ments. One point is that they almost always contain some element which is not in human milk, such as starch, which in very young babies may seriously disturb digestion. Another harm that I refer to is that they encourage carelessness of the selection of the milk supply which is used in connection with them, in that the parents are led to believe that the chief virtue of the food lies in the food itself, and not in the milk which is added to it. Mothers and nurses are therefore very apt to use a poor milk supply, and are indifferent to the fundamental principles of scientific feeding, since they believe that the important point is the selection of some particular brand of patent or proprietary food. It would be just as sensible to attribute the entire success of an infant that is thriving on a substitute food to the milk-sugar which happens to be used in an ordinary modification.

If the physician wishes, for a definite purpose, or as he naturally would, during the tenth, eleventh and twelfth months of life to use starch in his substitute food, the starch should be presented in definite percentages. Thus cereal solutions can be added in place of the boiled water in any combination without altering the percentage of fats and proteids, and the percentage of starch thus added may be accurately determined if the percentage strength of the cereal solution is known. For instance, if fourteen ounces of a 1 per cent. solution of barley water is added in place of the boiled water, to a twenty-ounce mixture, the percentage of starch in the mixture is:

$$\frac{14 \times 1}{20} \text{ or } 0.70 \text{ starch.}$$

It is well here to state my views concerning the use of starch in any form in the early weeks and months of life. The amylolytic function of the infant is but very little developed at birth. It gradually increases as the infant grows older and has attained the stage of development by the tenth, eleventh or twelfth month which warrants its being used at that time for the conversion of starch into sugar. If it is used in the early months of life, we are taxing an undeveloped function, and we are liable to have results as deleterious to the infant's welfare as we should were we to tax the function of walking and standing at too early a period. It is quite as illogical and detrimental to the infant's welfare to tax an undeveloped function as it is not to make use of such function when the time for using it has arrived. Thus we should make use of the amylolytic function in the latter part of the first year, since at that time it is ready for use, just as we allow the child to stand at the same period.

The advantage which used to be claimed for the use of starch in a substitute infant's food was that it broke up the caseinogen in the milk, and thus made a finer coagulum, and one more similar to that which occurs in human milk. This action of the starch on the coagulum is, however, required in comparatively few cases, as we have learned to deal with the coagulum in a different way since we have instituted a method by which the caseinogen which forms the coagulum can be reduced to a very small percentage, while the whey-proteids, which are almost as nutritious as the caseinogen, can be used in quite a large percentage. By this method we can obtain a total proteid percentage as high as 1.15 per cent., of which 0.90 per cent. is whey-proteids, and 0.25 per cent. is caseinogen.

The following Table 7 shows the combinations possible in these whey-cream mixtures, when prescribing from the milk laboratories:

Whey proteids, per cent.	Caseinogen, per cent.
0.25.....	0.25
0.50.....	0.25
0.75.....	0.25
0.90.....	0.25
0.90.....	0.50
0.75.....	0.50
0.90.....	0.60
0.90.....	0.75
0.90.....	1.00

Any percentage of sugar from 4 to 7 can be combined with these percentages.

If we thoroughly grasp the sense of what has just been said in connection with human breast milk, and the milk of animals, especially that of the cow, which seems to be the animal whose product is most adapted to substitute feeding, we shall at once see that the main question regarding substitute feeding is how to obtain the best milk, how to treat it, and how to modify it accurately. The importance of the milk supply is, however, great, and there is no doubt but that the breed of the cow, the care of the cow, of the stables and of the milkers on the farms especially used for the purpose of obtaining a proper milk supply for substitute feeding, is the keynote to success.

(To be continued.)

Original Articles.

ALBUMINURIA IN DIABETES MELLITUS.

