When the patient recovered consciousness, she expressed great relief, which improvement was maintained for two days, when her condition became worse than it had been before. Suspecting, then, a venous dilatation, and, also, as the vision of that eye was lost, a hypopyon having appeared with the additional symptoms of sympathy in the other eye, I proposed enucleation, which was agreed to.

On January 30, Dr. Duggan again administered the chloroform, and I availed myself of the Volkmann spoon No. 1, to load and overset the ocular globe, because the nippers failed to grasp either conjunctiva or the tendon which was tearing it, and the spoon enabled an easier enucleation. I next proceeded to the examination of the cavity, and ascertained that there was dilatation of the ophthalmic vein of about one centimeter. I then abandoned the idea I had of binding the vessel that formed the aneurysm, as, having been prevented from giving the other treatment proposed by the authors, I was forced to leave matters as they were, which I particularly regretted, because, not only would the dilatation increase greatly through need of compression, but also the troubles consequent thereupon would follow, and probably result in the need of a further posterior operation. Fortunately, however, both for my patient and myself, I remembered the advice of Dr. G. Laurens, when the lateral breast comes open when ascending the mastoid prominence, and this seemed to be a similar case, notwithstanding the fact that he referred to the opening of the breast by accident, while in this instance it was through intent, besides which I had the advantage of being able to compress it by reason of a bony surface behind the vessel. The resolution was quickly followed by the operation. With a compress of bichlorid gauze in my left hand, and the spoon in my right, the dilatation was pulled out, producing a copious flow of blood. This, however, did not alarm me, as I expected and was prepared to control it, and did immediately, by compression with the gauze I held in my left hand. I immediately requested Dr. Duggan to assume charge of the compression, while I proceeded to thoroughly clean the operated part. I then raised the level of the compression above the borders of the orbit, placing over these a silver dollar to equalize the pressure at the center, and admit of free circulation in that locality. I then placed a thick wrapper of cotton and antiseptic bandage thereon.

After twelve days I removed these appliances, which had been retained all the while without producing any disagreeable odor, through my having kept them dampened with a solution of formol. There was a sudden hemorrhage, caused by the tearing of some fleshy blood clots. I applied a little glutol with fresh wrappers of gauze and cotton to the outside, which I held in place with a tight bandage. On February 17 I again removed this, without further flow of blood, applying a little glutol as before in the socket of the eye, with a gauze covering, but without a compressing bandage.

On February 20 I was again called in, the patient complaining of a slight pain, which she feared would increase. With the object of relieving this, and hastening the cicatrix retraction, I prescribed the insertion of fifteen to twenty drops of a weak solution of tannic acid and cocain. On the 28th I found everything progressing excellently, the patient informing me that she had discontinued the use of my last prescription after two days, because she had ceased to experience the trouble. Not a drop of pus was found after the removal of the bandages.

There remains at present, as the only trace of the severe illness, a slight humming noise in the ear, which she states has already greatly diminished, and during a good portion of the day disappears entirely, enabling her to sleep without difficulty.

As another incident, which may be of interest, I would mention the following: On Oct. 17, 1896, two partners and myself were called into consultation with Dr. Duggan, who was attending a brother of the patient referred to above. Having discovered that he was suffering from venous varices of the floor of the mouth, we suggested intervention, to which the family would not agree. He died the following day of asphyxia. He was 32 years of age.

Deductions.—It is undoubtedly true that the members of this family are predisposed to vascular dilatations. It is probable that the dilatation of the vein was somewhat lengthened in the interior of the skull, which produced the humming of the ear mentioned before. The cure was doubtless due to the suppression of the more extended part of the vein, and the formation of coagulated blood in the balance of the dilatation, and the presence of the gauze in the open extreme, and the lengthening of the coagulation to the walls of the vessel, which was proved by the complete cessation of the humming noise.

