absent. In connection with the above the examination will show a hinge-like motion at the site of the lesion, which is typical of these fractures.

A simple yet complete classification of fractures is of value both to the student and teacher, and by reference to the following table we see just where this type of fracture belongs. This table is based on the fractures treated by us at the Roosevelt Hospital Dispensary during five years ended Sept. 1, 1898, aggregating 1029 cases, of which 841 were fractures of the long bones, and of these 423 occurred in patients under 16 years of age.

Fractures.
- Simple, 423.
  - Complete, 353.
  - Incomplete, 70.
    - Greenstick, 57.
    - Sub-periostal, 13.
- Compound.

When we bear in mind that these are hospital statistics, I feel I am justified in stating that the subperiosteal fracture occurs more frequently than indicated by this table. Many of our fracture cases, and this is especially true of the obscure ones, have been subjected to harsh and unnecessary manipulation before they reach us, and in this way cases have been converted from the subperiosteal type into the simple complete fracture.

It will be of interest to compare the subperiosteal fracture with the simple complete one, which I have done in the following table.

	Subperiosteal.	Simple complete fracture.
1. Trauma.	Usually not severe. Direct.	More severe. Direct or indirect.
2. Pain.	Absent or slight.	Marked. Often severe.
3. Function.	May be only slight impairment.	Usually marked loss, often complete.
4. Deformity.	Usually none.	Present or easily produced.
5. Edema.	Slight or absent.	May be marked.
6. Ecchymosis.	Usually absent.	Often present.
7. Crepitus.	Absent.	Present.
8. Mobility.	Hinge-like motion.	Complete.

At one time I was of the opinion that in the younger children we were more apt to have subperiosteal fractures, while in older ones the green-stick variety would preponderate, taking into consideration all incomplete fractures brought to us, but this opinion was probably due to the greater liability of subperiosteal fractures occurring in rhachitic bones before the stage of sclerosis.

I think it may be said that a "false diagnosis," or rather a lack of appreciation of the true state of affairs, is more often due to the neglect of a systematic examination than any other factor, and this is certainly true as regards the recognition of the subperiosteal fracture. As a rule in these injuries, the parts are favorable for a complete examination, but this must be systematic, the manipulations must be made in the proper manner and with due gentleness, and the anatomy of these fractures must be borne in mind.

Where these cases are overlooked they will develop undue pain and possibly excess of callus formation, with some angular deformity later on, due to sagging at the point of fracture. In fact, we have had cases brought to us where the first sign noted by the parents has been either bony thickening, or a slight angular deformity, and many of these were undoubtedly subperiosteal fractures following a slight trauma, and not recognized or treated. I can bring to mind several cases where children have received injuries, have concealed their existence from their parents, and where later a slight angular deformity followed. In each case loss of function was absent or trifling.

As regards treatment, simple fixation has been practiced, either by means of lateral splints, the starch dressing, or the plaster-of-Paris cast. In my hospital cases I have continued the fixation for four weeks, as a rule, though I am satisfied that in many cases repair has occurred somewhat sooner. The amount of provisional callus thrown out is always less than in the complete fracture, and in some cases it has been impossible to detect callus formation during the process of repair.

Various forms of incomplete fractures have been recorded and described. Some of these are frequent, have certain anatomic features, and may be recognized by certain clinical symptoms. Others occur more rarely, may be due to some peculiar combination of force, attitude, and constitutional dyscrasia, and do not give rise to any diagnostic group of clinical symptoms. Of the simple incomplete fractures of the long bones in the young, we have two principal types, the green-stick and the subperiosteal.

CONCLUSIONS.

Subperiosteal fractures usually present certain typical symptoms, and we should be able to recognize these lesions and differentiate them from the complete fractures.

Their union is characterized by the formation of a slight amount of callus, and solid union takes place in considerably less time than in the complete fracture.

In every contusion to the limbs, in the young, no matter how slight, a systematic search should be made for this particular lesion.

In rickets there is a greater liability toward the occurrence of incomplete fracture, and especial liability during the stage of softening for the production of the subperiosteal fracture.

Many cases of subperiosteal fracture are undoubtedly converted into simple fracture through improper manipulation or by too forcible examination.

