden (Ward A.): *Jour. of Ment. and Nerv. Dis.*, 1898, xxv, 529-560.
2. *Pathology of the Eye*, ii, 546.

compress the trachea sufficiently to obliterate its lumen. It does disprove Friedleben's dictum—*es giebt kein asthma thymicum*—though it would seem more accurate to call it thymic tracheostenosis.

In view of my tracheoscopic findings, it is easy to understand sudden deaths from thymic hyperplasia, for we all know how suddenly the end comes in any form of tracheal stenosis and how impossible it is to get respiration started after it has stopped when there is even slight obstruction, unless tracheotomy be immediately done. And in these thymic cases a long cannula reaching below the obstruction would have to be inserted. With a tracheal lumen diminished to a chink, the slightest engorgement, as from a cough, would be sufficient to cause temporary swelling and momentarily to shut off the passage of air. The coughing and gagging during an examination would do the same thing. Once the air is shut off the usual phenomena of asphyxia are sufficient to engorge the thymus gland and all the vessels passing through the upper thoracic opening, which prolongs the obliteration of the tracheal lumen until death supervenes. Then the recession of the blood and the sagging of the viscera, together with the usual autopsic technic, would serve to allow the trachea to assume its normal lumen. Thus the pathologist finds the gland hypertrophied, but not compressing the trachea.

Patient.—E. L., aged 4, was admitted to the Western Pennsylvania Hospital, in the service of Dr. Ogden M. Edwards, Aug. 30, 1906.

History.—The child is the youngest of eight; one brother died a year ago with typhoid fever, another with tuberculosis. Two others died in infancy. The boy has always been well, except for a mild febrile attack a few months ago; he was not bedfast. Six weeks before admission he went to bed apparently well, but was awakened in the night by a sudden croupy attack. Since that time the difficulty in breathing has been growing steadily worse and is now very marked and stridorous. Dr. John W. Boyce, who examined the child, reported: "Child manifested considerable expiratory dyspnea of obstructive type, made worse by sitting erect. Good color and a fair general appearance. No signs of 'lymphatic constitution.' Pulse rapid but of good quality. Tympanitic note over sternum and at sides, extending farther to right than left. Over same area increased voice conduction, tracheal breathing and a whistling tracheal r le. Over rest of lungs normal puer-

ile breath sounds, the same on both sides. Diagnosis: Obstruction in trachea. Tubes dry; lungs normal."

R ntgen Examination.—I was called to the case and, finding the larynx free from obstruction, suspected a foreign body, and ordered a radiograph. A radiograph by Dr. R. H. Boggs showed the shadow of an enlarged thymus gland (Fig. 1).

Operation.—On September 5, assisted by Dr. J. J. Schoening, I opened the trachea under infiltration anesthesia. The rings seemed of normal resiliency, and the thymus gland was seen to extend abnormally high in the neck, reaching to the thyroid gland, and rising somewhat when the patient coughed. On passing one of my bronchoscopes (Fig. 2) I discovered a scabbed trachea with a chink of not over 2 mm. on inspiration and 1 mm. on expiration, the obstruction extending from the second to the fourth ribs. The tracheal mucosa was collapsed from before backward almost into contact. The collapsed walls opened up ahead of the tracheoscope not unlike the cervical

esophagus, and, like the esophagus, the trachea tended to close instead of to open on inspiration. The mucosa looked slightly congested, but was not edematous. The passage of the bronchoscope relieved the dyspnea instantly and when it was withdrawn no air entered and asphyxia threatened, so a long tracheal cannula was inserted in the wound, giving partial relief. From measurements taken with the tracheoscope I had made a special long tracheotomy cannula extending to within 1 cm. of the bifurcation. This gave absolute relief and was worn four weeks, until the operation on the gland could be done.

