Regulation of Diet

The diet will require supervision, especially in those who are well above the normal in weight. Every unnecessary pound in weight adds to the load the heart has to carry.

The first essential is to diminish the intake. As one patient put it, after a heart-to-heart talk, "It seems to me that I must keep my head out of the nosebag." I do not believe it wise to give the patient detailed instructions as to diet. He should grasp the main principle—namely, that the diet must be simple, wholesome, and small in amount. "No second helping" is a good rule, and he will do well to remember that "a good cook may be more dangerous to a man in health than a bad doctor when he is sick." He must keep an eye on the scales. If the weight keeps up, the intake of food must go down.

He will probably find it easier to eat less if he takes his meals with a minimum of fluid. Water can be taken on rising, and again before retiring. The starches and the fats should be eaten sparingly, and it is probably wise to refrain from a diet rich in extractives. Gravies, soups, rich stews, and hashes should be avoided, especially when there is evidence of renal damage. Fresh fruits, salads, lettuce, celery, vegetables, milk, and honey should find an honoured place in the menu; and fish, cold boiled bacon, lean ham, and chicken may be allowed. Butcher's meat should be eaten moderately, and beyond the salt used in cooking none should be taken. Alcohol is better deleted from the diet, and any excess in tea, coffee, and tobacco avoided. The diet should be such that the patient likes what he eats, though it is obvious that he should not be allowed to eat just what he likes.

As someone has aptly said, "In few conditions are both the dish of herbs and contentment more essential, or the stalled ox and strife more injurious."

When possible, an occasional day in bed is helpful. In the more advanced cases, with indications of commencing cardiac failure, it is invaluable. During that day the patient should drink plenty of water, lemonade, orangeade, barley water, raisin tea, or weak tea, and refrain from solid food, with the exception of a few biscuits and honey, or an apple.

The Use of Drugs

Drugs, although of secondary importance, have their honoured place in treatment, more especially in the earlier stages. Later they are useful in treating various distressing symptoms—cardiac, cerebral, or renal. The benefit to be obtained in the earlier phases from bromide has already been mentioned, and it is of great service in menopausal hyperpiesia. Mercury, as a blue pill, or a dose of calomel, followed the next morning by a small but concentrated dose of saline, is undoubtedly of benefit. Such a pill should be taken once a week and the saline every morning—not to procure free purging, but one loose rather watery motion each day. Iodine and the iodides are still popular and widely prescribed, either in very small doses, such as 1/10 grain of the iodide, or in the larger dose of 10 grains. Iodine is acceptable either as collosol iodine or as the tincture—five to seven drops of the latter stirred up in an ounce or two of milk is not unpalatable, the small fraction of potassium iodide in the British tincture being an advantage rather than otherwise. If iodism is produced then the French Codex tincture can be used. The myocardium suffers from the toxæmia accompanying any infective illness, especially influenza, and during convalescence (which should be prolonged) hæmaturinics are essential—simple iron preparations with or without arsenic.

There are three drugs which have come more prominently into notice in the last few years: luminal, acetyl-

cholin (acecoline, pacyl), and the sulphocyanates of potassium and sodium. Their tendency is to lower the blood pressure, and their action is evanescent. They are more likely to be of service in the earlier than in the later stage, before gross pathological changes have taken place. Luminal has, in addition, a helpful sedative effect. The sulphocyanates are prescribed in doses of 1½ to 2 grains three times daily after food, the dose being increased gradually day by day. I question whether the nitrites are of any value, except to bring about an immediate and temporary lowering of the pressure.

It is well to remember that the blood pressure is constantly varying both in health and in disease, and that, within certain limits, the normal has been established. When the figures pass beyond this normal there is a corresponding diminution in the expectation of life. It is therefore a physical sign worth investigating in every patient, especially when there is a known familial tendency to cardio-vascular disease. There is here a wide field for study, more particularly for the general practitioner.