In submitting these preliminary observations I wish to express my appreciation of the many courtesies extended to me by Dr. Alexander B. Johnson and Dr. John McGaw Woodbury.

ALBUMINURIA; ITS PROGNOSTIC VALUE IN CHRONIC NEPHRITIS.*

BY CHARLES ALLING TUTTLE, PHB., M.D.
NEW HAVEN, CONN.

The mass of evidence which has come to us of late, from the autopsy table, shows conclusively that chronic nephritis exists and is an unrecognized cause of death in a proportion of cases far beyond ordinary belief, and the comparison of carefully kept records of cases before death with autopsy findings shows that little reliance can be placed on the mere urinary examination, either positive or negative, as a means of absolute diagnosis or prognosis of Bright's disease. It will be the purpose of these notes to show that the result of the ordinary examinations of the urine, either in our offices or laboratories, are of little if any value in very many cases, from a diagnostic or prognostic standpoint, and that we are forced upon the very varied and numerous clinical manifestations and the absolute toxicity of the urine as offering the best and safest means of diagnosis.

That chronic insterstitial nephritis can exist without albumin, without casts and without uremic symptoms is beyond question; and that such conditions do exist and are brought out only at the autopsy is not an unusual circumstance and, further, that albumin of measurable quantity, casts, either of a hyaline or granular variety or both, can exist and the health of the individual be unimpaired throughout many years is the experience

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of the writer. "Formerly—a single observation of albumin in the urine meant albuminuria; albuminuria meant disease of the kidney; disease of the kidney meant, in a great majority of the cases, chronic Bright's disease; Bright's disease meant steady progress toward a more or less remote fatality; interrupted at times by various accidents which would still more accelerate it."[1] In this both the premises and the logic are in error and are in general as untrue as would be the opposite statement that chronic Bright's disease is never attended with albuminuria, or that albuminuria never meant nephritis. My experience leads me to believe: 1. That Bright's disease may exist without the ordinary urinary manifestations, viz.: albumin or casts. 2. That positive urinary indications, viz.: albumin and casts, may be found in the normal urine and hence do not necessarily mean Bright's disease. 3. That, given a case of chronic Bright's disease attended with albuminuria, the fact of its presence, its constancy or its amount has absolutely no prognostic significance.

1. As to Bright's disease without urinary manifestations, there can be no question to-day that it may begin, run a more or less lengthy course and terminate fatally without at any period showing other urinary symptoms than continued, low specific gravity or frequent micturition, and cases also occur which at one period of their course have both albumin and casts, but in which these disappear and the patient has comfortable health throughout an indefinite number of years. Austin Flint, as early as 1886, says:[2] "the small granular kidney may sometimes secrete urine which in no way differs from normal," and quotes Bartel as mentioning a case in which albumin was absent throughout the whole course of the disease. He says, further, "that casts may be likewise absent." Saundby,[3] in 1889, said: "We should absolutely give up the idea still current in practice that albuminuria must always coexist with nephritis," and Cheberly,[4] speaking of the symptom albuminuria, says: "Often the only symptom of Bright's disease, it may be entirely wanting in a pronounced case and its intensity varies without direct reference to the degree of renal alteration." Coquet,[5] in a thesis on the same subject, divides the cases into three classes: 1. Those from which the symptom—albuminuria—is absent but a portion of the time. 2. Those from which it is absent all the time. Anders,[6] speaking of interstitial nephritis, says: "The existence of this condition may be unknown, as frequently happens when the post-mortem examination shows the characteristic kidney in one who, during life has no symptoms indicating renal disease." Stewart[7] writes: "There is a non-albuminuric nephritis exclusive of cases of typical fibroid kidney. In this form of nephritis albuminuria may be completely absent while signs of renal insufficiency and even uremia may appear." Fienga,[8] Denby,[9] Brandyth Symonds[10] bear witness to the same thing, and E. R. Edwards[11] also writes: "I am certain we overlook both acute and chronic nephritis in regarding albuminuria as a certain or constant symptom. Nephritis without albuminuria certainly exists." Qurirolo,[12] Lecorche,[13] Talamon[14] and Lancereaux each report many cases and agree with D. D. Stewart,[15] who says that he believes that albumin may be constantly absent and casts be detected only after much search if at all, and states frankly the conviction which he feels regarding his own experiences that he has overlooked many cases of nephritis without albumin. In summing up his article, Edwards makes this pertinent remark: "Hence as we fear instinctively, as it were, the existence of nephritis before we examine the urine, so we may still fear its existence after negative urinalysis," and he also says that casts should be searched for, as they are more constantly found than albumin, but are not invariable in nephritis.