ITS IMPORTANCE AS A FACTOR IN THE CAUSATION OF DIABETIC COMA.*

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Among the secondary tissue degenerations which result from diabetes none is so frequent as lesional changes in the kidneys. No extensive autopsy reports are at hand to establish the relative frequency of renal changes in diabetes, but such as are forthcoming demonstrate in a striking manner the association. Thus out of Griesinger's 64 autopsies, renal alterations were found in 32. In the main, the changes were degenerative in character, either fatty or hyperplastic. Seegen found such changes in 20 out of 30 cases brought to autopsy, and Dickinson in 25 out of 27. No special form of renal defect in diabetes can as yet be looked on as typical, the changes being variable in kind and degree, although almost always present. The form which they assume will depend on the duration and intensity of the irritation, to which the renal gland structure is subjected. As the toxemia differs very greatly in character and degree in the different forms—mild and severe—of diabetes, so we find the secondary changes in the kidneys differing widely in their essence and microscopic character.

The most interesting histologic feature of the diabetic kidney is the hyaline degeneration of the tubular epithelium first described by Armanni, and now generally known as the cellular necrosis of Ebstein. This condition of the tubular epithelium is encountered only in severe toxic forms of diabetes. Dreschfeld found these necrotic changes in 6 out of 10 cases of diabetic coma. Other renal alterations occasionally met with in severe

* Read at the Fifty-fourth Annual Session of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. J. M. Anders, Frank A. Jones and W. S. Thayer.

forms of the disease are hypertrophy and the so-called lesion of Ehrlich, which, according to Frerichs and Ehrlich, consists in glycogen degeneration of Henle's loop and the straight tubes. All these renal changes may be ascribed to the action of toxic bodies of highly irritating character, such as acetone, diacetic and oxybutyric acids, etc., which are present in the blood and urine of such cases.

In the mild long-continued form of diabetes the renal changes which develop are usually those of chronic nephritis. The development of chronic nephritis under these circumstances may be explained by prolonged hyperfunction, combined with persistent but low-grade irritation of the renal structure by the saccharin urine, which lead first to hyperemia, and if long enough continued, finally to inflammation of the parenchyma and overgrowth of the interstitial tissue. In this type of the disease the renal gland changes are nutritional and degenerative, whereas in the more rapidly progressive and toxic form of the malady they are inflammatory or necrotic. In the majority of cases presenting chronic organic processes in the kidneys the nephritis is probably of the mixed type, presenting both parenchymatous changes and interstitial hyperplasia. In aged diabetes arteriosclerotic renal atrophy is frequent.

Whatever form the renal alterations may assume, they sooner or later give rise to the presence of albumin in the urine. The frequent association of albuminuria with diabetes has received considerable attention, and much statistical evidence is available.

Pollatchek	found albumin in	37	per cent. of cases.
Von Schmitz	"	63.4	" "
Lecorché	"	50	" "
Bouchardat	"	45	" "
Von Noorden	"	23.5	" "
Grube	"	40.4	" "
Stokvis	"	40	" "
Bouchard	"	43	" "
Von Dusch	"	28.5	" "
Rokitansky	"	66	" "

These statistics represent an average of 43.68 per cent in which albuminuria was present in diabetes.

Attempts have been made to classify the albuminuria of diabetes. Goudard distinguishes two nosologic varieties, which he names functional and lesional, the former associated with renal fatigue with phosphaturia or with organic disassimilation, whereas the lesional is produced by some form of renal lesion. Jacobson, on similar grounds, separates albuminuria of diabetes into benign and severe.

Grube divides diabetic albuminuria into five varieties, viz.: 1, terminal albuminuria associated with diabetic coma; 2, cardiac albuminuria met with in connection with myocardial weakness; 3, senile albuminuria of arteriosclerotic origin; 4, functional albuminuria due to irritation of kidneys by the saccharin urine; 5, albuminuria associated with organic renal changes. Salles recommends an equally complicated classification.

It seems to the writer unnecessary and confusing to adopt any elaborate classification for this condition. With the exception of the rare cases of albuminuria produced by circulatory failure in patients of asthenic type, the albuminuria of diabetes may, for general clinical purposes, be placed in one of two categories.

