CONTENTS

CALCULUS DISEASE OF THE KIDNEY ........................................... 3
    Frank Capobianco, M.D.

DENSE CATARACTS IN 6 YEAR OLD ........................................... 11
    Robert R. Muschlitz, M.D.

THE ROLE OF PLASTIC SURGERY IN THE TREATMENT OF
CHRONIC LEG ULCERS ..................................................... 13
    Kerwin Marcks, M.D. and Allan Trevaskis, M.D.

A CASE OF PEMPHIGUS ...................................................... 17
    Peter J. Miraldo, M.D.

* THE TREATMENT OF THE PREVIOUSLY DISCUSSED CASE
  OF PEMPHIGUS ......................................................... 19
    Clyde H. Kelchner, M.D.

INTRAOCULAR USE OF ANTIBIOTICS ..................................... 24
    Robert Shoemaker, M.D., F.A.C.S.
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CALCULUS DISEASE OF THE KIDNEY
FRANK CAPOBIANCO, M. D.

The problem of Urinary Calculi presented itself as early as 7000 years ago. Recently in an unearthed 7,000 year-old grave of an Egyptian boy a vesical calculus was discovered in his bony pelvis. This stone had a uric acid nucleus and was surrounded by a thick layer of Calcium Oxalate and Ammonium Magnesium Phosphate.

The English Professor Elliot Smith concluded that the problem of stone was uncommon in Ancient Egypt since in examining 9000 mummies he found only 2 stone cases. However, this is questioned since only the rich could afford embalming and in all likelihood the problem of stone was more prevalent among the poor.

The Hindoos of India were considered in their time expert surgeons. They described four types of renal stones; the first three types simulated phosphatic, oxalate and uric acid calculi. Somewhere about the commencement of the Christian Era the operation of suprapubic lithotomy was described in the Ayur Veda. Celsus of Rome, in the First Century A.D., described an account of the operation of lithotomy in his encyclopedia of medicine “De Medicina”. Galen described symptoms of renal and vesical calculi; preferring “stone solvents” rather than surgery for their treatment. Avicemia, brought the knowledge of calculus disease to date about 750 A.D. This occurred during the rise of Arabic Culture. In his work “Canon Medicine” he recognized that renal and vesical calculi had a common origin. He enlarged on the difficulties and dangers of lithotomy.

In Greece, in the years 460 to 370 B.C., both renal and vesical calculus were described by Hippocrates. He left the surgery of renal calculi to the specialists. The latter was mentioned in the Hippocratic oath. “Neither will I cut them that have stone, but will leave this operation to those who are accustomed to perform it.”

Tratula, a European female physician about the middle of the 12th century and famous for her work on pelvic diseases, described stone in the male bladder and was familiar with the lithotomy operation.

The first suprapubic cystotomy in Europe was performed by Pierre Franco, born in 1500.

In the 17th century, and for eight generations, the family of Collots were expert lithotomists. They handed down closely guarded secrets from father to son. During their time, the government of France was petitioned to have all stone patients treated only by them. The Hotel
Dieu was the hospital started by the Collots for the purpose of treating stone cases. Frere Jacques performed many lithotomies and in direct contrast to the Collots he was always ready to demonstrate his methods to others.

In the 18th century, Cheselden was the most distinguished surgeon attached to St. Thomas Hospital. He did more than any other man of his day to advance the surgery of vesical calculus. John Douglas, at the same time a surgeon to Westminster Hospital, was the first surgeon in England to perform suprapubic lithotomy.

Frere Come had 316 cures out of 330 operations and in 1753 opened a hospital for stone in Paris, in which over 1000 patients were operated on for stone. He performed both perineal and suprapubic surgery. He was the last of the lithotomists. Surgery of the stone was then eagerly taken up by general surgeons in the 18th and greater part of the 19th centuries. This operation was so important at that time, it took precedence over all other surgery. Calculus disease ran rampant in Norfolk and along the east coast of England most likely due to defective diet, especially so in childhood.

Many prescriptions were written and concocted for dissolving stones but without much success. Uric acid was first isolated from urine in 1776 and found in urinary concretions in 1797 by Wollaston. Phosphates had previously been discovered by the alchemists by distilling urine with lime. Cystine was discovered in certain calculi by Wollaston in 1810.

With the above knowledge of the composition of urinary calculi, many solutions were attempted for the dissolution of the stones, but the latter only resulted in injury to the bladder.

Attempts were then made later to break off small fragments of the stone for chemical analysis. This was more successful and ultimately led Civiale, 1818, to introduce the first lithotrite. In 1878 Bigelow of Boston revolutionized the operation by improving the method for the extraction of fragments. The first positive x-ray diagnosis of renal calculus was made by MacIntyre in 1896.

FACTS ABOUT RENAL CALCULI

One of the commonest and the most important lesions of the kidney is stone formation. Renal stone occurs as a primary lesion or as a complication of many conditions. The underlying causes for the formation of most urinary calculi are unknown. Stones seem to form in the urinary tract as the result of changes or defects in body metabolism. Specific types of infections result in some urinary calculi.
Stone has been produced in the urinary tract of experimental animals under the influence of dietary changes that include (1) vitamin deficiencies (2) the ingestion of large amount of crystalline material. These observations on animals have been applied to man and have shed some light on the problem of urinary calculi, especially the geographical incidence and periodic prevalence of calculus disease in man.

None of the theories for etiology of urinary calculi, however, satisfactorily accounts for the different kinds of stones that form, nor the diverse conditions under which urinary calculi develop. More conclusive study of the body chemistry and body metabolism must be carried out to more definitely understand the cause for urinary calculi.

Many theories have been advanced to account for the genesis of renal calculi. No one theory has been accepted generally. Some facts are known about how a stone in the kidney grows, but little is known concerning the origin of a renal calculus.

