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TREATMENT OF STRABISMUS

By ROBERT R. MUSCHLITZ, M.D.

Early treatment of Strabismus, squint or "cross eyed" as it is sometimes called, is extremely important. To limit the length of this paper I will mention primarily the lateral deviations.

To consider squint we must first consider the function of the eyes.

There are four sensory components.

1. Useful vision in each eye.
2. Similar images in each eye.
3. Normal retinal correspondence.
4. High degree of awareness.

To elaborate: The eye must be able to resolve or have an image form on the retina, as a camera would, and the nervous mechanism to carry these stimuli to the brain.

The image must be quite similar in size and shape. For example, one quite far sighted eye and one quite near sighted eye can produce on the retina a 20% to 50% difference in the size of the image. Obviously such images cannot be fused. Likewise a diagonal astigmatism of high degree will produce slanted images, or images compressed in one axis, and if not the same in each eye will not be fused, or fused only with difficulty producting anisoconia. Frequently, in such cases, and in monocular aphacia, contact lenses will correct this.

These images must focus on corresponding parts of the retina. The image falling on non-corresponding parts produces a diplopia. If the macula areas correspond, then the other portions are out of the plane of the object being viewed and will produce a normal physiological diplopia which will help us determine the sensation of depth or distance.

The fourth factor, awareness is essential. All of us using the monocular microscope know how we learned to suppress the other eye, with only a slight degree of training. To secure binocular vision we must have a high degree of awareness in each eye, and little tendency to suppress.

There are also four motor components to the function of the eyes.

1. Focusing of the eye by the intraocular muscles to produce clear image.
2. Shifting of the visual axis to bring the eye to focus on an object.
3. Shifting of the visual axis of the eyes so they intersect.
4. Accommodation developing in relation to convergence.

The third function is to bring the visual axis of the eyes to intersect, these movements are called vergences. Convergence, divergence, cyclovergence and vertical vergence. These are sometimes called fusional movements.

This forms the background of studying squint cases.

How can we check the possibilities of squint?

Qualatively, if large in degree it is obvious. If small in degree there are several methods; also several pitfalls. The most frequent of the errors is that of the child with a prominent epicanthal fold which makes the parents think the child is cross-eyed. The cover and uncover test is the most convenient, especially to those not equipped to do a complete examination. This can be combined with noting the corneal reflex or Hirschberg reflex test. In doing this test, the physician secures the attention of the patient by having it look at a bright flashlight or something, then noting the position of each eye, and the position of the corneal light reflex. The examiner then covers and uncovers one eye, then alternately covers one and the other, noting the motion of the eyes, and the position of the reflexes.

Quantitatively we have the prism and cover test, in which we put prisms in front of the eyes until there is no deviation of the eye when covered and uncovered.

We can also use the perimeter directly and use either the Hirschberg reflex test, having the patient fix on one light with one eye, and carrying the other along the arm of the perimeter until the reflex is at the center of the pupil, and we can check this by cover and uncover at this time.

In all reflex tests the possible error introduced by the angle gamma (or kappa) must be considered.

This angle is due to the macula of the eye being slightly more nasally or temporally rather than at the exact posterior pole. This can give an appearance of divergence with a positive angle gamma or of convergence with a negative angle gamma when in each case there is no squint present. In these cases the cover test will show no motion on uncovering the eye and we can make our diagnosis.

In measuring squints we must consider the rotation of the eyes in the six cardinal directions to determine the muscles involved and whether there is a paretic or a comitant type of squint or a combination.

In taking rotations in the cardinal directions one can have the patient turn the eyes in the directions desired. However, by tilting the head in the directions one keeps the eyes toward the examiner and I believe the information easier to obtain.

The following is a case of left abducens palsy.

R. A., age 7. This boy had a paralysis from birth. Prior to the operation he presented a lack of deviation of the left eye to the left. On this boy I did a modified Hummelsheimer operation which consists of splitting the superior and inferior recti muscles, inserting the lateral parts at the insertion of the lateral rectus, transplanting the medial parts of the superior and inferior parts to the points of insertion of the lateral parts, advancing the lateral rectus, and recessing the internal rectus. It may be interesting to see his progress after operation:

One month later he had 5D L Hypotropia 15D Exotropia. 5 month P.O. he had 4D L Hypotropia, 4D Exophoria. 9 month P.O. he had no hypotropia, and no exophoria or esophoria at 20 feet. While he needed the hypotropia corrected, I corrected it with prisms in the glasses, but did not correct the exophoria. He now turns his eye quite well to the left and only in extreme left version does the left fail to turn as much as the right.

I would like to mention here in discussing suppression that he was not aware of diplopia when looking to the left beyond the point where the visual axis no longer intersected or intersect.

In comitant strabismus we find that such patients have misalignment of the visual axis and also some abnormalities of the sensory system such as suppression, amblyopia exanopsia and anomalous retinal correspondence.

When dissimilar objects are presented to corresponding retinal areas retinal rivalry exists, and suppression must result as I mentioned earlier when using the monocular microscope. Hence, when the eye is turned, or has a squint, suppression takes place. In many cases, where the refractive error is similar, there is alternation of use of the eye and alternating suppression. While the eye is deviating, we must note that suppression is helpful On the other hand, if, and when we desire binocular vision that suppression becomes very difficult to overcome. If one eye focuses easier than the other, then that one is used continuously and we have developing a greater suppression which becomes amblyopia exanopsia, or blindness from disuse. A panel of the American Academy of Ophthalmology and Otolaryngology has recently defined amblyopia exanopsia as "A reduction in visual acuity in one eye which sometimes may be reversed by appropriate means, and for which no ophthalmoscopicly demonstrable cause can be found."[2] It does not have to be due to strabismus but may also be due to marked difference in refractive errors. This panel "considers 20/40 vs 20/20 to be an am-

blyopia."[2] In other words it is the result or consequence of disuse rather than of inhibition, except that the inhibition results from disuse

While suppression prevents diplopia, another adaptive mechanism also prevents diplopia that is anomalous retinal correspondence or formation of a false macula. This is an attempt of the individual to restore binocular cooperation on the basis of an abnormal position of the eye. Ophthalmoscopically, one finds a normal appearing macula. The false area is never as sensitive as the macula, for physiological reasons with which you are familiar. In cases with well developed anomalous correspondence we have the so called paradoxic diplopia. In such cases when normal corresponding parts of the retina are stimulated diplopia results. I saw one such case in the Army in which one of the service men was operated, and with his eyes straight he projected the image to one side. This became gradually less but was still present nine months after operation.