Postoperative History.—The day after the tracheotomy, discharge from the tracheal wound was very profuse. Numerous moist r les of large size were heard all over both lungs, but within a few days diminished, as expectoration of purulent material became more profuse. There was no bronchi-

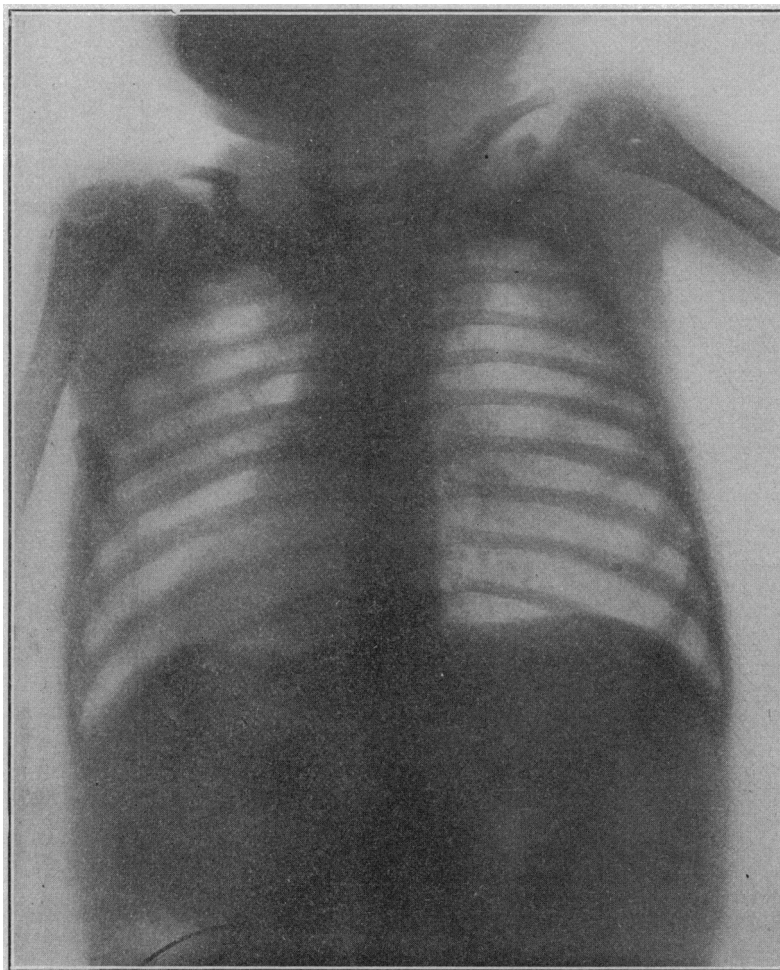


Fig. 1. Radiograph showing enlarged thymus gland.

tis (Boyce), the r les being produced by pus that had run down from the tracheotomy wound.

On September 8 the child developed a mild tonsillar diphtheria, for which he was transferred to an isolation cottage and placed under the care of Dr. G. W. Grier. The following day 3,000 units of antitoxin were given, and on the next day 1,500 more. By September 11 the membrane had almost entirely disappeared.

After a negative culture the child was released from quarantine on September 28, and was found by Dr. Boyce to have a mild bronchitis of the middle-sized tubes. There was some pallor and a rapid pulse. The abdomen was tympanitic and the stools were of foul odor. These intercurrent affections deferred the operation until October 2. During these four weeks the breathing was normal, except, of course, the usual stridor serraticus. September 28 a blood examination showed hemoglo-

bin 75 per cent. (Tallquist); red cells, 6,780,000; white, 18,000; polynuclear, 73; small mononuclear, 18; large mononuclear, 3; transitional, 8/10; eosinophile, 3 8/10.

Second Operation.—Assisted by Drs. J. H. McCready, J. B. Penrose and John W. Boyce, and with chloroform skilfully administered through the tracheal cannula by Dr. I. H. Alexander, on October 2, the second operation was performed. On removing the cannula for tracheoscopic examination, absolute stenosis followed, owing to collapse of the trachea. The tracheoscope showed a mild acute tracheitis, without edema or ulceration. The cannula was then reinserted. A curved transverse incision was made just below the upper border of the sternum to avoid the tracheal wound. The skin was retracted upward, and the incision carried throughout down to the thymus gland, the sternal attachments of both sternomastoids being severed. The thymus bulged into the wound with each coughing effort. I passed my finger down into the medias-

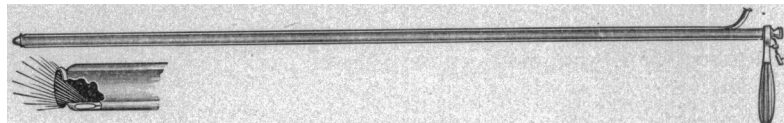


Fig. 2. Bronchoscope.