SOME OF THE ASPECTS OF RENAL INADE- QUACY FROM A NEUROPATHIC STANDPOINT.*

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The subject of renal inadequacy, a term first used, I believe, by Sir Andrew Clark, has become of more and more importance as we have grown increasingly familiar with the effects of autointoxication, and have learned to recognize how great a part the retained products of metabolism—especially those which result from incomplete retrograde change—play in the inception of diseased conditions heretofore attributed either to other causes or looked upon as arising *de novo*. This condition of the kidneys, I believe to be represented in the inability of these organs to completely eliminate the waste products of the body, either because they are themselves the seat of disease or because the products of destructive metabolism come to the kidney in such form chemically, as to be unable to pass through the renal epithelium or to complete their elaboration into those compounds which can be secreted and excreted by the functional portion of the tubules. There are many ways in which this inadequacy can be brought about, but let us first consider the function of the kidney and its relation to the welfare of the rest of the organism. If we accept the current teaching of physiology as to the structure of the epithelial lining of certain portions of the tubule, the kidney is a secretory as well as an excretory organ and has something to do with the elaboration and reduction of the compounded elements which are brought to it by the blood. Next to the brain, the kidney receives a proportionately larger and more direct blood-supply than any other organ, and the blood-vessels are so arranged in their final distribution as to expose the largest possible surface in contact with the functional part of the organ. It receives its nerve-supply from the same sources, both ganglionic and spinal, as the other abdominal viscera, and is surmounted by a glandular structure of whose function we know nothing accurately,

except that in its blood and nervous supply it is intimately associated with the kidney. It is also worthy of note that the suprarenal body receives, as a part of its nerve-supply, filaments from the pneumogastric and phrenic nerves. Finally, it is important to remember that each pyramid, with the cortex above it, is practically a separate kidney, capable, so long as its blood-supply is not cut off, of performing all of the functions of the organ as a whole. Like the brain, the kidney has a function which is absolutely essential to existence, and this function can not be vicariously performed except to an extremely limited extent. Besides, from the nature of the secretion and excretion from the kidney, any interference with the normal performance of its function will necessarily have a more disastrous effect on the rest of the organism than would disturbance of any other one of the vegetative processes. Furthermore, the intimate association of the kidney through its nerve-supply, not only with the abdominal viscera, but also with the more directly vital processes of circulation and respiration, shows how important its function is to the general welfare of the organism. Without going into the details of the chemistry of the urine and the nature of its constituents, it suffices for my purpose to state that, in both health and disease, those constituents whose elimination is most essential to the welfare of the organism appear in combination with uric, phosphoric, sulphuric, and hydrochloric acids, and as urea representing nitrogenous waste, the relative amounts of these substances eliminated during the twenty-four hours, and their proportion to each other, indicating not only the degree of functional activity of the kidneys, but also the nature and completeness of the changes going on in the organism as a whole. When we take into consideration the intimate association of the function of the kidneys with the vegetative organs, as shown by their intimately related nerve-supply, and indirectly by their association with the general nervous system, we can realize how important the function of these organs is to the welfare of the general organism, and conversely how they in their turn must necessarily be subject to disturbance of their function and normal rate of activity by extra demands resulting from derangement of some one of the vital functions due to visceral disease, excessive somatic change resulting from overwork or overstrain, involvement of the nervous system through impaired nutrition or excessive mental activity, or all of these causes acting together to interfere with metabolism and render incomplete the formative process in the products of retrograde change which it is the function of the kidneys to eliminate. It is a well-known fact in physiology that the activity of the function of the kidneys is greatly influenced by the amount of blood circulating through them, and that the amount of blood in them may be so considerable as to materially increase the size of the organ; while at the same time the tension of the blood-vessels may be so great as to seriously reduce the functional activity of the kidney or check it altogether. The same result may also be produced by vasomotor paresis. It has also been shown experimentally that the functional activity of the kidney may be checked or arrested by disturbance in the nervous system, without the presence of any diseased condition in the kidney. The aberration and sometimes abeyance of function in the kidneys as seen in hysteria, is well known, in some cases the secretion of urine being completely suppressed for considerable periods, while in others there will be polyuria, and both of these conditions occurring without apparent physical cause adequate to produce them, unaccom-

panied by any of the symptoms usually associated with anuria or polyuria.

