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SPEAKING OF NASAL SINUS OPERATIONS.*

By J. W. JERVEY, M. D., Greenville, S. C.

"Speaking of Nasal Sinus Operations" may sound rather Irvin Cobbish, but I can re-assure you that I am not going to give you my personal experiences as a patient. So far I have not been a victim of any of my rhinological brethren. I shall attempt to recount to you, in a general way, my observations covering these conditions as they have presented themselves to me in nearly thirty years' practice in a climate not very dissimilar from your own.

First of all, you note, of course, that I refer to "climate" in this connection. Why shouldn't I? Those of us who are engaged in this work in the more rigorous northern and western climates evidently see many more, and much more violent, cases of this sort than those with which we in the "Sunny South" come into contact. The same is true in mastoid infection—and, after all, this mastoid is virtually but another, though somewhat segregated, nasal accessory sinus.

To begin at the beginning (which is the proper place to begin even in such as discursive discussion as this promises to be): Why is sinus disease? And we shall, by mutual consent, throw out of consideration the so-called "catarrhal sinus", and confine ourselves to actual sinus infection.

In October, 1925, the American College of Surgeons held its annual meeting in Philadelphia. I was invited to open the discussion of Joseph C. Beck's paper on nasal sinus infections (I have forgotten the exact title of the paper). In the course of his remarks he stated that tonsil infections almost invariably followed sinus infections. In my discussion I mildly suggested that he may inadvertently have put the cart before the horse, for it has been my observation that in little less than one hundred per cent of the sinus cases I have seen in thirty years' practice there was either present, or *had been present*, a pre-existing tonsillar infection. (This is, of course, exclusive of those antrum cases of frankly dental origin, and those which seem definitely to be sequelae of influenza—though most of these latter will be found to have existent or pre-existent tonsil infection).

Furthermore, my experience has taught me that it is rather likely to lead to a foolish feeling, if one tries to eradicate a sinus infection and leaves infected tonsils "as is". Think that over! Every time the unfortunate proprietor of infected tonsils coughs, or sneezes, or blows his nose, a hurricane of pus-saturated air breezes through his post-nasal and nasal channels. Why wouldn't the sinus outlets gather in their share of it? And, remembering your anatomy, which of the nasal sinuses are most likely to bear the brunt of this polluted air attack?

Most of this vicious current is gathered in by the overhanging posterior end of the middle turbinate and is swept directly across the hiatus semilunaris where are the mouths of the frontal, ethmoid and maxillary sinus orifices. The sphenoid orifice is on the posterior wall of the nasal chamber. The effect of the blast is rather to create a sphenoid vacuum and cleanse this sinus, without direct contact of the impregnated stream with the sphenoid orifice. But the others suffer from the contact in varying degree. The frontal has a long downward drainage trend, and is not often implicated. The ethmoid has a downward and forward drainage trend, but it is a much shorter route; while the maxillary antrum has its yawping, dangerously placed, orifice ready to take in any stranger that comes its way. These are anatomical facts. What are our pathological observations?

The antrum is by far the most frequently infected sinus. I believe we are all agreed on that point. Next, certainly in my observation, comes the ethmoid labyrinth. Then comes the frontal, and, finally, and seldom seen in our climate, the sphenoid. Exactly the order we might expect in consideration of the mechanical formation and forcible expulsion of infection from behind—that is, from the pharynx up to the posterior choana and forward.

I do not for a moment deny the influence of

*Read by invitation at the eighth annual meeting of the Virginia Society of Oto-Laryngology and Ophthalmology, in Charlottesville, Va., April 30, 1927.

the teeth. Sufficient pressure is applied to effect a perceptible "blanching" of the gingival tissues. As the pressure is released there is a rapid inrush of blood causing a marked "blushing." This act is repeated several times about all the teeth. The result is a cleansing of the teeth and a distinct massage to the soft parts. It supplies the highest degree of cleanliness and health to the teeth and surrounding tissues.