I. TOXIC ALBUMINURIA.

To the class known as toxic albuminuria belong the cases of the severe form of diabetes which are entering the terminal stage of the disease and in which the highly irritating acid toxins of endogenous formation have produced hyaline changes of the tubular epithelium—the so-called cellular necrosis of Ebstein, already referred

to. This albuminuria is never absent in the stage of organic disassimilation and in diabetic coma, although it may fail to make its appearance until the terminal stage is entered on and toxinuria becomes established. This albuminuria is never of severe grade, seldom amounting to more than a mere trace, or at most, 1 to 1,000. It usually shows some increase just before death, and sooner or later becomes associated with large numbers of hyaline tube casts. After it makes its appearance, this form of albuminuria seldom disappears again completely. As a rule the albuminuria and glycosuria bear no direct relation to each other, and in occasional cases may even stand in inverse proportion to one another. It will usually be found, however, that a direct relationship exists between the toxinuria and the albuminuria, the latter increasing or diminishing with increase or diminution of acetone and acid toxins in the urine.

This form of albuminuria constitutes what has been sometimes known as the acute albuminuria of McGuire. Universal clinical experience associates this form of albuminuria with the advent of coma, which gives it a grave prognostic significance.

II. DEGENERATIVE ALBUMINURIA.

The second class of albuminuria in diabetes includes the vast majority of cases of this condition, for notwithstanding the greater proportional frequency of the first variety to the class of cases in which it arises, the severe type of diabetes, these constitute but a small per cent. of the great bulk of diabetes. This form of albuminuria is also the product of renal irritation, but irritation of an entirely different nature and severity. In this class of cases the sugar in the urine is the offending agent. Its potency in this respect is much less than that possessed by the acid toxins of the severe grades of the disease, but its continuous action over long periods of time, combined with renal hyperfunction, which attends the associated polyuria, in the end inevitably initiate changes in the kidney which, in their full development, constitute nephritis. In proportion as the renal epithelium is affected, albumin appears in the urine, and as parenchymatous changes are usually a first result of the hyperemia following irritation, albuminuria is a pretty regular attendant on the renal change. In many cases for considerable periods this albuminuria is not attended by any very demonstrable evidence of nephritis, and consequently it has been misnamed functional, to distinguish it from albuminuria, associated with definite and clinically recognizable renal lesions. It has been considered a development of little importance and in the presence of conflicting therapeutic indications, as between the glycosuria and albuminuria, the latter is usually neglected. After a time, however, this at first apparently accidental albuminuria is found to merge imperceptibly into a true lesional albuminuria, with impaired renal function and secondary cardiovascular developments. Hyperfunction and persistent irritation have done their work, and a nephritis of a mixed type becomes established and adds to the difficulties of the case.

It will be seen from the foregoing brief considerations regarding albuminuria in diabetes that fully a third or more of all cases of the disease manifest this symptom at some time during their course. Although by no means of itself always a sign of established renal mischief, it is never devoid of importance. It may appear so frequently without accompanying disturbance that many writers falsely regard it as a harmless

sign. As pointed out, it may occasionally arise as the apparent product of irritation of the kidneys by the saccharin urine, but should the glycosuria continue, definite renal and secondary systemic changes eventually result. In aged diabetes with arteriosclerosis albuminuria may be present from atrophic changes in the kidneys of sclerotic origin. In the late stages of diabetes, of whatever form, albumin may appear in the urine as the result of venous stasis from cardiac asthenia.

The conclusion forced on us by these considerations is that the development of this symptom is always a significant event, indicative in the mild, long-continued form of the disease of developing nephritis, and in the severe toxic stages of diabetes we are warranted by experience in regarding it as one of the terminal developments.