This is also the experience of the writer. In my last twenty cases, all in private practice, where the diagnosis of nephritis was made, two only are of interest in this connection. In one, A. G. (Case 12), there was neither albumin nor casts at any time, the diagnosis being made on the edema and uremia, and in the other, A. T. (Case 10), albumin was found in one-third of all examinations, seventy-five in number, extending over approximately three months. In two only of the twenty cases could autopsies be secured, one, A. G., who had presented only negative uranalysis, and yet the pathologic condition of the kidney was unquestionable.

2. Positive urinary indications, viz., albumin and casts, may be found in the normal urine, and hence do not necessarily mean Bright's disease. By means of the recent and more exact methods of uranalysis, it is made possible to show a much smaller amount of all the elements of the urine than formerly. This is particularly true of albumin and casts until to-day quantities of the former amounting to only 1 in 300,000 can be detected and approximately measured, and casts found with comparative ease where formerly none were believed to exist. I shall attempt to show, largely by reference, that not only albumin but casts occur in the normal urine—that is, if we accept as normal the urine passed by indviduals in whom it is impossible to detect anything but perfect health and strength and harmony of physiology at the time, and whose physical condition remains unimpaired throughout many years. It is not my purpose at this time to go far into the so-called "functional" or physiologic albuminurias. That albumin, both constant and intermittent, may occur in an individual and not be renal in its origin, or if supposedly renal be consistent with perfect health and longevity, will in this day hardly be questioned. The writer has at present no less than seven cases under observation, of healthful individuals in whom albumin has existed for from two to nine years, notably one of a man about 60 years old, in whose urine, twenty years ago, was detected a considerable quantity of albumin which still exists. He is hale, hearty, and with but slight arteriosclerosis. Saunby,[16] as early as 1889, answered the question: "Does albuminuria occur in healthy persons?" in the affirmative, and based his reply on the examinations of 461 cases of supposedly healthy individuals, in 118 of whom he found albumin.

Senator,[18] of Berlin, holds that urine is a product of transudation and normally contains a certain amount of albumin, as do all transudation fluids, and that the normal amount of albumin in the urine may be increased without fault in the kidneys to the production of so-called physiologic albuminuria. The writer agrees with Richardson[19] (London), who says: "It seems now to be accepted that there is a form of albuminuria in which there is no renal disease whatever, nor actual organic disease of any organ of the body, but a physiological state during which albumin passes by the urine as if the act were natural and dependent upon some simple physiological changes." The significance of the presence of casts underwent similar changes as to opinions, to that of albumin, in estimated importance, and for similar reasons, until recent writers—Haines and Skinner[22]—claimed that by their method of careful search casts might be found in samples of urine even from persons in perfect health. If, then, we accept this as true, and allow also the statement and later statistics of Shattock

and Mitchell, showing that they have found casts for years in healthful individuals, we must be impressed with the idea that little significance can be attached to them. In concluding this section, let me refer to one writer, Edwards (q. v.), who says: "Our conception has been broadened concerning the significance of casts, especially hyaline, now regarded as occurring in urine otherwise normal," and to Symonds,[23] who speaks of casts of "functional origin," or as I would prefer to call them, *physiologic casts.*

