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Western University

Mediscope

FOUR YEAR CHECK-UP:

Man would be happier if he lost track of time. But life itself does not let him forget. It was some five to ten years ago, we one day envisioned of finishing our medical UNDERGRADUATE curricula. The ROAD at that time appeared long, distant, and not within reach—almost non-attainable. And yet this finest hour has arrived. The means to an end has been m-ended.

As we look back, we think of the cries. and the sighs that we have met, the things we have sacrificed both in tangible possessions and in intangible happiness. Many have married and set up home—to others that privilege was to be denied for a few more years. Essentially, the course has been a long road—and many have said, "I've been loking forward to this day for ten years". Prognosis has been excellent, but now that healing stage is over, what now?

For many this is the end of his formal training. With a year or two of internship (till he feels reasonably comfortable about his capabilities) he will then face the public. For others, as the Trend is becoming, he will take a few more years at a recognized training centre to add further letters, so that he won't be singing "I know a little bit about a lot of thing but . . ." rather a lot about a 'little field of things'. But whatever our goal, we have a responsibility—either to replace the present leaders, or to add to the present knowledge. Why the latter? The answer is manifold. It may be to satisfy the innate curiosity of man in one field of work, or his incessant desire to create novelty, or to be inspired by SOME WOMAN as many men are—all in search for a better way of life, to show others and by doing so, to help himself.

And so, on this lonely pinnacle, the medical graduate can survey more history, that he has seen before. During the past four years, he has rediscovered his personal links with man and his membership in the vast animal kingdom. Somewhere in the turbulent years, he has accepted a responsibility for the fate of his land, for the mother and father of its past. And COSMIC MAN — what of him? The Medical man frustrated in DRUG RESISTANCE of microbials, his dead-end in the search for the panacea of malignant neoplastic growth, his words banned by doubt, fear, and limited apprehension of mother nature—still seeks, and tries to find, to help. And sprinkled in its midst is a bit of paternal desires of marriage, of children, and of comforts of life. His life is prematurely ended in many cases. (Is this man's life?)

His dreams of boyhood, manoeuvering his way across this vast and yet uncrowded earth, has but sometimes little hope of accomplishment. Yet we strive. And though we cannot do all what we would like to do, as we have dreamed in our boyhood midnight travels, today, we have a wider scope in which to make up our minds to follow certain paths. We very often take such opportunities for granted, but such a possibility has been the nature of the North American Dream, that dream of land in which life should be better and richer and fuller for every man, with opportunity for each according to his capabilities or achievements.

Yes, this North American Dream that has lured our parents, our grandparents, and our forefathers to our shores in the past century has not been a dream of merely material plenty, though that certainly must have counted plenty. It has been more than that. It has been a dream of being able to grow to the fullest extent as a man and as a woman, unhampered by the barriers which has slowly been erected in OLD CIVILIZATIONS, unrepressed by social orders which had developed for the benefit of classes rather than for the simple human being of any and every class, and that dream has been more fully reached in actual life here than anywhere else. This opportunity is

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further extended as he fully realizes that he is in a field whose scope is closer and broader than any other profession on earth. And yet at this threshhold, many of us do not feel much like a medical graduate.

However, since on us alone as a segment of leaders of tomorrow (as we are North Americans) has fallen the awesome responsibility of holding open the door of history against the forces of evil, disease, and desolation until FREEDOM, health and wealth is born anew over the lands. And thus we must consider what Whitman once said, that "Our New World, I consider far less important for what it has done, or what is than for the results to come".

But in our attempt to attain health freedom and reasonable wealth for all concern, are we to seek the problem by giving medical aid to under-developed countries. If so, we would be lengthening twenty miserable years of human existence to forty miserable years. Is this justified? I would say not. Our responsibility in this path lies in the attack from another angle. It comes in educating these poverty stricken folks whose life centres only in seeking the basic joys of life—matrimony, and its desires. When it comes to attainment of a good standard of living for these people, it requires men who are not only interested and familiar with the problems at hand, but in whom power has been vested so that he is able to make changes. Even then the re-FORM-ation of such under-developed countries is not a one year, a five year, a ten year plan; but it is an undertaking requiring generations for its completion. It has taken medieval Japan eighty years to reach WESTERN standards from the time Commodore Perry opened the gates of feudalistic Nippon to let her see the modern western world. She was one of the more ambitious countries. How long will it take others?

We have been prepared, by OUR PROFESSORS, by our friends, and by ourselves. We have learnt to appreciate life from a segment unappreciated by factions of other professions. We will remember to be kind, gently, and individualistic as only Dr. Brien[1] could have taught us; we will be concise, terse, and time-saving as Dr. McLachlin[2] exemplified in his daily work; we will be incessantly careful of minute details in the maze of gross pathological features as only Dr. Fisher[3] can do it; we will never fail to keep an open mind, that it could be some other disease as only Dr. "Differential Diagnosis" Rathbun[4] could have hammered into us on more than several occasions, we will remember other teachers, each for something that they gave to each of us, to help us in our difficulties. Out of this we will certainly find new ones to replace our old.

In this Age of jet aircraft and guided missiles capable of carrying destruction to the other of MEGATONS, we are only THIRTY minutes away from destruction at any one time. And thus we seek answers over the conference table in the council of nations as the civilized method to solve our problems. But remembering that it is the biochemistry and physiology of the man which destines him to make such destructions as mass destruction or total annihilation, we, as medical guardians are responsible in their actions whether evulsed "physio-pathologically", or sustained psycho-somatically. By understanding the bio-pathological changes hitherto uneexplained, we may find an answer to the rational for the actions of good men, and greedy, avaricious, jealous, envious, cruel, dishonest, and selfish man—the epitome of a fearful man.

Or will it take a perilous force outside the sphere of the planet earth to awaken us, that only then shall we forget the

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[1]Professor of Medicine, University of Western Ontario Medical School.
[2]Professor of Surgery, *ibid.*
[3]Professor of Pathology, *ibid.*
[4]Professor of Pediatrics, *ibid.*

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idiosyncrasies of communism, nationalism, imperialism, momism and all the other -isms and that man shall dispense with fear, frustration, and fighting to replace it with fraternity, fatherhood and fighting force to fraught the feverish force from far. Or is the present CAMP SYSTEM between the forces of the proletariat and of the nouveau riche be good enough for both sides to look for long range guided missiles, for space platforms, or for peaceful utilization of man's environment. We can only conjecture the answers, but we must constantly keep an open mind if are to maintain our standards in the council of united nations.

Man must allay the "dark" night for much higher endeavour, by finding means to protect himself from the unknown. And thus with our superior education, it is a challenege presented to us that as MEDICAL GRADUATES we are to be the guardians of the populous. Fear hits us as well as to others, it is inevitable in life, as it depends upon tension, and tension easily becomes anxiety. As greater understanders of biochemical, physiological and pathological entities destining actions of men, we have an awesome responsibility in utilizing our appreciation of these processes to make this a safer and more satisfying, and finally a more stimulating type of existence in our r a t h e r short tenure on global earth. In this attempt, we may fail sometimes in our efforts. But we will be guided by a few famous words, once said:

"There's the courage that nerves you in starting to climb
The amount of success rising sheer:
And when you've slipped back, there's the courage sublime
That keeps you from shedding a tear.
These two kinds of courage, I give you my word,
Are worthy of tribute—but then.
You'll not reach the summit unless you've the third—
The courage of try-it-again."

Initially when we set out on life we will have problems. But once the frontier stage is passed, that is the acquisition of a bare living and the setting up of a fair economic level, the North American dream itself opens all sorts of questions as to value. It is easy to say a better and richer life for all men, but what is better and what is richer? Just so long as wealth and power are our sole badges of success, so long will ambitious men strive to attain them. The prospect is really discouraging, but not yet hopeless. It has occurred to me that the working out of the Dream into a reality can only be wrought by using the means provided by the accumulated resources of the people themselves, a public intelligent enough to use them, and men of high distinction, themselves a part of the greater freedom from fear, law and religion, devoting themselves to the good of the whole.

These are but few thoughts in the multitude of dreams which flash across the p. q. r. s. t. complex. Amongst it we realize that the formal part of our education is at a close. We leave thee O WESTERN U, with hopes, with dreams, and armed with YOUR IMPRESSIONS that you have given us. We now face the world. How we use it, will mean the difference between more comforts for our loved ones, and for our own satisfaction. We have accepted that magnanimous responsibility. We hope to sincerely justify your confidence in us.

—Masashi Kawasaki '57

Medical News and Views

Venereal Disease Legislation

The World Health Organization in a survey of 40 countries has found that legislation in control of Venereal Disease is greatly varied and disputed. Denmark, since the end of the 18th century and Sweden from 1918 provide for free and

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Rheumatic Fever

MARCIA C. SMITH, '57

INTRODUCTION

By our present definition, rheumatic fever is a post-streptococcal non-suppurative inflammatory disease, affecting especially the heart, vessels, and larger joints. It sometimes shows lesions in the skin, subcutaneous tissues, periarticular tissues, brain, lung and serous linings. It is a generalized systemic disease characterized by frequent recurrences or exacerbations.

Rheumatic fever is important as one of the leading causes of chronic illness, invalidism, and death in children. Its effects are projected into adolescence and beyond.

ETIOLOGY AND PATHOGENESIS:

The cause of this disease is unknown. However, it is generally agreed that both initial and recurrent attacks follow an infection with group A beta-hemolytic streptococci.

The usual sequence of events can be described in three phases:

(1) The acute septic phase, where the patient is infected with group A beta-hemolytic streptococci.

(2) An asymptomatic phase.

(3) The late non-suppurative phase.

The acute septic phase lasts three to seven days. The patient is ill with fever and constitutional symptoms. If he is susceptible to the erythrogenic toxin he develops a rash. There are local signs of inflammation such as tonsilitis and pharyngitis, scarlet fever, erysipelas, or wound infection.

After the toxic phase, the patient recovers and is symptomless for a period of from one to eight weeks. This latent period is followed by another illness of differing symptomatology. The patient may develop fever with no other signs of infection and this has been called "streptococcal fever". He may develop acute glomerulonephritis. Or, he may develop rheumatic fever.

The mechanism by which these late non-suppurative complications of group A beta-hemolytic streptococci are produced is unknown. Investigations into this problem may follow four lines of thought:

(a) The streptococcal theory: The symptoms of the disease were thought to be the result of direct action of the streptococcal organisms themselves, or their products or circulating toxins.

(b) The viral theory: Unsuccessful attempts were made to find a filterable virus.

(c) The allergic theory: Proponents of this theory believe that the disease is an allergic response of tissues previously sensitized by a streptococcal infection, especially of the respiratory tract. There is evidence to support this.

(d) Since 1940 investigation has centred on the hemolytic streptococcal infection and its relation to rheumatic fever. This work is based on epidemiological data indicating, (i) that prophylaxis of upper respiratory infection due to streptococcus results in a substantial decrease in incidence of recurrences of rheumatic infections, and (ii) that early and adequate treatment by antibiotics of upper respiratory infections with

streptococcus in their initial stages reduces substantially the occurrence of rheumatic fever.

To date investigations have established that there in an intimate relationship between group A beta-hemolytic streptococcal respiratory infection and rheumatic fever. Probably, rheumatic fever develops only after multiple exposure to the organism. It is likely, but not yet proved, that an inappropriate immunological reaction to some part or product of the organism is the basic pathological mechanism involved.

EPIDEMIOLOGY OF RHEUMATIC FEVER:

A. Incidence: The difficulties in obtaining data on the incidence, prevalence, and general importance of rheumatic fever have stemmed from the facts that,

(a) It is not a reportable disease.

(b) Often it cannot be definitely diagnosed in the presence of mild symptoms.

(c) Reports of the occurrence of heart disease often include the cardiac sequelae of syphilis and arteriosclerosis as well as rheumatic fever.

In formation must be drawn from sources which are not strictly accurate but are the best available. These include mortality statistics, morbidity surveys, postmortem examinations, compulsory notification, special rheumatic clinics, hospital admission rates, and rheumatic heart disease among special groups such as college students and military personnel.