A matter of great interest in connection with the albuminuria of diabetes is the frequency with which the glycosuria is observed to diminish as the albuminuria becomes more pronounced. In exceptional cases the glycosuria may even entirely disappear, thus constituting the substitutive albuminuria of Salles. Maragliano is skeptical of a true substitutive process, and explains the fact on the theory that albuminuria appears on improvement of the diabetes under treatment, because the polyuria lessens *pari passu* with diminution of the glycosuria and albumin already present but masked by the previous dilution of the urine is revealed. This view can not be seriously accepted as an explanation, because in most cases diminution of the glycosuria does not precede the advent of albuminuria, but follows it, and albuminuria is, moreover, not usually the result of treatment. A diminution in the glycosuria, even its entire disappearance, has been observed to occur as the apparent result of the hyponutrition of such complications, as pulmonary tuberculosis, etc., when they arise during the course of a diabetes. This can not be considered a similar process to that under consideration, for in the great majority of substitutive albuminurias the nutrition is not materially depressed. Naunyn's contention that this peculiar conduct of the glycosuria is analogous to the behavior of that symptom in the presence of cachexia and marasmus, from whatever cause they may arise in diabetes, is stoutly opposed by Kleen, who observes that he has repeatedly seen cases in which nephritis, though advanced, has left a fairly good state of health, with no marasmus or cachexia, but with marked diminution of glycosuria. Such cases are within the experience of most observers.

A number of very suggestive experimental observations are at hand which have some bearing on this question of the diminution of the glycosuria in the presence of albuminuria.

Achard has suggested that in carrying out the test for alimentary glycosuria we must not forget that the failure of sugar to appear after large amounts of glucose have been swallowed, may be due not solely to the vigor of the sugar-converting power as has been supposed, but wholly or partly to renal impermeability. In confirmation of this idea he reports a case of apoplexy with a small amount of sugar in the urine but a very large amount in the blood (5.1 grams per liter). This hyperglycemia he attributes to a lack of renal permeability, which was shown to be present in this case by the methylene blue test. McGuire declares himself inclined to ascribe the diminution of the glycosuria to inefficiency of the kidneys, and he suggests that the albuminuria

and its accompanying renal changes may be concerned in the production of coma.

Schupfer observed in cases of combined albuminuria and glycosuria experimentally produced that more sugar was excreted by the kidney which eliminated the least amount of albumin, the excretion of urea corresponding to that of sugar. When the kidney lesion was one-sided, the injured kidney excreted albumin and much less sugar than the uninjured one.

Ellinger and Seelig demonstrated that dogs from which the excretion of sugar was constant, after injection of cantharides showed a diminution in the output of sugar, the excretion falling in one case from 7.6 to 2 per cent. At the same time they were able to establish that the sugar content of the blood was markedly increased. They think that their results prove that the kidney lesion caused by the cantharides led to a diminution in the glycosuria, while the glycemia still existed. Along the same line as the foregoing are the experiments of Richter, who, after previously damaging the kidneys, produced glycosuria by means of phloridzin and observed that the excretion of sugar almost constantly began later than in control animals, and did not reach so high a point.

In an attempt to solve the question of the existence of a renal form of diabetes Klemperer administered as much as 10 grams of phloridzin to each of ten subjects having granular kidneys, and there was no reaction in seven. That three of his patients developed glycosuria he thought might be due to portions of the kidneys having been still unaffected by the morbid process. Klemperer gave phloridzin by the mouth. Pognat and Ravilliod administered phloridzin subcutaneously to twenty-one cases of renal disease, and in no case was the excretion of sugar normal in amount, and they conclude that with renal disease phloridzin glycosuria is either diminished in amount or entirely absent. In thirteen of the cases a marked diminution was observed, and in eight cases it failed entirely to appear. The experiments of Achard and Delamarre, performed in the same manner as the above, gave somewhat similar results. In five cases of interstitial nephritis four showed a marked hypoglycosuria, and in one case no glycosuria resulted. In four cases of acute tubal nephritis three showed hypoglycosuria, and in one case no glycosuria resulted. Of nine cases of chronic tubal nephritis five were hypoglycosuric, and four non-glycosuric.

