an infinite variety of attractive scenery. The excellence of the steamers of the United Fruit Company and their swiftness have made an excursion to this island very popular during the winter and spring months. The usual passage from Boston occupies from four to four and a half days.

Thomas Lathrop Stedman.
Revised by *Edward O. Otis.*

JAMBUL.—This is the local name applied to the bark and seeds of *Eugenia Jambolana* Lamarck, a tree belonging to the natural order Myrtaceæ. It is indigenous to tropical Asia and the neighboring islands, where it grows to a large size and bears a crop of edible fruit in July and August. It is also known as rose apple and Java plum. The *Eugenia pimenta*, the common allspice tree, and *Eugenia caryophyllata*, from which cloves are obtained, are both closely allied species. The fruit varies in size from a cherry to a pigeon's egg, and when ripe is olive-shaped, smooth, juicy, and purplish-black in color. It contains a single seed, which is enclosed in a thin, papery shell. All parts of the tree are astringent, and the bark furnishes a beautiful brown dye. The bark is smooth and whitish, and the cell structure contains a number of characteristic pitted cells which are visible to the naked eye. A white crystalline substance has been obtained, termed jamborine, which is tasteless, insoluble in cold water, soluble in alcohol, ether, and chloroform. It is said to possess the active properties of the seeds, but its composition and properties are uncertain. The seeds are cylindrical in shape, about one-third of an inch in length, hard and dry, and almost tasteless. The following analysis of the seeds has been furnished by Mr. Thomas Christie, of London: Essential oil, a trace; chlorophyll and fat, 0.27; resin soluble in alcohol and ether, 0.30; gallic acid, 1.65; albumin, 1.25; coloring matter, 2.70; moisture, 10.0; insoluble residue, 83.73.

The plant is highly esteemed in India for its medicinal properties, and is used by the native physicians in the treatment of many diseases. The sap or juice expressed from the leaves and bark contains the astringent properties of the plant, and when mixed with goat's milk is thought to be particularly beneficent in the intestinal disorders of children. The juice, and an infusion of the bark, are also employed in dysentery and diarrhœa, and in leucorrhœa. A liquor, jambava, is prepared from the fruit by fermentation; it possesses a stimulating and tonic action, and is a favorite beverage of the Hindoos. The most important use of jambul is as a remedy for diabetes, and it is in the treatment of this disease that it has acquired notoriety and attracted the attention of the profession during the past few years. It has long been employed for this purpose in the East, where it has the reputation of producing a rapid and, in many instances, a permanent cure. The quantity of sugar and urine is reduced, the many distressing symptoms are relieved, and a return of health and strength is said to follow its administration. This treatment was brought to the notice of the English physicians in 1883, by Banatvala, a medical officer in the service of the Madras government, and has been the subject of numerous clinical and experimental researches.

Von Mehring and Graser[1] performed an important series of experiments to demonstrate its power to check the production and lessen excretion of sugar. They produced artificial diabetes in animals by the administration of phloridzin, and carefully estimated the amount of sugar excreted when phloridzin was given alone, and when it was given in combination with jambul. The diminution was found to be invariable and very decided. The following figures indicate the results in three experiments:

Sugar excreted without jambul	12.2	10	10
Sugar excreted with jambul	2.1	1	1.5

They also proved that it was devoid of any toxic action, as very large quantities were given without producing any ill effects.

Experiments have also been made to show its inhibitory action on saccharine fermentation by adding it to a solution of starch and malt, and it has been clearly shown that the quantity of sugar is reduced in accordance with the amount of jambul present. In one instance it was found that a solution of rice starch with a definite proportion of malt produced 27.4 parts of sugar; when fifteen grains of jambul were added the amount was reduced to 9.4 parts, and when twenty-five grains were used only 1.3 parts of sugar were formed.