I have presented a great many details relating to the causes, pathology and treatment of pyorrhea at the risk of becoming tedious to a body of physicians, but they are facts essential to a discussion of this subject because the minor details are the small irritants that are "like unto the mustard seed, which is the least of all seeds, but when it is grown it is the greatest among herbs and shooteth out great branches."

Conclusions

(1) The term periodontoclasia appears more appropriate than any other title for describing diseases of the investing tissues of the teeth.

(2) The term pyorrhea is more generally used in describing these conditions.

(3) The manifestations of this disease are to be seen in almost every adult mouth.

(4) Its importance as a focalized infection is generally conceded.

(5) Its successful treatment depends upon early recognition.

(6) Early recognition is dependent upon careful co-operation of clinicians and roentgenologists.

(7) It can often be diagnosed by a clinical examination alone, but its extent can never be determined or proper operative treatment applied without an X-ray examination.

(8) An X-ray examination will frequently show evidence of pyorrhea which cannot be seen upon clinical examination.

(9) An X-ray examination alone will sometimes fail to show an active pyorrheal process.

(10) The safest method of examination is a combined X-ray and clinical examination.

(11) In the prevention as well as in the treatment of pyorrhea, correct brushing of the teeth and gums is the core of the whole subject.

INFECTIOUS JAUNDICE.

By WYNDHAM B. BLANTON, M. D., Richmond, Va.

Infectious jaundice is a disease that has been recognized as a clinical entity since 1849[1]; a disease that has assumed epidemic proportions both in war and in civil life, where individuals have been forced to live among insanitary surroundings. It is likewise a disease that occurs sporadically, and as such is probably encountered more frequently than is generally supposed. Whether we are justified in classifying infectious jaundice into a single, or into several separate categories remains to be seen, and awaits the final demonstration of the infective agent in the case of epidemic icterus, and doubtless also in those cases of so-called catarrhal jaundice. It seems likely from what is already known of this whole class of icteric diseases that the common etiological organism when demonstrated will be a spirillum.

Since Schaudinn demonstrated the *Treponema pallidum* (Syphilis) in 1905, the number of infectious diseases recognized as due to spirochetes has steadily increased. Under this heading are now classed the *Spirillum of Vincent* (Vincent's Angina), *Spirochaeta Obermeier* (Relapsing fever), *Spirochaeta morsus mura* (Rat bite fever), *Spirochaeta pertenuis* (Yaws), *Leptospira icteroides* (Yellow fever) and *Leptospira icterohemorrhagiae* (Weil's disease).

I. Spirochaetosis Icterohemorrhagica (Weil's Disease)

Weil's disease was first recognized by Larrey and Ozanam (1849). It was first described accurately by Weil (1886)[2]. The first demonstration of the causative organism was made by Inada, (1913)[3], and since then it has been successfully cultured on artifical media, using the method by which Noguchi for the first time grew the treponema of syphilis.

This type of infectious jaundice occurs widely throughout the world. It was recognized among the troops on every battle front in the recent war—French, Japanese, Canadian, British, Rumanian and Italian. Epidemics of it have been studied and described in Japan,[4] especially in mines where the adjacent soil is alkaline. Sporadic cases are everywhere met, but in this country very few instances are on record in which the causative organism has been isolated. In January of

this year, Hayman[5] reported the fifth case. Cushing[6] has recently demonstrated it in two cases in Bellevue Hospital, New York City.