From these collected observations the inference seems clearly justifiable that increased tolerance for carbohydrates and diminution of glycosuria in the presence of albuminuria is due to lesion of the kidney, which operates to cause diminished permeability of the renal secreting structure, not alone to sugar, but also to urea and other extractives, with accumulation of these bodies in the blood.

In the light of this deduction, an entirely new importance becomes attached to albuminuria and its associated renal changes in diabetes. The development of albuminuria in diabetes at once assumes a new and greater prognostic significance, especially in grave cases of the disease, where it may be of importance in the production of coma, by causing a heaping up of sugar and toxins in the blood. We have seen that there are constantly present in fatal cases of diabetic coma inflammatory and necrotic changes in the renal gland structure, comprising most frequently the epithelial necrosis of Ebstein, and less frequently hypertrophy with hyperemia and the degenerative lesion of Ehrlich. That these changes are not merely accidental is attested

by the constancy of their occurrence, some form being almost invariably present. We have also seen that albuminuria invariably develops before the advent of coma. Grube, Williamson, Stokvis, Kleen and others regard its occurrence at this stage as constant, and their opinion is verified by practical experience. Personally, I have never seen coma develop without albumin being present in the urine, usually associated with casts. Kuelz first pointed out the frequent association of renal casts with diabetic coma, and they are now regarded as a sign of decided significance. We may regard the albuminuria and casts, under these circumstances, as the visible outward evidence of the renal gland changes. The results of experimentation already detailed point to the fact that the kidney lesions are inimical to the glycosuria, and frequently interfere completely with transudation of the sugar. Equal proof exists that urea, and presumably other organic extractives, suffer a diminution in the urine. It is a matter of common observation that immediately preceding the development of coma, a marked fall in renal efficiency takes place. Not only does the sugar diminish in quantity, but the quantity of urine, the urea and other urinary solids suffer a decided and material reduction. Dreschfeld has observed in two cases that at the outset of coma acetone and the ferric chlorid reaction entirely disappeared from the urine. No corroborative evidence is at hand regarding this interesting observation. Certain it is, however, that preceding coma we invariably have renal conditions established with albuminuria and casts, and with decided reduction in renal permeability. To the writer abundant proof exists in these associated renal changes and clinical developments for the belief that the kidney alterations are an invariable antecedent of diabetic coma, and frequently its determining factor, by causing a sudden and marked diminution in renal permeability and a heaping up in the blood of the diabetic toxins. This view of the etiology of diabetic coma does not substitute a uremic for a purely diabetic origin, for the coma is certainly not uremic, although uremia may be a secondary contributing factor. It is frequently observed in grave diabetes that acetone and diacetic acid may be present for long periods of time without the incidence of coma, and not until the evidence of renal alterations make their appearance, does the patient develop profound evidence of coma. Experimental evidence regarding the toxicity of these bodies demonstrates that acetone, diacetic and oxybutyric acids can not be said to be toxic in the ordinary sense of the words, and that all of them can be taken by a healthy person in comparatively large amount without ill effects. It is shown, however, that if they are introduced into the blood of animals in large enough quantities they will produce symptoms closely resembling those of diabetic coma, and that they possess the power of producing albuminuria. It has been observed that acetone, diacetic and oxybutyric acids occurring in certain other states than diabetes occasionally give rise to all the graver symptoms of the coma of diabetes. It can not be doubted that these toxins are the cause of coma in diabetes, but it is the renal impermeability produced as a result of the irritation of these bodies in the blood and urine which is the final determining factor, by so increasing the toxemia through accumulation of these bodies in the blood as to initiate the coma:

CONCLUSIONS.

The following conclusions seem warrantable in the light of the facts here collected:

1. Albuminuria is a frequent development during the

progress of diabetes, over one-third of all cases manifesting this symptom at some time during their course.