This like suppression is a considerable obstacle to overcome when treating the patient to secure straight eyes and binocular fusion.

I have been discussing comitant strabismus. By this term we mean strabismus in which the angle of deviation is about the same regardless of which eye fixates though it many vary near or far and in different positions. The incomitant type are due to insufficient action or over-action of the muscles, and includes the paralytic type.

The physiology behind this is briefly: In infancy the fusion free position of the normal eyes is convergent, which changes to divergence with age.

Versions develop after the first month, and vergence not until three to six months. However, if vergences are excessive, they may be noted quite early, even at four to six months. By using a bright light to attract attention, any doctor by covering and uncovering the eyes can detect excessive vergence, and refer it for study. In my opinion this may be and should be done any time after nine months when it is noted, and even after six months if the child is normal or above normal intelligence. During the first four months accommodation is not possible, and the foveas are not fully developed. Accommodation develops slowly according to the degree of visual acuity which begins to approach adult level at two years.

In addition to excessive convergence of childhood, the infant usually has hyperopia, hence there is excessive accommodative convergence.

Someone may ask, why do some children with fairly high degree of hyperopia not squint while others with less do so? Why is there no correlation between the angle or squint and the amount of hyperopia?

The only logical answer is that the convergence mechanism is more or less sensitive in some than in others, and tonic convergence reflexes are more excessive in some, also the desire for single vision is more acute in some than in others.

In our examination we must remember that comitant strabismus may also be due to sensory disturbances, and we must not overlook these. Among these may be microphthalmos, coloboma of the macula, choroidal changes, congenital cataract, or marked difference in refractive errors.

The case of congenital cataracts which I reported in this Journal several years ago developed a stribsmus after removal of the first cataract, and became straight after I removed the second cataract.

Accommodative esotropia has the following characterstics:[3]

1.   Usually begins after age of two years, usually between two and four.
2.   Magnitude varies at first.
3.   Deviation seldom greater than 40 D.
4.   Near deviation is greater than far.
5.   Deviation has no relation to visual acuity of the eye.
6.   Majority have normal retinal correspondence.
7.   Hyperopia is greater than average of the non-squinting child.
8.   Majority correction of refractive error results in cure.
9.   Hereditary factor is about 50%.

J. P. – Age 2 years 9 month

History: Parents noticed eyes turning in for over a year. Left eye usually turned in, but at times the right.

Retinoscopy with mydriatic. + 6.00 — .75 x 180
+ 7.25 — 1.00 x 180

I prescribed the spherical equivalent as we call it, since it was the excess hyperopia which was our concern.

On checking the rotations, all were good, with the eye up and in when fixing with the other eye. This is seen normally and is due to overaction of the inferior oblique.

After prescribing the glasses we did patch the right eye continuously for two weeks, then the left for two weeks alternating for eight weeks. At the end of four months he was straight in the distance with Rx on, and no eso with cover test but on close 8D eso. He fused with Rx.

A year later I gave him + 5.50 — .75 x 180 both eyes and some prism base out to encourage fusion at all times. At a later date I will have

him use the stereoscope to overcome some of the remaining esophoria. Age itself will weaken the esophoria, as well as the hyperopia.

This next case is one of accommodative convergence excess.

J. M., female, age 11 when I first saw her.

History: At about two years of age her parents noticed that her eyes crossed. Went to ophthalmologist. Was refracted, and wore glasses which corrected her squint for distance, but she still converged at near, especially when tired. (During this time, prior to my seeing her, I would have used bifocals in such a case)

Unaided vision 20/20 and 20/20 each eye with or without glasses.

Was wearing O.D. +4.75 and O.S. +3.75 Spheres.

Her vergences were:

Without glasses Eso 44D at 20 feet 40D at 14 inches
With glasses 34D at 20 feet 30D at 14 inches

I gave her training on the stereoscope with graduated cards, until she could diverge 20D or more than I can, and I have a slight exophoria. A year and a half later, under cover and uncover test she still has 28D of Esophoria at 20 feet and 20D at 14 inches. But since she can diverge to 20D or a total of 48D of fusional divergence I would not consider operation. She can now go swimming without her glasses, though she uses them at all other times.

This case amply shows the advantage of conservatism as long as fusion can be maintained, and suppression or the like does not tend to develop. I will admit, that had I seen her at the age of five, with this amount of squint remaining after the accommodative factor was corrected with glasses, I would probably have advised operation. At that, I will say that I feel it would not have done any harm to have done so.

Concomitant Esotropia other than accommodative have the following characteristics:

1. Develop at or shortly after birth
2. The deviation is usually large, often over 40D
3. The angle of deviation is approximately the same for distance and near.
4. Fixation alternates, and hence amblyopia is rare.
5. The majority have anomalous retinal correspondence if not treated
6. Refraction parallels that of non-strabismus children
7. Correction of refractive error has no effect on the squint.

In the divergent deviations we have the same classifications and problems. These deviations can be sensory or accommodative. The accommodative exotropia is usually associated with myopia.

Here is a case of exotropia with myopic astigmatism which I would class as a sensory type.

P. E., male, 10 months. There is a convergence of between 20D and 40D.

The retinoscopy shows: OD –3.00 x 75   O.S. –3.00 x 95. This had to be corrected to secure sharp images on the retina. After wearing glasses for a month he became straight, though without them even at age three years, he does converge. Incidentally he does not want to go without them, and when broken he stayed in bed most of the day until they were repaired.

In Conclusion: The management of comitant strabismus has to do with the management of binocular vision. Like any conditioned reflex, the longer the improper reflex is permitted to develop, the more difficult it is to retrain; and if well developed, for example by age of four to eight years, it is almost impossible to retrain, and operation can only produce cosmetic results.   The emphasis must be in preventing the formations of suppression and other adaptive responses by treating the patient as soon as possible, even at the age of six to twelve months.

This is done by:

1.   Proper refraction (by careful retinsocopy under mydriatic) and by supplying glasses to overcome the accommodative factor.

2.   The non-accommodative factor usually requires surgery.   Braley[4] believes in operation as soon as the diagnosis is made.