The incidence of rheumatic fever varied from 1 to 3 per 1,000 population. About 3 to 6% of those infected during a streptococcal epidemic consequently develop rheumatic fever. Evidence of rheumatic heart disease exists in 1 to 6% of the population. In the U.S.A. rheumatic fever with heart disease is the leading cause of death during the 10 to 15 year age period (Gould: "Rheumatic Diseases of the Heart", 1953). At present the mortality rate due to rheumatic heart disease seems to be declining. Many observers are of the opinion that attack rates of both initial and recurrent episodes are also decreasing. This apparent decline in morbidity and mortality of rheumatic fever may be due to changes in the natural history of the disease, to the improvement of living conditions, or to better control of upper respiratory infections. Rheumatic fever is still an important public health problem, and it may be that present prophylactic measures may further reduce the sickness and death it causes.

B. Geographical Prevalence: Rheumatic fever is common and severe in temperate zones; it is less common in warmer, subtropical and tropical climates. It seems to flourish best under conditions where there is cold and damp weather. The effect of climate may be the effect of crowding susceptible people within doors under conditions which tend to spread the etiologic agent.

C. Seasonal Influences: In temperate climates both primary and recurrent attacks occur more frequently during the colder and wetter months of the year. This is also true of other diseases, e.g. measles, acute upper respiratory infection of viral and bacterial origins. The ratio of winter to summer cases is 3 to 1. The peak incidence of cases occurs in March or April on the east coast of this continent, in January and February on the west coast, and in November in England. Some other interesting findings are that prolonged cold rather than severe cold weather seems more likely to be responsible for the increased incidence of rheumatic fever, and that the greatest frequency of rheumatic fever precedes rather than follows a period of heavy precipitation.

D. Age: The disease is more prevalent in childhood. First attacks have a peak incidence at 6 to 7 years of age. The di-

sease is rare under 2 years and after 30 years of age.

E. Sex Prevalence: There is a higher incidence of chorea among girls (2 to 1).

F. Race: No definite difference in racial susceptibility have been established. Among Negroes the age distribution is different, with peak incidence of attacks in the 10 to 14 years period. Immigrants of any race going from an area of low incidence to one of high incidence develop the disease at a greater rate.

G. Physical Characteristics: It has not been shown that red hair and freckles make one more vulnerable to development of rheumatic fever.

H. Familial Incidence: Some investigators have found a higher rate of attack of rheumatic fever among siblings of rheumatic parents. Others have shown that the attack rate is 3% regardless of the rheumatic state in the parents. So, there is conflicting evidence concerning a hereditary predisposition to rheumatic fever.

L. Living Conditions: Rheumatic fever is most prevalent where there is poor housing, dampness, crowding, inadequate food, and other circumstances of poverty. These conditions may affect the spread of streptococcal infections and in this way produce an increase in incidence of rheumatic fever. In one survey it appeared that the degree of deficiency of the diet was related to the incidence and degree of heart damage. On the other hand, rheumatic fever frequently occurs among military personnel who have adequate diets.

PATHOLOGY OF RHEUMATIC FEVER:

The lesions of rheumatic fever are proliferative and degenerative. The Aschoff body is a microscopic granuloma, pathognomonic of rheumatic carditis. It occurs in the myocardium, the valves, the pericardium and the aorta. As the lesion heals a scar is formed.

The subcutaneous rheumatic nodule is another proliferative lesion. It resembles that of rheumatoid arthritis except that it is painless, and disappears in 4 to 6 days. The degenerative lesions of rheumatic fever are fibrinoid swelling of collagen and fibrinoid necrosis. These changes in collagen mark rheumatic fever as a "collagen disease".

The heart, blood vessels, and larger joints are principally affected. The mitral and aortic valves are often thickened, distorted and scarred. Vegetations form on the mural endocardium. The chordae tendinae become shortened with fibrosis. There is a rheumatic aortitis and arteritis especially in the small vessels of the myocardium.

Rheumatic arthritis chiefly involves the periarticular tissues and synovium. Inflammatory edema and synovial effusion produce swelling.

Lesions in the brain associated with chorea occur in the ganglia and cerebral peduncles. They consist of thrombosis of small vessels, tiny focal hemorrhages, infiltration of lymphocytes around the vessels and chromatolysis of nerve cells. These lesions regress in a few weeks.

Rheumatic pneumonitis is sometimes seen. Pericarditis and pleurisy also occur.

CLINICAL MANIFESTATIONS OF RHEUMATIC FEVER

There are three clinical phases of rheumatic fever, each presenting a problem in diagnosis:

I The acute attack.

II Chronic or subacute rheumatic activity.

III Rheumatic heart disease.

I The Acute Attack: The attack may come on suddenly or gradually. Its signs and symptoms may be grouped according

to (a) those of infection, (b) those of involvement of the heart and great vessels, and (c) those of involvement of other organs and tissues.

(a) Signs and symptoms of infection:

1. Fever.

2. Leucocytosis up to 15,000 or 30,000 per cu. mm.

3. Elevated sedimentation rate.

4. Secondary anemia.

5. Loss of weight, or failure to gain; fatiguability.

Fever, leucocytosis and elevated sedimentation rate tend to follow the pathologic process. They are also useful guides in chronic rheumatic activity. Antipyretic medication will influence them.

(b) Signs and symptoms of involvement of the heart and great vessels:

1. According to Harrison "it is now generally accepted that involvement of the heart is a practically universal phenomenon of rheumatic fever". Active carditis is the cause of death in those succumbing in an acute attack. The patient with severe involvement of the heart is pale and prostrated, with weak rapid pulse and possibly concomitant symptoms of heart failure.

2. Pericarditis is indicated by a to-and-fro friction rub. Pericardial effusion must be differentiated from dilatation. It is diagnosed by lack of apical impulse, distant heart sounds, and the typical radiographic globular cardiac silhouette.

3. Enlargement of the heart is often seen in children, but if this is not marked only serial x-rays will show it.

4. Diastolic murmurs mean cardiac damage, but not necessarily permanent valvular damage. They may be produced by acute dilatation of the chambers, or by edema of the cusps.

The above signs appearing during an attack of rheumatic fever are certain evidence of acute carditis of undoubted severity.

5. ECG changes and disturbances of cardiac rhythm indicate myocardial involvement. A prolonged P-R interval is most frequently found.

(c) Signs and symptoms of involvement of other organs and tissues:

1. Migratory polyarthritis: This can be a very confusing symptom since many conditions may cause it. Large joints are most often involved. One or more of them becomes reddened, hot, swollen, tender and painful. The process may affect the joints in succession, flaring up in one area as it dies out in another. It lasts from a few hours to a few weeks. Complete restoration of function follows subsidence of the acute inflammation.

2. Chorea, also called Sydenham's chorea or St. Vitus' dance. It is rare in adults. It develops slowly over a period of one to two weeks. The patient is restless, nervous, emotionally unstable, and shows incoordinated and purposeless movements of limbs. Choreic patients rarely show the systemic signs of infection, but most eventually show other signs of rheumatic fever.

3. Subcutaneous nodules: These are hard, shot-like bodies seen or felt over the extensor surfaces of joints, usually elbows, knees, and wrists, in the occipital region or over the vertebrae and scapulae.

4. Erythema marginatum: This is a rash in which the color gradually fades away from the scalloped edge leaving a reddish gyriform margin. It is found mainly on the trunk. It is transient and migrates from place to place.

5. Recurrent attacks are characteristic of rheumatic fever. The history of a previous attack is helpful in diagnosis since 75% of all patients will develop a recurrence.

6. History of a preceding streptococcal infection is established by, (a) history of scarlet fever or a typical clinical picture of other streptococcal infection in the preceding four weeks; positive culture of beta-hemolytic streptococcus in the nose

or throat. (b) elevated or rising antistreptolysin - 0 titre.

7. Abdominal pain occurs frequently and may simulate an acute abdominal condition. This may be pain referred from the pleura or pericardium.

8. Pleurisy accompanies acute rheumatic fever but does not last longer than a few days.

9. Pneumonia may develop in acutely ill individuals.

10. Epistaxis is common and may be profuse. Its exact cause is not known.

II Evidence of Chronic or Subacute Rheumatic Activity:

1. Arthalgia: In children "growing pains" are frequent, due to synovitis of tendons such as the hamstring tendon or of the bursa of the Achilles tendon. Fleeting joint pains not accompanied by the signs of inflammation are complained of during wet weather. They are usually relieved by going to warm bed.

2. Fever of low grade, 99 to 101° F.

3. The sedimentation rate is quite a sensitive index of rheumatic activity.

4. C-reactive protein determination: C-reactive protein or acute phase protein as it is also called can be demonstrated in human serum in certain acute conditions of disease, e.g. heart infarction, hypernephroma, acute cholecystitis, rheumatoid arthritis, rheumatic fever, and intra-uterine fetal death. The test is simply done, incubating human serum with rabbit antiserum, and measuring the degree of activity by the amount of precipitation. Available evidence seems to indicate that the presence of C-reactive protein is probably the most sensitive test for the presence of rheumatic activity that is now available. The test is of real value once the diagnosis has been made of rheumatic fever. C-reactive protein has not been present in sera of patients with Sydenham's chorea.

The American Heart Association has published criteria for guidance in the diagnosis of rheumatic fever. These are modified Jones criteria (in 1944 Dr. T. D. Jones published his original ones). The diagnostic features of the disease are divided into major and minor categories dependent upon their relative occurrence in rheumatic fever and in other disease syndromes for which this disease must be differentiated.

The major criteria:

1. Carditis
2. Polyarthritis
3. Chorea
4. Subcutaneous nodules
5. Erythema marginatum

The minor criteria:

1. Fever
2. Arthralgia
3. Prolonged P-R interval in the ECG
4. Increased ESR, WBC, or presence of C-reactive protein
5. Preceding beta - hemolytic streptococcal infection
6. Previous rheumatic fever or inactive rheumatic heart disease

These criteria are for diagnostic purposes only in the identification of those persons who have had or are having an attack of rheumatic fever. The presence of two major criteria or one major and two minor criteria indicates a high probability of the presence of rheumatic fever. In questionable cases the diagnosis of rheumatic fever should be restricted to those illnesses which meet acceptable criteria. **Differential Diagnosis:** Diseases to be ruled out are rheumatoid arthritis, septic arthritis, disseminated lupus erythematosus, subacute bacterial endocarditis, nonspecific pericarditis with effusion, leukemia, sickle cell anemia, serum sickness, tuberculosis, poliomyelitis, undulant fever, and septicemias, especially meningococcemia.

III Evidence of Chronic (inactive) Rheumatic Heart Disease:

The diagnosis of chronic rheumatic heart disease rests on the recognition of the various valvular lesions. Mitral stenosis and aortic insufficiency are the classical ones. Aortic stenosis may also be of rheumatic origin, as may aortic insufficiency. A past history of rheumatic fever establishes the origin definitely. In the absence of this congenital heart disease and functional murmurs must be considered in the differential diagnosis.

COURSE AND PROGNOSIS:

1. About 4% of patients die in the first attack, due to active carditis.
2. A few patients recover with no clinical evidence of rheumatic activity.
3. About 75% develop recurrent attacks of rheumatic fever, and about 5% of these will die of carditis.

In general, serious cardiac damage results from repeated attacks.

In the adult, chronic valvular disease leads to heart failure, auricular fibrillation, embolic phenomena, or bacterial endocarditis.

TREATMENT

This has 3 aspects:

1. Prevention and treatment of streptococcal infections.
2. Prevention and treatment of acute attacks of rheumatic fever.
3. Treatment of the complications of chronic valvular disease.

Acute attacks of rheumatic fever are treated by rest in bed and symptomatic measures. (a) Rest in bed is recommended until clinical and laboratory evidence indicate quiescence of the rheumatic process; (b) Salicylates relieve fever and arthritis; (c) Barbiturates are useful in the management of chorea; (d) Heart failure is treated by digitalis, diuretics, and diet. Recently, antibiotics have been given during acute attacks to eradicate any hemolytic streptococci which the patient may be carrying, and to prevent reinfection with the same organism. Cortisone and ACTH are very useful in suppressing the symptoms of polyarthritis, and it is thought that large doses given early may suppress to some degree rheumatic inflammation in the heart.