Two individuals, apparently equally healthy, may sit down to a hearty meal, each one eating of such dishes as he enjoys. Both may be called upon immediately afterward for some unusual exertion, either mental or physical. The one will have an attack of acute indigestion, accompanied by the usual symptoms; the other may or may not have these symptoms, but he will have others. He may become stupid, suffer from intense headache with conjunctival injection, slow pulse with flushed face and body surface, respiration more or less labored. Mentally the patient will be confused, irritable, restless, unable to think or apply himself. After a time he falls into a heavy sleep and awakens comparatively free from discomfort, with the exception of some dizziness and soreness of the scalp. Instead of this symptom-group, there may be another. In the midst of some occupation he will become dizzy, the action of the heart will become rapid and feeble, the surface of the body become pale and covered with sweat, and the patient tumble over in a syncopal attack. He may recover rapidly from this or remain weak and confused for several days; the heart's action continuing rapid and weak, and the pulse feeble and of low tension. In either case there will be, during the persistence of the symptoms, diminution of the secretion of urine, and their disappearance will be accompanied by copious urinary discharge. The examination of the urine will possibly disclose the presence of albumin and, microscopically, a few leucocytes, but usually all that is found is an increase in the amount of phosphoric acid and sulphates, with diminution in the quantity of chlorids and urea. There may or may not be an increase of indican. If the symptoms disappear within forty-eight hours, and the constituents of the urine assume normal proportion, it is safe to assume that we are dealing with a temporary form of renal inadequacy, resulting from imperfect performance of the function of the kidney on account of renal congestion, or engorgement following vasomotor peresis. It can be easily understood how the blood, overloaded with the products of retrograde change, some of which are not completely reduced, will act as an irritant to the general nervous system, producing either vasomotor stimulation with increased tension, or paresis to be followed by blood stasis, and either of these affecting the kidney with its relatively large blood-supply, thus interfering with elimination and adding to the intoxication. If the kidney structure is intact, there will be a gradual resumption of function with increased elimination. But should the kidney be the seat of degenerative change and the vitality of the organism lowered by disease, the effect of intoxication will be more profound, the interference with the function of the kidney greater, with the resulting convulsion followed by paralysis, from which there is slow recovery; or there may be, instead, coma, vasomotor paresis, dilatation of the right heart, pulmonary edema and death. In some people these outbreaks are occasional and short-lived, but there are many others in whom they are of frequent occurrence. In this class of cases the renal inadequacy is habitual and accompanies any unusual or extended effort, either mental or physical, but especially mental. The patients do not seem to be so much the victims of nervous instability as lack of nervous force, with a limited potentiality for sustained effort. In these people the renal inadequacy may be said to be congenital. I have never had an opportunity to follow such a case to the end as yet, because in all with whom I have been familiar I have either