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RIGHT PARADUODENAL HERNIA

By E. JOHN STAHLER, M.D. and
CLIFFORD H. TREXLER, M.D., F.A.C.S.

A case of right paraduodenal hernia is presented because of its rarity and because it was correctly diagnosed by X-ray preoperatively.

A paraduodenal hernia is a condition in which all or part of the small intestine is incarcerated behind the mesocolon and the entrance of the incarcerated intestine is in the area of the duodenjejunal junction. It is the most common of all the internal hernias. Two types of paraduodenal hernia occur, a right and a left and the former is more rare in the ratio of one to three.

Anatomically a right paraduodenal hernia has the opening of its sac facing toward the left with the superior mesenteric artery on the anterior border of the neck. The left paraduodenal hernia is open toward the right and has the inferior mesenteric vein in the neck of the sac anteriorly.

There is a voluminous amount of literature on this comparatively rare hernia. The lesion was first recorded 175 years ago and described in detail by Treitz 100 years ago (15). Up to 1939, as reported by Collins,[7] 200 cases of left paraduodenal hernia had been reported in the literature, 62 on the right, with a mortality rate of 25% upon the operated cases.

The etiology of these hernias is obscure. There are numerous theories as to etiology, but in general there are two schools of thought: (1) that they are acquired and (2) that they are congenital. The point is definitely not settled. The earlier theory of Treitz contended that a loop of small intestine insinuated itself in one of nine described fossae at the duodenojejunal junction and that owing to variations of intra-abdominal pressure the fossae becomes enlarged until a retro-peritoneal hernia is formed. In 1923 Andrews[1] developed a more widely accepted theory in that the abnormality occurs during the third stage of return of the intestinal tube from its temporary extrusion into the yolk sac — in the the 5th to 11th week of embryonic life.. If the cecum is incompletely rotated during the 10th week when the intestine returns to the abdominal cavity, it would be inferior to the small intestines and as it grows to the right in its normal path of rotation it could partly or completely imprison the small intestine behind the mesentery of the colon. The same type of explanation is given for left paraduodenal hernias. A slight reverse

rotation would be present at the return of the small bowel, with the cecum to the right and small bowel to the left. As the colon gains in length and forms a loop the small bowel would be imprisoned behind the mesentery of the descending colon. Callender[5] and his associates believe these hernias are the result of an evagination of an area of the descending mesocolon by the jejuneileum in the 10th or 11th week of fetal life, when the descending mesocolon was mobile. He does not believe faulty rotation is necessary.

Preoperative diagnosis of a paraduodenal hernia is very rare. Many patients have no symptoms, and the anomaly is only found at autopsy. There may be sudden onset of symptoms indicating an acute intestinal obstruction. Bile is usually present in the vomitus, fecal vomiting would be unlikely because of the usual high level of obstruction. Most cases give an indefinite history of colic like abdominal discomfort with or without vomiting, and an excess of borborygmi and gasseous eructation may be present. This may progress to the clinical picture of acute intestinal obstruction. At physical examination there may be some eccentric distention of the abdomen with a vague soft to firm mass palpable and auscultation may reveal obstructive bowel sounds.

The diagnosis is usually made by roentgenograms, and before 1933 only 2 cases (Auerbach)[2] of 40 right paraduodenal hernias had been diagnosed pre-operatively. The roentgen diagnosis is being made with increasing frequency. Parsons[15] in 1953 reported 12 new cases all diagnosed by X-ray examination. This leads one to believe there is an increasing awareness of the condition. X-ray findings are described in detail in Exner's[8] paper. The typical findings are that the intestines appear as in a bag, the coils cannot be displaced and the axis is to the right of the midline. The encapsulated loop shows coarsening of the mucosa, dilatation and stasis. If only a small amount of intestine is herniated the rest of the small bowel shows normal distribution.

At operation practically all the small bowel may be present in the sac. If so, no free small bowel will be seen and instead a smooth peritoneal mass will be encountered. The sac is usually opened in an avascular area and the neck of the sac located from within and the bowel may be able to be returned to its normal position. If only a small amount of bowel is present it may reduce very easily. Enlargement of the neck of the sac may be difficult due to the location of the superior mesenteric artery in its anterior border and so any enlargement would have to be downward. In late obstruction cases most of the small bowel may be

(11)

gangrenous. The mouth of the sac is usually closed although some writers claim that closure is not necessary and recurrence extremely rare (Giles).[9]

CASE REPORT

A 32 year old white female was admitted to the hospital on July 11, 1954 complaining of right side abdominal pain of at least six months duration. She first noticed it in January when she was bending forward. The pain was intermittant, colicky, and associated with considerable gasseous eructation, borborygmi and nausea. There was no relation to types of food or to meals. Discomfort was present daily, but during the two weeks before admission pain was severe. She had several episodes of watery stools during this time. There was no weight loss, jaundice or evidence of G.I. bleeding. During this time the family doctor felt she exhibited signs of partial intestinal obstruction or perhaps cholecystitis since some tenderness was present in the R.U.Q.

Past history was negative except for an uncomplicated appendectomy 15 years ago and biopsy of the left breast five years ago.

The relevant physical findings were that of an asthenic type female in no acute distress, with apparent elevation of the right side of the abdomen showing active visible peristalsis that did not cross the midline. The mass which was compressable was apparently bowel and some tenderness was present in the right colic area. High pitched bowel sounds were present.

Initial laboratory work, including urinalysis, complete blood count, blood sugar and BUN, were essentially normal except for moderate elevation of polymorphonuclear cells. The clinical working diagnoses were (1) partial intestinal obstruction, possibly due to adhesions or (2) regional ileitis.

Chest X-ray and barium enema were negative. X-ray of the stomach and duodenum revealed an unusual position of the jejunum. The duodenal loop was not normal and did not approach the ligament of Treitz. The jejunum was grouped in the upper right abdomen and could not be displaced. The barium progressed normally toward the ileum. A diagnosis of a right paraduodenal hernia was made. It was felt that exploration was warranted due to the prolonged history of partial obstruction although the patient had no acute symptoms at this time.