Great progress in prevention of attacks has been made since antibiotics were introduced for the prompt eradication of hemolytic streptococcal infections. Penicillin is most used for this purpose, and schedules of treatment have been published by the American Heart Association. Penicillin has also been given prophylactically to rheumatic subjects, to prevent infections with hemolytic streptococococci. To be effective such a program must be continued indefinitely, or well on into adult life.

SUMMARY

Rheumatic fever is a disease entity which is closely related to beta-hemolytic streptococcal infections. Its epidemiology seems to be parallel that of such infections. Its diagnosis may be a problem. Recurrences and their undesirable sequellae can be prevented by prompt diagnosis and adequate treatment of streptococcal infections, and by prophylactic measures aimed at preventing reinfection in rheumatic individuals.

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Toxemia of Pregnancy

JOSEPH HACKNEY, '57

INTRODUCTION

The term toxemia of pregnancy is used in reference to one or both of two clinically recognized entities: pre-eclampsia and eclampsia, which occur late in pregnancy, during labour or in the early post-partem period. At the present time, the word toxemia is a misnomer, because investigation has not been able to implicate a true toxin in either of these syndromes. Pre-eclampsia is characterized by the development of one or all of the following: marked fluid gain in body weight, with or without clinical edema; hypertension, and proteinuria. Eclampsia is characterized by one or all of the signs of pre-eclampsia, plus convulsions. Rarely does true toxemia appear before the twenty-fourth week of pregnancy.

INCIDENCE AND MORTALITY

The incidence of toxemia is difficult to evaluate as it varies geographically, as well as between private and hospital practise. For example, the figures given for Denmark are considerably lower than those given for the United States, and the world incidence is given as 1%, which is considerably higher than either the United States or Denmark. One survey predicts that the physician who averages one hundred deliveries per year may expect to encounter three cases of toxemia, and in a three year period will encounter one which is dangerous to life. Naturally the hospital rate is much higher, because it includes the abnormal cases from a selected sample of the obstetrical population.

Toxemia vies for the first place with hemorrhage and infection as a cause of maternal death, and is slowly assuming the lead. This is due to the fact that the etiology and prophylaxis of hemorrhage and infection are better understood, and, therapy, therefore is more effective.

Toxemia accounts for at least four maternal deaths per ten thousand live births in the United States. Deaths from pre-eclampsia are rare, whereas the case fatality rate for eclampsia is still 10%.

At the present time, early diagnosis of this disease is probably the most important reason for adequate prenatal obstetrical care.

PREDISPOSING AND CONTRIBUTING FACTORS

There are conditions which have been statistically shown to bear more than a coincidental relationship to the occurrence of the disease.

1) *Body Build.* Toxemia is most common in the girl who is short and stocky with a tendency to obesity, and is relatively uncommon in the tall, thin individual.

2) *Weight Gain.* Pre-existing obesity is very important as a pre-disposing cause. However, there is very little evidence to support the concept that true caloric weight gain during prgnancy pre-disposes to toxemia.

One series seems to indicate that among patients who were 20% or more underweight at the beginning of pregnancy, the incidence of toxemia increased for those who failed to gain an average amount before the third trimester.

3) *Age Groups.* The incidence is higher in those under twenty and those over forty years.

4) *Multiple Pregnancies.* Increases the incidence two and a half times.

5) *Hydatidiform Mole.* Increases the incidence eight times.

6) *Pre-existing Diabetes.* Increases the incidence by twenty times.

7) *Pre-existing Hypertension.* Pre-clampsia occurs in 1/3 of these patients.

8) *Nutrition.* Although much has been written on this subject, we actually know very little except that the incidence increases when patients are on a high carbohydrate, high sodium diet. One recent survey indicates that geographic areas with Vitamin B Complex deficiencies have a higher incidence of eclampsia, e.g. Southwestern U.S., Philippines and China.

9) *Emotional Factors.* Acute emotional episodes precede the onset of fulminating toxemia sufficiently often to warrant serious reflection about casual relationship. Also, girls with certain types of fears or frustrations such as those with illegitimate pregnancies are reported to have a high toxemia rate, even when they receive adequate prenatal care.

10) *Acute or Chronic Renal Disease.* Increases the incidence many times.

11) *Severe Anaemia.* The incidence rises as the anemia increases.

12) *Toxemia in Previous Pregnancy.* It is recognized that a patient who has had a previous pregnancy complicated by toxemia has a greater chance of developing toxemia in a subsequent pregnancy than other multigravida. Figures have been given which imply between fifteen and thirty per cent recurrences.

13) *Gravida.* The incidence is much higher in a primigravida.

THESIS OF ETIOLOGY

Since the true etiology is unknown, the following will be a summary of some of the more important present and past theories.

1) *Uremia.* This was a 19th century theory. The objection is that no significant rise in N.P.N. occurs.

2) *Injection.* The criticism of this theory is based upon the fact that no bacteria are demonstrable in blood, tissues or urine.

3) *Biological Incompatibility Between Mother and Fetus or Fetal Metabolic Products.* The disease, however, occurs in the presence of hydatidiform mole, where no fetus is present.

4) *Increased Maternal Intra-Abdominal Pressure.* This situation is present as a common denominator in some of the outstanding pre-disposing causes of toxemia. For instance: multiple pregnancies, as term approaches, and in prima gravidae where the abdominal muscles are stronger than than those of multiparae.

This increased abdominal pressure was formerly blamed for the lesions found in liver, kidney, and placenta. However, toxemia occurs with hydatidiform mole at a time when the pressure of the uterus on surrounding organs is negligible and thus seems to discount the former theory. At the present time, most workers believe that the increase in intra-abdominal or intra-uterine pressure, i.e., hydatidiform mole, produces uterine ischema by closure of intermuscular blood vessels. This causes in turn a decreased blood supply to decidua and placenta. The latter sequence would explain the fetal anoxia, placental infarcts and high incidence of abruptio seen in toxemia. It does not, however, explain the widespread vascular, visceral, or hypertensive changes.

5) *Hormonal Theories.*

(a) Posterior pituitary secretions which are increased during pregnancy might act on a susceptible vascular tree to cause increased blood pressure. By the same token, since the normal placenta seems to produce an anti-posterior pituitary enzyme, its degeneration in toxemia might be followed by a decrease in anti-enzyme, which might account for the hypertension.

(b) Chorionic gonadotrophin which increases in the blood and urine of patients with toxemia has not yet been related to the disease.

(c) Page & Glendening, working with rats showed that the administration of desoxycorticosterone acetate (D.O.C.A.), renin and high sodium diet produced an eclampsia-like syndrome with fairly typical P.M. lesions. This work has received considerable attention. In the paper he suggests that sodium intake plays the final determining role in a susceptible patient. At present we have no experimental animal that develops true toxemia under natural conditions. Therefore, we must view the above results with skepticism.

(d) Steroid hormones and estrogens are no longer thought to be an etiological factor, as they usually decrease, coincident with placental degeneration.

6) *Trueta Shunt.* Trueta, working with rabbits showed that distention of the uterus produces a reflex shutdown of renal blood vessels with hypertension and albuminuria. This theory has also been discarded as the disease does not occur in pregnant rabbits.

7) *Uterine ischemia.* At the present time this seems to be the most acceptable theory.

PATHOGENESIS

After reviewing the literature, it is evident that there are many gaps or missing links in the pathogenesis of toxemia. There seems, however, to be certain facts or occurrences which are common to many cases of the disease. This fact leads me to outline, with the aid of the literature, what I feel is a possible sequence of events.

The inter-muscular blood vessels of the myometrium undergo a decrease in lumen size and blood flow. This may occur as a result of:

(1) Increased intra-abdominal or intra-uterine pressure.

(2) Forceful uterine contractions as in labor.

(3) Obstructions by atheromatous plaques.

(4) Neurogenic constriction via sympathetic nerves.

(5) Any combination.

This causes placental anoxia and degeneration.

Then in response to one or more humoral substances the basement membrane of the renal glomerular tufts thicken and obstruct the filtration of water and salts, allowing plasma proteins to pass into the urine. This selective filtration is difficult to comprehend unless we accept the explanation put forth by E. W. Page,

"To illustrate in a homely fashion, one piece of intact filter paper in a funnel will separate water from lamp black quite neatly and at a definite rate; whereas, two thicknesses of paper, both damaged slightly with a pin will allow the leakage of lamp black at the same time that the water filters through at only one-half the rate."

In normal pregnancy, there is an increase in the sodium retaining and possibly antidiuretic hormones produced. These hormones in the toxemic patient act on the renal tubules causing increased sodium and water reabsorption from a decreased filtration fraction. The result of this is sodium and water retention with production of clinical edema. The urine output is decreased and it contains a varying amount of plasma protein.

Next, one or more humoral substances act on blood vessels in the various organs to cause spasm with resultant hypertension, and later blood vessel degeneration distal to the spasm, with production of typical visceral lesions in the susceptable individual. This humoral substance or substances might be a hormone or antihormone released or secreted by the degenerating placenta and/or a renin-like substance produced by the kidney.

The hypertension is likely a manifestation and not a cause of the disease as it has, on rare occasion, been absent or late in appearance in advanced disease.

In certain individuals with a suitably low convulsive threshold, the vascular spasms cause cerebral tissue anoxia, hemorrhages, and exudate which produce supra-added convulsions and full-blown eclampsia is seen clinically. These convulsions may further confuse the clinical and laboratory picture by production of acidosis and shifts in body water.

The hemorrhages and exudate is in heart, liver, intestines, adrenals and other organs usually are silent, but, if severe enough may cause symptoms and signs referable to any one of the affected organs.

Cardiac failure, when it occurs, is likely not due primarily to hemorrhages and exudates, but to hypertension, shock, and other factors.

POST MORTEM FINDINGS

With the exception of the placental lesions and possibly the glomerular basement membrane thickening, most workers feel that most, if not all of the visceral lesions have a common origin. This is arteriolar and capillary spasm with subsequent degeneration of the vessel distal to the site of spasm. Then, plasma or frank blood is released into the tissues.

The following are the organs which most commonly show specific pathology on post mortem.

Liver: In fatal eclampsia, liver lesions are usually present. The lesion is characteristically a hemorrhagic necrosis of the periphery of the hepatic lobule, with thromboses in smaller vessels, rupture of capillaries and considerable exudate which has compressed and destroyed surrounding hepatic cells. The hemorrhage and necrosis may be minimal or may on occasion involve the entire hepatic lobule and liver parenchyma.

Kidney: The characteristic lesion in this organ is a thickening of the basement membrane of the glomerular capillary, with a reduction in lumen size. There are no polymorphs or epithelial crescents as seen in acute glomerulonephritis. The lesions in the nephrons are not usually as advanced as those of the glomeruli unless supra-added on former nephron disease. There is usually some degree of albuminous degeneration of the tubules, some of which may be due to an attempt at reabsorption of plasma proteins.

Some cases show bilateral cortical hemorrhages and necrosis with associated complete renal failure.

Brain: The picture here may vary from simple cordical ischemia to frank hemorrhages, exudates and edema. If hemorrhages are present, they are usually petechial. It is not unusual, however, in a fatal case to find a cerebral hemorrhage which involves a large area of cortical substance.

Heart: Small areas of hemorrhage and necrosis of the myocardium are frequently seen. It is felt by most workers that these lesions are not adequate to account for loss of myocardial function.

Lungs: They usually possess various degrees of edema, and petechiae may be present. In many cases, bronchopneumonia caused by aspiration during coma is seen.

Adrenals: Lesion are frequent and consist of hemorrhages and necrosis in varying degrees.

Placenta: There is considerable disagreement as to the significance of the lesions found here, since any full term placenta will show a wealth of pathology. There is however, a greater degree of degeneration seen in the placenta of the toxemic than in a placenta from a normal pregnancy at or before term. One worker observed that up to 90% of the syncytial coats of the villi are lost, even in the areas of placenta which appear normal to gross inspection. The pathological picture

varies greatly from patient to patient and may be complicated by pre-existing disease, co-existing disease, or complications of toxemia.