tinum, breaking up numerous fine fibrous attachments to sternum, care being taken not to wound the pleura or pericardium. The gland was gradually drawn up with tissue forceps until its tip, which was seen to be drawn in with each inspiration, was turned out of the wound. Several fibrous attachments were ligated and divided, and the lower extremity of the gland was ligated and removed. These manipulations with the finger in the mediastinum were rendered possible by the tracheal cannula, which prevented compression of the trachea; it was noted at this time that the lower tracheal rings were of normal resiliency. All ligatures were attached to the upper border of the sternum and one transverse stitch was passed through the pretracheal fascia and secured to the manubrial periosteum. The sternomastoid tendons were sutured into position and silk worm drainage was passed from end to end of the wound, following which the skin was sutured. A wet bichlorid dressing covered with rubber tissue was applied, and then a bib of oiled silk was placed under the tape holder of the tracheal cannula to try to prevent the coughed-up secretions from infecting the wound. The child stood the operation fairly well, but showed some weakening and rapidity of pulse, while the finger was in the anterior mediastinum.

Postoperative History.—"Patient passed a fair night. Pulse very rapid (over 150), but of good volume. Breathing rapid and irregular. Rattling of tracheal pus prevented satisfactory examination of chest" (Boyce).

The attempt to prevent infection of the wound by tracheal discharge proved futile, as the two wounds were less than 1 cm. apart. The thymic wound healed kindly by granulation, leaving a depressed scar. At each dressing bubbles of clear serum were seen exuding from the mediastinum on expiration, and a probe could readily be passed downward for a distance of 3 cm., but strange as it may seem, this cavity never became infected, which was due, I think, to dressing the wound with wet bichlorid gauze every three hours. This kept the entire wound free from pus. The tracheal cannula was removed on the eighth day after operation. The boy made a slow recovery, due, in my opinion, to the intercurrent affections rather than to the operation. He never showed the slightest stridor after the thymectomy. His last blood examination, three months later, showed: Red, 6,440,000; white, 12,480; hemoglobin, 85 per cent. He was plump and well nourished (Fig. 4).

Histologic Examination.—Dr. Joseph H. Barach examined the gland microscopically and reported as follows: "Section of the specimen showed it to be made up of about equal parts of thymus gland tissues and changed fibrous tissue. Half of the space occupied by the fibrous tissue was composed of hemorrhagic areas, and the lobe as a whole was enveloped in a fibrous tissue capsule under which there was some fatty infiltration. The lymphoid half was divided into lobules separated by fibrous septa; the lobulæ each contained on an average

about five corpuscles of Hassal. From this it would seem that the glandular enlargement was due, not to a marked increase in any one element, but that it was naturally large and undergoing the usual changes excepting the hemorrhage."

The upper thoracic aperture in this case was not narrower than normal, as was at first thought possible on account of the old fracture of the clavicle discovered on the radiograph. This fracture must have occurred prior to the child's learning to talk, that is, about two years before the tracheal wound.

The upper thoracic skeletal aperture in a child of this age is very small. Through it must pass the trachea, esophagus, thoracic duct, the innominate, left carotid, left subclavian, internal mammary and superior intercostal arteries, the right and left innominate and inferior thy-