**ETIOLOGY OF RENTAL CALCULI**

The etiology of renal calculi is not clearly understood. The varied physiological states and the diversity of the chemical composition under which they occur suggests that many different factors are responsible for stone formation. It seems quite evident that renal calculi are the result of the precipitation of salts normally present in the urine, although again the initiating mechanism or process within the kidney is unknown.

Crystalline deposits are considered to be the result of the physical changes in the relationship of crystalloids and colloids in the urine. But this theory does not explain how this phenomenon occurs. The theory that there exists in the urine a protective colloid mechanism which permits substances to remain in solution considerably in excess of their normal solubility and that the derangement of this mechanism allows the precipitation of these substances, carries with it little, real supportive evidence.

Urinary stasis or obstruction to free flow of urine was considered a factor in stone formation, but obstructive lesions so commonly are absent in the presence of stone that it has been argued that stasis is perhaps more often the result of stone rather than the cause.

Urinary calculi can readily be produced experimentally in animals on deficient diets. Also, the incidence of stone increases when in man there is evidence of dietary deficiency. But the difficult thing to under-
stand is why, then, stones should be prevalent in individuals who exist on apparently perfectly adequate diets.

Infection likewise produces urinary tract stones, as is evident when urea-splitting organisms are present in the urine. These urea-splitting organisms break up the urea secreted in the urine and produce ammonia which when formed alkalizes the urine and facilitates the precipitation of crystals of calcium phosphate and magnesium ammonium phosphate and calcium carbonate. In the presence of urea-splitting organisms the incidence of stone formation is very high. However, with all the foregoing evidence of stone formation in the presence of infection what is the reason for stone formation in culturally sterile urines? This is not known, or at any rate no one has as yet been bold enough to explain it. The urine is a good culture medium for bacteria but most organism will grow less rapidly when the pH of the urine is low.

Changes in the Urinary Organs and Stone Congenital abnormalities of the urinary organs favor the retention of urine, appearing more frequent in these cases than where organs are normally developed. Stone is six times more common in horse-shoe kidney than in normal organs. Stone is found in pelvic kidneys. Acquired or congenital changes in the bladder have also an influence over the frequency with which stone is met within that organ. Vesical calculi were found in 67 out of 728 cases of prostatic enlargement. This is 9.2%. Stone was found in 12.6% of 220 cases of vesical diverticulum. In both of these conditions infection soon sets in and persists until a radical operation is performed, so that the condition of sepsis, plus stagnation, are always present, and this combination is most favorable for stone formation.

Age and Stone Stone is the disease of extremes of life. They are most common in childhood; comparatively more common in middle life, and frequent in old age. More recent by a more rational diet for children made stone almost non-existent in childhood, especially so in this country.

Sex Vesical calculi are more common in the male than the female. Renal calculi is about the same in both sexes. The important difference is that severely infected calculi are more common in the female than in the male.

Para-thyroid disease is an example of a condition which so disturbs calcium metabolism that urinary calculus complicates many of the cases of hyperparathyroidism. Excessive amounts of calcium in the diets, decalcification of the bones from prolonged recumbency may increase the output of calcium through the kidneys and this seems to favor stone formation. Careful microscopic studies of kidneys removed
post-mortem have shown tiny calcium plaques beneath the mucous membrane of the renal papillae in many cases.

These plaques are believed to be the result of cell injury and have to be regarded the initial lesion from which a stone grows.

Renal calculi have been divided into two categories: (a) those derived from proteins such as cystine, xanthin or uric acid; and (b) those containing calcium in some combination as calcium or magnesium ammonium phosphate or calcium with oxalic acid as calcium oxalate.

Under (a) the knowledge concerning the formation of stones derived from proteins is only theoretical.

Under (b), stones containing calcium, we have some evidence as to their cause from metabolic states.

The geographical incidence of urinary calculi seems to be controlled by areas in which vitamin deficiency diets are existent. Stone incidence increased in Europe during the 16th, 17th and 18th centuries after every protracted war and decreased after the establishment of peace. This was also due to the vitamin deficiency diets prevalent during the time of wars and the improvement in the diet during peace. The incidence of urinary calculi in children decreased in direct proportion to the improvement of their early diet.

Urinary calculi have been produced experimentally in animals which have ingested large amounts of a crystalline material called oxamide or calcium oxalate and calcium carbonate. Animals kept sufficiently long on diets deficient in Vitamin A formed stones quite consistently. Higgins was able to produce bladder calculi in 80% of rats maintained on diet deficient in Vitamin A for 265 days. Concretions in the kidney were present at autopsy in 42% of these animals. The calculi so produced were composed of calcium phosphate. If Vitamin A was added to the diet the stones disappeared.

Keratinization of the mucous membrane of the urinary passages has been considered a factor in this dietary deficiency theory of stone formation. Desquamated epithelium is supposed to be the nidus upon which crystals are deposited. The primary effect of Vitamin A deficiency is on the epithelial structures and consists of atrophy of the epithelium and the substitution for it of a stratified keratinizing membrane. The entire genito-urinary tract, as well as organs of the body, is involved in this keratinizing metaplasia. The lesions disappear rapidly after Vitamin A is restored to the diet as has been previously mentioned.
Hyperparathyroidism is a rather unusual condition which is accompanied by such severe disturbances in the body metabolism of the calcium and phosphorous that urinary stone is a frequent complication. This disease is due to a functioning adenoma of the parathyroid glands whose chief abnormalities are hypercalcemia, hypophosphatemia, hypercalcinuria and hyperphosphaturia. The kidney lesion of this disease consists of (a) calculi in the renal pelvis with secondary pyelonephritis; (b) an acute form of parathyroid poisoning with deposits of calcium in the renal parenchyma and in the other organs but without renal changes; and (c) chronic nephritis with calcium deposits in the renal parenchyma.