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MESOTHELIAL REACTIONS IN CHILDHOOD*

BY

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I confess I find it a difficult thing to choose an appropriate subject for a lecture such as this. It is a privileged opportunity to address a gathering of men who are engaged in general practice, and I would like to justify that opportunity by presenting to you a subject which will be both interesting and helpful. It is in that very ambition that my difficulty lies, for I would like to leave the beaten tracks of stereotyped subjects and venture into fields that are as yet but little understood and largely unexplored. I know that when we leave the broad highway our footsteps often tread on sinking sands, and the foundation that we fondly thought was firm and sure may crumble into fragments when we rest upon it. None the less there is an interest—an adventure—call it what you will—in venturing into new territory, and in a very small and modest way the subject I propose to present to you may be said to justify the description, so alluring to the mind of the average man, of "terra incognita." If by these introductory remarks I seem to weave a veil of mystery where no secret really lies, it is because I appreciate that what I have to say is based, in part at least, upon hypothesis, supported, it is true, by a certain measure of experimental evidence and a framework of clinical observation. The purpose of my paper is to present to you certain reactions of the mesothelial spaces of the body, and more especially of the great mesothelial space—the peritoneal cavity.

A CASE HISTORY

Some months ago a boy of 6 years was admitted to the Edinburgh Royal Infirmary. The summary of the case history was as follows: Ten days before admission he complained of a sore throat, there was a rise of temperature with general illness, and a diagnosis of acute tonsillitis was made. Four days later, under appropriate treatment, the local infection subsided, and the boy appeared to be convalescent,

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but at the end of a further forty-eight hours there was a recurrence of illness, the temperature rose again, there was abdominal pain and vomiting, and considerable constitutional disturbance. The usual simple remedies failed to produce improvement, and further examination showed that an acute peritoneal infection had developed. When the child was admitted to hospital, ten days after the original onset of symptoms, he was found to be suffering from a generalized peritonitis, and it was our view that the infection had arisen secondary to an inflamed appendix.

Operation revealed an interesting state of affairs. The peritoneal cavity contained a considerable amount of purulent fluid, but we were unable to discover any localized infection from which the peritonitis could have arisen. The appendix was normal, and a careful exploration of other regions yielded negative results. The condition might be described as an idiopathic generalized peritonitis—an unsatisfactory diagnosis, and we preferred to regard the peritoneal infection as secondary to a generalized blood infection originating from the tonsillitis. The organism recovered from the peritoneal fluid was a streptococcus, and, though no blood culture investigation was done preceding the operation, the clinical history suggested that a septicaemia existed. It was therefore our view that the tonsillitis had induced the septicaemia, and that the peritonitis arose as a secondary blood-borne infection. Such was the position, but it is my purpose to elaborate the problem and to present other possibilities for your consideration.

THE STRUCTURE OF THE PERITONEUM

When we examine the structure of the peritoneum we find that it is arranged on an essentially simple plan; a layer of flattened cells rests upon a connective tissue basis which carries the lymphatic and blood vessels (tunica propria). On surface outline the cells appear to be pavement epithelium, but on profile they are seen to be imperfect pyramids with truncated apices and sloping slides. Such being their outline, there are ditches or fossae between the individual cells, and contiguous cells are in contact only at the fringes of their respective edges. There has been much surmise on the *raison d'être* of this peculiarity in structure, and it has been suggested that the relative insecurity of contact between individual cells permits the passage of leucocytes and connective tissue phagocytes from the deeper tissues to the surface, and vice versa. It will be recalled that when the peritoneum is stained by the silver nitrate method a network-like arrangement interspaced with punctate points is evident. It was thought at one time that this appearance was produced by stomata, apertures which established continuity between the peritoneal surface and the underlying lymphatics; in all likelihood deposits of silver stain in the ditches explain the appearance, and it is doubtful if actual stomata exist. I would add that the peritoneal cells, while capable of great reparative power, so that wounds of the surface are rapidly healed, have relatively small phagocytic activity, this function being mainly assumed by phagocytic leucocytes and connective tissue corpuscles.

THE ORIGIN OF THE PERITONEUM

What is the source from which peritoneal tissue has arisen? The peritoneum originates as a split in the mesoblast, and the method of its production is similar to that which occurs in the formation of certain joint spaces and in the pleura. It is therefore obvious that the cells which line the peritoneum are specially adapted mesoblastic cells, and, because they adopt the function of space-lining, they are appropriately termed "mesothelium." The terms "epithelium" and "endothelium" are often applied to the structure, but, as Professor Mall has shown, they are misnomers.

THE FUNCTION OF MESOTHELIAL SPACES

We may now inquire why this special adaptation of the mesoblastic area arises. Why should a mesoblastic split occur, and what is the reason for this peculiar structural

metaplasia of the cells? In other words, what are the functions of mesothelial spaces, and of the peritoneal space in particular?