Following the introduction of the drug there appeared a number of reports of cases treated, and in nearly all it was found that the desired effect was produced in a greater or less degree. Among these are reports by such observers as Kingsbury,[2] Saundby,[3] E. H. Fenwick,[4] Mahomed,[5] Egasse,[6] Villy,[7] Lewaschew,[8] Lawrence,[9] and Britto.[10] In some of the cases in which it was used it failed altogether, in others the sugar reappeared immediately the remedy was discontinued, and frequently it would only lessen the symptoms in a slight degree; but the general tenor of the reports is in favor of the remedy. Notwithstanding the favorable reports it has not come into general use, although occasional reports of its successful employment still appear.

The seeds and the bark both possess the antidiabetic action, but the seeds are the more active of the two. A paper presented by Dr. T. Stevenson, of Bombay, to the Pharmaceutical Conference, held at Edinburgh, 1892, states that the fresh seeds, or an extract prepared from the fresh seeds, is the most serviceable and the only certain method of securing the medicinal properties. Under any circumstances, it is recommended that the seeds should be carefully preserved and reduced to powder only as required. Some such variation in the active properties of the drug may account for the uncertainty of its action and the difference in the quantity administered. The usually recommended dose is five to ten grains of the powder, or five to ten minims of the extract, three or four times a day. This, however, appears to be inadequate, and much larger doses are now advised. Dr. Britto, who reports from India his successful treatment of a number of cases, gave it in doses of one drachm of the powder, or one fluidrachm of the extract, three times a day; and Professor Lewaschew, who reports his experience of two years, in which he employed the drug with marked success, advocates it in doses of as much as from 20 to 40 gm. (3 v.–x.) in the twenty-four hours. No toxic action follows its use, but instances of nausea and depression have been reported from its continued use.

Beaumont Small.

[1] The Lancet, p. 902, 1889.
[2] British Medical Journal, March, 1887.
[3] The Lancet, October, 1887.
[4] *Ibid.*, October, 1888.
[5] London Practitioner, December, 1888.
[6] Bulletin Général de Thérapeutie, July, 1890.
[7] *Ibid.*, January, 1891.
[8] British Medical Journal, March, 1891.
[9] The Medical News, January, 1893.
[10] The Therapeutic Gazette, February, 1893.

JASMINE, YELLOW. SEE GELSEMIUM.

JAUNDICE.—(*Icterus; Morbus regius; Gelbsucht; Ictère*). Jaundice is a syndrome and not a disease, a condition marked by staining of the skin, conjunctivæ, and urine by bile pigment. Since the first formulation of theories by Frerichs and Kuehne, there has been interminable discussion of what may be termed the pathological physiology of jaundice. The liver was long regarded as a separator rather than a producer of bile pigment; given a cause of blood destruction, bilirubin could be formed in the blood stream or in tissues; the hepatic cell might prove unequal to the demands of elimination or its function be suppressed and jaundice resulted. Of late years there has been unity in abandoning such a view, and the paramount importance of the liver cell in the manufacture of bile pigments has been unquestioned. The idea of hæmatogenous jaundice has become obsolete and all

jaundice is regarded as of liver origin. It is true that small amounts of bilirubin may be found in other places than in the liver; bile pigment has been found in apoplectic foci, blood extravasations, and hemorrhagic infarcts in the form of hæmatoidin crystals which are identical with bilirubin. Loewit, in frogs, showed that leucocytes could take up fragments of red cells and elaborate them in different tissues to granules of bile pigment. Naunyn and Minkowski, in the course of their experiments with hæmolytic poisons in geese, demonstrated leucocytes in the liver containing fragments of red corpuscles and granules of bile pigment. Within a few months Croftan, in the course of his experiments on the bile acids, has emphasized anew the extrahepatic origin of bilirubin and of bile acids. Clinically the facts are of no import. The experiments of Kunde and Moleschott with cold-blooded animals, of Stern with pigeons, of Naunyn and Minkowski with geese, have shown conclusively that it is the liver cell above all that is concerned in the elaboration of bilirubin from hæmoglobin.