**Patient Immobilization and Stone**

Individuals with chronic osteomyelitis or arthritis and those with spinal cord injuries, those with extensive fractures, all should be watched carefully for evidence of urinary calculi, as the prolonged immobilization of the body resulting from these conditions sometimes causes sufficiently great metabolic disturbances so that urinary stones form. The high incidence of renal calculi in convalescent patients was revealed by physicians serving with the troops in the armed forces; bony decalcification plays an important role here, also there is increased urinary excretion of calcium; other factors in stone formation here are concentration of urine from low fluid intake, alkaline urine high in phosphatic deposits and urinary stasis in the renal calyces.

**High Calcium Intake and Stone**

It is believed that people who have high calcium intake are prone to stone formation. Yet in a baby with high calcium intake in the form of milk, stone incidence is low, but a baby must have high calcium intake for bone formation and the adult does not, hence that taken in by the adult in excess of body requirement is excreted in urine. Ulcer patients of alkali and large milk intake fulfill requirement of stone formation.

Certain substances which the body is unable to metabolize normally as oxalic acid, uric acid, cystine and xanthine are related to calculus disease.

In the characteristics of compositions of urinary calculi, calcium oxalate is found the most frequent constituent. The next most common are stones composed of calcium phosphate or carbonate. Uric acid stones no doubt are more common than is generally supposed. There seems to be a close correlation between infection and the presence of magnesium in urinary stones. These contained magnesium and am-
monium in addition to calcium phosphate and calcium carbonate. Urinary calculi are often composed of more than one substance. One material usually composes 90 to 95% of the calculus.

In a study carried on at Massachusetts General Hospital an analysis of 1078 cases of urinary calculi showed as follows:

<table>
<thead>
<tr>
<th>Substance</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cal. Phosphate</td>
<td>355</td>
</tr>
<tr>
<td>Cal. Oxalate</td>
<td>288</td>
</tr>
<tr>
<td>Uric Acid</td>
<td>75</td>
</tr>
<tr>
<td>Cystine</td>
<td>9</td>
</tr>
<tr>
<td>Xanthine</td>
<td>1</td>
</tr>
<tr>
<td>Mixed Phos. &amp; Oxalate</td>
<td>295</td>
</tr>
<tr>
<td>Phos. &amp; Urate</td>
<td>43</td>
</tr>
<tr>
<td>Oxalate &amp; Urate</td>
<td>12</td>
</tr>
</tbody>
</table>

1078

Magnesium stones do not require any description as they occur with Calcium Phosphate or Calcium Carbonate as mixed stones associated with infection. Pure Calcium Carbonate stones are uncommon, if they do occur. They are the hardest variety of urinary calculi, pale in color and extremely radio-opaque. Calcium Phosphate in combination with Magnesium and Ammonia are associated with infection, alkaline urine and urea-splitting bacteria. These may dissolve in an acid urine. These stones are multiple and tend to recur, form in layers and often have a laminated structure, may be deposited as a cortex about a central core of crystals of another variety. Inheritance seems to play no part.

In the presence of excessive amount of oxalic acid in the diet you have oxaluria. This substance is found in large quantities in chocolate, cocoa, tea, spinach, beet tops and rhubarb. The amount of crystals of calcium oxalate may be so high in the urine that they may cause hematuria and severe renal colic. Since diagnosis is difficult by X-ray, it must be made by identifying these crystals under the microscope and by chemical analysis of the urinary sediment. These oxalate stones are the most common urinary calculi. They form in acid, alkaline or neutral urine and when in acid urine occur in pure form. These stones are usually small and easily fall into the ureter from the pelvis, hence constituting a large per cent of ureteral calculi.

Since these oxalate stones have a rough surface, often with spicules, they frequently cause hematuria and severe colic. In the bladder they assume such bizarre form as mulberry or jackstone calculi. They are very opaque to x-ray.
The diagnosis of cystinuria is made by identifying cystine crystals in the urine with the microscope and by chemical tests of the morning urine. These stones have a low density but may be detected by X-ray.

During attacks of gout, urates are present in renal tubules in an abnormally high concentration and may be precipitated as crystals. Uric acid stones always form in acid urine. They may dissolve in urine of sufficiently high pH. These stones are frequently invisible on X-ray examination.

The rarest variety of urinary calculi is composed of Xanthine. This is the end product of the metabolism of nucleic acid. Sources of Xanthine are from ingested protein as glandular meats and alkaloids of tea and coffee. The calculi are frequently radio-opaque.
DENSE CATARACTS IN 6 YEAR OLD
ROBERT R. MUSCHLITZ, M.D.

J. T., six years old, white, female.

This child was first seen in January, 1951, after the school nurse and teacher insisted she could see very poorly. At that time, the pupillary areas showed gray color, which on closer inspection was floculant in appearance. This was more dense in the right eye than in the left.

The history, due to lack of any accurate observation on the part of the parents, was not too satisfactory. The parents noticed nothing amiss until she started to school, when she told them and the teacher that she could not see the blackboard, except when close to it. When examined by the school nurse in November, she could get no Snellen Acuity Test in the right eye, and 20/200 in the left eye. Toward the middle of December the child began to fail in school work and would "bump into things when she would run and play".

In January, when I first saw her, her vision was hand movements in the right eye, and 10/200 in the left eye. Fundus details could not be seen in either eye. External ocular rotations were normal, though an occasional intermittent exotropia of 10 degrees was noted. Pupils reacted promptly to light and accommodation.

Due to the speed of loss of vision, apparently within six months, blood calcium and blood sugar tests were taken. These were normal.

Because of the speed of change and the normal blood calcium and sugar findings, I was inclined to view this as a cataract due to malnutrition factors or atopic, rather than congenital. However, I was unable to perform the tests to prove or disprove any allergic basis.

Physical Examination: She was a small, thin, very active child, appearing to be suffering from some malnutrition. Ears and nose normal. Throat showed enlarged tonsils with red streaks and caseous material in the crypts. The glands of the neck were enlarged. Teeth were poor, showing many caries. Other physical findings normal.

She developed several colds during the next few months. Following this on March 7, 1951, a T. and A. was performed to prevent the possibility of infection from that source, complicating the cataract extraction.