Operation: Under endotracheal anesthesia a right upper paramedian incision was made. On entering the peritoneal cavity the stomach and

(12)

transverse colon appeared to be in normal position, however above and to the right side of the abdomen a mass of small bowel presented itself with a thin peritoneal covering. This mass was located over the right kidney, beneath the liver with the transverse mesocolon anterior. Inspection of the small bowel from a point beneath the transverse mesocolon was then done. The ligament of Treitz appeared to be to the right of the midline. Just distal to the origin of the jejunum an opening was found in the mesentery through which several loops of jejunum had entered and presented in the upper right quadrant beneath the previously mentioned peritoneal covering, with the opening having the superior mesenteric artery on its anterior margin. The neck of the hernia was not tight and was about two inches across with the opening facing to the left, and so by simple traction the hernia was easily reduced. After reduction of the hernia, simple over and over suture of the opening of the sac was performed. Approximately 1/3 of the small bowel was present in the sac. The patient had an uneventful recovery and has been seen twice at follow-up examinations and is asymptomatic.

The prognosis in general of a high obstruction in an acute case varies with the condition of the patient and the state of the circulation of the involved intestine. The mortality of the operation in acute obstruction was previously very high, but more survivals are being reported (Giles).[9]

COMMENT

The case presented had rather typical X-ray findings of paraduodenal hernia. In retrospect it could have been possible to diagnose this internal hernia on physical examination since the history suggested partial intestinal obstruction and with a soft compressable mass in the upper right abdomen and visible peristalsis limited to this area. The operative procedure was simple. The high mortality in the event of acute obstruction made diagnosis of this condition desirable in the prodromal stage. There were no problems in the operative correction of this hernia.

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A MEIGS' LIKE SYNDROME OCCURRING WITH FIBROMYOMA OF THE UTERUS

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Meigs' Syndrome has been defined as being limited to cases with a solid tumor of the ovary such as a fibroma, teratoma, granulosa cell tumor or Brenner tumor. These are associated with ascites and hydrothorax, followed by cure after removal of the tumor. The lesion should be made up predominantly of fibrous tissue and it is obvious from statistics that fibromas of the ovary are much more commonly associated with Meigs Syndrome.

In a recent article Meigs[1] states that some leiomyomas of the uterus are accompanied by abdominal and chest fluid. It would be interesting to know why these are few since some of the pedunculated variety are not very different in position and make up from the fibroma of the ovary. He further states that if all such cases associated with ascites and hydrothorax were included in the Meigs syndrome, the group would be confusing since there would be too many divisions. Heart disease, renal failure, pancreatic cysts, pancreatic injury, cirrhosis of the liver, cancer and other lesions would greatly confuse the syndrome that appears to be so definite.

Meigs says that there is nothing distinctive about the laboratory findings in the true syndrome. In no case were tumor cells found in the chest fluid. The specific gravity of the fluid varies but is usually in the transudate level. The serum protein in the fluid is definitely low, ranging from 5 mg. per 100 cc or lower. The color of the fluid is usually amber, yellow or colorless and the cell count is low.

There are a number of theories as to the source of the fluid. The best explanation so far is one suggested by Gibell, Miller and Rubin.[2] They state that these tumors (fibromas), by their very nature excrete fluid through their own lymphatics and always show edema between their cells. The covering of the fibroma is only one cell thick. It often shows areas of cystic change, or necrosis, due to pressure from the inside. This pressure can squeeze fluid from the edematous tumor and secrete it into the abdomen. These fibromas after removal have been placed in dry containers and in 24 hours they will have secreted a large amount of fluid. This, then, may be the reason for ascites in the true

syndrome. The effusion in the chest, most likely is due to the flow of fluid through the lymphatics of the diaphragm or through small openings between the cells of the diaphragm. That the current of fluid is from the abdomen to the chest has been proven by placing india ink in the abdomen and later recovering it in the fluid in the chest.

Meigs' final conclusion is that it is important to recognize this syndrome since if the diagnosis is not made patients who are considered hopeless may be curable.

From an article in the American Journal of Obstetrics and Gynecology, May, 1954 [3] by Dr. Meigs some of the historical background is available concerning this syndrome. Speigelberg in 1866 first reported a patient who died without operation. She had swelling of the abdomen, a fluid wave, and at autopsy showed left pleural effusion. Associated with this was pain, anorexia and emaciation. Her fibroma weighed 60 lbs. and measured 51 x 46 x 23 cms. Many of the early investigators reported ascites and hydrothorax associated with cystic tumors of the ovary, but this is not the true Meigs syndrome. There are lesions other than those already mentioned which may simulate the Meigs syndrome. In a series of 122 cases reported by Meigs, 84 were typical cases of Meigs syndrome, 69 were reported as fibromas, 8 as Theca cell tumors, five as granulosa cell tumors, one as a Brenner tumor. There were 16 cases of benign overian cysts, five patients had leiomyomas of the uterus and there were two teratomas of the struma thyroid type. There was one papilloma of the Fallopian tube and one case had a pelvis so confused with inflammatory process that the pathologist did not know what to call it, tuberculosis was suspected.

In summary, again from Meigs' article, there are four groups of patients that may have fluid in the abdomen and the chest. After the removal of the tumor the fluid will disappear. These are (1) the true Meigs' syndrome, (2) benign cysts of the ovary, leiomyomas of the uterus and teratoms; (3) malignant lesions of the ovary with peritoneal seeding; and (4) cysts and traumatic injuries to and carcinoma of the pancreas. Diseases of other organs such as that of the heart, kidney and liver, certain diaphragmatic and inflammatory lesions will cause fluid in the abdomen and the chest. These, however, cannot be cured by removal of any pelvic tumor. Thus Meigs states that is seems best to include only the solid tumors of the ovary as the true Meigs syndrome. The case which we will present from the Allentown Hospital must be entitled A Meigs' Like Syndrome Associated with a Leiomyoma of the Uterus.

CASE PRESENTATION

The patient was a 43 year old white female who is a gravida III para III. She was admitted to Allentown Hospital on March 23, 1954. Her chief complaint was menorrhagia, with dysmenorrhea of one year's duration. The onset of illness was insidious, about one year prior to admission. Her menses occurred every 28 days, flowing for seven days and requiring from 6 to 8 pads per day, associated with passage of clots. Her last normal menstrual period prior to admission started March 19, 1954. She had had metrorrhagia for four months prior to admission. Her past medical history is essentially negative. On symptom review positive findings were exertional dyspnea. The patient had been sleeping in the orthopneic position for three months. She had no ankle edema, no cough or hemoptysis. She had nocturia occurring 4 to 5 times a night. There was stress incontinence present with pruritis vulvae.