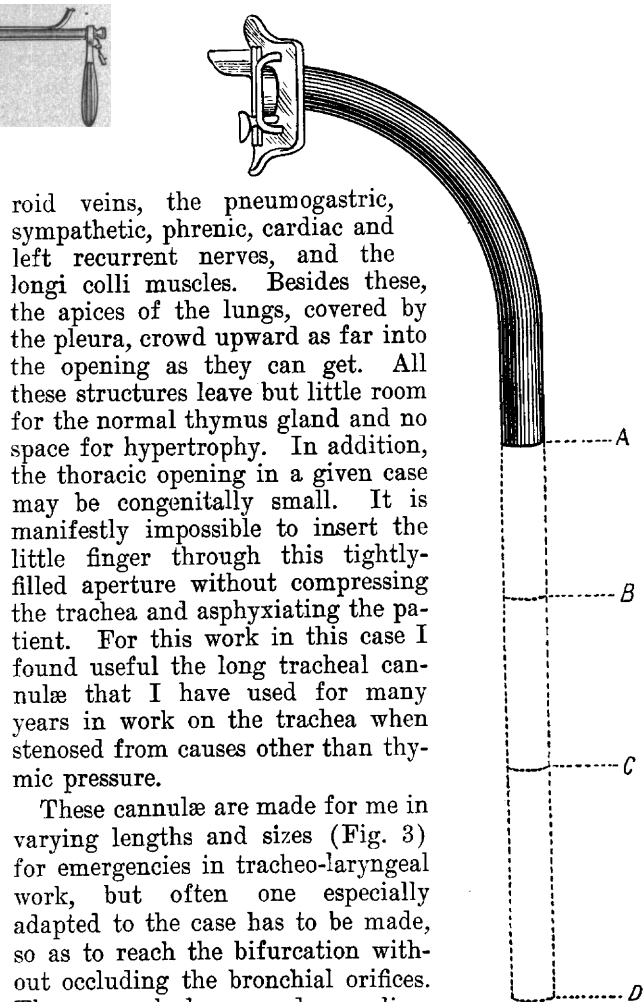


Fig. 3.—Tracheal cannulae of various lengths.

roid veins, the pneumogastric, sympathetic, phrenic, cardiac and left recurrent nerves, and the longi colli muscles. Besides these, the apices of the lungs, covered by the pleura, crowd upward as far into the opening as they can get. All these structures leave but little room for the normal thymus gland and no space for hypertrophy. In addition, the thoracic opening in a given case may be congenitally small. It is manifestly impossible to insert the little finger through this tightly-filled aperture without compressing the trachea and asphyxiating the patient. For this work in this case I found useful the long tracheal cannulae that I have used for many years in work on the trachea when stenosed from causes other than thymic pressure.

These cannulae are made for me in varying lengths and sizes (Fig. 3) for emergencies in tracheo-laryngeal work, but often one especially adapted to the case has to be made, so as to reach the bifurcation without occluding the bronchial orifices. These cannulae have saved many lives that must have been lost had the ordinary cannulae been used.

As to whether the pressure caused by crowding the finger through the already full upper thoracic opening caused a laryngeal spasm or not, I am unable to say, but it did not cause any laryngeal paralysis, as an examination the next day showed the glottic movements to be normal.

SUMMARY.

This is the seventh case on record of the cure of "thymic asthma" by thymectomy, the first case demonstrated radiographically and the only case in which the mechanical pathology of the disease was proved by direct tracheoscopic examination of the living patient.

Thymic tracheostenosis is a more accurate term than

thymic asthma and is acceptable, now that the mechanical nature of some cases has been proved. Let thymic asthma be applied to cases supposed to be associated with neuropathic, convulsive, lymphatic, rachitic, hemic or other pathology, if desired, until their exact pathologic mechanism shall have been demonstrated, as has been done beyond doubt in the tracheostenotic case herewith reported.

No patient with thymic tracheostenosis should die of asphyxia if a surgeon be at hand in time, with a very long tracheal cannula, bronchoscope or similar tube which will reach below the obstruction. All the patients who have died after tracheotomy seem from the reports to have died from the want of these long tracheal cannulae. These cannulae may be worn indefinitely, but a better procedure in thymic cases is to extirpate the thymus gland, partially or totally, or an exopexy may be done. The long tracheal cannulae prevent asphyxia from tracheal compression while the finger of the surgeon is passed into the mediastinum to break up adhesions and

4. An absolutely positive diagnosis can be made with the tracheoscope. Upper tracheoscopy is probably not safe in these cases. Tracheotomy should be done under infiltration anesthesia, and should be high, so as to be as far as possible away from the thymectomy wound.

5. A long tracheal cannula, reaching to within a centimeter of the bifurcation, renders the breathing free and the thymectomy safe from risk of asphyxia.

6. Thymectomy is indicated, and is best done by the insertion of the little finger from above downward behind the sternum through a transverse incision after double sternocleidomastoid tenotomy. The insertion of the finger should be of brief duration, as, though the patient with the long cannula inserted is safe from asphyxia, there seems to be serious cardiac inhibition, probably from compression of nerve trunks about the esophagus. One should be careful not to injure the pleura.

7. An almost complete thymectomy is without effect on either the blood or nutrition.

Special Articles

WATER SUPPLY AND PUBLIC HEALTH.