Practically every icterus is an icterus from absorption of bile from the liver. Without liver function there is no icterus. The mechanism of absorption is plain in the jaundice termed obstructive or mechanical, the jaundice of stasis, "ascending jaundice"; stasis in the ducts overcomes the pressure of the bile secretion (not more than 200 mm. of water), bile is secreted at higher pressure and passes from the intercellular duct to the lymphatic vessels near the bile capillaries, thence to the larger lymphatics, thoracic duct, and to the blood stream. The liver cells are continuous with the walls of the bile capillaries, and Kuppfer has demonstrated prolongations of the capillaries even within the liver cell. It is possible under certain conditions that functionally disordered liver cells may send bile to the blood capillaries and not to the bile ducts, or may allow passage of bile from the ducts back to the blood-vessels and not to the lymphatics. This is the so-called parapedesis of bile (Minkowski), diffusion icterus (Liebermeister), paracholia (E. Pick).

The investigations of Charcot, Legg, and others have broadened our views of mechanical jaundice. Under the influence of increased hæmolysis from toxic or infectious cause, excess of hæmoglobin is brought to the liver and elaborated into bile. There is increase in quantity of bile, but particularly an increase in viscidity and pigments, a polycholia and particularly a pleiochromia. This thick, viscid bile leads to stasis in the small bile channels, to irritation and swelling of the mucosa, to obstruction and to icterus by absorption; again an icterus of obstruction, but a descending rather than an ascending icterus. Even without increased amount of viscidity of bile, the eliminated poisons in course of an intoxication or infection may lead to catarrh of the bile terminals, to swelling with mechanical blocking and absorption. Though all cases of jaundice may be classed as obstructive, the classification of Hunter is a convenient one for descriptive purposes.

I. Obstructive Jaundice.—Causes acting from within or without the bile ducts. The obstruction is obviously mechanical and independent of changes in the blood or bile. The following is substantially the table of Murchison:

A. Obstruction by Foreign Bodies within the Duct.
1. Gall stones, inspissated bile, blood clot.
2. Foreign bodies from the intestines.
3. Parasites—hydatids, distomata, lumbricoids.

B. Obstruction by Catarrhal Swelling of Large or Small Ducts.

C. Obstruction by Stricture or Obliteration of Ducts.
1. Congenital deficiency or stricture of ducts.
2. Stricture from perihepatitis, from ulcer of the duodenum, from ulcers or scars in the bile ducts.
3. Spasmodic stricture (icterus psychicus).

D. Obstruction by Tumors at the Papilla or of the Bile Ducts.
Fibroma, lipoma, gumma, papilloma, xanthoma, sarcoma, carcinoma.

E. Obstruction by Pressure from without.
(1) Tumors of the liver; (2) tumors of the gall bladder; (3) enlarged glands in the fissure of the liver; (4) tumors of the stomach or duodenum; (5) tumors of the pancreas; (6) tumors of the kidney; (7) floating kidney; (8) omental tumors; (9) retroperitoneal tumors; (10) aneurisms of abdominal aorta, hepatic artery; (11) fæcal tumors, especially of the hepatic flexure; (12) pregnant uterus, tumors of the uterus; (13) ovarian tumors.

II. Toxæmic Jaundice.—Jaundice dependent on changes in the blood and bile; the end cause is obstruction dependent on increased viscidity of bile or on catarrhal swelling of the bile ducts. This is the group formerly called hæmatogenous. Hunter classifies causes as follows:

1. *Definite Poisons.*—Phosphorus, arsenic, toluylendiamin, snake venom.

2. *Poisons of Infectious Fevers.*—Yellow fever, malaria, pyæmia, typhus, typhoid, relapsing fever, scarlatina, pneumonia.

3. *Special Icterogenic Poisons.*—These are of probable infective nature. Various names have been given to the jaundice, as epidemic, infectious, febrile, malignant, septic, Weil's disease, icterus typhosus, icterus gravis, acute yellow atrophy of the liver.