CLINICAL PICTURE AND LAB FINDINGS

Pre-Eclampsia

The onset of pre-eclampsia is almost invariably in the last trimester of pregnancy, during labor, or in the first 12 hours post partum. The onset is often insidious with no subjective complaints by the patient. Commonly the sequence is:

(1) A sharp rise in body weight due to the fluid and sodium retention which may or may not cause demonstrable edema.

(2) Hypertension with a rise in both systolic and diastolic blood pressure. Some clinicians say the diastolic must exceed 90 mm. and the systolic 140 mm. Comparison of the rise to the normal for that patient is likely a more reliable yardstick to use.

(3) Proteinuria which ranges from a trace in mild pre-eclampsia, to four plus in the severe form.

Commonly, the first indication of pre-eclampsia is a weight increase of six to eight pounds in one month as indicated on the Doctor's weigh scales. Subjective complaints may be tight wedding rings, noticeable decrease in urine output, headache, vision disturbances or epigastric distress.

It cannot be stressed too firmly how important it is for the attending physician not to be lulled into false security because the patient feels well and objective findings are minimal. It is impossible to predict the course of the disease. It may remain static for weeks or progress through pre-eclampsia to eclampsia in a matter of days.

By examination of the optic fundi one may be rewarded by direct visualization of the same arteriolar constrictions that may be occurring in other organs. This spasm may involve the visible length of one arteriole or may be confined to one point, giving a sausage-like picture. Retinal edema may also be present but exudates are rare.

Urine studies in the pre-eclamptic show various degrees of oliguria and proteinuria. N.P.N. excretion is fairly normal but uric acid clearance is low.

The abnormal blood findings are increased uric acid, decreased CO_2 combining power, decreased serum proteins, and hemoconcentration may be present even in the grossly edematous patient. This is felt to be due to an intracellular shift of sodium and water.

Eclampsia

The onset of convulsions is usually preceded by severe pre-eclampsia, but not always. The Doctor may be warned in advance by complaints from the patient of a severe, boring frontal headache with accompanying visual disturbances. The seizure is usually heralded by twitching of one or more groups of facial muscles. The patient then goes into a generalized tonic seizure with cessation of respiration, froth from the mouth and cyanosis. The blood pressure is elevated to varying degrees. The tonic seizure stops and the convulsion becomes clonic with the entire body undergoing contortions for a minute or two. The cyanosis deepens in the clonic phase, then as convulsions gives way to coma, respirations are resumed. The patient usually regains consciousness between seizures.

Eighty percent of esclampsia cases will occur before or during labor. A few will occur within 24 hours after delivery but these are not usually as violent and are more easily controlled.

The Doctor must accept the fact that once convulsions have commenced, the patient may die at any moment, and therefore must have constant attention.

The blood picture during and after convulsions is as follows::

(1) Uric acid is increased.

(2) CO_2 combining power is down considerably.

(3) Urea and N.P.N. go up moderately.

(4) Lactic acid goes up because of increased muscular work.

(5) Hemoconcentration occurs soon after the seizure.

PROGNOSIS

Pre-Eclampsia

The immediate prognosis for the mother depends upon whether or not eclampsia occurs. The outlook for the fetus depends upon how early in pregnancy the disease appears. Generally speaking the later in appearance of the disease, the better the prognosis for the fetus.

Most cases will return to an apparent normal one to two weeks following delivery. A few, however, will retain varying degrees of hyptertension for an indefinite period of time. Dieckmann of Chicago believes that these are actually cases of essential hypertension which are triggered off by toxemia, and would have occurred regardless of pregnancy. Other workers believe, however, that these are really a post-toxemic hypertension.

Another few cases will return to normal soon after delivery, and then recur in subsequent pregnancies.

The question of sequelae or no sequelae will doubtless not be answered until our knowledge of etiology and pathogenesis is more complete than at the present time.

Eclampsia

If the convulsions are brought under control by medical management, the mother will usually recover completely. If the reduction in placental blood flow has been severe or prolonged, the baby often dies.

The following situations indicate a poor prognosis:

(1) A pulse rate of 120 and rising.

(2) A temperature of 103° F. and rising.

(3) Failure to regain consciousness between convulsions.

(4) A blood pressure above 240/120.

(5) Pulmonary edema or other signs of cardiac failure.

The overall case fatality rate once eclampsia has developed is 10%.

Differential Diagnosis

Under ordinary conditions where good prenatal care is available and where the patient is known to have no history of renal disease or hypertension prior to the 24th week of pregnancy, the diagnosis is usually quite straightforward, and depends upon the criteria outlined above. In some cases, however, the Doctor does not see the patient before the third trimester and, at that time she may already have advanced to the eclamptic stage. Also, although rare, the patient may develop a concurrent disease such as acute glomerulonephritis.

The following are conditions which may be confused with toxemia in a pregnant woman:

1. *Essential Hypertension*: This will usually have been present to some degree prior to the third trimester. If severe, it may produce retinal hemorrhages, and exudates. Toxemia rarely does.

2. *Chronic Nephritis*: The patient will have a history in most cases of acute or chronic disease before the third trimester. Urea clearance may help, as it is decreased in chronic nephritis and relatively normal in toxemia. Retinal hemorrhages and exudates may be present. It must be remembered that both chronic nephritis and

essential hypertension are predisposing causes of toxemia, so the clinical picture may become very confused. In some cases, the final answer may not be available until four or five months post partum.

3. *Acute Glomerulonephritis*: This may present with frank hematuria, loin pain, and a history of an upper respiratory infection. The blood N.P.N. is usually elevated in this disease in contradistinction to toxemia.

4. *Meningitis*: The patient will usually have a stiff neck and a positive Kernig's sign. Examination of the cerebro-spinal fluid for cells and bacteria may settle the question.

5. *Epilepsy*: Seizures will usually have occurred before pregnancy began. If no history is available, an electro-encephalogram may help. The attending physician must be careful that he does not treat the epileptic as an eclamptic and cause her death through overtreatment.

6. *Encephalitis*: In this case, the spinal fluid picture will usually help to differentiate.

7. *Sub-Arachnoid Hemorrhage*: Some workers believe that the hormone relaxin may cause changes in the connective tissue in the wall of the aneurysm, causing it to become weaker and more liable to rupture. The urine findings of toxemia will not be present with sub-arachnoid hemorrhage and spinal fluid examination in most cases will reveal frank or microscopic blood.

MANAGEMENT

As in most other diseases, an ounce of prevention is worth a pound of cure. There is no substitute for close co-operation between patient and Doctor.

Pre-Eclampsia

Prophylaxis

The most important factor in the prevention of pre-eclampsia is early recognition. This means frequent prenatal visits to the Doctor and recording of weight, blood pressure, and urinalysis on each visit. Also, the patient must be casually but firmly warned to notify the Doctor in the event that she should develop such symptoms as persistent headache, visual disturbance or puffiness of hands and face.

The Doctor should outline a high protein, low carbohydrate diet, and advise the patient how to restrict her sodium intake, especially in the final trimester.

In the event that the woman possesses one or more of the predisposing factors outlined above, she should be handled as a special case and seen more frequently. As an extreme example, the short, stocky, emotional primigravida, who has a history of diabetes or chronic nephritis should be seen very often indeed, as she is almost sure to develop some degree of disease.

One must also be aware of a high incidence of placental separation and hemorrhage in the pre-eclamptic. In the event that it does occur, the patient must be treated promptly and as an abruptio in any pregnancy.

Active Treatment

The object here is to get the patient as close as possible to term, prevent convulsions, and then deliver her in the most natural manner possible so that the end result is a normal, healthy, mother and baby.

If the disease is mild in nature the patient should be treated by decreasing calories and sodium, increasing protein intake and by getting rid of fluid retention, possibly with the aid of a mild diuretic.

If pre-eclampsia is more severe and causing symptoms, the patient should immediately be placed in hospital on the following regime:

1. Bed rest with visitor restrictions.

2. Low calorie, low salt, adequate protein diet.
3. B.P. taken twice daily.
4. Daily urinalysis and measurement of intake and output.
5. Daily weight measurement.
6. Mild sedation.

If, in spite of the above, the disease continues to progress, the only specific treatment for her pre-eclampsia is termination of the pregnancy, regardless of the stage, as nothing is gained for mother or fetus by temporizing. The principal reasons for terminating the pregnancy are:

(1) The prevention of convulsions.

(2) The prevention of residual hypertension.

(3) The fetus, though premature, is safer outside of the uterus than within and dependent upon an inadequate blood supply.

The method of termination must be chosen for each individual case. If the head is engaged in an adequate pelvis and the cervix favorable, the patient should be induced by rupturing the membranes.

If the membrane rupture is not followed promptly by labor, the patient should probably be given intravenous pitocin in 5% dextrose solution. One method described is to add 0.25 cc. of pitocin to one litre of 5% dextrose in distilled water and give intravenously at a rate which is rapid enough to maintain adequate labor. Pituitrin is absolutely contraindicated, as it contains pressor and anti-diuretic principles.

If the head is not fully engaged and the cervix unripe, the patient should be delivered by Caesarian section. If possible, a regional anaesthetic should be used.

Eclampsia

Prophylaxis

In the case of eclampsia, this consists of adequate prevention or control of pre-eclampsia and if both of these fail, prompt termination of the pregnancy.

Active Treatment

This is directed towards controlling convulsions and rapid termination of the pregnancy twelve to twenty-four hours later. It must always be remembered that an untreated eclamptic woman fares better than one who receives an operative termination of pregnancy during the acute stage of her illness.

General Measures:

(1) The patient must be hospitalized and placed in a quiet room in a bed with side boards. A nurse should be in constant attendance.

(2) Place a retention catheter in the bladder. This will avoid repeated catheterization and will allow hourly output measurement.

(3) Record T.P.R. at regular intervals.

(4) Record blood pressure q. 1-2 h.

(5) Record fetal heart rate q. 4 h.

(6) Do a complete urinalysis b.i.d.

(7) The patient may take nothing by mouth while the threat of convulsions is imminent due to danger of fluid aspiration. 1500-2500 cc. of hypertonic glucose in distilled water in a 24 hour period. This must be regulated to urine output, cyanosis, and pumonary edema. If there is embarrassment of respiration by fluid, give 250 cc. of 25% glucose in distilled water q. 6-8 hours.

(8) A suction apparatus should be present in the room for prompt removal of mucous and fluid.

(9) Enemas should not be given unless the patient is suffering distress as a result of constipation.

Measures to Decrease Body Water:

(1) Limit sodium intake to 1 gm. or less per day.

(2) Give hypertonic glucose to cause diuresis.

(3) Give a mild diuretic (ammonium chloride).

(4) An electric blanket may induce sweating and fluid loss.

Measures to Control Hypertension and Convulsions:

(1) Magnesium in the form of magnesium sulfate will stop convulsions in adequate blood concentration. One method is to give 20 cc. of a 10% solution intravenously, repeated at intervals which depend upon the blood pressure. Since magnesium ion is excreted only by the kidneys, it must not be given in the presence of anuria.

(2) Paraldehyde is a safe and efficacious drug. It is usually given per rectum in an equal amount of olive oil; 30-40 cc. may be given at one time. Paraldehyde is very useful in this disease because unlike morphine and some barbiturates it has no anti-diuretic effect.

(3) Barbiturates should not be used in the presence of impaired hepatic or renal function because of the danger of drug accumulation. At times, however, the drugs may produce dramatic results in therapy of convulsions and hypertension.

(4) Opiates are only mentioned to condemn them. In doses large enough to prevent convulsions, they cause depression of respiration, suppression of urine output and increased spinal reflexes.

(5) Veratrum may be given intravenously at a rate sufficient to keep the blood pressure within normal limits. One method is to give 5-10 minim doses of veratrone at 20 minute intervals to keep the systolic blood pressure between 120 and 150, and the pulse rate below 80. Veratrum must be employed with extreme caution due to the danger of causing circulatory collapse.