The family history was non-contributory. There was no history of carcinoma, tuberculosis or diabetes in the family

Physical examination revealed a pale, well hydrated, cachectic appearing white female in moderate respiratory distress sitting in the orthopneic position. She was 5' 5" tall and weight 142 lbs. Examination of the head, eyes, ears, nose were not revealing. The chest showed flatness to percussion bilaterally posterior extending up to T8. The breath sounds were absent below T8 with decreased absent vocal and tactile fremitus. The heart did not seem enlarged clinically. The P.M.I. was in the fifth interspace within the midclavicular line, the supra-cardiac dullness was normal and there were no murmurs. Rate was 88 and the rhythm was regular sinus rhythm. The abdomen was pendulous with slight flaring at the flanks. The liver was not palpable and the impression was that a fluid wave was obtainable.

Pelvic examination: External genitalia were multiparous and otherwise negative. The cervix was situated anteriorly and the corpus uteri was anteverted and enlarged, firm and globular to the size of about three months pregnancy. This was slightly mobile and non-tender.

Laboratory studies on admission revealed a Hgb. of 48% with an RBC of 2,560,000 WBC — 8,500 with 59% polys. There was hypochromia with slight anisocytosis and poikilocytosis present. A catheterized urine specimen showed a light cloud of albumin with six white blood cells and 20 RBC. The serum albumin total was 3.19 total protein 6.35 with an A/G ratio of 1/1. The hematocrit was 27; cholesterol 135 mgs.%. Urea was 15.2 and the prothrombin time 100%.

Due to this patient's orthopneic condition on admission, a cardiac consultation was ordered immediately. A note by the hospital cardiologist was as follows: "The patient has been dyspneic as presented in the history. This had increased in severity in the last two weeks. There is no history of edema. The apex beat is near the midclavicular line with no thrills or murmurs. There is some dullness of both bases with poor transmition of breath sounds. The abdomen is too distended to palpate the liver. In conclusion, the pulmonary findings are not probably due to cardiac origin. I do not know the etiology". The electrocardiogram was normal. Chest X-ray confirmed the presence of fluid in both lung cavities. Two days after admission, this patient was subjected to a thoracentesis and 850cc of clear straw colored fluid was removed from the right chest. The laboratory studies of this fluid revealed a negative smear. The protein was 3.3. gms. The specific gravity was 1.010 and the cell count was 100 WBC differential being 60 polys and 40 lymphocytes.

This patient was placed on a low salt diet and was started on 500 cc of whole blood daily plus Feosol grs. 5 four times a day and Ascorbic Acid 100 mgs. four times a day. She was weighed daily and given thiomerin 1/2 cc daily. On March 27th her RBC was 3,220,000. By now it was found that the fluid in the chest was re-accumulating very rapidly. During the period March 28th to March 31st the patient received 500 cc of whole blood daily and on April 3rd her hemoglobin was up to 90% with RBC of 5,600,000 and the patient was scheduled for surgery. The night before operation the thoracentesis was repeated in order to give this patient the maximum pulmonary capacity. On the right side 800 cc of clear straw colored fluid was again obtained and 300 cc from the left side. Laboratory studies on this fluid were similar to the first. At operation a large myomatous uterus about the size of a 3 months gestation was found. Both ovaries were normal and there was some ascitic fluid. A total abdominal hysterectomy and bilateral salpingo-oophorectomy was performed.

The patient withstood the operative procedure very well. Her postoperative course was uneventful. A chest film on April 12th revealed a slight fluid level at the base of the right lung. On April 14th the patient was discharged from the Allentown Hospital.

The x-ray films included in this report show the admitting film and the final film. The final film was taken May 26, 1954 six weeks after the patient was discharged. There has been no re-accumulation of fluid. At present the patient is in a very good state of health with no clinical signs of re-accumulation of the fluid. This is a case of leiomyoma

of the uterus, bilateral hydrothorax and ascites. This patient was first considered and properly so to be a fundal carcinoma, after cardiac disease was ruled out, but the fluid found to be in her chest was a transudate. Thus metastatic carcinoma was not a likely diagnosis since. Even with correction of the anemia and hypoproteneimia, the fluid re-accumulated rapidly prior to removal of her pelvic tumor and it did not recur after its removal.

In conclusion, this case is one of a Meigs-like Syndrome associated with fibromyoma of the uterus. According to Meigs' latest classification this cannot be considered in any sense a true Meigs Syndrome, however it is very gratifying to be able to find a patient who appears so chronically and seriously ill with pleural effusion and ascites and have her respond so remarkably well to a surgical procedure. We wholeheartedly agree with Dr. Meigs when he states that the value of knowledge of this syndrome is to cure the apparently hopeless looking case.

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A DIABETES SCREENING PROJECT

STANLEY E. ZEEMAN, M.D. and RICHARD W. JACKSON, M.PH.

Screening projects for the detection of diabetes have been conducted widely throughout the United States. It has been estimated that the results of such surveys will yield from 0.5% to 1.0% identification of previously unknown diabetics among the adult population. (1)

Under the auspices of The Lehigh Valley Diabetes Association and with the co-operation of the Lehigh County Tuberculosis and Health Society such a screening program was instituted at the Allentown County Fair from Sept. 16, 1956 through Sept. 22, 1956. The purpose of the survey was three fold: (a) As a public health measure for the detection of previously unknown diabetes; (b) To stimulate interest in the detection of diabetes among the population of the Lehigh Valley; (c) To determine if the time, effort and money required to conduct such a survey is justified on a periodic basis in terms of the number of previously unknown diabetics discovered and brought under treatment.

METHODS

The survey was conducted at The Allentown Fair, a county fair attracting people from a large surrounding area of both urban and rural communities. There were no rigid restrictions regarding age of the persons studied, but those under 30 were not encouraged to participate. Those individuals who stated they were known diabetics were discouraged from participating but were not excluded completely if they insisted on a blood test. Individuals who stated they had previously been diabetics but were no longer receiving medical care and those who stated they were classified as possible diabetics were encouraged to participate.

Capillary blood was screened by the Wilkerson-Heftmann method with the aid of the Hewson Clinitron machine. Persons who had eaten no food for 1 hour prior to the test were screened at 130 mgm/100 cc but if food had been eaten within the past hour, the screening level was 180 mgm/100 cc.