failed to get a post-mortem or other conditions have existed which have modified the changes found in the kidney. Reasoning from analogy, however, such patients should present a purely atrophic change with shrinkage of the kidney, destruction of the epithelial lining of the tubules and capillaries, but no increase of connective tissue; approximately, the same conditions that exist in the so-called granular kidney, which to my mind represents the congenitally defective kidney. According to West[1]: "Granular kidney is a disease of great importance on account of its frequency—a frequency which is by no means adequately recognized. Post-mortem it is often discovered when not suspected. It is often in itself a cause of death, even of sudden death, and it often explains why death has happened in other diseases which otherwise might not have proved fatal. During life it is often discovered unexpectedly if looked for, it is often overlooked if not suspected, and it often explains a case which has been a puzzle until granular kidney gave the key. For all these reasons granular kidney is not only one of the most interesting of diseases but also one of the most important." My own experience coincides with this declaration, but my observations suggest a different explanation of the origin and variations in the extent and nature of the changes in the kidney. The probability of primary defect in the structure of the kidney is shown in the persistence of lobulation in the kidneys of the adult. It is quite a common occurrence in our post-mortem work to find lobulated kidneys, and in some the lobulation is as marked as that usually found in the calf or pig; and further, the cortex of these lobulated kidneys is disproportionately thin. What the exact histologic difference between such a kidney and a perfectly developed one is, it is practically impossible to determine, because it would be only by the merest accident that an opportunity might occur to study such a kidney free from gross secondary changes. Another argument in favor of the existence of primary renal inadequacy of varying degree is the fact that while the conditions of life are practically similar for all people subject to the influence of a uniform environment, only a comparatively small number out of any given group will suffer either from simple renal inadequacy or the more conspicuous forms due to degenerative changes in the kidneys. Only a small proportion of the cases in any given epidemic of one of the exanthematous diseases suffer from nephritis, and they are not necessarily the severe ones. Even those conditions resulting from the exigencies of modern civilization, with the strain of industrial and social competition, which are reckoned as the most common causes of degenerative disease of the kidneys, while practically universally operable, really disastrously affect only a comparatively small number of those who are subject to their baneful influence. Again, in the form of renal inadequacy which results in puerperal eclampsia, how few women there are out of the whole number confined during a given period who will have convulsions! It would also be interesting to know the relative number of deaths following puerperal convulsions, occurring among primiparæ and multiparæ, and the number of cases in each class in which the kidney showed evidence of antecedent degenerative change. From an analysis of the cases coming to post-mortem in this hospital during the past seven years, I have found that next to the brain the kidney is more often and seriously involved in the degenerative process than any other organ, and that the extent of the degeneration in the kidney as well as the persistence of

lobulation is always in proportion to the same change in the brain; also that the more marked the defect in the brain development in the individual, the earlier will be the changes in the kidney and the more certain the patient is to suffer from the effects of renal inadequacy. Again, the law governing the form in which the degenerative process will be manifested, as stated by me in another connection[2], applies equally well and explains why in one case we find a purely atrophic change involving the functional portion of the kidney—in another a structural change resulting in connective tissue increase primarily, with involvement of the functional portion of the kidney, secondarily. This is why, in so-called "granular kidney," the kidney is sometimes small and at others large, while the clinical picture and result of uranalysis is the same. In one case the change is atrophic, especially involving the tubules with only apparent increase of connective tissue, and the other hypertrophic with connective tissue increase. These are the same changes as are found in the kidneys of old people, the difference being that in the one case there is a normal senescence more or less modified by the way the life of the individual was conditioned, and in the other a premature senescence modified by the degree of defect in the individual and its influence in limiting the functional and structural potentiality of the organ. In considering the availability of the class of cases found in an institution of this kind for study, it seemed to me that they offered an opportunity to elucidate functional and degenerative changes in the kidney not to be found among the population of a general hospital, because the existence of degeneracy and its results would exaggerate the changes found in the more normal individual, thus magnifying them and making them more conspicuous, while at the same time the régime of the hospital, uniformity of diet and freedom from excess, together with the simple vegetative existence led by this class of patients would free the subjects of study from the complications resulting from the exigencies of social and industrial competition; and the prolonged existence of the patient would allow of a more comprehensive and well-defined development of the pathologic changes, thus making the clinical picture more clear and definite and the morbid anatomic changes more complete.

During the four years from Aug. 1, 1894, to Aug. 1, 1898, there were admitted to this hospital, as new patients, 618 men and 488 women, total 1106, and in all of these a careful examination was made at intervals during the first month of the residence of the patient in the hospital. The following table contains an analysis of the changes in the urine:

	Men.	Women.	Total.
Specific gravity increased	338	210	548
Specific gravity decreased	76	104	180
Urea increased	26	12	38
Urea decreased	337	260	597
Indican increased	203	162	365
Albumen present	268	204	472
Sugar present	138	62	200

A complete quantitative estimate of the amount and relative proportion of the different salts in the urine was not made during the entire quadrennial period, so I have reserved for a separate table the analysis of a series of cases in which changes in the kidneys are known to have taken place. When it is taken into consideration that in the large majority of these patients there was nothing in the history or symptoms at the time of admission to

[1] West: "Lettsomian Lectures," London Lancet, Feb. 11, 1899.