On July 7, 1951, I operated on her. The possibilities in my mind were to do multiple decisions and allow lens material to absorb, or to attempt a linear extraction.
Because of the flocculation of lens material, and the difficulties attendant with repeated anaesthetics and hospital admissions, I decided to do a linear extraction. I used a modified Barkan\(^1\) technique.

The pupil was dilated with atropine 1%, the day before operation and neosynephrine 10% solution just prior to operation. Under general anaesthetic, I inserted a superior rectus bridle suture to help fix the eye. I made a narrow keratome incision about 2 mm inside the limbus parallel with the iris, and inserted one suture in the center after the incision was made. I took a capsulotomy forceps and entered the eye with it closed, and opened it as wide as the wound would permit, then grasped the capsule. As I did this the capsule ruptured before I closed the forceps, some fluid escaped and the lens substance became milky. I closed the forceps and tore out part of the anterior capsule, then removed the forceps. With an anterior chamber irrigator, I began to wash out what lens substance was possible, intending to leave the residue to absorb. All washed out but a small nucleus and I then extracted that by pressure below, moving over the cornea with counter pressure on the lower lip of the incision. There remained some capsule material which was lying across the pupillary apperture and I took the smooth capsule forceps and grasped the most anterior tab. I was able to extract the entire capsule remains in this way, ending with an extraction that for all practical purposes and end result was equal to that of an intracapsular extraction. Suture was tied, a small bubble of air introduced into the anterior chamber, and the eyes bandaged. Recovery was uneventful.

Six weeks later a temporary lense was provided with vision of better than 20/40.

An interesting fact is that the unoperated eye is now in a position of divergence of 20 degrees, and hypertropia 5 degrees, yet on near vision testing even with the bifocal the eye converges about 10 degrees.

The child is doing very well in school and we plan to remove the other cataract at the end of the present school term.

THE ROLE OF PLASTIC SURGERY IN THE TREATMENT OF CHRONIC LEG ULCERS

KERWIN MARCKS, M.D., F.A.C.S. and ALLAN TREVASKIS, M.D.

The chronic leg ulcer is the bane of existence for countless individuals, and because of the seeming futility of treatment, the doctor hates to see such a patient walk into his office. All of us have seen many cases subjected to saphenous vein ligations, diet restrictions, medical sympathectomies, and even surgical sympathectomies. We have put many people to bed, applied countless Unna boots, powders, lotions, and greases. And, in spite of it all, we have seen the chronic leg ulcer continue on its clean or necrotic way.

The causes of leg ulcers are many and do not fall in the scope of this paper. But all of the corrective causes have a common denominator in circulatory inadequacy. Ulcers with a neurogenic background, on the other hand, still remain an unanswered therapeutic problem.

The procedures which strive to correct the circulatory inadequacy, as cited above, are of unquestionable value in the treatment of selected leg ulcer cases. In many other selected cases they are entirely unnecessary. But what is more, if the ulcer is long standing these procedures, even though indicated, are little more than preliminary.

The normal leg is notoriously poor in blood supply between the ankle and the lower third of the tibia. This fact, together with a focal cause (such as injury), is sufficient to explain many chronic ulcers in an otherwise relatively "normal" leg.

An old ulcer, regardless of the initial cause, has a clinical picture of its own. The surrounding skin, sometimes circling the entire leg, is of poor quality. This ranges from an eczematous illness, to edema, to pigmented induration. Its subcutaneous tissue is grossly infiltrated with scar. This scar may be so thick as to give the appearance of edema. The ulcer bed too has been replaced by the same scar, its depth sometimes extending as far as the periosteum.

Scar is notoriously poor in blood transportation. Vessels in such an area which once might have done their share of the work are now squeezed, obliterated and thrombosed by this tissue "concrete", through which even sympathectomized vessels can not dilate. We may picture the edge of the ulcer as the "end of the line" in the ever constricting vascular system. As the scar increases, so the "end of the line" is moved and the crater edge goes with it.
The only conclusion which we can draw from this is that in order to correct the ulcer the scar must be removed. After doing this the resulting extensive defect, now bordered by normal tissue, is then closed in a manner as befits the particular case. Where possible, skin grafting is done. Occasional defects, such as those exposing bone or tendon, will demand closure by flaps.

We do not mean to indicate that this is a panacea for all leg ulcers, nor do we mean that this program is a complete substitute for the other surgical procedures mentioned previously.

We do mean that excision of the ulcer scar with grafting is a proven method of high success in the treatment of chronic leg ulcers. This is to be used alone or in combination with vascular surgery of a type dictated by the individual case.