2. Albuminuria in diabetes may, for clinical purposes, be divided into two varieties, toxic and degenerative.

3. Toxic albuminuria is generally of acute onset, arising during the later stages of the severe forms of the disease, as the result of irritation of the tubular epithelium, producing hyaline degeneration thereof, and due to the presence of acid toxins in the blood and urine.

4. Toxic albuminuria is an invariable precedent and accompaniment of coma, and may be regarded as of the gravest prognostic significance.

5. Toxic albuminuria with its associated renal changes, is the final determining cause of coma diabeticum, by producing a heaping up of sugar and toxins in the blood, through diminished permeability of the renal secreting structure.

6. Degenerative albuminuria occurs with great frequency during the progress of the mild type of the disease, and is of renal origin and produced by a gradually developing nephritis, which arises from prolonged hyperfunction and impairment of renal nutrition.

7. The appearance of albumin under these circumstances, while of little immediate significance, is of the greatest eventual importance, as indicating the intrusion of chronic nephritis into the already manifold clinical difficulties.

8. A few cases of albuminuria in diabetes may be produced by venous stasis due to cardiac asthenia, and under such circumstances will be associated with other evidence of heart failure.

9. Albuminuria in diabetes is never devoid of importance. It may be of the gravest significance, as we have seen, and is always of sufficient import to receive the earnest attention of the clinician.

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DISCUSSION.

DR. WILLIAM KRAUSS, Memphis, Tenn.—The percentage of cases of diabetes with albuminuria is perhaps greater than stated by the essayist, at least this has been my limited experience. I recall a case that no longer eliminated sugar on account of lost permeability, only albumin and casts. He had a horror of Bright's disease, and so I assured him that he was suffering from secondary nephritis, and under proper regimen he did fairly well. A student one unfortunate day told him nephritis and Bright's disease were one and the same thing. This depressed him so that he took to his bed and never recovered.

DR. F. R. WEBER, Milwaukee, Wis.—There is one point of great importance in this connection, the great influence of intercurrent febrile affections in cases of diabetes. Symptoms of intercurrent febrile affections, lasting three or four days, have a wonderful influence on these patients. I recall an instance

occurring in a young girl of eight years, who had diabetes in childhood, which was not a typical case at all. One evening the child was stricken with tonsillitis and died within twenty-four hours. In this case a physiologic albuminuria rapidly developed into a pathologic albuminuria, coma appearing before death.

DR. ARTHUR R. ELLIOTT, Chicago—My purpose in presenting the paper was to draw attention to the importance of albuminuria in diabetes; the association of the two having never been regarded hitherto as having any special prognostic significance. I believe albuminuria occurring in diabetes is never without significance. In acute cases it ought to be interpreted as a sign of approaching danger, and it should under all circumstances receive the earnest attention of the practitioner. The association of albuminuria with developing diabetic coma is too invariable to be mere coincidence. The conclusions of this communication, if they be correct, make the development of albuminuria one of the most important incidents of the diabetic state.

FRACTURE OF THE UPPER THIRD OF THE FEMUR SUCCESSFULLY TREATED WITH PLASTER OF PARIS WITHOUT EXTENSION.*

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Fractures of the shaft of the femur are very common accidents, despite the strength of the bone. The lower end of the bone is more frequently broken than the upper. Fractures of the upper third are usually due to indirect violence, and of the lower third to direct injury. The course of the fracture is usually oblique. Marked angular, rotary and longitudinal displacements usually attend these fractures. The lower fragment is usually drawn to the inner side and behind the upper; this tendency to deformity is greatest when the fracture occurs in the upper third.