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(Continued from page 1671.)

CHAPTER IV.

THE SOLVENT ACTION OF WATER ON METALS.

Lead.—The development of chronic lead poisoning among the users of water which has passed through lead pipes is a well-known and a not very infrequent occurrence. It is also known that certain waters act more energetically on lead than others. In communities supplied with such waters, cases of chronic lead poisoning are occasionally very numerous. A celebrated case is that of towns in Lancashire and Yorkshire in England, that used water derived from peaty moorlands and delivered through lead pipes. The citizens of these towns experienced widespread and mysterious bodily derangements for some years, and it was finally discovered that throughout a considerable period lead poisoning had been prevalent among the inhabitants, although its real nature and cause had remained long unrecognized. In many other places (e. g., Somerfeld, Germany, and Lowell, Mass.), numerous cases of lead poisoning, due to the action of water on lead pipes have been reported. The cumulative action of lead on the system and the varying degree of individual susceptibility to its action conspire to render highly dangerous the use of water containing even small amounts of this substance.

It is undoubtedly true that some waters attack lead more vigorously than others, but all natural waters have some solvent action. Among waters that act with especial solvent power on lead are those containing oxygen or carbon dioxide in solution, oxygen being the more actively corrosive. The peaty acids, which sometimes give a strong reaction to waters from swampy regions, are strongly solvent for lead, and some of the most severe cases of chronic lead poisoning have been traced to waters of this character. It sometimes happens that waters coat the inside of lead pipe with a closely adhering film of mineral substance, but the degree of protection conferred by this deposit is somewhat problematical. In general, soft waters have a greater solvent action than waters containing considerable mineral matter in solution, but there are so many exceptions to this statement and the action of the various factors that determine the corrosive action is so complex that no general rule can be formulated. The only sure method of determining to what degree a given water is plumbo-solvent is by testing the question experimentally, and establishing the amount of lead actually taken up.

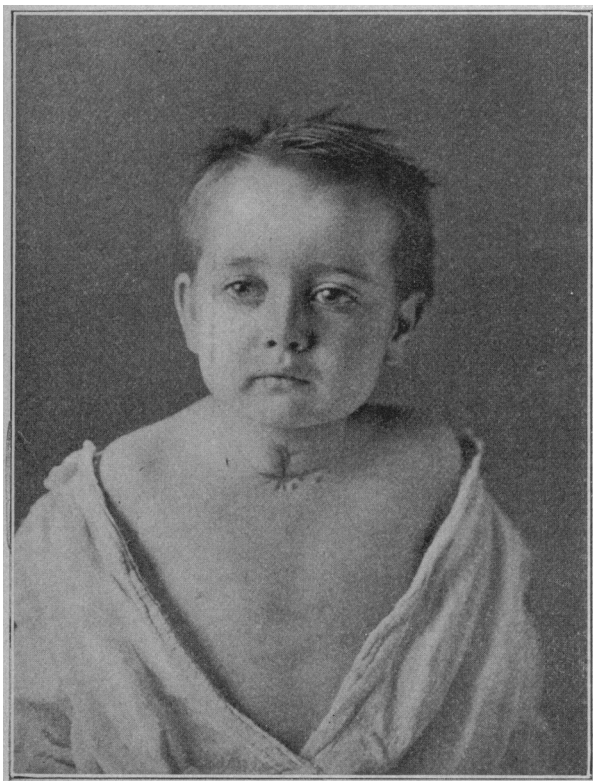


Fig. 4. Patient three months after operation.

remove the gland. This removal can not be done satisfactorily by pulling the gland upward with forceps, as the gland would be torn.

CONCLUSIONS.

1. Friedleben's dictum, *es giebt kein asthma thymicum*, is an error. The thymus gland in this case did compress the trachea sufficiently to diminish and to obliterate momentarily its lumen. Thymic tracheostenosis seems a better term for this class of cases.

2. The dyspnea in thymic tracheostenosis is worse in the erect position, and it is expiratory, as might be expected from the increased intrathoracic expiratory pressure, and as demonstrated tracheoscopically in this case. The mechanism of this was demonstrated by the flopping out and in of the elevated gland before it was severed.

3. A radiograph is a valuable diagnostic aid.