Persons who screened positive were mailed notification of the result of the test and urged to visit their family doctor for more definitive study. At the same time, the family doctor of the individual was also notified of the result of the test. One month after notification, the family physician was asked to fill out a questionnaire stating whether or not the person had visited, and if so, the tests used to establish or rule out a diagnosis of diabetes and the results of such tests. The

methods employed by various physicians in establishing a diagnosis of diabetes varied. The physicians opinion as to whether or not diabetes existed was accepted regardless of the method used. Those classified by their physicians as possible or potential diabetics were included in the statistics as negative.

RESULTS

The results of the survey are summarized in Table I. 1,250 subjects were tested, but 23 of the tests were unsatisfactory leaving a total of 1,227 on which this report is based. Of these, 101 (8.2%) screened positive and were referred to their family physician for diagnosis. Reply was received on 89. 26 (29.25%) of the 89 individuals on whom we received a follow-up were found to have diabetes. 17 of these individuals had previously been known to have diabetes and 9 were previously unknown diabetics.

The 26 cases of diabetes detected represents 2.1% of the 1,227 individuals screened. The incidence of previously unknown diabetes (9 cases) was 0.7% of the total population screened.

The number and percentage of those screened positive by decades is given in Table II. Generally, the older age group the higher the percentage of cases who screened positive and also, the higher the percentage of those subsequently found to have diabetes. The small number of cases studied in the older age groups, above the age of 70, makes statistical comparison impossible for these groups.

When the incidence of positive screening tests is correlated with sex, no significant difference is observed although there is a slight preponderance of males (9.6%) who screened positive compared to females (7.5%). On the other hand, the incidence of subsequently proven diabetes was higher among females (2.1%) than among the males (1.5%).

The incidence of positive screening tests among those who had a family history of diabetes was slightly higher (9.5%) than those with no family history of diabetes (7.7%). Likewise the yield of proven diabetes was higher among those with a family history of diabetes (2.1%) than those without (1.7%).

DISCUSSION

The primary aim of a diabetes screening program is to detect previously unknown cases of diabetes. In this respect, the present study uncovered 9 cases of previously unknown diabetes, or 0.7% of the total number screened. This figure is similar to those obtained in other

(21)

	Total		130 mg. %		180 mg. %	
	No.	Percent	No.	Percent	No.	Percent
Number Screened	1,227	100%	547	43.8%	680	56.2%
Previously Known Diabetics	34	2.8%	18	3.3%	16	2.4%
Screened Positive	101	8.2%	61	11.2%	40	5.8%
Previously Known Diabetics	18	1.5%	9	1.7%	9	1.3%
Referred to M.D. for Diagnosis	101	8.2%	61	11.2%	40	5.8%
Reported by M.D.	89	7.2%	55	10.1%	33	4.9%
Diabetes Diagnosed	26	2.1%	14	2.6%	12	1.8%
Previously Known Diabetics	17	1.4%	9	1.7%	8	1.2%
Previously Unknown Diabetics	9	0.7%	5	0.9%	4	0.6%

TABLE I

studies conducted throughout this country. Generally, the rate of discovery of previously unknown diabetics varies between 0.5% to 1.0% depending upon the methods used and the incidence of diabetes in the population studied. Since an attempt was made to exclude known diabetics the results reported cannot serve as an index to the incidence of diabetes in the general population in this area. 34 individuals with previously known diabetes however, were included in the study, either because of their insistence to be tested or because they deliberately withheld the fact that they were diabetics in order to be tested. That this is so, is indicated by the fact that 12 individuals who screened positive stated that they were not known diabetics; but when the report was received by the family physician, the presence of previously known diabetes was indicated.

That the sensitivity of the methods employed in this study were far from perfect is indicated by the fact that 16 individuals screened negative who stated they had diabetes. These individuals were not taking insulin at the time of the study. It is possible of course, that many of them were controlled by diet or that their diabetes was very mild. The exact number of false negative screening tests is not known since we have already demonstrated that the subject's statement about whether or not he has diabetes cannot be completely accepted and also because information was not requested from the family physicians of those individuals who screened negative. Kurlander, etal (2) also found the sensitivity ratings low in this type of screening study.

The laboratory procedures used by various physicians to determine the presence of diabetes in those individuals who screened positive varied considerably. Some physicians used a fasting blood sugar to determine whether diabetes was present. Others were satisfied with a urine examination while still others used the glucose tolerance test. Unless standard procedures are used to determine the presence of diabetes in accordance with modern knowledge, screening procedures such as this lose considerable measure of their potential value.

In any screening study a primary objective is to keep the number of false positive and false negative results at the irreducible minimum. By using two blood levels depending upon the interval after the last meal, we hoped to achieve this objective.

The proportion of individuals screening positive was almost twice as high among those tested at 130 mg.% (11.2%) as compared to those tested at 180 mg.% (5.8%). Despite this, the number of cases of diabetes subsequently diagnosed was not proportionately greater in those screened at 130 mg.%. 2.6% of those screened at 130 m.g.%

Age	No. Screened	Screened–Positive		Diabetes–Detected	
		No.	Percentage	No.	Percentage
10 to 19	5	0	—	0	—
20 to 29	16	0	—	0	—
30 to 39	324	19	6%	4	1.2%
40 to 49	294	23	8%	4	1.4%
50 to 59	322	32	10%	6	1.9%
60 to 69	210	21	10%	7	3.3%
70 to 79	46	6	9%	5	10.9%
80 to 89	5	0	—	0	—
No age given	5	0	—	0	—
Totals	1,227	101	3.2%	26	2.1%

TABLE II

were found to have diabetes compared to 1.8% in those screened at 180 mg.%. Since there was a slightly higher percentage of previously known diabetes in the 130 mg.% group, this difference becomes even less striking. It is obvious therefore, that capillary blood tested at 130 mg.% one hour after food intake is too sensitive a test for a screening program where a large number of false positives is not desired. Future studies using a less sensitive test for the longer postprandial intervals would probably sacrifice little specificity. In this regard, it has been suggested by Wilkerson that projected surveys using capillary blood be conducted using a determination of 160 mg.% if over three hours since food intake and 180 mg.% if less than three hours has intervened. (3)

It should be remembered that in evaluating the results of this study, there is a difference of 20-30 mgm.% between the capillary and venous blood levels during the first two hours after eating. Generally, capillary blood contains a higher percentage of glucose than does venous blood. The high incidence of false positive reactions recorded at 130 mg.% is understandable when transposed to figures for venous blood. This would mean that individuals, who had their last meal more than one

hour before the test, were screened positive at levels over 100 to 110 mgm.% for venous blood.