The Japanese,[7] who have added most to our knowledge of this disease, describe it as presenting three phases. *First*—Febrile stage, 7 days. There is a sudden onset with chill, fever, injected eyes, herpes, hemorrhages, headache and muscle pains. There is albuminuria, enlargement of the liver and tenderness of the abdomen. Organisms (leptospira) occur in the blood, liver, spleen and adrenals. No immune bodies are demonstrable. *Second*—Icteric stage, 7th to 13th day. Jaundice usually begins on the fourth day. There is a peculiar greenish tint present in the skin. The pulse is slow, itching and other symptoms of jaundice exist. The leptospira now leaves the blood stream, liver and adrenals. It is still present, however, in the kidneys and urine. Immune bodies can now be demonstarted in the blood. This is the period during which death, if it occurs, is to be expected. *Third*—Convalescent stage, 13th to 16th day. Jaundice begins to fade. Anemia and wasting are now important features. Immune bodies reach their height in the blood serum. Organisms are present in the kidneys and urine.

CASE REPORT.

A university student, aged 20, presented himself because of jaundice of three weeks' duration. He had taken part in a swimming meet and almost immediately became suddenly and acutely ill. He entered the University of Virginia Hospital as a suspected case of grippe. Headache, fever, pains in the back, nausea and vomiting were distressing and continued off and on for the first week. He had no appetite and was quite constipated. His temperature is said to have reached 104° F. On the second day of his illness he became jaundiced, his stools were observed to be light and his urine dark. There were no hemorrhages.

Examination three weeks after the onset showed that he was still markedly jaundiced, though he was up and about. His temperature was normal. The stools were light though not clay colored. The urine was dark and showed bile. There were both albumin and casts in the urine. The icteric index was 75. Several duodenal taps got quantities of dark bile. The liver and spleen were not palpable. He continued to be constipated, showed little appetite and lost weight. The blood serum was sent to Dr. Noguchi, of the Rockefeller Institute, who found that it completely destroyed leptospira icterohemorrhagiae, establishing the diagnosis of Weil's disease.

On the twenty-eighth day of his illness he began to complain again of headache, loss of appetite, constipation and general malaise. His temperature rose to 101°-102° F. After one week in bed he became markedly better, the jaundice began to clear and five weeks after the onset he was able to return to his work. Though still jaundiced, the urine was bile free. At the time of his second febrile reaction, urine was injected intraperitoneally into a guinea pig. This pig did not develop jaundice and has remained well.*

Correspondence with Dr. H. B. Mulholland, of the University of Virginia Hospital, who saw this patient when he was first taken ill, disclosed the fact that blood withdrawn at that time was injected into guinea pigs from whom, he reports, the leptospira icterohemorrhagiae was isolated. This was an interesting verification of Pfeiffer's reaction already demonstrated in the blood serum by Dr. Noguchi. Dr. Mulholland plans to report this case and his work upon it during this stage in a separate paper. His findings were made entirely independent of the subsequent history.

II. EPIDEMIC INFECTIOUS JAUNDICE

Jaundice of an epidemic nature occurred in 22,509 soldiers during our Civil War. There were 161 deaths in this number. 5,648 cases were reported as occurring in the South African war. In 1917, in Mesopotamia alone, there were 4,171 cases. Colonel W. H. Willcox[8] has admirably reported this epidemic in his Lettsomian Lectures on jaundice. There have been civilian epidemics of jaundice in the United States since 1812. As Blumer's[9] studies showed, practically every state in the Union has been affected. There were small epidemics in Norfolk in 1812, at Halifax Court House in 1856, and in Richmond in 1860. In 1922 Williams[10] reported an epidemic of 300 cases among school children in New York State. Symmers[11] and Witt[12] both described small outbreaks from Bellevue Hospital.

Willcox from an enormous experience gives the symptoms as follows: a prodromal period of abdominal discomfort, anorexia, nausea, occasional vomiting, diarrhea and constipation. The onset is marked by a mild pyrexia and chilliness. The tongue is furred. There is vomiting, headache and "pains all over". About the fourth day jaundice gradually begins to appear. The liver, spleen and right heart are enlarged. After a mild course—usually three weeks—the patient is convalescent. Severe and fatal cases were occasionally encountered. Yellow atrophy of the liver was noted in a few of the fatal cases. The mortality was far below that of Weil's disease—0.4 per cent as against an average mortality of 30 per cent. No organisms were found after elaborate and thorough laboratory investigation of the Dardanelles' epidemic. Other workers in similar outbreaks have been unsuccessful in establishing the causative organism.