(6) Apresoline may be given orally, intravenously, or intramuscularly, and according to some is a relatively safe agent for lowering blood pressure in hypertensive disease of pregnancy. Tachycardia, headache, and nausea are frequent side effects. The drug is said not to decrease renal blood flow.

(7) Continuous regional block anesthesia is an effective method of lowering blood pressure. The procedure, however, carries with it a great deal of risk unless carried out by a skillful anesthetist.

(8) Venesection has, in the light of modern day thinking only two indications. One is pumonary edema, and the other is congestion in the peripheral circulation.

Miscellaneous Drugs:

(1) Digitalis is indicated for heart failure associated with toxemia. The dosage and preparations used are the same as for any other heart failure.

(2) Cortisone has been used with apparent beneficial results in certain critical cases of eclampsia. It is not known for certain what action the drug may have other than possibly substitution for a damaged adrenal cortex.

Termination of Pregnancy:

This should not be considered until 12-24 hours after the patient is apparently free from convulsions. The methods have been described above.

SUMMARY

1. Maternal mortality in toxemia is almost always associated with convulsions. Therefore, the disease must be controlled at all costs before pre-eclampsia becomes eclampsia.

2. The best treatment for pre-eclampsia which is progressing in spite of medical management is an empty uterus.

3. The most effective treatment of toxemia is carried out in early pre-eclampsia. Therefore, successful treatment is based on early diagnosis.

4. Early diagnosis is accomplished by a combination of

a. a recording at each visit of the patient's B.P., weight and urinalysis,

b. a casual awareness of subjective findings that she must report immediately,

c. an awareness by the doctor of anything that might predispose that patient to toxemia.

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"The woman whose anatomy I demonstrated was named Sancta, and a month earlier she had given birth to twins which unfortunate creatures had scarcely come into the light when she deprived them of it by suffocation, and the judges ordered that she too be suffocated for as the poet said 'There is no law more just than that murderers perish by their trade.' Therefore in this woman named Sancta, but in truth a demoniac and sorceress . . . spectators were able to understand through what veins the menses flow and that they do not cross through the uterus."

Demonstration of the Uterine Veins, Realdo Colombo, 1545.

Portal Cirrhosis

DONALD CAMPBELL, '57

INTRODUCTION

This condition is also known as Laennec's Cirrhosis, Alcoholic Cirrhosis, and Atrophic Cirrhosis. The stony hard liver with dropsy was described nearly three centuries B.C. by Erasistratus of Alexandria. The first adequate account of portal cirrhosis was written in 1819 by Laennec who was attracted to the fawn color of the projecting nodules on the liver and thereby named the disease "cirrhosis", from the Greek word "Kirrhos" meaning orange coloured—or tawny. The fibrosis and nodular regeneration of the liver, however, are considered to be more important since the color simply depends upon the amount of hyperemia, hemosiderosis, necrosis, fat and jaundice; but the term cirrhosis has become too universally established to be displaced.

INCIDENCE

Portal Cirrhosis is by far the most common form of cirrhosis. In the U.S.A. it is discovered in 2-3% of all post-mortem examinations. It occurs most commonly between the ages of 45 and 65 years, and occurs in men two to three times as frequently as in women. Portal Cirrhosis occurs most often in occupational groups with ready access to alcoholic beverages although the relationship between cirrhosis and alcohol still is not definite. It has been seen that 70% of all patients with cirrhosis have a history of chronic alcoholism but at a maximum only 30% of all chronic alcoholics develop the disease.

ETIOLOGY AND PATHOGENESIS

Portal Cirrhosis has been generally regarded as a response to injury of some type. This primary injury may take one of the following forms:

1. Repeated infections such as acute virus hepatitis, malaria and syphilis.
2. Toxic agents such as lead, copper, arsenic, chloroform, ether, carbon tetrachloride, and alcohol.
3. Clinical studies of recent years have placed more importance on the co-existence of chronic alcoholism and nutritional deficiencies.

The three basic pathological processes going on in the liver are:

1) Necrosis of hepatic cells
2) Fibrosis
3) Regeneration

The initial necrosis may be due to any of the above toxic materials which interfere with tissue respiration causing anoxia of cells. When there is anoxia of the cells, efficient carbohydrate metabolism cannot take place and the cells become infiltrated with fat which replaces the normal glycogen. Marked fatty infiltration appears to be an antecedent of cirrhosis and the liver which is initially fatty, later shows fibrosis. The essential cause appears to be a diet deficient in *lipotrophic* substances which is aggravated by their defective absorption from the gastro-intestinal tract. A direct toxic action of alcohol is as yet to be proven but the drinking of excessive amounts of alcohol contribute indirectly to the condition by leading to loss of appetite and decreased food intake and by setting up a gastro-enteritis, thus interfering further with absorption.

A lipotrophic substance is a substance which appears to reduce the amount of

fat in the liver. These lipotrophic materials are the lipoproteins, choline and substances which promote choline synthesis such as methionine and lecithin. The mode of action of the lipoproteins is unknown. The important point however is that choline appears to prevent and cure fatty liver before it has gone on to the stage of fibrosis.

Fatty liver leads to necrosis of cells and fibrosis. The scarring, by interfering with hepatic blood supply, aggravates the condition of anoxia and sets up a vicious circle leading to more necrosis and scarring.

PATHOLOGY

The size of the liver varies. It is usually smaller but may sometimes be considerably larger. In general it is large in the early fatty stages but becomes smaller as fibrosis takes place. The consistency is very firm due to the connective tissue. The surfaces are roughened by nodules varying in diameter from 1 mm. to 1 cm. The cut surface of the liver shows nodules and the lobules of liver cells can be seen separated by bands of connective tissue.

Microscopically the important feature is the absence of the normal lobular architecture of the liver. Varying sizes of groups of liver cells are separated by broad bands of fibrous tissue running between portal areas.

SIGNS AND SYMPTOMS

The liver possesses so great a reserve that 80% or more of its substance must be attacked before symptoms and signs of hepatic insufficiency develop. Cirrhosis is a disease of great chronicity and the symptoms usually are very gradual and sometimes a bout of acute alcoholism, or an intercurrent infection such as the common cold or pneumonia, may precipitate a state of acute hepatic failure.

Sooner or later, before hepatic insufficiency develops, the fibrous tissue begins to obstruct the return of portal blood, resulting in partial portal obstruction causing congestion of the stomach and intestinal mucosa and the early gastrointestinal symptoms develop. These are, dyspepsia, anorexia, nausea, vomiting, weight loss. These symptoms may be aggravated by the gastroenteritis which often follows consumption of alcohol. Bowel irregularities, alternating diarrhea and constipation, may result from edema and congestion of the lower bowel.

In an attempt to get around the portal obstruction the anastomoses between the portal and systemic systems of veins enlarge and a collateral circulation is established which is most evident at the following sites:

1. The veins of the lower end of the esophagus become varicose and anastomose with those of the stomach forming esophageal varices.

2. The inferior mesentric veins anastomose with the hemorrhoidal veins forming hemorrhoids.

3. The formation of a caput medusa due to anastomosis between para-umbilical and superficial epigastric veins.

Abdominal fullness due at first to flatulence and later to ascites is also an early symptom. Not uncommonly, the patient may complain of abdominal pain which has the quality of a dull dragging ache in the right hypochondrium. Signs of biliary obstruction with frank jaundice are uncommon in the early stages of cirrhosis although a very mild jaundice with a subicteric tinge to the sclerae may be seen.

Sooner or later signs of liver failure appear and the more specific signs and symptoms of cirrhosis become obvious.

1. *Fluid Retention*: Ascites, pleural effusion and edema.

Ascites is the fost frequent and characteristic sign of cirrhosis and appears in 80% of patients. Fluid retention is thought to be due to:

1) Low serum albumin levels due to lack of production of albumin in the liver.

2) Increased pressure in the portal system causing increased capillary permeability.

3) Salt retention by the kidney for some unknown reason. This is referred to as cirrhotic nephrosis.

4) Lack of breakdown of antidiuretic hormone.

2. *Angiomata or Spider Naevi*: These are seen in 2/3 of cases most frequently over the head and neck. They consist of a central red point with radiating strands. These blanch on pressure. Liver palms is an erythema of the thenar and hypothenar eminences. These are thought to be due to failure of the liver to metabolize estrogens. For the same reason it is common to find impotence and loss of libido in male although true gynecomastia and testicular atrophy are infrequent. Oligomenorrhea and amenorrhea are common in the female patient with cirrhosis and this is also thought to be due to high estrogen levels.

3. *Bleeding Phenomena*: These can be related to low prothrombin and fibrinogen levels due to a lack of production of these substances in the liver. Massive hematemesis of a litre or more is not unusual due to ulceration and rupture of esophageal varices. The latter may be the presenting symptom in 10% of cases.

4. *Low Grade Fever* unaccompanied by leukocytosis and lasting for weeks or months has been observed in about ½ of these patients. The reason for this is not known. This phenomenon is particularly apparent in patients with ascites.

5. *Clubbing of the Fingers* is infrequent in portal cirrhosis but is more common in biliary cirrhosis. The explanation for this is unknown.

6. *Neurological manifestations* of cirrhosis are usually those of peripheral neuritis on the basis of the accompanying nutritional deficiency and chronic alcoholism.

7. A peculiar *sweet odor of the breath* commonly occurs in cirrhosis. This is called "liver breath" and may develop at any time during the course of the disease. If it appears suddenly in association with worsening of the patient's clinical state, the prognosis is poor. Frequently however, the odor develops gradually and many of the patients continue to be ambulatory for a number of years.

8. *Coma* usually appears terminally and may be ushered in by a brief pre-coma period of inappropriate behavior, progressive mental confusion and euphoria. The patient drifts off to deepening coma as contact is lost. While in coma, pyramidal tract signs, choreiform movements and athetoid postures are evident. Twitching and coarse fasciculations are usually observed but delerium is not common.

9. *A palpably enlarged spleen* occurs in more than half the cases of cirrhosis and has been reported in as many as 80% in some groups. This finding along with anemia, leukopenia, thrombocytopenia, and attacks of gastrointestinal bleeding constitutes what is known as Banti's Syndrome and is produced on the basis of portal hypertension due chiefly to cirrhosis.

LABORATORY SIGNS OF HEPATIC FAILURE

1. Normochromic anemia is common but occasionally a macrocytic hyperchromic anemia is seen and is thought to be due to failure of the liver to form or store the anti-pernicious anemia principle. Hypochromic anemia may be present if there has been blood loss from leaking esophageal varices.

2. One of the most constant findings is alteration of the serium proteins. In early hepatic insufficiency due to cirrhosis, the globulin will rise producing

little change or a slight rise in total proteins. Later albumin decreases thus lowering the total protein values unless the globulin has shown a proportionate rise.

3. The prothrombin time is prolonged and is unresponsive to parenteral administration of vitamin K.

4. A rise in icteric index and serum bilirubin occurs with jaundice and the serum bilirubin is usually the direct reacting fraction.

5. Cephalin cholesterol flocculation tests frequently show a 3 plus or 4 plus reaction but the thymol turbidity test may be only slightly elevated unless the globulin levels are elevated.

6. One of the most useful tests is the Bromsulphalein excretion test. In cirrhosis more than 5% of this dye is usually retained at the end of a 45 minute interval.

DIAGNOSIS

Diagnosis is obvious when the disease is well advanced and is difficult during the early stages. It is most important to make the diagnosis early since proper therapy if instituted, will slow progression of the disease and prolong the life of the patient. Aside from the history, the clinical picture of the patient and the laboratory examinations as mentioned above, the following procedures may be helpful in the more puzzling cases.

1. Abdominal paracentesis. The purpose of this is twofold as far as diagnosis is concerned.

   (a) It relieves distension of the abdomen so that the liver and spleen can be palpated.

   (b) It permits an examination of the ascitic fluid for characteristics of of the transudate and a study of the sediment to rule out malignancy.