While the primary purpose of this type of screening procedure is to uncover unknown diabetes, the total value of the project exceeds this limited objective. The dissemination of knowledge about diabetes can only be fostered by conducting such a program in a community. The spread of knowledge regarding diabetes even to those who did not participate in the testing but who were made aware of it, would be expected to bring many individuals to their doctors for periodic examinations. Those who screened negative on the study reported herein would probably be more receptive to periodic testing in the future. Even the physicians in a community will be encouraged by the response of their patients to suggest and perform periodic tests for diabetes more often. Furthermore, individuals with previously known diabetes who had neglected periodic testing and who screened positive on the survey were made aware of the realities of their illness and brought back under the supervision of their physicians.

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APPRECIATION

FRACTURE-DISLOCATION OF TALUS
— EXCISION AND REPLACEMENT

KENNETH R. WESTON, M.D., F.A.C.S.

A case of fracture–dislocation of a talus with excision and replacement is presented because of the difficulty encountered in attempting both closed and open reductions.

CASE REPORT

R.L., a 20-year-old, white male, was admitted into the hospital on 8-23-52 with an injury to the right lower leg and ankle following a motor accident. He was a passenger on a motorcycle which was involved in a collision with an automobile.

On admission into the hospital, he was conscious and complained of pain in the right lower leg and ankle. He was found to have abrasions of the right lower leg and foot with extreme swelling and deformity of the ankle. Temperature was 98.6°; pulse – 88; respiration – 20; blood pressure – 132/80. He did not appear in any acute systemic distress and was well oriented and cooperative. Examination of the right foot revealed considerable swelling and cyanosis over the area of the medial malleolus. Six hours after admission, he was referred to the Orthopedic Department where immediate closed reduction was attempted. X-rays obtained on admission revealed a fracture-dislocation of the talus with complete posterior displacement of its body.

Under general anesthesia, the abrasions of the right lower leg and foot were toileted. There was no evidence of compounding at the fracture sites. The wounds were dressed and an attempt at reduction carred out by manipulation with the leg at a right angle and traction on the os calcis and counteraction on the thigh. This was not successful. However, following attempt at closed reduction, the circulation of the foot improved. A posterior mold was applied. The following day, under spinal and pentathol anesthesia, open reduction was effected. The dorsalis pedis artery was palpable prior to surgery and the foot was warm. It was impossible to palpate the posterior tibial vessels because of the intense hematoma and swelling. The fractured area was approached through a six-inch Henry type incision over the postero-medial aspect of the ankle joint. Portions of the flexor digitorum

communis and the flexor hallicis longus tendons were found severely torn and the entire body of the talus was found lying outside the joint under the muscle plane. It was impossible to reduce the talus and further exposure was necessary. The Achilles' tendon was severed — using Z-plasty procedure and severing through the deltoid ligament and detaching it. By strong inversion of the foot, the ankle joint was completely exposed and the fragment of the neck and portions of the head were found floating in the joint. A large portion of the neck was replaced; and after removing the body of the talus, this was replaced into its normal position in the joint. The joint appeared stable; thus, no internal fixation was used. The wound was closed in layers, instilling a half-million units of penicillin into this area. A long leg padded plaster cast was applied with the foot in equinus and the knee at 20 degree flexion. He received 500 c.c. of whole blood during this procedure. He was given a prophylactic dose of T.A.T. and was placed on 800,000 units of Abbocillin, b.i.d. until 9-3-52.

His post-operative course was uneventful. The wound was healed by primary intention. He was discharged from the hospital on 9-17-52 using crutches. Cast change and x-ray studies were carried out at various intervals. A walking iron was applied on 2-5-53. Plaster immobilization was employed until July 1953.

When last seen on 1-6-55, he stated he was steadily employed. However, he changed his job from roofer to automobile mechanic. Pain, tiredness and soreness of the foot were present toward the end of day at times. He walked with a moderate limp. This, he stated, became worse in the evening after being on his foot all day. The ankle joint was moderately thickened. The foot was held in slight equinus. He stated it was almost impossible to walk without a shoe having a heel. Motions of the ankle were restricted — dorsi-flexion being restricted to 95°; planter flexion at approximately 120°; and subastragalar motion was almost completely restricted.

Comment: Subtalar fusion was considered as was a stragalectomy at open operation. However, it was felt that he may have revascularization of the fragment and also there would be no shortening if the talus could become viable and that disability would be less than if one of the procedures such as pantalar or subtalar arthrodecis or astragalectomy was performed.

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Ankle on admission — fracture dislocation.

Post-operative result following open reduction.

Last x-ray of ankle.

THE SIALOGRAM AND THE PAROTID GLAND

By EDWARD WEINER, D.D.S.

The parotid gland is the largest of the three pairs of the major salivary glands. Each parotid gland lies in the space packed between the mastoid process and the ramus of the mandible. It overflows onto the face below the zygomatic arch from which its duct (Stensons) runs parallel to and just below the zygomatic arch running across the masseter muscle piercing the buccinator muscle to open into the buccal vestibule of the mouth opposite to the maxillary second molar tooth.

Histology of the Parotid Gland

The parotid gland is enclosed in a well defined fibrous tissue capsule and is considered a compound-tubulo-alveolar gland of a serous type. It is characterized with many and prominent intralobular ducts. Accumulation of fat cells in the connective tissue septa is also characteristic of this gland.

Fig. 1
Structure of tubulo-alveolar gland.

The sialogram is a roentgen visualization of the larger salivary glands such as the parotid, and the submaxillary glands. This article deals mainly with the parotid gland, since its disfunction and disease leads to surgical problems which are fraught with facial nerve injury partial or complete, temporary or permanent. Esthetically and psychologically this type of postoperative sequelae is fraught with tragedy and unhappiness on the part of the patient. Because of this disfiguring complication to parotid surgery, it is advisable that a preop evaluation, by every means we have, be done. Such a preoperative evaluation is a sialogram.