The authors wish to present, on the following pages, a series of cases treated under the principles as described above. Those who have been lost from our follow-up care for one reason or another, have not been included in this study.
<table>
<thead>
<tr>
<th>No.</th>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis and Associated Disease</th>
<th>Duration</th>
<th>Previous therapy</th>
<th>Special Facts</th>
<th>Hospital Days</th>
<th>Results Immediate</th>
<th>Follow Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>240,526</td>
<td>M</td>
<td>49</td>
<td>Stasis ulcer left medial malleolus</td>
<td>2 years</td>
<td>Sub. Vein Lig.</td>
<td>Excision and Split graft.</td>
<td>29 days</td>
<td>100% coverage</td>
<td>Same.</td>
</tr>
<tr>
<td>2</td>
<td>270,842</td>
<td>F</td>
<td>62</td>
<td>Varicose ulcers rt. Lower third leg</td>
<td>2 years</td>
<td>Sub. Vein Lig.</td>
<td>1 Excision 2 Split graft</td>
<td>20 days</td>
<td>100% coverage</td>
<td>Same.</td>
</tr>
<tr>
<td>3</td>
<td>280,844</td>
<td>F</td>
<td>78</td>
<td>Three stasis ulcers both lower legs.</td>
<td>10 months</td>
<td>Bed Rest Topical</td>
<td>1 Excision 2 Split graft</td>
<td>4 months bed rest pre-op.</td>
<td>25 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>4</td>
<td>275,937</td>
<td>M</td>
<td>68</td>
<td>Ulcer rt. Sec to compound fracture</td>
<td>6 years</td>
<td>Topical</td>
<td>1 Excision 2 Split graft</td>
<td>Bone avascular</td>
<td>17 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>5</td>
<td>273,094</td>
<td>M</td>
<td>40</td>
<td>Ulcer lower third secondary to burn</td>
<td>16 years</td>
<td>Topical</td>
<td>1 Excision 2 Split graft</td>
<td></td>
<td>22 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>6</td>
<td>245,702</td>
<td>F</td>
<td>58</td>
<td>Stasis ulcer left medial Malleolus</td>
<td>1 year</td>
<td>Topical</td>
<td>1 Excision 2 Split graft</td>
<td></td>
<td>20 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>7</td>
<td>245,484</td>
<td>F</td>
<td>66</td>
<td>Stasis ulcer left medial malleolus</td>
<td>11/2 years</td>
<td>High Sub. Lig.</td>
<td>Excision and primary split graft.</td>
<td>Ulcer appeared immediately after sub. lig.</td>
<td>13 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>8</td>
<td>232,335</td>
<td>F</td>
<td>69</td>
<td>Bilateral Varicose ulcers and dermatitis</td>
<td>3 years</td>
<td>Topical and Postural</td>
<td>1 Excision 2 Split graft</td>
<td></td>
<td>21 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>9</td>
<td>236,885</td>
<td>M</td>
<td>49</td>
<td>Ulceration in large burn scar left lower leg.</td>
<td>20 years</td>
<td>Topical and Medical</td>
<td>1 Excision 2 Split graft</td>
<td>Falsely diagnosed osteomyelitis, unable to get insurance</td>
<td>23 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>10</td>
<td>211,356</td>
<td>M</td>
<td>67</td>
<td>Stasis ulcer lower medial third</td>
<td>8 months</td>
<td>Topical</td>
<td>1 Excision and split graft</td>
<td></td>
<td>51 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>11</td>
<td>270,278</td>
<td>M</td>
<td>23</td>
<td>Stasis dermatitis and ulceration lower third</td>
<td>10 years</td>
<td>Topical Sub. Lig.</td>
<td>Excision and Split graft</td>
<td>Tendinous scar exposed No home</td>
<td>70 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>12</td>
<td>242,299</td>
<td>F</td>
<td>01</td>
<td>Ulcer lower secondary to injury</td>
<td>6 weeks</td>
<td>Topical</td>
<td>Excision and Split graft</td>
<td></td>
<td>16 days</td>
<td>100%</td>
</tr>
<tr>
<td>13</td>
<td>293,542</td>
<td>M</td>
<td>27</td>
<td>Ulcer in burn scar proximal third, right.</td>
<td>1 year</td>
<td>Topical</td>
<td>Excision and Split graft</td>
<td></td>
<td>21 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>14</td>
<td>247,214</td>
<td>M</td>
<td>64</td>
<td>Post traumatic, left lateral malleolus</td>
<td>3 months</td>
<td>Topical</td>
<td>1 Excision 2 Split graft</td>
<td>Symptoms indicating Boeck's disease</td>
<td>69 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>15</td>
<td>240,888</td>
<td>F</td>
<td>41</td>
<td>Bilateral varicose ulcers</td>
<td>4 years</td>
<td>Bil. Sub. Lig.</td>
<td>1 Excision 2 Split graft</td>
<td></td>
<td>49 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>No.</td>
<td>Case</td>
<td>Sex</td>
<td>Age</td>
<td>Diagnosis and Associate Disease</td>
<td>Duration</td>
<td>Previous therapy</td>
<td>Operations</td>
<td>Special Facts</td>
<td>Hospital Days</td>
<td>Results Immediate</td>
</tr>
<tr>
<td>-----</td>
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<td>---------------------------------</td>
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<td>---------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>16</td>
<td>278,329</td>
<td>F</td>
<td>35</td>
<td>Varicose ulcer medial malleolar</td>
<td>4 years</td>
<td>Topical and vein</td>
<td>Excision</td>
<td></td>
<td>19 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>17</td>
<td>273,474</td>
<td>M</td>
<td>58</td>
<td>Ulcer caused by compound fract. tibia</td>
<td>3 years</td>
<td>Topical</td>
<td>Excision and sympathectomy</td>
<td>Split graft</td>
<td></td>
<td>32 days</td>
</tr>
<tr>
<td>18</td>
<td>G1 173,207</td>
<td>F</td>
<td>44</td>
<td>Large ulcer right foot dorsum, X-ray dermatitis</td>
<td>4 years</td>
<td>Topical and X-ray</td>
<td>Excision and</td>
<td>Split graft</td>
<td></td>
<td>50 days</td>
</tr>
<tr>
<td>19</td>
<td>270,632</td>
<td>F</td>
<td>59</td>
<td>Varicose ulcer right lower anterior</td>
<td>8 months</td>
<td>Topical</td>
<td>Excision</td>
<td></td>
<td>23 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>20</td>
<td>251,352</td>
<td>M</td>
<td>55</td>
<td>Varicose, right lateral malleolar</td>
<td>2 years</td>
<td>Topical</td>
<td>Excision</td>
<td>Hi vein lig. with excision</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>272,324</td>
<td>M</td>
<td>49</td>
<td>Large ulcer lower tibia &amp; foot with extensive dermatitis, X-ray dermatitis.</td>
<td>10 years</td>
<td>Topical</td>
<td>Excision</td>
<td></td>
<td>32 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>22</td>
<td>245,488</td>
<td>M</td>
<td>46</td>
<td>Ulcer left heel secondary to burn</td>
<td>25 years</td>
<td>Topical</td>
<td>Excision</td>
<td>Cross-leg flap</td>
<td>3 days 5 Admissions</td>
<td>61 days 100% coverage</td>
</tr>
<tr>
<td>23</td>
<td>225,573</td>
<td>M</td>
<td>53</td>
<td>Large ulcer secondary to fracture and osteo.</td>
<td>3 years</td>
<td>Topical, Sympathectomy</td>
<td>Split graft</td>
<td>Osteo still active</td>
<td></td>
<td>90% coverage 100% but will probably break-down.</td>
</tr>
<tr>
<td>24</td>
<td>228,423</td>
<td>F</td>
<td>70</td>
<td>Diabetic ulcer medial ankle both legs</td>
<td>29 years</td>
<td>Topical</td>
<td>Excision</td>
<td></td>
<td>88 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>25</td>
<td>227,423</td>
<td>F</td>
<td>72</td>
<td>Ulcer medial ankle both legs, same</td>
<td>1 year</td>
<td>Topical</td>
<td>Excision</td>
<td></td>
<td>26 days</td>
<td>100% coverage</td>
</tr>
<tr>
<td>26</td>
<td>227,423</td>
<td>M</td>
<td>54</td>
<td>Chronic ulcer amputation stump</td>
<td>8 months</td>
<td>Topical</td>
<td>Excision</td>
<td>Some scar left at base</td>
<td>24 days 95% coverage 3 cm ulcer persists</td>
<td>9 days 90% coverage</td>
</tr>
<tr>
<td>27</td>
<td>275,389</td>
<td>M</td>
<td>41</td>
<td>Osteomyelitis, ulcer entire shin</td>
<td>4 years</td>
<td>Previous graft</td>
<td>Excision and</td>
<td>Split graft</td>
<td></td>
<td>48 days</td>
</tr>
</tbody>
</table>