[2] Tomlinson: "Insanity and Phthisis, their Transmutation, Concurrence and Co-existence. Jour. of Nervous and Mental Disease, October, 1895.

the hospital to indicate that there was any renal disease present, that about 60 per cent. of them either entirely recovered mentally or sufficiently to enable them to get along outside of the hospital, that the death-rate among them was approximately 3 per cent., while out of the whole number considered—1106—the specific gravity of the urine was decreased in 180, urea was diminished in quantity in 597, albumin was present in 204 and sugar in 200 cases, it will be seen that apparently considerable interference with the function of the kidney may be present without serious degenerative change in the organ and that these diseased conditions may entirely disappear, leaving behind them no evidence of chronic renal change. At the same time it is a fact in our experience that when a patient comes to the hospital, in whom uranalysis shows marked evidence of renal inadequacy, the disappearance of the acute mental symptoms almost invariably goes on *pari passu* with the restoration of the constituents of the urine to their normal relation, both as to quantity and quality. In all of the cases of acute delirium admitted to the hospital during the past two years, the uranalysis has shown marked evidence of renal inadequacy; while in those cases where the termination has been fatal, either uremia has been the immediate cause of death, or else bronchopneumonia or pulmonary edema, with marked degenerative change in the kidneys. When it is taken into consideration that the symptoms associated with uremic poisoning, independent of mental disease, are practically the same, including restlessness, insomnia, muscular twitchings, headache, low muttering delirium, and if fatal, coma, pulmonary edema and death, it seems to me a rational conclusion that while in the one case the involvement of the nervous system antedated the renal inadequacy, and in the latter the disease of the kidneys brought about the involvement of the nervous system, yet in both cases death resulted from the inability of the kidney to perform its function. The following table shows a comparison between a series of cases in which there was marked evidence of nephritis clinically and another series in which no such evidence was apparent. In the thirty-eight patients presenting clinical evidence of nephritis, the symptoms varied from emaciation with indigestion, constipation, edema of the eyelids and ankles, to convulsions, coma and paralysis. In one patient, in whom a recent examination of the urine discovered nothing abnormal, there had just been recovery from a severe uremic attack, ushered in by convulsions and these followed by a hemiplegia with profound stupor. In another, who has since died and where the necropsy disclosed extensive degenerative changes in the kidneys, the urine was comparatively normal for a month before death, although he was helpless from a hemiplegia which the post-mortem examination showed to be uremic and not hemorrhagic in its origin.

The following table shows the result of a careful examination of the twenty-four-hours-urine in eighty-three selected cases. In all of these the diet and general environment of the patients was practically uniform at the time when the observations and examination of the urine was made:

EXAMINATION OF URINE.

	Clinical evidence of nephritis. 38 cases.	No clinical evidence of nephritis. 45 cases.
Quantity increased	2	3
Quantity decreased	29	28
Quantity normal	7	14
Specific gravity increased	19	16
Specific gravity decreased	9	9
Specific gravity normal	10	20
Urea increased	4	8
Urea decreased	7	11
Urea normal	27	25
Phosphoric acid increased	7	7
Phosphoric acid decreased	4	9
Phosphoric acid normal	26	29
Sulphates increased	24	25
Sulphates decreased	13	20
Sulphates normal	1	0
Chlorids increased	14	12
Chlorids decreased	4	12
Chlorids normal	20	21
Albumin	3	0
Sugar	7	0