2. Visualization of the lower esophagus by fluoroscopy and X-ray following a swallow of thick barium in an effort to determine the presence of varices.

3. The expert use of the punch or needle biopsy is not often necessary but is the ultimate means of establishing the diagnosis.

PROGNOSIS

Cirrhosis of the liver does not necessarily imply a poor prognosis and a considerable number of cases remain latent throughout life. However when clinical signs of hepatic failure develop the outlook is grave. Ten years ago, after the onset of ascites, approximately 80% of these patients would be lost within two years and an additional 10% by the end of three years. Jaundice or hematemesis in the presence of ascites exaggerated these statistics. Today these figures have been greatly altered by the application of nutritional therapy.

CAUSE OF DEATH IN CIRRHOSIS

1. Death is most commonly due to cholemia with a picture of deep coma and intense jaundice.

2. Esophageal bleeding may be the cause of death either directly or by precipitating coma. Since coma is thought to be related to high ammonia blood levels, the cholemia may be due to breakdown of blood in the gastrointestinal tract with resulting absorption of ammonia.

3. The patient with cirrhosis in his weakened state is subject to infections and before the era of antibiotics, the commonest cause of death was pneumonia. Today, however, cholemia and hemorrhage are more important causes.

TREATMENT

There is an abundance of literature on treatment of cirrhosis. This fact speaks for itself. That is, a curative treatment

and the proof of its efficacy are wanting today.

Since the modern concept of cirrhosis is a nutritional deficiency syndrome, therapy should be aimed at measures which ensure an adequate food intake of the widest type.

1. Primarily, appetite should be stimulated in an anorectic patient. This can be carried out in several ways.

(a) Withdrawal of alcohol in a patient who has been drinking liberally often restores appetite. The discontinuance of alcohol is probably advisable in any case since it is considered to be hepatotoxic.

(b) Large amounts of parenteral Vitamin B or crude liver extract IM are useful supplements.

(c) Ten units of insulin twenty minutes before each meal has been of some aid but should be used with caution.

2. Opinions vary as to the type of food to be given. As far as proteins go, it seems logical that in the presence of a poorly functioning liver deamination of proteins is poor and the accumulating nitrogenous products may push a borderline case into hepatic coma. However, proteins of all kinds appear to be alright in the absence of signs of hepatic failure.

It has been the tradition to prohibit fat in the cirrhosis diet because of the possibility of further liver injury. Today it is thought that the inclusion of fat actually carries no unusual risk if balanced by the protective action of protein. This fat improves the taste of the diet and also supplies the essential fatty acids.

Carbohydrates should be supplied up to amounts of 250-350 G. per day. Supplementary vitamins have been used a great deal in the treatment of cirrhosis. In the presence of marked malnutrition and vitamin deficiency syndromes, there is a rationale for their use; but ordinarily, they need not be continued long after the time necessary for the reversal of the deficiency syndrome.

The use of the liptrophic substances is controversial. Theoretically if the patient has a hypertrophic fatty liver they may be useful but in the scarred atrophic stage there does not appear to be any indication for their use.

3. In the presence of decompensated liver disease, it is important to reduce the demands on the liver. Thus absolute bed rest is indicated. The use of morphine and short acting barbiturates is contraindicated in patients with liver disease. Sedation may be necessary in acute alcoholic episodes and paraldehyde or chloral hydrate is preferred in these cases.

4. Antibiotics may help because they inhibit the bacteria in the liver and the gastrointestinal tract and thus slow the absorption of ammonia. Also, the detoxification of bacteria which goes on in the liver is impaired in cirrhosis, thus the use of antibiotics will protect the patient from infection.

5. Ascites should be removed by paracentesis if it interferes with gastric filling or breathing but not more often than necessary because of the accompanying protein and electrolyte loss. Solutions of human serum albumin have shown great promise in the control of ascites and edema and correction of hypoproteinemia. Until albumin solutions are generally available they are not included in the general management.

Mercurial diuretics produce freedom from ascites in an occasional patient but they usually act indifferently or not at all. Ammonium chloride is contraindicated because of the gastric irritation which ensues thus aggravating the anorexia. Some investigators consider that ammonium chloride may precipitate coma due to a rise in blood levels of ammonia.

It is wise to restrict salt intake in patients with ascites. The more persistent the ascites, the greater the salt restriction should be.

6. Numerous surgical measures have been devised to control esophageal bleeding.

(a) in acute episodes, esophageal tamponade using a triple lumen rubber tube has been life saving.

(b) spleno-renal shunt offers a reduction of at least 25% of the portal pressure.

(c) injection or ligation of varices is occasionally successful.

7. The treatment of hepatic coma is unsatisfactory. In general it consists of:

(a) large amounts of intravenous glucose with added Vitamin B complex.

(b) As in many other conditions where cortisone and ACTH have been tried, the results are conflicting.

The appearance of cholemia is often the terminal stage of progressive liver failure, although it has been shown to be reversible, but only about ¼ of these patients have recovered long enough to be discharged from the hospital.

"One will only really understand how a person thinks about a definite point when one knows the general constitution of his mind. This is true if we are desirous of apprehending exactly the opinions concerning scientific matter, whether of simple individuals or whole schools or centuries. For this reason the history of the sciences is intimately bound up with the history of philosophy, but equally so with the history of the lives and characters of individuals and of people."

Goethe, Zur Farbenlehre, 1881.

Spinal Cord Trauma

FAY INMAN, '57

The first reference to spinal cord injury is found in the Edwin Smith Papyrus published in 2500 B.C. where it is stated: "One having a crushed vertebra of his neck, he is unconscious of his two arms and two legs and speechless. An ailment not to be treated." It is not surprising that it was then thought unwise to perpetuate a state of misery which could not be relieved. But it is perhaps surprising that over forty centuries should have elapsed before any alleviation of this tragic fate could be offered to victims of traumatic paraplegia who so often are affected in the prime of life.

In fact it has been in the past fifteen years that the greatest advances have been made in treating the patient with spinal cord trauma. Of the American soldiers who sustained traumatic paraplegia in World War I, one alone was alive 20 years later. So it was in Britain and elsewhere. They all died miserably. Watson Jones gives a very graphic description of these patients. "They lay rotting in bed with large stinking bedsores which increase in size as emaciation got worse until the bones were exposed, lacking sensation, incapable of movement, often with distressing involuntary spasm and secondary contraction of joints, incontinent of urine and faeces, with infection of urethra, bladder and renal tract. They were doomed to die within a few years of inanition, uremia and pneumonia."

Now a new hope has been born. Monroe of Boston— a great pioneer in this field said: "If they have good upper limbs and are intelligent they can be assured of ability to walk, though perhaps with appliances. They will gain complete 24 hour control of bladder and bowel. And they will earn their own living and lead an almost normal life." This hope has now become a proved experience. Unlike the sole survivor of World War I, of the 4000 American soldiers paralysed from spinal cord injury in the Second World War, more than 2000 are surviving, and of these 80% are able to walk, and are in jobs or training for jobs.

MECHANISM OF INJURY

The causative forces of spinal injuries may be placed in three main categories:

1. *Indirect Violence*: This is the most frequent cause of civilian injuries. It occurs as a result of falls, diving accidents, birth injuries produced by the traction accompanying breech extraction, automobile accidents and the like. The applied force whether it causes rapid acceleration or deceleration enforces exaggeration of the normal movements of the spine in flexion or extension. Thus the neck may be whipped forward or jack-knifed as a result of sudden stop occasioned by an automobile accident; or again it may be acutely flexed as in a fall on the head. Overflexion is the most common mechanism.

2. *Direct Violence*: This occurs in falls directly onto the back—particularly across a rigid object. It may be produced by falling objects and by crushing injuries so common in automobile and airplane accidents. As a result of direct and indirect violence the vertebra are fractured and dislocated, thereby injuring the cord.

3. *Penetrating Wounds*: This type of injury is seen most often among the casualties of modern warfare and may be produced by missiles of any types, high explosive fragments or stab wounds.

PATHOLOGY

There are three major pathological entities:

1. The first of these is *Concussion.* This category includes all of those injuries which fail to give permanently detectable neurological signs. Grossly there are no changes observable except perhaps some engorgement of blood vessels. Microscopically there are vaguely defined cellular changes in the grey matter—the white matter being completely intact. Clinically there is a momentary disturbance of function which disappears rapidly and completely.

2. Under *Contusion,* the second category, are included those injuries which exceed concussion to the point where small vessels are ruptured within the substance of the cord. Grossly there may be no early external evidences of the lesion for the vascular net of the grey matter is more often affected. However subpial hemorrhage may be seen. Microscopically extravasation of blood and cellular destruction are evident, and in more severe cases the fibres of the white matter are disrupted. Functionally there is always a residual of this type of injury, ranging from scarcely detectable motor signs to complete loss of all cortical connections.

3. The final category is *Laceration.* There is actual incision of the cord material well beyond the pia. Grossly the damage is obvious and ranges from incisions such as are imparted by the scalpel to wide bands of blunt tearing by dislocated bone. Anatomical transection is included in this group. As long as the pia mater is intact, laceration is excluded. Microscopically, the disruption of pathways in the white matter, destruction of cells in the grey and hemorrhage are observed. Functionally, the gamut from scarcely detectable changes to partial lesions (for example the Brown-Sequard Syndrome) on to complete loss of function are observed. There are always permanent changes from such injuries, but they are not always permanently disabling.

CLINICAL FEATURES AND PHYSIOLOGY

Complete Transection of the Cord

Immediately following the accident if there is no associated head injury the patient will be entirely conscious and rational. He frequently experiences the sensation of movement below the level of the lesion.

On examination there is flaccid paralysis and absence of reflexes below the level of the transection. As a result of vasoparalysis, sweating is abolished, the skin is dry, pale and lifeless, the blood pressure drops precipitously. Suppression of bladder reflexes accompanied by intense spasm of the bladder sphincters results in urinary retention. The rectal sphincter is likewise spastic, peristalsis is absent, and the abdomen silent. Abdominal distention soon appears. This is a description of spinal shock which usually lasts 3 to 6 weeks this phenomenon is apparently due to sudden suppression of impulses descending from the upper centres to motor neurones in the spinal cord.

With time the cord regains its primitive powers of activity. As the stage of shock passes off functional activity returns first to smooth muscle. Tone returns to bladder and bowel walls, and blood vessels—the blood pressure rises to normal levels. Tone returns to skeletal muscles, especially of the flexor variety, and the state of decreased reflex activity is replaced by one of heightened activity. On stimulation withdrawal reflexes return first—in the form of the Babinski response. Gradually dorsiflexion of the foot is added and eventually withdrawal of the whole leg. In the exaggerated form the withdrawal reflex is known as the mass reflex. When fully developed, it may arise spontaneously or be evoked by cutaneous stimulation.

Concussion of the Cord

Concussion of the cord may produce a clinical picture which in no way differs

from that seen after complete anatomic severance. However some return of function is usually apparent within 24 to 48 hours, and eventually there is complete recovery.

Partial or Incomplete Spinal Cord Lesions

The clinical picture of incomplete spinal cord lesions is extremely varied. The paralysis below the level of the lesion is incomplete, asymmetric and although present immediately after the trauma may be flaccid or spastic in type. There are varying zones of sensory loss below the level of injury. Loss of pain and temperature sensation usually extends to a higher level than other sensory modalities. The effect on the more discriminative sensations is more profound than the cruder forms of sensation. Irritative phenomena such as areas of hyperalgesia and hyperesthesia are common and radicular pains occur frequently. Bladder function may be intact from the onset, or if absent initially will return relatively early.

With return of reflex activity there are certain differences between complete and incomplete transections of the cord. With incomplete transection some descending fibres from the brain escape injury and therefore connections between brain and cord exist. The recovery of functional activity is more marked in the extensor group of muscles so that the paralysed lower extremities tend to assume a fully extended position—rather than the flexed position of complete transection.

A discussion of the clinical picture in specific segmental levels of injury is beyond the scope of this paper. Such information is readily obtained in any standard neurologic text.