*Table continues...*
A CASE OF PEMPHIGUS

By PETER J. MIRALDO, M.D.

This 68-year old white female, native of Austria, was admitted to the Allentown Hospital on July 17, 1952 because of large superficial ulcerations and vesicles involving the upper and lower extremities and all of the back.

Her illness dates back to the second week in April, three months before admission. She first noticed an inflammation of the right eye, then shortly afterwards involving the left eye, but both cleared without medication. A week later she had an upper left molar removed because it was badly decayed. At that time the patient spoke to the nurse in the dental office stating that she had a small ulceration of the buccal mucosa near the area of the extracted tooth. She was informed that it was insignificant. While she had the ulcer in her mouth she also developed a cold and a very severe sore throat. Later her entire mouth became filled with blisterlike lesions. At this time, which was the latter part of June, she consulted and ENT man who gave her lozenges, mouth washes and penicillin without much change.

During one of her visits, the doctor noticed a vesicle on the dorsum of her hand. For the first time it was revealed that she had similar lesions all over her body. The patient stated it began with a marked pruritis of her toes and gradually ulcerations appeared involving her legs, then arms and back. The lesions began as small blisters and later reached the size of lemons, especially in the groin. The bullae ruptured, expelling clear yellow fluid and then leaving a raw surface. None of these lesions had healed at the time of admission. Approximately three weeks elapsed from the time of the first vesicle and the time of admission.

In addition the nails of her toes fell spontaneously and those of her hands darkened. She was also aware of an offensive odor from her legs. Pruritis was quite severe and the opened areas were painful. Her appetite had been poor since the onset of her illness.

There had been no previous hospitalization nor any serious illnesses. The patient lived on a farm on which there were many domestic animals. There was no history of animal bites. She had remained in the same locality since her immigration from Europe.

Physical examination revealed a well nourished female in no acute distress, but very apprehensive. Throughout the body there were many erythematous ulcerated lesions which had coalesced. The inner aspect of her thighs were more severely involved. Vesicles the size
of peas and golf balls were scattered in the ulcerated areas. The nails of her toes were all absent, those of her fingers were discolored and elevated. A musty odor was present even at a distance from the patient. In the buccal cavity there were numerous ulcers of the mucosa and tongue. None of these appeared infected. The lips were also involved. The chest was clear to auscultation and percussion. Normal sinus rhythm was present. No murmurs were heard. There were no axillary, clavicular or inguinal nodes. No liver, spleen or other masses were palpable in the abdomen.

Throughout her hospitalization her temperature remained within normal limits. However, for a week there was some elevation in the evening. Her blood pressure was within normal limits. Her appetite was good. Some of the vesicles were aspirated for smear and culture. These were sterile. Red blood counts were not remarkable. Blood sugar and urea were normal. Wassermann and Kahn were negative. A low protein was found with a reversal of the A/g 6:1. The sedimentation rate was 50 mm after one hour. One month later it had returned to normal — 6 mm. With the elevated temperature it rose to 42 mm. Pemphigus in this patient has been proven by Macht at Mt. Sinai Hospital in Baltimore, giving “a markedly positive Macht-Pells test for Pemphigus”. This test is reported to be 90% accurate. A positive Nikolsky sign could not be elicited.

With treatment, the patient showed remarkable recovery. New vesicles appeared, but did not reach the size noted on admission. The new bullae gradually dried without rupturing. In the groin, where the lesions were more severe, there was evidence of epithelialization. The mouth lesions were less painful. This improvement was seen within one week after therapy was started. In two weeks no new vesicles could be found. Healing took place from the center of the lesion while the periphery continued to ooze. In three weeks, there was 75% improvement in the patient’s condition. Complete epithelialization occurred after five weeks, leaving a brown pigmentation of the skin. The mouth lesions cleared except for a small ulceration at the tip of her tongue.
THE TREATMENT OF THE PREVIOUSLY PRESENTED CASE OF PEMPHIGUS

By CLYDE H. KELCHNER, M.D., F.A.C.P.

Pemphigus need not be fatal. Antibiotic and, recently, hormonal therapy have changed the old dictum — the "mortality" is "nearly 100% in six months to three years". The patient whose case is presented in this journal has been controlled for a survival period of one year to date of going to press.