This study will demonstrate the morphology of the gland on film by means of a radiopaque substance as lipiodol. The main duct, the duct tree, and the parenchyma of the gland can be studied, thereby aiding in the differential diagnosis of diseases of this gland including stones, or other malfunctions of this gland. This study may decide on surgical or nonsurgical treatment.

(29)

Indications for this study:

1. Demonstrate the relationship of the normal parotid gland and duct to other adjacent structures.

2. Aid in demonstration of foreign bodies and calculi.

3. Demonstrate salivary fistulae.

4. Demonstrate strictures of the duct.

5. Confirm clinical diagnosis of chronic parotitis.

6. Determine whether the tumor in the parotid gland is encapsulated, or whether the tumor is infiltrating the duct system and gland.

7. Determine the proximity of the retromandibular process of the gland to a mixed salivary tumor in the tonsillar region.

8. Aid in the planning of an operative procedure in the parotid area, thereby differentiating tumors from chronic inflammation in the parotid gland.

Technique

The patient is placed in a dental chair in the sitting position. Anesthesia used may be local, however general anesthesia is most desirable during the probing of the duct, and the injection of the lipiodol. Preoperatively intravenous meperidine with intravenous atropine sulphate are used. The anesthesia was intravenous sodium pentothal. The patient is kept in a light plane of anesthesia. The X-rays are taken as the patient responds to commands. It is important to note that since the main duct or auxiliary ducts may be partially blocked with thick serous exudates, the addition of intravenous atropine sulfate is a good adjunct in the easier passage of the Lipiodol from the cheek back and down to the gland proper.

Once the patient is anesthetized it is much simpler to probe Stensons duct since there is no movement of the patient due to pain. Dilatation of the main duct whose orifice is in the cheek until a canula approximating a twenty gauge needle can be easily inserted. A twenty gauge needle (blunted) is easily inserted into Stensons duct, and 2-2½ c.c. of lipiodol is inserted downward and backward into the parotid gland. The gland will be seen to expand on completion of the procedure. The injection of the lipiodol should be done slowly and carefully. Extreme care should be exercised so as not to cause injury to the oriface in the cheek resulting in scarring and stricture of the oriface in the cheek.

Roentgen Examination

Prior to injecting the lipiodol solution, an occlusal plane lateral jaw, and antero-posterior films should be taken. These films will be compared with the films taken postoperatively following the sialogram.

Once the lipiodol solution has been expended into the parotid gland repeat the occlusal plane, lateral jaw, and antero-posterior films. If it is possible take lateral jaw films in sterioscopic views.

After Care

The patient should be instructed to eat or drink foods which stimulate the flow of saliva. This will aid in the washing out of the opaque substance in the gland and the duct system of the parotid gland. This procedure using this radiopaque iodine solution may in itself help reduce some residual infection in the gland. The gentle massage of the gland may stimulate the flow of the lipiodol out of the duct system and the gland. Normally, the complete discharge of this solution takes several days.

Evaluation of Normal and Abnormal Sialograms
Normal Sialogram*

Reveals a duct running almost horizontally backwards sometimes slightly downward from the oriface in the buccal mucosa. The main duct then divides and subdivides producing a tree like effect. In the

antero-posterior view it is oftimes possible to outline the retromandibular lobe of the parotid and to determine how close to the pharyngeal wall it lies. This is a very important evaluation for frequently tumors are found in the pharyngeal wall lateral to the tonsils. Also, at times, large tumors extend from just beneath the skin back of the mandible and push the pharyngeal wall medially.
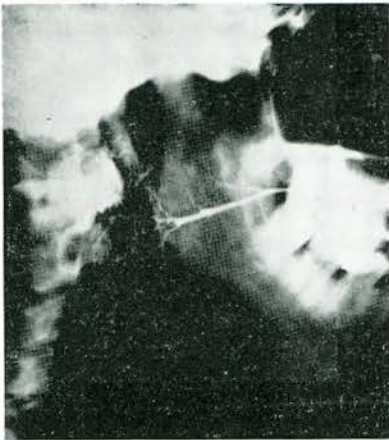
Fig. 2
Normal Sialogram

Abnormal Sialogram

In cases of chronic parotitis there is sacculation or puddling of the duct, accessory ducts within the gland resulting in a snowflake appearance.

Tumors-benign

In cases of benign tumors of the parotid gland, the tumor is circumscribed and therefore any filling of the defect in the duct tree will be regular due to the displacement of the ducts surrounding the tumor. Each duct is visualized throughout its entirety and the configuration of the defect varies with the size of the tumor. The main duct may be displaced or there may be considerable displacement of various ducts in the gland depending on the location of the tumor.
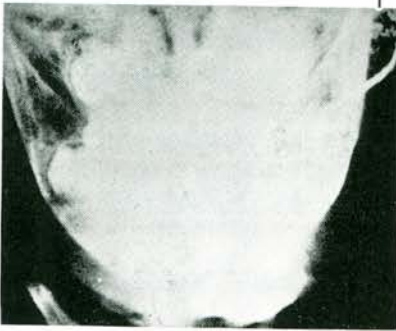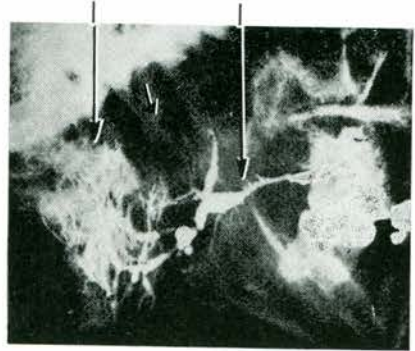

Fig. 3 Puddling of the duct.

Fig. 4 Benign tumor, constriction of parotid duct.

Tumors-Malignant

Since Carcinoma and other malignant tumors are invasive, the various ducts are destroyed in an irregular fashion. One cannot visualize the parotid duct system in its entirety. The main duct system is abruptly stopped, and there may be irregular filling defects in the duct system. Also there may be localization or puddling of the opaque solution due to erosion of the ducts and their distortion by the invasive malignant process.

SUMMARY

The sialogram and its careful evaluation is an extremely valuable diagnostic aid in Parotid Gland surgery. Since this surgery is fraught with catastrophic esthetic sequela due to facial nerve injury partial or complete, temporary or permanent, it behooves the surgeon to use this preoperative diagnostic aid in his overall evaluation of Parotid gland disfunction and disease.

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