We may learn something if we consider the question from the morphological standpoint. In the most primitive types of organism the digestive tube courses through a mesoblastic field. It is in the Annelidae that we find the first suggestion of a peritoneal space. It has arisen as a split in the mesoblastic tissues which surround the digestive tube, and it is significant that it contains large numbers of leucocytes suspended in fluid, the function of which is to destroy parasites which have passed through the gut wall. From evidence such as this it is reasonable to argue that one of the reasons for the formation of the peritoneal space is to develop a protective barrier between the dangerous area of the digestive tube and the tissues which lie in relation to it. The earliest and original function is the basic and primitive one of body protection. I agree that it is difficult to ascribe such a function to the mesoblastic spaces of joints, and even of the pleura, but, where the peritoneum is concerned, the argument is appropriate, and, as we shall see, evidences are apparent of the same inherent quality when we consider the reaction of other mesothelial spaces to disease.

The second function of the peritoneal space is to permit freedom of mobility and motility of the viscera which lie within its confines. At first sight this would seem to be the most important and obvious function. It is one which has become elaborated as the complexity of the digestive tube has increased, but it is probably correct to regard it as acquired and secondary rather than primitive and original. Clinical experience tells us that the space capacity of the peritoneal area may be obliterated by a widespread adhesive process while comparative efficiency of the related viscera is retained, a demonstration of the fact that the "space mobility" function is not as essential as might appear. The peritoneal space and tissue exercise a third function, which is physiological. They are concerned in the secretion and absorption of fluid, changes which imply the physical processes of osmosis and dialysis. We know comparatively little regarding the stimuli which induce these changes or the ways in which the reactions are beneficial, but the processes are being constantly exercised, both in health and in disease.

Such are some, probably the most important, of the functions of the peritoneum and of its associated space; it will be noted that they may be grouped as protective, mechanical, and physiological.

THE NATURE OF THE PROTECTIVE FUNCTION

Let us now focus our interest on the protective function. Mesoblastic tissue would appear to be essentially supporting in purpose and design; it forms the bulk of the body tissues, the bone, and the muscles, but where it splits and where certain of its cells become adapted as a lining substance or mesothelium, such portions of the mesoblast appear to exhibit changes in the existence of infection which I believe we shall some day regard as protective in character. Let me recall experimental evidence which has a bearing on this matter. In 1929 Herrmann published in the *Bulletin of the Mayo Clinic* the results of an experimental inquiry on peritonitis. He encountered the difficulty which previous investigators had experienced in inducing an experimental peritonitis similar to the state which we meet with clinically. The intraperitoneal injection of a small quantity of infected material produced no apparent result, owing to the exercise of the phagocytic and antibacterial influences of the peritoneal area, while the injection of a larger quantity resulted in death from septicaemia before local evidences of peritonitis were appreciable. Herrmann attempted to overcome the difficulty by increasing the immunity of the subject prior to

the inoculation. For this purpose he employed a combination of the *Bacillus coli communis* and the *Streptococcus viridans*, and a vaccine prepared from this symbiosis was injected on successive occasions over a period of some weeks. Animals so prepared, when injected intraperitoneally with an amount of infection which under ordinary circumstances would have proved fatal from septicaemia, developed a purulent peritonitis virtually similar to that which we encounter clinically. These experiments, while not fully conclusive, indicate that a peritonitis develops in coexistence with a certain degree of immunity, that in the absence of immunity no local reaction develops, and death ensues from general infection, while in the existence of a fully developed immunity there is no local reaction, because the infection is overcome as soon as it is introduced.

THE MEANING OF IDIOPATHIC PERITONITIS

It is now appropriate to recall the circumstances of the case I outlined at the beginning of this address. If we correlate the case history with the various points I have discussed, we may build up an interesting, though hypothetical, explanation.

Our little patient developed a septicaemia secondary to the acute tonsillitis which introduced his illness. He might have succumbed to such an infection, but fortunately he possessed sufficient resistance to develop a specialized field of immunity. That field was located in the largest and most active mesothelial region of the body—the peritoneum. He developed, in fact, what the older clinicians termed a "fixation abscess," a state which they used to induce by the subcutaneous and intramuscular injection of turpentine, and that which was regarded in the case under discussion as a most regrettable development, a purulent peritonitis, was really an attempt on the part of the body to induce a local field of reaction from which a general immunity would result. I appreciate that the infection was conveyed to the peritoneal area by the blood stream, but so it was to every other portion of the body; the significance lies in the specialized reaction which the mesothelial tissues demonstrate.