TREATMENT

The treatment of injury to the spinal cord can be arbitrarily divided into several phases: Immediate or emergency measures; the stage of initial recovery while the patient is bedridden; operative treatment; and finally the stage of rehabilitation.

Immediate or Emergency Phase of Treatment

Treatment of the patient should begin at the scene of the accident. A patient who has been partially or totally paralysed by acute spinal cord injury should not be hastily moved from the scene of the accident as blood loss and shock are rarely important factors in the patient's condition. First, a survey of the situation should be made. Then if at all possible removal of the injured person should be supervised by a physician. The following instructions may be used as a general first-aid guide.

Primarily, do not flex or hyperextend the patient's back. Do not allow the patient to try to sit up. Rather keep him lying flat on his back, in the perfectly extended position.

In moving the patient to a stretcher, if possible 5 persons should be in attendance. One person, preferably the doctor, applies traction to the head and neck; another applies traction to the legs. The other 3 persons lift the trunk. The stretcher should be firm and flat—not made of canvas since it tends to allow flexion of the patient's back. A flat plywood board or door is suitable if nothing else is available. Sandbags should be placed behind and on either side of the neck in the case of cervical injury. In transportation do not bounce or jostle the patient.

In hospital allow as little movement of the patient as possible. X-rays of the spine may be taken on the original stretcher. Great care must be taken not to dislodge loose bone fragments of the vertebra which might convert a partial to a complete transection.

Stage of Initial Recovery

This is the period immediately following the injury while the patient is still

bedridden. Here there are 4 major categories to consider: The care of the bladder; care of the bowels; maintenance of a healthy skin; general and nutritional care.

1. *Care of the Bladder*

Care of the bladder is one of the most important problems in spinal cord injury. A knowledge of bladder physiology and the development of the neurogenic bladder is essential for intelligent therapy of bladder dysfunction in these cases.

Immediately following serious trauma to the spinal cord, spinal shock ensues with depression of spinal segments caudal to the lesion. The bladder is immediately affected as shown by atonicity of the bladder wall, absence of reflex contraction of the detrusor muscle, failure of sensory impulses to the brain and the motor impulses from it. The bladder distends to a huge size; overflow incontinence occurs in 36 hours. This atonic bladder represents the first of three merging but fairly well defined stages of spontaneous recovery. This first stage may last 1 day to 18 months. Overflow incontinence is the only type of urination to be expected.

In the second stage, lasting a variable period of time from days to months, there is a return of reflexes caudal to the injured segment of the cord. There is an increase in intravesical pressure. Reflex contractions of the bladder wall occur, but they are of insufficient strength or duration to produce efficient emptying. The clinical picture is that of periodic overflow incontinence. This is an autonomous neurogenic bladder.

Progression to the third and final stage of recovery depends on the extent and level of injury. In incomplete transection of the cord, a normal cord bladder under voluntary control may ultimately be attained. In complete transection an uninhibited reflex bladder is possible. When the patient experiences such symptoms as sweating, flushing of the face, or headache he knows that his bladder is distended. Micturition can then be initiated by some method of extravesical stimulus such as massaging the lower abdomen.

Treatment of the bladder in these patients is directed at the prevention of overdistension, infection and the formation of calculi. The ultimate goal is a "social bladder".

On admission to hospital a urethral catheter should be inserted to prevent bladder distension. As soon as it practicable tidal drainage should be instituted. This is the most effective method of maintaining bladder capacity and tone, and guarding against infection. M and G solutions, composed of citric acid, magnesium oxide, and sodium bicarbonate, autoclaved before use, are the most popular irrigating agents. Attempts to establish an automatic bladder are begun as soon as possible. In general, this is after the period of spinal shock has started to disappear. This is best accomplished after removal of the catheter by the use of the Crede maneuver every 4 hours. The patient himself is instructed in how to express the urine manually and supervised until he does so properly.

Urinary infections are a tremendous hazard in these patients. Care should be taken to prevent contamination of the catheter. The use of solutions M and G as bladder irrigants do much to discourage sepsis. Chemotherapy and antibiotics are reserved for clinical infections and the prophylactic use avoided. The attending physician should be constantly on guard for signs and symptoms of infection. Routine urinalysis should be performed once a week as a safety check.

Because of prolonged bed rest which tends to promote stasis of urine, and demineralization of bone with resultant high urinary excretion of calcium, and the high incidence of urinary infection, paraplegic patients are prone to develop urinary calculi. This complication is best avoided by early ambulation of the patient. Other prophylatic measures are a high

fluid intake, and an acid diet with low calcium content. Since many of these patients suffer no pain with development of calculi, some authorities advise the taking flat films of the abdomen at monthly intervals as a measure for early detection.

2. *Care of the Bowel*

Care of the bowel is simpler than the problems presented by the bladder disturbance. During the stage of spinal shock the bowel is atonic, peristalsis is absent, and the rectal sphincter is in spasm. Enemas at this time fail to stimulate contraction or initiate emptying and may merely aggravate the distension already present. Therefore for the first few days after injury it may be advisable to withhold all food and fluids by mouth and to maintain fluid balance by parenteral routes. Distension may be combated by the use of the rectal tube and Wangensteen suction. Prostigmine may occasionally be beneficial.

The passage of flatus and return of peristalsis herald the return of bowel contractability and decrease of sphincteric spasm. At this point bowel training may begin. Enemas are given every 2 to 3 days until return of reflexes indicates that evacuation is possible without the use of enemas. It then becomes desirable to place the patient on the bedpan at regular intervals and endeavour to initiate spontaneous evacuation by stimulation of the abdomen, thighs, or perineum. Laxatives if used at all should be mild.

3. *Maintenance of a Healthy Skin*

Decubitus ulcers are the most common complication in paraplegic patients, occuring in 85% of such cases. A large amount of body protein may escape from their weeping surfaces, thus depleting the patient's nutritional state. Their prevention requires nursing care of the highest degree. The patient must be turned every two hours. Use of the Stryker frame make this maneuver much easier for patient and attendants. Flannelette sheets should be used, and kept free of wrinkles. The skin must be kept dry at all times with special attention given to the perineum after bowel movements. Hot water bottles should never be placed next to unprotected skin. The knees should be partly flexed, and support placed beneath thighs, knees, and calves, leaving the heels free of any pressure.

The better the general condition of the patient, the less prone to decubitus ulceration he is, so that every effort must be made to maintain a good nutritional state.

4. *General and Nutritional Care*

Maintenance of adequate nutrition presents a major problem. An adequate intake must be enforced, if necessary by supplement to the diet by other methods of feeding. This is especially true when protein is being lost through exudation from bed sores. When the plasma level falls, a 4000 Calorie diet with 150 grams of protein is recommended.

Anemia not only further delays healing processes, but also enhances malnutrition. Thus if the hemoglobin falls below 80% transfusion of whole blood is indicated. Similarly if the plasma protein level falls to a value of grams or less, transfusion of whole blood, plasma or serum albumin is recommended.

The muscles of the shoulder girdle and arms are essential if a permanently paraplegic patient is to be ambulatory. These vital muscles rapidly undergo marked atrophy from disuse. Therefore, physical therapy in the form of resistance exercises for the unaffected upper limbs must be instituted early, while the patient is still confined to bed. In addition, the paralysed lower extremities should be carried passively through a complete range of movement twice daily.

The paraplegic patient faced with a life of chronic invalidism has every reason to be depressed. Yet, every effort must be

made to maintain his morale. Though prognosis for return to normal function may be hopeless, the doctor should stress the possibilitie of rehabilitation.

Operative Treatment

This discussion will be confined solely to neurosurgical considerations. Orthopedic measures are not included.

The first subject that must be considered is decompressive laminectomy. During the early phase of the post-traumatic period it is necessary to decide whether laminectomy is indicated. The question has been controversial for the past 25 years.

Watson-Jones states that indications for laminectomy exist in only 3 types of cases:

1. In the rare case of depressed fracture of the lamina with protrusion of the bone into the spinal canal.

2. In the incomplete lesion where there there is a progressive increase in neurological signs suggesting the development of an epidural hematoma.

3. In complete or incomplete paraplegia where there is no X-ray evidence of fracture or dislocation, but there is a block to the flow of spinal fluid as proved by lumbar puncture.

In contrast Covalt and associates of the New York University College of Medicine advocate laminectomy in every early case of paraplegia that is due to injury of thoracic and lumbar spine. Every patient in whom there is a cervical vertebra dislocation should have it reduced. Skeletal traction should be tried first and if not successful it should be followed by laminectomy.

Covalt presents the following arguments in defense of his line of therapy:

1. At the present time there is no way of ascertaining by neurological examination whether there has been an anatomic transection of the cord. Although there may be clinical evidence of complete physiological transection, an examiner is not justified by clinical examination in stating that the cord has been irreparably damaged and in concluding that laminectomy would be of no avail. Therefore unless surgical exploration reveals a transected cord, one cannot conclude during the early weeks after injury that a patient is permanently and irreversibly paraplegic.

2. Although X-ray is of considerable aid in estimating neurological damage, complete reliance on this examination is not justified.

3. Covalt and workers found that a small percentage of patients who undergo laminectomy even as long as one year after injury will benefit from surgery. Particularly at the critical levels, such as at the level of origin of brachial and lumboscacral plexuses, one can usually anticipate at least a recovery of 2 to 3 roots following laminectomy. This is often sufficient compensation to the patient.

4. The complications of pain and spasticity in chronic paraplegia occur less frequently in those patients who have undergone laminectomy. The removal of bony spicules, foreign bodies, hematomas and dural scars will remove factors favourable to development of pain and spasticity.

5. Laminectomy carries a low mortality and morbidity rate. From the mortality standpoint it compares favorably with such operations as tonsillectomy.

6. Psychologically paraplegic patients adjust to their disability better if they feel everything possible in the way of definite treatment has been done for them.

Freeman of Indiana, also an advocate of early laminectomy, states his argument strongly thus: "The ideal time for surgery would be in the first 20 minutes but failing that, every minute lost could mean further circulatory embarrassment and one would think that if decompression would

be of any value at all, it would relieve pressure so that circulation could be restored.

The control of pain and mass spasms may also be attempted by neurosurgical procedures. However time does not permit any discussion of these problems.

The Stage of Rehabilitation

Rehabilitation is the principal means of reaching the ultimate goal whereby the paraplegic accomplishes reorientation to his disabled state and enters society again as a useful citizen. Since World War II paraplegic centres have been developed to carry out this final phase of treatment.

The five hour daily schedule carried on at the New York University-Bellevue Medical Centre is an example of the well organized rehabilitation program. General conditioning exercises are performed for one hour. Especially important are strengthening exercises for the muscle group of the upper extremities, and such abdominal and trunk muscles as are present. General exercises are followed by an hour workout in the ambulation room. The patient is fitted with braces and taught first to stand and eventually to walk. A 2 hour period per day is spent in "Activities of Daily Living" where the patient is taught to care for his own daily needs such as cleansing and dressing himself. He must learn how to put on and take off his braces; to get up from his bed to a wheel chair and back again; and to rise to a standing position, ambulate and return to his chair. The fifth hour is occupied by vocational testing and guidance, social welfare problems including psychological help if needed.

Conclusion

As the complexity of our civilization increases with further advances in transportation in particular, it is probable that the incidence of spinal cord injury will increase. With proper emergency handling and early laminectomy some patients will escape permanent cord damage. In those suffering irreversible damage, meticulous care of bladder, bowel, skin and nutrition in the early phase of treatment will help to prevent the complications which so often threaten the patient's life. Despite the desperate difficulties which must first seem insurmountable, paraplegics with undying spirit, aided by doctors, nurses and physiotherapists no longer go quickly and miserably to their graves. They make useful service to the community and they make it happily. Surely this is one of the miracles of modern surgery.

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"History maketh a young man to be old without either wrinkles or grey hairs; privileging him with the experience of age without either the infirmities or inconveniences thereof."

Thos. Fuller, History of the Holy War, 1639.