*Inada denied the occurence of relapses. He speaks instead of after-fevers due, he thinks, to disintegrating toxins at the height of immunity. He bases this on the fact that at this time there is no return of the main symptoms and the blood is not infectious.

A small epidemic of infectious jaundice was recently encountered in Richmond school children. Three children, each eight years old, occupying adjacent seats in a school room, simultaneously developed a similar train of sypmtoms, the prominent features of which were anorexia, languor, mild gastric and intestinal complaints, slight temperature, enlargement of the liver, jaundice, clay colored stools and bile in the urine. After one week all of the stools again showed the presence of bile. A study of the urine sediments under the dark field revealed no spirochetes. Guinea pigs were inoculated intraperitoneally with 6 c.c. of fresh urine from each case during the first week. The pigs were bled from the heart after forty-eight hours and the blood serum searched under the dark field. No organisms were found. All of the pigs survived. After ten days the urine was again inoculated into guinea pigs, also with negative results.

Three weeks after the development of these three cases, other cases began to appear. In the home of one of the children a younger brother and sister each developed the disease, the only other brother escaping. Another of the original cases infected her younger brother. The third did not infect any of his three brothers and sisters, who were older than he. At this time three other cases developed, one in the same class room, two others from a different class. In all, eight cases of jaundice appeared, three simultaneously; and three weeks later, five others who were definite contacts. Six of these cases were seen and studied by us. It is to be noted that home contact between school children and children who were not in school was a prominent feature of this outbreak. The disease is evidently carried from person to person, though in this instance the contact was usually an intimate one—those first affected sat side by side in school and the secondary cases were brothers and sisters. The great majority of the other children in the same school room were unaffected. These observations are at variance with the assertion of Willcox that "the epidemic character appeared due to a common cause rather than a spread from person to person". He asserted that instances of infection from patient to patient in the hospital were not common.

CASE REPORTS.

Case 1. An eight year old boy, who had been perfectly well except for tonsillitis a month previously, became listless, constipated and lost his appetite. The next day he complained of pain in the epigastrium and nausea, and vomited moderately. Jaundice of the skin and mucous membranes, clay colored stools and dark bile-containing urine were noticed. Pule 72. Temperature 99.5° F. The liver margin was firm, but not tender, and extended one inch below the costal border. After two days, most of the symptoms subsided, but jaundice, palpable liver and characteristic stools and urine continued for one week. On the seventh day a distinct yellow color began to show in the stool.

Case 2. A previously healthy girl of eight years was given calomel by her mother for constipation. She appeared well for a week. Her mother then kept her in bed because she refused food and was again constipated and very languid. There was no pain, nausea or vomiting. No symptoms referable to the head, heart, lungs, kidney or nervous system. Examination disclosed moderate jaundice of the mucous membranes and skin. The liver was palpable one inch below the costal margin, not tender. Spleen not felt. Slight elevation of temperature. Physical examination otherwise negative. Urine contains considerable bile, stool light grey. After several days symptom-free, except for jaundice. Color returned to the stools after one week.

Case 3. A boy of eight was noticed to be pale, irritable, and without appetite. He was put to bed after having a large foamy loose stool. No temperature. Next day he had no complaint except languor. No pain, nausea, vomiting or other symptoms. On the second day jaundice of the skin and mucous membranes was noticed. Urine dark and stool light. Spleen and liver negative. Remaining physical examination negative. After one week color began to return to the stool.