Symptoms. I. *Obstructive Jundice.*—1. Staining of the tissues is most striking and is due to bilirubin. Connective tissue has particular affinity for the pigment. The conjunctivæ usually show the earliest tinge; the color is well seen in the mucous membrane of the hard palate, especially on pressure; color of the skin varies from sulphur, lemon, or saffron hue in slight cases to greenish, bronze, or even greenish-black in chronic cases with complete obstruction. The pigmentation is most marked over the forehead, temples, scalp, upper extremities, and thorax. The pigment lies in granular masses in the deepest layers of the rete Malpighii, and may persist ten or twenty days after it has disappeared from the blood. The cornea, peripheral nerves, cartilage, hair, and teeth escape stain; the brain is not colored, except in the newborn; the fœtus may be lightly jaundiced. 2. Nearly all secretions show presence of bilirubin. It can be demonstrated in the urine, sweat, exudates, amniotic fluid, and pus; it is inconstant in the milk, rarely present in the sputum except in pneumonia, has been demonstrated exceptionally in saliva, but is not found in the tears. The urine is usually dark yellow or brown, sometimes reddish or greenish. The foam is yellow, immersed filter paper is stained yellow, and the presence of bilirubin can be shown by a number of tests. In the Smith-Rosin test, 3 c.c. of a solution of tincture of iodine diluted ten times with alcohol is added to 10 c.c. of urine; a green ring forms at the zone of contact. Gmelin's test: Fuming nitric acid is poured beneath a layer of urine in a conical glass; a play of colors occurs at the zone of contact—yellow, green, blue, violet, to red; the green color is most characteristic. The Schwerdtfeger-Huppert and Gluzinski tests are equally delicate but less convenient. If the serum contains only slight quantities of bile pigments, the urine may contain only urobilin and no bilirubin. The urine is yellowish-red and only rarely brownish-red. Urobilin occurs in small quantity in normal urine, occurs in the fæces as stercobilin, may occur alone in the urine in slight jaundice, at the beginning or end of severe jaundice, usually disappears from the urine when bile is totally shut off from the intestine. It is a reduction product of bilirubin or hæmoglobin, and reduction may occur either in the intestine or in the tissues. There is no true urobilin icterus; the staining of the skin is always by bilirubin. To test for urobilin, water is poured carefully over the urine in a test tube; urobilin diffuses more rapidly than bilirubin, and may be recognized with the spectroscope (Hayem). The following table of Quincke, little modified from that of Hayem and Tissier, shows the shifting relations of the bile pigments in urine, fæces, and skin in the course of ordinary obstructive jaundice:

Skin.	Serum.	Urine.	Fæces.
1. Very slight yellow	Bilirubin 0	Bilirubin 0 ..... Urobilin 0 or little	Normal color.
2. Light yellow.....	Bilirubin +	Bilirubin − ..... Urobilin + ...	Colored.
3. Yellow	Bilirubin +	Bilirubin + ... Urobilin + ...	Pale.
4. Deeply yellow ...	Bilirubin +	Bilirubin + ... Urobilin + or 0..	Clay color.

Besides bilirubin and urobilin the urine contains bile acids and at times nucleo-albumin and albumin. Bile-stained cells and hyaline and finely granular casts are found in all jaundice urines.

3. Pruritus is frequent, and in marked jaundice may be severe and tormenting. It is an intoxication symptom, and largely modified by individual peculiarity; it may precede the jaundice, and in fact be present for long periods without jaundice, as in hepatic cirrhosis; but as a rule it is not intense save in complete and long-standing obstruction. It may cease when bile reappears in the stools, though the skin still remains colored. Urticaria, eczema, fissures, and boils occur as a result of scratching. Xanthoma, xanthelasma, or vitiligoidea is a peculiar condition characterized by formation of yellowish flat patches or tubercles; the flat variety occurs on the eyelids, the tubercular form elsewhere on the skin or in the viscera. The association with icterus is not a necessary one. Sweating is frequent and may be confined to the back or abdomen. Telangiectases may develop in chronic cases in the skin and occasionally in the mucous membrane of the tongue and lips. Clubbing of the nails has been occasionally observed, and periosteal nodes may form.