The comparison made in the table apparently negatives the assumption with which I begin this paper, but really it does not! In the first place we must consider that these patients were all placed under practically uniform conditions before the examination of the urine. Next, in placing them in one or the other category, only those symptoms which are recognized as significant of nephritis were considered. I quote from a paper of Dr. Richard C. Cabot[3], as follows: "It will be readily admitted that the means at present at our disposal for the diagnosis of nephritis are far from satisfactory. On the one hand the autopsy may reveal a nephritis in a case which during life showed no albumin in the urine; on the other hand, we may have albumin and casts and yet the autopsy reveal no nephritis. The estimation of urea or of total solids can no longer have for us that clean-cut significance which once it had. For it has been abundantly proven that urea may be greatly below normal for years and yet no uremic paroxysm occur, while on the other hand a uremic convulsion may come upon a patient out of a clear sky when the urine contains a normal amount of urea."

Finally, in all of the patients suffering from renal inadequacy, in whom there has been a definite, well-marked attack of uremia, there was, in the beginning of the attack, decrease in the quantity of urine, decrease in the percentage of urea and chlorids with increase in the percentage of phosphates and sulphates. Besides, several placed in the category of those furnishing no clinical evidence of nephritis showed this same disturbance of the relative proportion of the solid constituents of the urine, and one of these has since died. At the necropsy, marked evidence of advanced degenerative change was found in the kidneys, although there were no clinical evidences of nephritis before death.

The following cases illustrate the varying aspects of renal inadequacy in its graver forms:

M. W., aged 58 years, a widow, was the victim of circular insanity. She had spent the greater part of the past twenty years in the hospital and was last admitted Sept. 4, 1896, said to have just recovered from typhoid fever. The action of the heart was irregular and intermittent, ankles edematous, and appetite and digestion impaired. She was constipated and could not sleep. At this time the urine was decreased in quantity, contained 1 per cent. of albumin, urea 1 per cent., chlorids 8 per cent. Microscopically there was renal epithelium. She improved rapidly, however, and was in fair health until in June, 1898, when she became weak, stupid, took nourishment poorly and complained of precordial distress. The skin was cold and clammy, the lips and finger ends cyanosed. The twenty-four hours' urine at this time amounted to 900 c. c., sp. gr. 1031, a trace of albumin,

³ Cabot: "The Use of Methylene Blue in the Diagnosis of Renal Disease." St. Paul Med. Jour., February, 1899.

urea 3 per cent., phosphoric acid .2 per cent., sulphates 20 per cent, chlorids 5 per cent. The microscope showed granular epithelium and leucocytes. The patient grew weaker, the area of cardiac dulness increased, there was a murmur at the apex, systolic in time and transmitted to the axilla, also a systolic murmur at the base, and she complained of pain over the sternum. The abdomen was distended and tympanitic, but not tender, the tongue heavily coated, the lips blue, and there were petechiæ scattered over the body and limbs. The temperature was 101 F. in the evening, the pulse rapid and feeble, and she was very stupid. Her physical condition did not change materially during the next month, except that the stupor increased and there was occasional twitching of the facial and flexor muscles of the limbs. The urine became still further reduced in amount, but there was no material change in the proportional amount of the constituent elements. Granular and hyaline casts, however, began to appear. On August 4, the week she died, the sp. gr. of the urine was 1023, there was no albumin, urea was 3 per cent., phosphoric acid .2 per cent., sulphates 1.5 per cent., chlorids 9 per cent. During the last week of her life she had frequent attacks of dyspnea and there was marked decrease in the amount of urine with retention. She took nourishment well, however, until an hour before she died. At 9 a.m., August 9, she became rigid, and passed rapidly into a tonic convulsion. The pupils were dilated but unequal, the urine was passed involuntarily, there was expulsion of frothy saliva from the mouth, and she was cyanosed. In about twelve minutes the muscles relaxed, she gasped once or twice and was dead.