"Sexual" Reproduction in Bacteria

DORIS NICHOLLS, '59

INTRODUCTION

For many years it was thought that bacteria did not have nuclei, or genes, or the ability to reproduce sexually. However, in other ways, they were thought to be similar to other forms of life. Knight (1936) and Lwoff (1938) had proposed ideas that the nutritional requirements of bacteria (and other micro-organisms) were related to an evolutionary loss of synthetic ability. Such losses would thus be based on mutation of genes and subsequent natural selection. These ideas lead Beadle and Tatum (1941), working on the fungus *Neurospora,* to show that the biochemical reactions leading to amino-acid and vitamin synthesis were controlled by genes. The genes could be artificially mutated by agents such as X-rays. Tatum (1944) applied mutation-inducing techniques to bacteria and produced a number of mutants of several species of bacteria. They were mutants with certain growth factor needs, virus resistance, antibiotic resistance, and sugar utilization. This work indicated that most bacterial characteristics were controlled by genes just as in other organisms.

EXPERIMENTAL METHOD

Next, steps were taken to see if the mode of inheritance of bacterial characteristics was similar to the Mendelian process in plants and animals. Tatum and Lederberg (1947) built up stocks of *Escherichia coli* with several nutritional deficiencies by successive mutational treatment. Thus, cultures of one mutant required added threonine, leucine and thiamine for growth but not biotin and methionine; while the second mutant required biotin and methionine but not the other three amino acids. They proposed that if "sexual reproduction occurred in a mixed culture containing both strains, there would be a random recombination or reshuffling of the five growth requirements. The most easily detected recombinant or "daughter cell" would be the one with requirement for none of the 5 amino acids, since it would grow up on a minimal medium with no added amino acids.

RESULTS

Such "daughter" cells were found. In fact, one in every 10,000,000 cells plated from the mixed culture to the minimal medium grew to form a colony. It is important to realize that other explanations of the observations were also suggested. These explanations were not reasonable, on the basis of further experiments which were carried out. The only satisfactory explanation was that there was "sexual" reproduction followed by a reassortment or segregation of genetic material.

CONFIRMATORY EVIDENCE

Further confirmation of this has come from other laboratories where it has been found that direct cell contact of the "parents" is necessary. The number of "progeny" depends on the frequency of contact of "parents". The latter is controlled by the relative and absolute concentration of

the two "parent" types in the mixed culture.

MORPHOLOGY

The morphological details of mating are still obscure although a number of workers have sought evidence in fixed and living preparations. Genetic evidence suggests that there is a mating factor akin to "maleness" and "femaleness".

GENE AND CHROMOSOME NUMBER

It has been found that the genetic particle representing any particular trait is only represented once in the "parent" and "daughter" cells. Thus such bacterial cells have the haploid numbers of chromosomes and genes. Genetic maps of *Escherichia coli* have been constructed. One-half the known genetic markers have been located in a single linear linkage group, that is, on one chromosome. The other genetic markers have a more complex behavior which is believed to be due to complex chromosomal aberrations. It was long thought that *E. coli* had only one chromosome. Now it is believed that there are at least two, the segregation of which is partially dependent.

Certain spontaneously mutated cultures of *E. coli* gave rise to cells with a double number (diploid) of chromosomes. The genetic factors of the two "parents" are found together within this single cell. It has been impossible to count the chromosomes, but the genetic evidence favours this interpretation. Such diploid cells support the idea of "sexual" recombination in *E. coli.*

It is of interest that the diploid cells of *E. coli* show that streptomycin and bacteriophage (that is, virus) sensitivity is a dominant characteristic. The role of mutation in other bacterial species is well known. The existence of recombination in species other than *E. coli* has not been established. In other words, it is not known whether pathological species of bacteria undergo "sexual" recombination. It is possible that recombination may result in highly virulent strains of pathogenic bacteria. Burnet (1951) has suggested such a mechanism to explain the influenza pandemic of 1918.

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"It is the curse of talent, that, although it labors with greater steadiness and perseverence than genius, it does not reach its goal, while genius, already on the summit of the ideal, gazes laughingly about."

Robert Schumann.

Abstracts

TREATMENT OF ACUTE AND CHRONIC LEAD POISONING WITH DISODIUM CALCIUM VERSENATE—

Wade, J. F., Jr., and Burnum, J. F. (1955), Ann. Int. Med. 42: 251.

Until recently, the treatment of lead intoxication has been generally unsatisfactory. However, a relatively new metal chelating agent has recently been found to enhance the urinary excretion of lead; and offers, for the first time, considerable promise in the treatment of lead intoxication. This agent, disodium calcium ethylenediamine tetra-acetate, is also known as Ca E.D.T.A. or disodium calcium versenate. The present report deals with experience in the treatment of five cases of lead poisoning with Ca E.T.D.A. All patients were hospitalized, and the diagnosis of lead intoxication was confirmed by quantitative urinary lead analysis. Daily 24-hour urine collections were assayed for lead before, during, and after treatment. Other appropriate laboratory tests were carried out.

The patients received an average dose of 15 G. of the drug, I.V. in normal saline drip, over a 10-day period of therapy. With this form of treatment, the urinary excretion of lead was increased more than fifteen-fold in all patients. In two cases of acute lead poisoning, all signs and symptoms disappeared dramatically within two hours of injection of the drug. No toxic consequences were observed during or following treatment. It appears, therefore, that Ca E.D.T.A. affords a safe and effective means of treatment of acute and chronic lead poisoning.

—James Goodwin, '57.

INVOLVEMENT OF THE SPINAL CORD IN INTERVERTEBRAL DISC PROTRUSIONS — J. E. A. O'Connell,

(1956), British Journal of Surgery, 43: 225.

Protrusion of an intervertebral disc into the spinal canal is a space-occupying lesion, but differs from a spinal tumor in several ways. It is not neoplastic. It may develop suddenly, and its accommodation is not easily achieved, especially because of its greater rigidity. It, thus, may produce acute distortions in small areas of the cord, or slowly compress it. It may interfere with blood supply, or section nerve fibres to produce permanent loss of function. Volume for volume, disc protrusions at operation appear to have produced a more severe neurological disturbance than benign, extramedullary neoplasms. The differential diagnosis of cervical spondylosis and cervical disc protrusions is emphasized, because they differ in pathology, clinical features, and therapy. The author reviews and analyses fourteen cases of compression of the spinal cord in the cervical, dorsal, and lumbar regions by intervertebral disc protrusions.

—James Goodwin, '57.

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"Thus is Man that great and true *Amphibium,* whose nature is disposed to live, not onely like other creatures in divers elements, but in divided and distinguished worlds: for though there be but one to sense, there are two to reason, the one visible, the other invisible; whereof Moses seems to have left description, and of the other so obscurely, that some parts thereof are yet in controversie."

Sir Thomas Browne, *Religio Medici,* circa 1671.

INTERN PLACEMENTS FOR CLASS OF 1957

OF UNIVERSITY OF WESTERN ONTARIO MEDICAL SCHOOL

NAME	HOSPITAL*
ANDERSON, John	General, Hamilton, Ont.
ARCHDEKIN, Fennel	General, Toronto, Ont.
ARMSTRONG, Ronald	Victoria
BARNES, David	General, Vancouver, B.C.
BERRY, Ronald	St. Joseph's
BURGESS, Glenn	General, Vancouver, B.C.
BURT-GERRANS, Norman	St. Joseph's
BUTLER, William	St. Joseph's
CAMPBELL, Donald	Victoria
CLUNAS, Daniel	General, Hamilton, Ont.
CODE, Thomas	Victoria
COLLYER, James	General, Vancouver, B.C.
COLLYER, John	Victoria
DAVIDSON, Ronald	General, Vancouver, B.C.
DODICK, Joe	Grace, Windsor, Ont.
GOODWIN, James	St. Joseph's
GREEN, Albert	New Mount Sinai, Toronto, Ont.
HACKNEY, Joseph	St. Joseph's
HAMILTON, James	General, Montreal, P.Q.
HOGG, William	Royal Victoria, Montreal
HOWLETT, Lyall	St. Joseph's
HOLLAND, Eleonore	Kitchener-Waterloo, Ont.
INMAN, Fay	General, Montreal, P.Q.
JOHNSTON, Keith	Victoria
JORDAN, Graham	Sisters of Charity, Buffalo, N.Y.
KAWASAKI, Masashi	Victoria
KAYLER, Travis	General, Hamilton, Ont.

*Where city not indicated Hospital is located in London, Ontario.

KILLINGER, Donald ..... Victoria

KNIGHT, Charles ..... Victoria

KRAWETZ, Michael ..... General, Hamilton, Ont.

LAWSON, David ..... Victoria

LOHSTOETER, John ..... St. Joseph's

LUKK, Gertrude ..... New York, N.Y.

MacDONALD, William ..... Victoria

MacLACHLAN, Robert ..... Victoria

MALCOLM, Ian ..... Victoria

MAX, Paul ..... General, Toronto, Ont.

McGEE, Donald ..... Victoria

MILBURN, Lewis ..... St. Joseph's

MILLS, Donald ..... Victoria

ORT, Reginald ..... Victoria

PALMER, Eric ..... St. Joseph's

PATTEN, John ..... St. Joseph's

PLATT, Jonh ..... Victoria

PRIEBE, Victor ..... St. Luke's, Chicago, Ill.

QUINLAN, Vincent ..... Victoria

RAWLING, Edward ..... Victoria

RIX, Donald ..... General, Vancouver, B.C.

ROBINSON, Donald ..... Victoria

SCHISLER, Joseph ..... St. Joseph's

SHONIKER, Thomas ..... St. Michael's, Toronto, On.t

SMITH, Marcia ..... Victoria

SMITH, Newel ..... Victoria

SPEARS, John ..... Victoria

VAN HOOGENHUIZE, William ..... Oakland, Calif.

WALSH, Paul ..... Victoria

WAXMAN, Benny ..... Victoria

ZIELONKO, Walter ..... General, Hamilton, Ont.

Medical News and Views—(Continued)

compulsory treatment. Brazil requires only the collaboration of both patients and medical practitioners by "measures of persuasion". England and Wales make provision only for free treatment and the suppression of charlatism and publicity. Belgium, since 1791, has had regulations for the purpose of suppressing prostitution but a real international agreement for control of venereal disease was the 1924 Brussels agreement, which stipulated provision of facilities to merchant seamen for treatment.

The overall world rate appears to be stationary or even tending to increase. This latter aspect is especially true in areas of war and strife whereas improved living conditions in many countries has produced a noticeable decline. As for uniformity in legislation the main problem has been and probably will continue to be s o c i a l and moral implications. Should a person's rights be exercised to the point of endangering the health of a large group or should an afflicted individual become a public charge for the protection of his fellow man? Great differences of opinion are to be found throughout the w o r l d and only when continuity of thought on the subject is achieved can total eradication be possible.

Medical Education

A medical teacher who took part in the teaching of medical students in all of the four years of a course in an attempt to study the integration of the medical curriculum and the personal teacher-student relationship made some of the following observations.

In the first two years students leaned heavily on him for advice and moral support. In the last two or clinical years he was treated as a colleague with less need for his service as an advisor.

(Continued on Page 123)

Medical News and Views—(Continued)

The experimenter in this survey found that his knowledge was inadequate in many fields resulting in loss of confidence in his abilities. Only students with scholastic difficulties seemed to derive some moral support from this in a "we can't be expected to know what the teacher doesn't" attitude.

The efficiency of students was found to be tied up with emotional aspects and their general outlook. Those repeating subjects realized the difference that a change in attitude could do and as a result only one failure was reported and this due to circumstances revolving around a perfidious female.

The observations on personal relationships were interesting. The instructor and his wife were readily accepted, getting to the point of not being able to keep up with social invitations. The most disruptive incidence seen was the courting of two students in the Anatomy course, resultant marriage during the Physiology course and unfortunate divorce in the Pathology course.

Aging

One view of the biology of aging is that it is fundamentally an endogenous process. Internal and external stresses such as seen in the finding of relatively more cholesterol and calcium in the right side of a right-handed person are thought to be important but not basic.

Life can be defined as an orderly functioning of enzymes and aging as changes or continual adaptivity in an attempt to maintain homeostasis.