4. The color of the fæces may be modified. In total obstruction the stools may be pasty and more or less grayish-white or clay-colored. The color is due partly to absence of modified bile pigment, and partly to the large amount of undigested fat; according to F. Mueller, from fifty-five to seventy-eight per cent. of fat goes to waste when bile is wholly shut off from the intestines, instead of normally from seven to ten per cent. With partial obstruction the stools are more colored; return of color may be the first sign of relief from total obstruction. Formerly much stress was laid on the antiseptic properties of bile, but we now know that the bile has little influence on bacterial growth and controls but little the odor of the fæces.

5. Slow pulse is a common symptom, especially of catarrhal jaundice. There may be 60, 50, or even as low as 30 or 20 beats a minute. The phenomenon is variously attributed to action of bile acids on the vagus, heart muscle, or intracardial ganglia; irritation of the vagus is the most probable cause, for Wintrend has demonstrated a rise from 40 to 120 after atropine injection. Later, in a chronic jaundice, an originally slow pulse may rise to normal or increased rate.

6. Disturbances of vision are rare—xanthopsia, hemeralopia, nyctalopia.

7. Digestive disorders are common but equivocal. The usual complaints are of anorexia, bitter taste in the mouth, distaste for meats or fats, flatulence, constipation, or occasional diarrhœa.

8. Nervous symptoms are of many kinds. Physical and psychical weakness, depression, irritability are present in mild cases; with persistent jaundice severe disturbances may occur. The general condition grows worse, a typhoid state develops, dulness and stupor deepen into coma that proves fatal, coma alternates with states of excitement and delirium, or general convulsions of indefinite nature end the scene. The symptoms directly suggest intoxication, and the condition has long borne the name of cholæmia. The name is not a good one, as the same group of symptoms may terminate a cirrhosis of which jaundice forms no part. It is an auto-intoxication of complex kind, as shown by the variety and inconstancy of the symptoms. The term hepatic intoxication, proposed by Quincke, should supersede cholæmia.

9. Hemorrhage. The presence of bile constituents slows coagulation of the blood, and in long-continued icterus, instead of the normal time of three and a half to four minutes, coagulation may be delayed to eleven or twelve minutes (Osler). The tendency to bleeding is shown in spontaneous hemorrhages—purpura, suggillations, more rarely bleeding from mucous membranes, or by profuse and fatal hemorrhage after operations. It is well known that surgeons operate with dread in cases of long-standing jaundice.

II. *Toxæmic Jaundice.*—In this form obstruction depends upon increased viscidity of bile, due to blood changes or to catarrh of small bile ducts; no obvious obstruction is to be found in large ducts. Bile is never absent from the fæces; in fact, the stools may be very dark from excess of bile (polycholia). The bile acids are not constant in the urine, but this is of no clinical importance. Coloring of the skin is usually less deep, constitutional disturbances are as a rule decidedly more marked; the jaundice seems often merely a symptom of a general infection. All the cases of this group present about the same clinical picture; differences in the symptoms and course are due to the variety and especially to the intensity of the intoxication. There may be gradations from an apparently simple epidemic catarrhal jaundice to the syndrome of malignant jaundice or Weil's disease, or to the severest type of icterus gravis or acute yellow atrophy. For further discussion of this group, see articles on *Phosphorus Poisoning, Weil's Disease, Yellow Fever, Liver Diseases: Acute Yellow Atrophy.*

Morbid Anatomy and Course.—The pathological findings of icterus vary widely with the causes; they are sufficiently discussed in connection with symptomatology or in the sections dealing with the special diseases. The course and prognosis vary also with the cause; in general, prognosis is less good when obstructive jaundice has lasted three or four months; after eight to twelve months liability to hemorrhage or to sudden severe nervous symptoms renders the outlook unfavorable. Budd, however, cites recovery after four years; Murchison, Barth, and Bismer report a favorable termination after six years' duration; Legendre, Gailliard, and Debove mention cases of complete obstruction of twelve, twenty, twenty-five years' standing without much general disturbance!