Case 4. A boy of seven had a stomach upset, with foul breath and headache. Three weeks previously his sister had had jaundice. He temporarily improved and returned to school. At this time he showed no alteration in the color of his skin, stools or urine. One week later the conjunctivae became violently inflamed and fever developed—102° F. was the highest. No nausea or vomiting, no pain in abdomen. Anorexia. Constipated. Twelve days after onset, urine became dark. Parents had not noticed any jaundice. For a few days had had a cough and a head cold. Examination disclosed an acutely ill boy with slight jaundice of the skin and mucous membranes. Temperature 99.5° F. Pulse 110. The nose and throat were slightly red. The heart and lungs were normal. The lower margin of the liver could be felt one inch below the costal border, not tender. The spleen could not be felt. The urine showed bile. Icteric index was 15. The urine showed albumin but no casts. Blood count—W. B. C. 6,000; R. B. C. 4,130,000; Hgb., 74%. A guinea pig into whose peritoneum 5 c.c. of blood was injected, survived symptom-free.

Case 5. A boy four years old (brother of one of the first cases), was awakened at 5 A. M., vomiting phlegm. In bed all day, no temperature. Next day better. Next day had pain in his abdomen, which was relieved by enema. Temperature 102.5° F. Large foamy stool. Two more later. No temperature next day. Coughing, pain in stomach, no appetite. Three days later, dark urine (6th day). Next day vomited, pain in abdomen, no temperature, jaundiced, bile in urine. Next day stool yellow with brown streaks. Progressively better, but urine still dark on the 11th day of the disease. This case occurred three weeks after the outbreak of the contact case.

Case 6. A girl three years old (sister of one of the first cases). Severe attack of urticaria of one day's duration. Pain in abdomen, vomiting. Temperature 101° F. Two days later much pain in the abdomen, distention, belching. Temperature 103° F. Mucus in stool. Three days later, urine dark. On the 4th day, no temperature. On the 8th day, suggestion of clay colored stool. Slight jaundice noticed the same day. Bile in urine. Very yellow on the 9th day. Same day color began to come back in the stool. After the 12th day, much better. This case also occurred three weeks after the outbreak of the contact case.*

Etiology

Epidemic jaundice is recognized as contagious. A soldier sleeping in a barn with seven or eight other soldiers has been known to infect them all. However, we know neither the organism nor the method of conveyance.

Rats have been suspected of being the most important factor in the spread of Weil's disease. In Japan (40 per cent), in America (10 per cent), and elsewhere rats have been shown to harbor pathogenic leptospira icterohemorrhagiae in their kidneys, and to excrete them in their urine. Victims of Weil's disease are thought to become infected through contact with rat contaminated soil, (bare footed miners), or from articles of food to which rats have had access. These findings "seem to be particularly important in revealing a latent danger to which we have been constantly exposed, but from which we escape as long as sanitary conditions are not disturbed by untoward events." No contact with rats, however, could be shown either for our case of Weil's disease or for the eight cases of epidemic jaundic reported. Emulsions of the kidneys of a number of Richmond rats were injected into guinea pigs with negative results.

Several of our cases were tested for immune bodies. The serum of the case of Weil's disease completely destroyed leptospira icterohemorrhagiae. The sera of three of our other cases were likewise tested by Dr. Noguchi. None of them reacted to leptospira icterohemorrhagiae, but they did react to a leptospira recovered from ice box water. It was not possible to collect drinking water for examination to which our cases had access. These appear to be very suggestive results. As said, no one has yet demonstrated the organism of epidemic jaundice. A tremendous amount of work was done on it by the British during the recent war, with negative results. Dr. Noguchi's findings certainly indicate the need for further studies in epidemic jaundice, dark field examinations of blood and urine, and the inoculation of guinea pigs in all stages of the disease.

*Cases No. 7 and No. 8 were seen by Dr. J. O. Fitzgerald and presented similar symptoms.

Table I

A Comparison of Epidemic Jaundice and Weil's Disease Based on the Cases Here Reported.