This patient's treatment was both TOPICAL and SYSTEMIC. Topical therapy to the skin and mucosal lesions to afford the patient comfort and to prevent, or at least control, secondary infection, included:

- Potassium permanganate mouth wash, 1:10,000 q.4 h.;
- Potassium permanganate (1:100,000) body baths for 1 hour bid.;
- Zephiron, 1:1000, locally to all lesions daily;
- Sterile petrolatum gauze pressure dressings, applied to the skin lesions of the thorax, abdomen, and thighs, after the baths, with pressure bandages;
- Body cradle to keep the weight of the bed clothes from further traumatizing her skin lesions and loosening finger and toe nails.

Her systemic treatment was planned:

1. To control infection;
2. To overcome the stress and exhaustion of the severe toxemia;
3. To repair the damage done by the toxemia and to increase her resistance;
4. To include several drugs on a purely empirical basis, because they previously had been found of value in controlling pemphigus.

To control infection she was given aureomycin, 250 mg. q.4 h. from July 17th to Sept. 3rd and 250 mg. q.8 h. from Sept. 3rd to Sept. 7th, 1952. She had been given sulfonamide therapy and several million units of penicillin without any benefit during the three months preceding her hospitalization.

Sulfa and antibiotic therapy alone has NEVER completely controlled the lesions in a single case of pemphigus according to Sulzberger and Baer. However, Bettley and Fairburn believe that there results an increased cohesion of the cells of the epidermis, with the disappearance of Nikolsky's sign. Our patient showed no such increased cohesion until after ACTH therapy was started. However,
there was no secondary infection in spite of widespread, generalized, raw denudement.

Secondly, to overcome the stress and exhaustion of the severe toxemia, pituitary adrenocorticotropic hormone, ACTH, a stimulating rather than replacement hormone, was administered 20 mg. intravenously in 500cc. of 5% glucose, very slowly over a period of eight hours daily from July 21 to August 23, 1952. After the first five days of this therapy, no new bullae or other lesions appeared and healing began. ACTH was given intramuscularly after August 23, decreasing the dose by 5 mg. every three days until September 4, 1952, when she received 2.5 mg. as her final dose. She developed a gradually increasing P.M. fever, which rose to 101 and 102 F. on September 6 and 7, but no skin or mucosal lesions. Cortisone, 25mg. q.6h., was started on September 8 and her temperature immediately dropped to normal levels. Thereafter, she was put on a half tablet, 12.5mg. q.6h., the lowest dose on which control can be maintained. In October and November, whenever she stopped Cortisone or decreased the dose, she developed a fever. On one occasion, after a week without therapy, she developed bullae on her right great toe and on the buccal mucosae and gums, with a necrotic gingivitis around the lower central incisors. All of these lesions healed completely with Cortisone, 25mg. q.6h. The patient complains of a soreness of her mouth and tongue almost continuously, although all mucous membranes appear normal. This soreness is probably caused by her using her tongue to chew her food since she has only one upper tooth and a fair set of lower teeth. Her course clinically proves and substantiates the conclusion that “ACTH and Cortisone never 'cured' a patient” 5,6, but “alter the disease picture for the better”. Adequate clinical improvement is said to occur only after the eosinophilia is reduced to below 50 per cubic mm. in the circulating blood.5 The hormones also elevate the skin temperature and accelerate the peripheral flow in the capillaries.7

Various measures were instituted to avoid the undesirable side effects of ACTH and Cortisone therapy. She was given potassium chloride, gm.ss.,qid., and a low sodium, low carbohydrate, high protein dental diet. Sodium limitation is important, not only because the hormones cause sodium retention, but especially because the salt content of the skin is increased in pemphigus, a factor Robert regards as being of primary significance in pathogenesis.8 She was examined daily for evidence of edema and blood pressure elevation. After the ACTH was stopped her pressure dropped to 78/50. Her urine was checked for sugar weekly and her blood sugar monthly. Hormone therapy has been kept to the lowest effective dosage, remembering that ACTH intravenously equals 5 to 10 times the effectiveness of
the same dose intramuscularly, while ACTH intramuscularly is two
and a half times as effective mg. for mg. as cortone intramuscularly, and cortone intramuscularly is more effective than the same dose orally. This is of great economic importance as well. Her healing with marked pigmentation may be a side effect of cortone therapy or of arsenic therapy. Of course, such pigmentation may be racial, because of her Slavic origin.

Thirdly, to repair the damage done by the severe toxemia and to increase her resistance, she received four fresh blood transfusions of 500cc. each, and Mol-iron, gr.V.,tidac. Hocker in studying 40 cases in Munich found that of 20 surviving, only one had an erythrocyte count below four million, and that was 3,700,000 with Hb 77%. The 20 who died averaged 3,300,000 RBC and 62% Hb. Supplemental protein feedings of cottage cheese, gelatin, protenum and low fat milk are essential to make up the loss of protein through the denuded skin. The serum albumin is greatly reduced, often only one fourth of its normal value, while gamma globulins are increased, for while globulin is regenerated, albumin is not. Study of the contents of the bullae shows the total protein value lower in the blister fluid than in blood serum.

To avoid transfusion reactions she received pyrabenzamine hydrochloride, 50 mg.qid.

Her sore mouth and tongue combined with anorexia and a depressed certainty that she was going to die had led to a very low food intake for several months prior to hospitalization. Her one upper tooth made her mastication inadequate for years. Hence her treatment included Unicaps, 1.tidpc.; niacinamide, 25 mg.qid.; riboflavin, 5mg.qid.; and Endo Manibee, parentorally, 2cc. daily.

To further repair the damage of the toxemia some have advised the use of calcium pantothenate, liver extract, calcium paraminobenzoate, 2gms.tid., and huge doses of vitamin D, because of the reduced calcium content. None of these measures were instituted in our patient.

Finally, several drugs were given empirically during the period when her condition seemed critical:

1. Carbarsone, the least toxic of the arsenicals, 250mg. bid., one-half hour before meals. (Fowler's Solution and Acetarsone or "Stovarsol", 0.25gm. qid for four days and then skipping the next four days might have been substituted for the stimulating effect or arsenic on many functions of the adrenal cortex).