Varieties of Jaundice.—1. *Icterus Neonatorum.*—Jaundice of the newborn may be (*a*) severe, due to congenital stricture or absence of the bile ducts, to syphilis, or to sepsis; this form rapidly proves fatal; (*b*) mild or physiological. This occurs in one-third or two-thirds of all infants born in hospital, and in a somewhat smaller per cent. of private cases. It is more frequent in boys, in premature infants, in cases in which chloroform was used, or in cases attended with marked congestion. The jaundice appears on the second or third day, is most marked in the face and upper part of the body, as a rule is not deep, the conjunctivæ are stained only in severe cases, there is little or no general disturbance, the color fades in from a few days to three or four weeks. The urine, as a rule, is of normal yellow color and contains no soluble bile pigment; it frequently shows traces of albumin, and the sediment may show bile-stained kidney epithelium or cells enclosing granules or crystals of bilirubin. Bilirubin is held in the kidney in the form of infarcts. There is no urobilinuria; the fæces are of normal yellow color. Pathogenesis is obscure. The benign character shows the practical physiological nature of the process; it may depend in part on the increased blood destruction and consequent polycholia of the first days after birth, in part on the slow excretion of bile by the kidney (infarct formation), in part on lack of bile reduction in the intestine. More probable is the explanation of Franck and Quincke that the icterus depends on increased bile absorption from the intestines; the meconium contains bilirubin, bile secretion is increased with ingestion of the first food, bile is consequently absorbed in quantity into the portal blood, and in the first few days patency of the ductus venosus Arantii allows the bile constituents to enter in part the

vena cava and so reach the general blood stream without passing the liver.

2. *Icterus after Hemorrhages.*—Definite icterus has been observed after blood extravasations and internal hemorrhages. The staining appears in from three to ten days after the hemorrhage, is of slight degree, and fades in a few days or weeks. Urobilinuria accompanies and, in fact, precedes the jaundice; it is extremely rare to find bilirubin in the urine. Elaboration of the bile pigment probably does not take place locally; hæmatoidin crystals may form, but this is a slow process and the crystals show little tendency to solution. More probable is the solution of hæmoglobin *in situ* and its transformation into bilirubin in the liver; the jaundice is hepatogenous, an icterus pleiochromicus. With small extravasations no jaundice occurs, only urobilinuria.

3. *Inanition Icterus.*—Slight staining of the conjunctivæ or skin may occur in inanition or starvation. Trendelenburg observed a case with slight bilirubinuria. It is a common event to find bile in the urine of fasting dogs—the absorption occurs within the liver.

4. *Icterus Syphiliticus.*—This is the icterus syphiliticus præcox. It occurs in the secondary stage, is not frequent, occurs oftener in women (Fournier). The cases show generally severe secondary symptoms, eruptions, and marked glandular enlargements (Werner). It is a mechanical jaundice and due to swelling of the glands in the portal fissure (Lancereaux). In one case Quincke observed ascites and splenic tumor coming and going with the jaundice.

5. *Icterus Psychicus, Icterus Spasticus, Icterus Ex emotione.*—In the minds of the laity the emotions play a large part in liver pathology. The only cases of jaundice that can be ascribed to nerve influence are those which occur within a few hours or even minutes after a sudden nerve shock, as fright, anger, fear. There are two classes of such cases: 1. Jaundice coming on immediately after severe shock; in all literature examples of this class are extremely rare; two cases of Villeneuve (1818) are cited by Murchison. 2. Jaundice occurring a few hours after great nerve shock or strain. This is comparatively common; the jaundice is light and of short duration; in a few cases acute yellow atrophy has followed. Various explanations have been advanced—polycholia, lowered portal pressure (Frerichs). The most probable explanation is that of spasmodic contraction of the bile ducts with increased back pressure and quick absorption.

Herbert C. Moffitt.

JAWS, INJURIES AND DISEASES OF. — Injuries and Diseases of the Upper Jaw.—The upper jaw is peculiar from the fact of its possessing a large cavity, the antrum of Highmore. This cavity is situated in the body of the bone, and is lined with mucous membrane continuous with that of the nasal cavity through a small orifice opening into the middle meatus.