If we regard the problem in this way our conception of various infective conditions is modified. There is a polyarthritis associated with a streptococcal septicaemia, sometimes spoken of as Still's disease. In it we are probably witnessing a very similar process to that which we have been discussing—the joint changes are mesoblastic and mesothelial responses to a general blood infection, and in the adult the proliferative type of rheumatoid arthritis may well have a similar explanation. I believe that, as our knowledge develops, we shall increasingly appreciate the significance of the mesothelial response to general infection, but for the moment our interest is centred on the peritoneal mesothelium. From what we have seen, the response of this tissue is chiefly evidenced in childhood, and more particularly in the early years of that period. The protective association is then at its maximum, and, as age proceeds, the response tends to diminish, perhaps to disappear.

THE MESOTHELIAL REACTION IN PNEUMOCOCCAL PERITONITIS

Some years ago I was interested in the question of pneumococcal peritonitis, and the investigation which was carried out at that time in conjunction with Dr. MacCartney seemed to indicate that in female children the peritoneal infection probably arose in consequence of a spread through the genital tract into the pelvic peritoneum. In view of what I have since observed, I am inclined to believe that the majority of pneumococcal peritoneal infections are examples of a mesothelial reaction

to a pneumococcal septicaemia, and that the cases which survive are those which show the degree of peritoneal reaction which results in a purulent peritonitis.

Too often there is an insufficient immunizing response, and all that we discover is the deep blush of a congested peritoneum with but a scanty effusion of turbid fluid, findings which indicate the insufficiency of resistance and the hopelessness of the ultimate prognosis.

PERITONITIS IN RELATION TO NEPHRITIS

Let me recall one further point in relation to the part which the peritoneum plays in the general infections of childhood.

Last summer a boy of 5 years came under my care with the following history. Some weeks previously he developed acute nephritis; the urine contained a large amount of albumin, and there was considerable body oedema, with marked ascites. The general condition improved under medical treatment, but the albuminuria and ascites persisted. On the evening of July 12th acute abdominal pain was complained of, the temperature rose from 99° to 102°, and general abdominal tenderness was elicited on palpation. I saw the boy for the first time the following morning. The clinical features were those of an early generalized peritonitis. Dr. Charles McNeil recalled that Holt had described the development of a streptococcal or a pneumococcal peritonitis in association with nephritis and ascites, and in this case Dr. McNeil had made the diagnosis of a pneumococcal or a streptococcal peritonitis. A laparotomy was performed, and we found the abdominal cavity filled with turbid fluid; there was no localized infection, and from the fluid a pneumococcus was isolated and cultured. The findings were those of a generalized pneumococcal peritonitis. The real interest, however, lies in the sequel. Within five days the albumin vanished from the urine, the tissue fluid disappeared, and an uninterrupted recovery was made; the child to-day enjoys apparently perfect health. It is not unreasonable to recognize in this case an example of an immunizing response of the mesothelial tissues. The kidney error was local evidence of a general infection; the peritonitis was an attempt at an immunizing experiment; and, though we interrupted the development of Nature's intention, sufficient had been achieved to eliminate both the local and the general errors.

THE PROBLEM OF TREATMENT

If we accept the view that an immunizing peritonitis arises in childhood in association with certain types of general bacterial infection, the proper course to adopt in relation to it gives us anxiety. If we intervene too early we are interrupting a process which is protective, and therefore salutary; on the other hand, because the reaction arises in a closed space and in an area from which absorption will ultimately occur, a time comes when intervention in the form of drainage is essential if disaster is to be avoided. I suppose the truth is that, as we are unlikely to recognize an immunizing peritonitis until it is demonstrated by laparotomy, we shall continue to intervene; the time is not yet when we can say, "*Cunctando restituit rem*." I would add that, while there is no doubt a time of election at which operative intervention is most appropriate, less harm arises from early operation than from undue delay.

And now the demon of doubt has entered my mind. Have I erred in drawing attention to this question? Shall I be accused of the heresy of advising conservatism in peritonitis? I hope no such construction can be placed on what I have said, but perhaps I should insist that the condition I am discussing is the generalized peritonitis developing in the absence of any local inflammatory or perforating lesion, a peritonitis which is the result of the response of a certain type of tissue to a general infection, a response which, I believe, we may regard as protective and immunizing in its character, a phenomenon (and this is important) which is exercised in its maximum degree in the years of childhood.