Every kind and variety of fracture is liable to occur to the femur. On account of the large muscular attachments to the bone, displacements are nearly always present in fractures of the femur, and this is especially true in relation to fractures of the upper third, when the bone is tilted forward by the action of the psoas, and abducted and turned out by the gluteus maximus and external rotators of the thigh, at the same time the lower end is drawn upward and to the inner side by the hamstring and adductor muscles. The weight of the foot assists these adductors in the eversion of the foot, which is a symptom of this fracture. The proper appliances for treatment of fracture of the femur has caused wide and varied discussions with many diversified views; there are perhaps more special apparatus invented and discarded for the treatment of these fractures of the femur than for any other bone of the body, but the one point of extension is agreed on by all.

Recumbent position, a suitable bed with foot elevated, a hair mattress, not too soft, and immobilized retention of the fragments are indispensable to success.

The nates are prevented from sinking into the mattress by placing boards between the mattress and springs of the bed. Extension is usually obtained by cord and weight applied to the leg over a pulley attached to the foot of the bed. The foot of the bed is often raised six inches and the extension weight increased up to ten pounds in order to overcome the muscular contraction so that the fragments can be brought down together.

* Read at the Fifty-fourth Annual Session of the American Medical Association, in the Section on Surgery and Anatomy, and approved for publication by the Executive Committee: Drs. A. J. Ochsner, J. E. Moore and De Forest Willard.

Many surgeons have called attention to the difficulty in approximating the fractured ends, especially when they occur in the upper third of the femur, and in maintaining them in apposition after they have once been reduced. A casual glance through our different museums will prove that this is the seat of permanent deformities and usual shortening of the fractured limb, which has caused so many suits for damages and brought out so many cork-sole shoes and criticisms on surgeons.

Dr. Allis even considered this shortening and displacement so serious that he advised immediately cutting down on the fracture and wiring the bones together. In the case of a transverse fracture with lateral displacement and overlapping more than an ordinary extension apparatus to hold it in place is required. Direct lateral and anterior pressure is absolutely essential for the proper union and keeping in place of broken fragments. Under a general anesthetic, with the patient placed on a hard table and by making firm pressure on the upper fragment of the bone, while an assistant makes strong extension and counter-extension by pulling directly on the foot and body, the fragments can usually be approximated.

Owing to the mass of fleshy muscles which encompass the bone it is rather hard to say exactly when these fragments are in apposition. Good, all-around anterior lateral support for these fractured ends is best obtained by a plaster-of-paris dressing, after the bones have once been properly adjusted and swelling has abated. Owing to the thick muscular attachments of the bone, too much padding is not indicated at this point, provided the fracture is of the simple variety. The adipose tissue and muscles soon yield to the plaster dressing, which if at first seems rather tight, is soon loosened by the atrophy of the muscles and adipose tissue under rest and continued pressure, hence moderate support as can best be had by a plaster-of-paris dressing applied all around the limb, as a rule, gives better results than the long side splint, extension and sand bag and various other appliances in use. In the treatment of these cases the double inclined plane has been practically abandoned. Buck's extension by weight and pulley is still used by many surgeons. Dr. Hamilton's preference is for a long-sided splint extending from the foot to the axilla, much after the pattern of Liston's long extension splint. Valkman's sliding foot-rest is of great assistance in treatment of this fracture in many cases.

In a recent case of fracture of the upper third in a little boy 8 years old, with marked elevation of the upper fragment accompanied by eversion of the foot and drawing up of the lower fragment, I had a most successful result by the following simple method of one plaster-of-paris dressing which was not disturbed from the time of its application a few hours after the fracture until its removal four weeks after the accident, in which we found perfect union without any shortening. The little patient in pushing a swing that in its rebound came within six inches of the ground was caught under the swing-box seat, in which five or six other children were seated, the fracture being produced by direct violence, with some, though not very great, contusion of the soft parts.

The little patient was placed on a hard table with pelvic rest, the leg being held in extension; he was thoroughly relaxed with chloroform and the fracture reduced by extension, counter-extension and manipulation. While in this position a 2-inch Z. O. plaster was applied on both sides of the leg above the knee with a loop around the heel to provide for extension in the event of shortening; several roller bandages of padded cotton were ap-