On account of its structure the upper jaw is more subject to diseases than the lower. The affections of the bone calling for surgical interference are injuries, inflammation and abscess, cystic diseases, and tumors.

Fractures of the Upper Jaw.—Owing to the position of the upper jaw, protected on all sides by its outlying processes of bone—the malar bone externally and the nasal bones internally—fracture of the upper jaw is not a very frequent accident. Almost invariably fracture of this bone is associated with fracture of the more prominent bones of the face, with which it is articulated. Direct violence, such as blows upon the face, falls from great heights, etc., is usually the cause of the fracture.

The fracture may be of the penetrating variety, consisting of a small opening into the antrum made by a sharp-pointed instrument, which may enter by way of the orbit, the palate, the nostril, or the anterior wall of the cavity. Such wounds, as a general rule, heal rapidly, and require but little attention on the part of the surgeon.

Fracture may involve any part of the bone—the nasal, palatal, or alveolar process, or the body of the bone. As the result of falls upon the face from great heights, the fracture, in a few recorded cases, has been vertical in the median line, constituting a diastasis or separation of the two superior maxillary bones.

Comminuted fractures, attended with the most frightful deformity, as the result of gunshot wounds, are occasionally met with.

When the alveolar process is separated from the body of the bone there is usually marked displacement.

The anterior wall of the antrum of Highmore is sometimes crushed in by fragments of the malar bone driven down upon it by the force of blows.

The soft parts overlying the fracture are nearly always extensively involved. Hemorrhage from wounds of branches of the internal maxillary artery is occasionally very profuse—sometimes even requiring the ligation of the common carotid artery, or the application of the actual cautery to the bleeding point.

When a fracture of the nasal process of the upper jaw is complicated with a fracture of the nasal bones in which the mucous membrane of the nose has been more or less lacerated, extensive emphysema of the face may take place.

If the line of fracture runs through the infra-orbital foramen, causing contusion or laceration of the infra-orbital nerve, temporary paralysis of the parts supplied by that nerve may ensue.

Obstruction of the lachrymal duct, with a constant overflow of tears upon the cheek, may follow fracture of the upper jaw.

Symptoms.—In the majority of cases recognition of fracture of the upper jaw is not difficult. Deep-seated pain, increase of saliva, hemorrhage from the mouth, and the special signs of fracture, viz., crepitus, preternatural mobility, and deformity, are all present in greater or less degree. The accessible position of every part of the bone makes it usually an easy matter to detect a fracture of the upper jaw.

Treatment.—In the treatment of fractures of the upper jaw the indications are to replace, by manipulation, the fragments as accurately as possible, and, by suitable appliances, to render them immovable. Pressing the lower jaw firmly against the upper with a bandage will in most cases suffice.

If the tendency to displacement is great, as in fractures of the alveolus, it may be necessary to wire the teeth of opposing fragments together, or to adjust a gutta-percha or vulcanite interdental splint.

When the fracture is comminuted and compound, great care should be taken to preserve every fragment, however loosely attached, as the experience of a great many surgeons has shown that such fragments reunite very readily. Another point to be observed in the treatment of fractures of the upper jaw is not to extract loosened teeth, as, in addition to the fact that they most frequently become firm again, their extraction is attended with some danger of removing fragments of bone that might have been preserved.

Repair in cases of average severity takes place in from thirty to forty days with a scanty formation of callus, and not infrequently in less time. The vitality of the bone is exceptionally great; hence the rule laid down by Malgaigne and some of his predecessors, and repeated by all subsequent writers, to leave every fragment that is not absolutely and entirely detached. Although this rule is a sound one, it occasionally happens that fragments become necrosed and have to be removed.[6]

Inflammation, either acute or chronic, may attack the mucous membrane of the antrum or the periosteum of the bone. The cause of the inflammation is most commonly irritation set up by carious teeth, though it may originate from mechanical injury, from the poisonous effects of syphilis, scrofula, the exanthematous fevers, mercury, or phosphorus. Its tendency is to run rapidly on to suppuration, and in the majority of cases this process has been already established when the surgeon is called.

When the mucous membrane of the antrum is in-