2. Suramin, naphuride, Bayer 205, or Germanin, as C$_{51}$H$_{34}$N$_6$O$_{23}$S$_6$Na$_{61}$, so useful in trypanosomiasis has variously been known,
was given intravenously q. five days until a total of 8.25 gms. had been reached. Sulzberger and Baer insist that there is no excuse for undue delay in using naphuride, carbarsone, and huge doses, if necessary, of ACTH or sortisone, until the patient becomes moribund and beyond all aid.

3. DEPO-HEPARIN, 200mg. intramuscularly, daily for six days, after which it was discontinued because of a tendency of the raw skin lesions to bleed. Heparin inhibits the action of hyaluronidase which has been suspected of producing the acquired defect in the cement substance which leads to the formation of bullae. This defect may be an analogue to the induced defect in experimental rheumatic disease in which hyaluronidase inactivates the mucopolysaccharides, hyaluronic acid and chondroitin sulfate isolated from the interfibrillar substance of connective tissue.

Deep X-ray irradiation over the liver and spleen was administered according to the technique suggested by Macht to diminish the toxic reaction of pemphigus serum. The blood serum in pemphigus may exhibit "a specific phytotoxicity on the root growth of Lupinus albus seedlings, which is not given by the sera of most other dermatoses (Pels-Macht or Macht-Pels test)" (which was found by Macht to be positive in the patient. Exposure to specially filtered X-rays diminishes this toxic reaction of pemphigus serum, while sera from other dermatoses are not affected by such rays (Macht-Ostro reaction). Macht insists that these "two tests performed simultaneously lead to a reliable differential diagnosis of pemphigus", Other dermatologists are much less enthusiastic.

At any rate the course of these patients would tend to substantiate the viewpoint that at the present time there is no cure for pemphigus. Antibiotic, supportive and hormonal therapy clears up the skin lesions promptly, the mucous membrane lesions more slowly, but THE DISEASE REMAINS ACTIVE and relapses after such therapy is stopped. Hence, treatment must be continued for an indefinite period of time.

REFERENCES

11. Lynch, F. W., Current Therapy, 1951, ed. by H. S. Conn, Saunders, Phila., 1951, P.471
TWO of the recent major advances in medical therapeutics represent the two major advances in ophthalmology in the past fifteen years. Without doubt, the introduction of the sulfonamides—and now, most commonly—antibiotic agents in the control of ocular infection, and the more recent use of ACTH and cortisone in the control of inflammatory processes, represent together the two most significant contributions to present-day ophthalmology.

Considering the use of antibiotics, it should be remembered that in general antibiotics applied locally are not absorbed in effective concentrations in the anterior chamber and that none penetrates into the vitreous itself. However, the concentration of such therapeutic agents in the anterior chamber of the eye may be increased by such measures as iontophoresis and subconjunctival injection. Systemic administration of antibiotics will result in only fairly effective concentration of the agent in the aqueous, but the concentration in the vitreous itself is too minimal for therapeutic value.

The following case, published in detail elsewhere, is reviewed as a rather striking illustration of the sometimes sight-saving effect of antibiotic agents:

CASE REVIEW

A 26 year old Negro was admitted to the Allentown General Hospital after injury from an accidental dynamite explosion while he was drilling a hole in rock preparatory to blasting.

Injury was limited mainly to both eyes, examination of which revealed swollen lids showing numerous areas of penetration of rock dirt. The right eye showed a perforating wound of the cornea with prolapse of iris; the lens and visible portion of the fundus was clear; patient stated that he could see. The left eye showed multiple perforating wounds of the cornea with prolapse of iris; the anterior chamber was filled with blood; vision was reduced to light perception.

The patient was placed under systemic penicillin and tetanus and gas gangrene antitoxin administered. Initial surgery was undertaken four hours later.
The following day the right eye was apparently in good condition showing a clear, anterior segment although there was some vitreous haze. The left eye showed an intense inflammatory reaction with clouding of the aqueous.

On the second postoperative day the left eye showed a frank infectious panophthalmitis with pus in the anterior chamber. The right eye was beginning a similar picture—vision was reduced to counting fingers at 3 feet, the vitreous was turbid and fundus details could not be seen. The patient was taken to the operating room for removal of the left eye, cultures of the intraocular material being made. At the same time the anterior chamber of the right eye was irrigated with penicillin solution (5000 units per cc).

Two days later vision in the right eye was reduced to hand motions and a yellow exudate was grossly visible behind the lens. Episcleral injection was pronounced and the globe was tender. Culture from the enucleated eye was reported as a gram-negative bacillus. Systemic administration of streptomycin was begun (0.5 Gm. every four hours) and 500 micrograms of streptomycin was injected directly into the vitreous.

The following day the condition of the eye was worse, and vision was reduced to light perception. The intravitreal injection was repeated in larger dosage (1400 micrograms) and a streptomycin pack (20,000 micrograms per cc) was placed in the cul-de-sac for one hour.

Four days later the vision was reduced to nil but the inflammatory reaction of the globe began to show gradual improvement, the eye becoming grossly non-inflammatory ten days later. Light perception had returned; a secondary cataract prevented visualization of the fundus.

Subsequently, two months later the secondary cataract was removed.

Examination of the patient six months after his cataract extraction showed corrected vision of 20/200. The vitreous was clear but the retina showed degenerative scar formation involving the macular area such scar formation having been reported from experimental work when intravitreal doses exceeding 900 micrograms were used.

It appears obvious that direct intravitreal injection of streptomycin alone saved this patient's remaining eye after systemic penicillin had

1Shoemaker, R. E.; Arch. Ophth., 41:629, 1949.
failed. This is particularly significant and stresses the inadequacy of systemic administration, because later bacteriologic reports showed the infecting organism was a gram-positive spore-forming organism of the brevis group, and sensitivity tests proved the organism more susceptible to penicillin than to streptomycin in vitro.