3. Given a case of chronic Bright's disease attended with albuminuria, the fact of its presence or its constancy or its amount has absolutely no prognostic significance. For some time it has been the personal opinion of the writer, based on the observations of something over one hundred cases, that the amount of destruction in the renal parenchyma is, in chronic nephritis, a measure of the prognosis, but that the amount of albumin in the urine is in no sense indicative of such destruction, and, further, that we to-day have nothing, unless it be the absolute toxicity of the urine or the degree and character of the albuminuric retinitis, that at all serves as a measure of this destruction. Albumin may simply indicate a lesion of the glomerular filtering membrane, nothing else, and to form an idea of its profundity, extent, or gravity, other sources of information are to be searched. Lecorche and Talamon (l. c.), in speaking on this point, say: "It is impossible to attach any prognostic value direct or remote to this condition, as the percentage of albumin in the urine is not proportionate to the degree of renal lesion." In a recent address before the Academy of Medicine, Paris, Semola[25] expressed himself as follows: "There can be no exact relation of cause or effect between the severity of the renal lesion and the degree of albumin excreted, and one can influence the latter only by the state of the blood, albumins without any reference to the condition of the kidneys." Mesnard[26] writes: "The intensity and character of this condition (albuminuria) are without value to indicate the degree of pathological changes in the kidneys or the imminence of the grave occurrences common in Bright's disease." That a fatal issue in Bright's disease may be forestalled in some and the prognosis as to time materially modified in almost all cases, is beyond question, for those are not uncommon where the individual life has been prolonged fifteen, twenty, or even thirty years under the influences of judicious medication and dietary.

It would seem, from what has been written, that Bright's disease might be difficult of diagnosis, and I believe it is more often overlooked than any other common disease. Insidious in its approach, indefinite in its manifestations, and far-reaching in its effect, it often baffles the most acute of diagnosticians until one realizes the full force of what was written by Edes, who says: "If in such a case as is reported by Edwards, nephritis is *suspected* when there are no suggestive cardio-vascular findings, no albumin, no casts, no decrease in total solids, specific gravity or quantity, no edema mentioned, and the possible diagnosis is confirmed by autopsy, we can only congratulate the physicians on their acuteness and feel that when a diseased organ presents absolutely no symptoms of abnormality in its products and no evidence —except so very indefinite one as nausea—of impaired functions, nobody but a clairvoyant can be blamed for overlooking it."

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129 Whalley Avenue.

SIMPLE AND ETHEREAL SULPHATES.

A SIMPLE AND RAPID METHOD FOR THEIR SEPARATE DETERMINATION—THIRTY MINUTES.

BY G. W. McCASKEY, A.M., M.D.

PROFESSOR OF CLINICAL MEDICINE AND NERVOUS DISEASES.
FORT WAYNE COLLEGE OF MEDICINE.

FT. WAYNE, IND.

The clinical value of the estimation of the proportion of the ethereal to the simple sulphates in the urine is an indication of the extent of intestinal putrefaction from the most common forms of intestinal organisms and the consequent associated formation of intestinal toxins is very great. The importance of the estimation of the ethereal sulphates is based on the fact that they are the result of a combination of the simple sulphates of the urine with certain aromatic principles, the products of bacterial action in the intestinal tract. The principal compounds of this class of aromatic substances are phenol, indol, skatol, cresol, and pyrocatechin. Most of these substances are, so far as we know, the exclusive result of bacterial action, which may occur anywhere in the organism, as in an abscess cavity, but which as a matter of fact nearly always occurs in the intestinal canal. The quantity of ethereal sulphates, otherwise known as conjugate, aromatic, or combined sulphates, or ester compounds therefore becomes in large measure an index of the intensity of bacterial processes. These substances may have, some of them, considerable importance on account of their own toxic properties, but are perhaps more important as indicators of a process which would have associated with them other substances more toxic than these, but not so far as we know responsive to any chemical test after their elimination through the kidneys. It is perfectly well understood that there may be other toxic processes in the intestines which do not produce aromatic substances and would not therefore lead to an increase of ethereal sulphates; the absence or normal amount of the latter has not thus the same clinical importance as an excess.

It is the more unfortunate, therefore, that the methods in vogue are so cumbersome and time-consuming as to preclude their use in ordinary routine clinical work. My first attempt at a practical solution of the question in my private clinical laboratory was with the ordinary titration method. This is intolerably slow, owing to the fact that after each addition of barium solution the specimen under examination ought to be boiled in order to insure the combination of the sulphates with the barium, and then allowed to settle before the further addition of