Epidemic Jaundice		Weil's Disease
One week	*Duration*	Six weeks
Unknown	*Etiology*	Leptospira icterohemorrhagiae
	Immune Bodies	
For leptospira from ice box water		For leptospira icterohemorrhagiae
Intense and sudden	*Jaundice*	Slight and gradual
None	*Hemorrhage*	None
None	*Splenic Enlargement*	None
Moderate	*Hepatic Enlargement*	None
One week, slight	*Fever*	One week, high
Light	*Stool*	Variable
One week	*Bile in Urine*	Five weeks
6-18	*Icteric Index*	75
Absent	*Casts and Albumin*	Present
None	*Relapse*	One
0.4%	*Accepted Mortality*	30%

Epidemic Jaundice vs. Weil's Disease

A comparison of the two diseases here reported brings out the following differences. In our cases of epidemic jaundice the duration was one week, the icterus was of gradual onset and mild, the fever was slight, the urine was negative, the liver was enlarged and there was no relapse. No organisms were found though the sera reacted with a leptospira. On the other hand, in Weil's disease the duration was more than five weeks, the jaundice came on suddenly and was very intense, the urine showed casts and albumin, there was no enlargement of spleen or liver, a relapse occurred after four weeks, leptospira icterohemorrhagiae was demonstrated in the blood (Dr. Mulholland) and later the convalescent sera reacted to the Rockefeller strain of leptospira icterohemorrhagiae. The difference in mortality between the two diseases should be noted—0.4 per cent for epidemic jaundice against 30 per cent for Weil's disease.

III. Catarrhal Jaundice

Cocayne[15] believes that catarrhal jaundice is the sporadic or endemic form of epidemic jaundice. The French, Brulé[16] in particular, hold to the same view, regarding catarrhal jaundice as an infectious disease due to alteration in the hepatic cells and not the result of obstruction. Eppinger[17] and also Stadelmann[18] showed intrahepatic mucous, bile-stained plugs blocking the bile-canaliculi, and claimed to find them only in non-infectious icterus. The old idea of a mucous plug obstructing the ampulla of Vater is not tenable. Autopsy findings do not substantiate it and clinical experience, with gall-bladder drainage through a duodenal tube, shows the bile usually flows freely in cases of catarrhal jaundice. The occurrence of catarrhal jaundice in adolescence, the immunity conferred by one attack, the similarity of its symptoms to infectious jaundice, the occasional development out of it of Icterus Gravis, or acute yellow atrophy of the liver, point to an infectious disease involving the finer ducts and cells of the liver in varying degrees. This theory is not really in conflict with Eppinger's description of mucous plugs in the ducts, because such accumulations may easily form as a part of a diffuse cholangeitis.

There is need for more study of these very interesting outbreaks of icterus. Leptospira may be a comparatively recent invader of the human body as Noguchi suggests. As it gains in virulence by repeated animal passage, we may expect more serious consequences and a higher mortality. The relatively high mortality among the Japanese is explained also by Noguchi on these grounds. These spirochetal infections doubtless occur about us more frequently than we realize. It is important to know that though the organism may not be always demonstrable, the serum reaction for immune bodies can be done, and is as informing as the Widal reaction in typhoid fever.

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ON HEMORRHAGE AS A COMPLICATION OF TONSILLECTOMY.*

By WALTER A. WELLS, A. M., M. D., F. A. C. S., Washington, D. C.

There is probably no kind of medical information which it is so important that we should possess, in an accurate and complete form, as that which concerns the surgical emergencies; for its province is not the indefinite interwoven boundary between health and disease or between disease and death, but rather the slender, delicate thread that connects almost perfect health with sudden death.

Tonsillectomy is practically always an operation of election, done to prolong life and not to save it. The patient comes to us direct from school, the work-shop, the office, and we promise that within a week or so he shall be back again at his accustomed avocation, better fitted than before for life's activities. If it happen otherwise, the fault is naturally placed at our door.

The removal of tonsils when done by a competent laryngologist familiar with his ground, versed in all the technicality of his profession, and prepared for every emer-

*Read before the Section on Ophthalmology and Otology, Medical Society of District of Columbia, February 18, 1927.