The condition of the brain, post-mortem, was what we have come to consider characteristic in death from uremia. There was increase of cerebrospinal fluid, distension of the pial vessels with dark, fluid blood, the sinuses in the same condition. The membranes and brain were edematous and soggy, the ependyma in the ventricles edematous, especially in the floor of the fourth ventricle, while the blood-vessels were deeply injected and showed well-marked puncta on section of the medulla. The heart weighed 396 grams, the right ventricle was filled with fluid blood, was dilated and its walls thin; the left side of the heart was hypertrophied, the aortic valve incompetent, the other valves healthy. There was marked atheroma of the aorta and it was dilated. There was fluid in both chest and abdominal cavities, and the viscera showed evidence of degenerative change. The right kidney weighed 113 grams; the capsule was slightly adherent. The kidney was lobulated, its surface covered with small infarcts and cyst scars, the cortex very thin, the pyramids illy-defied and the pelvis dilated. The same physical conditions were present in the left kidney. The bladder was empty.

(To be continued.)

TREATMENT OF EPITHELIAL AFFECTIONS WITH FORMALIN.—In the *Deutsche Med. Woch.,* of December 5, Daniel states that he found that if formalin was applied to warts, they soon dropped off, leaving a smooth unscarred surface. Other epithelial affections were treated with complete success in every case: epitheliomata, condylomata, sycosis, etc. Further tests are now in progress, but Daniels considers himself justified in calling attention to formalin as absolutely reliable, easily managed, comparatively painless and leaving no scars, in the treatment of epithelial hypertrophies and the transformation of suppurating affections.

SHOULDER-HUMERO-SCAPULA ARTICULATION.

SOME OF THE COMPLICATIONS AND SEQUELÆ ATTENDING OR FOLLOWING REDUCIBLE OR IRREDUCIBLE DISLOCATIONS, WITH A BRIEF REVIEW OF THE VARIOUS MODERN OPERATIVE MEASURES NOW EMPLOYED FOR THEIR TREATMENT.

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RESISTANCE TO REDUCTION.

One of the remarkable phenomena of shoulder luxations is the inexplicable freak we note here in efforts in reduction. Occasionally we meet with cases which stubbornly resist all our efforts. We have seen some very skilful surgeons utterly fail, others fracture the humerus. It has never been my misfortune to fail to effect reduction when the case has come under my care any time during the first week. I have seen about a dozen, old irreducible cases with fairly good functional results in all.

Thorbone notes that it is a comparatively common experience in hospital practice, to meet with unreduced shoulders of old standing, the patient applying for relief at periods varying from eight to twelve weeks or more after the injury. The condition was not before discovered or the reduction had failed.

Brechet cautions us not to set a case aside as irreducible after our first efforts fail, lest, after muscular spasm passes off and the patient is composed, he may fall into the hands of another surgeon who may very easily replace the bone. Several times cases have come under my observation in hospital service, which had resisted every description of force and manipulation outside, where the bone was very easily reduced by gentle handling. Stimson records an instance in which a man was returned to bed, all efforts at reducing having proven fruitless. After a good night's rest he awoke to find that spontaneous reduction had occurred.

Platt, who has written the latest and best contribution on "Dislocations of the Arm," cites an instance in which manipulation and powerful extension utterly failed to reduce the bone. The man was then sent up stairs to the operating-room, that anesthetics might be given him and renewed efforts made; but when he reached the room, the bone had gone back into place. This was a recurrent case, the humerus having before been dislocated sixteen times.

Stimson advises us to "humor" the muscles in these cases, and never employ great force until all the milder means have been exhausted.[5]

FREQUENCY OF FAILURE IN REDUCTION.

It is interesting to note, in making a retrospect of the literature on shoulder reduction, that about the same proportion of failures to secure reduction occurs now as in preanesthetic times.

ON THE TIME LIMIT WHEN REDUCTION MAY BE SAFELY UNDERTAKEN.

The most noted surgeons of the past, with a few exceptions, recommended that it was unsafe to attempt a shoulder reduction after three months. In modern times, Delbet, Verneuil, and Broca adopt the same limit. Richet sagely observes that if "Dupuytren and Billroth succeeded at a much later date, it was well for their patients."

The number of published cases in which dire results