Pounds & Inches

Dr. Simeon's "Pounds and Inches"
Editor's note: Dr. Simeon's protocol was specific for hCG injections.

Pounds & Inches

A NEW APPROACH TO OBESITY
BY: DR. A.T.W. SIMEONS
SALVATOR MUNDI INTERNATIONAL HOSPITAL
00152 – ROME VIALE MURA GIANICOLENSI, 77

FOREWORD
This book discusses a new interpretation of the nature of obesity, and while it does not advocate yet another fancy slimming diet it does describe a method of treatment which has grown out of theoretical considerations based on clinical observation.
What I have to say is an essence of views distilled out of forty years of grappling with the fundamental problems of obesity, its causes, its symptoms, and its very nature. In these many years of specialized work thousands of cases have passed through my hands and were carefully studied. Every new theory, every new method, every promising lead was considered, experimentally screened and critically evaluated as soon as it became known. But invariably the results were disappointing and lacking in uniformity.

I felt that we were merely nibbling at the fringe of a great problem, as, indeed, do most serious students of overweight. We have grown pretty sure that the tendency to accumulate abnormal fat is a very definite metabolic disorder, much as is, for instance, diabetes. Yet the localization and the nature of this disorder remained a mystery. Every new approach seemed to lead into a blind alley, and though patients were told that they are fat because they eat too much, we believed that this is neither the whole truth nor the last word in the matter.
Refusing to be side-tracked by an all too facile interpretation of obesity, I have always held that overeating is the result of the disorder, not its cause, and that we can make little headway until we can build for ourselves some sort of theoretical structure with which to explain the condition. Whether such a structure represents the truth is not important at this moment. What it must do is to give us an intellectually satisfying interpretation of what is happening in the obese body. It must also be able to withstand the onslaught of all hitherto known clinical facts and furnish a hard background against which the results of treatment can be accurately assessed.

To me this requirement seems basic, and it has always been the center of my interest. In dealing with obese patients it became a habit to register and order every clinical experience as if it were an odd looking piece of a jig-saw puzzle. And then, as in a jig saw puzzle, little clusters of fragments began to form, though they seemed to fit in nowhere. As the years passed these clusters grew bigger and started to amalgamate until, about sixteen years ago, a complete picture became dimly discernible. This picture was, and still is, dotted with gaps for which I cannot find the pieces, but I do now feel that a theoretical structure is visible as a whole.
With mounting experience, more and more facts seemed to fit snugly into the new framework, and when then a treatment based on such speculations showed consistently satisfactory results, I was sure that some practical advance had been made, regardless of whether the theoretical interpretation of these results is correct or not.

The clinical results of the new treatment have been published in scientific journal[1] and these reports have been generally well received by the profession, but the very nature of a scientific article does not permit the full presentation of new theoretical concepts nor is there room to discuss the finer points of technique and the reasons for observing them.
During the 16 years that have elapsed since I first published my findings, I have had many hundreds of inquiries from research institutes, doctors and patients. Hitherto I could only refer those interested to my scientific papers, though I realized that these did not contain sufficient information to enable doctors to conduct the new treatment satisfactorily. Those who tried were obliged to gain their own experience through the many trials and errors which I have long since overcome. 2

Doctors from all over the world have come to Italy to study the method, first hand in my clinic in the Salvator Mundi International Hospital in Rome. For some of them the time they could spare has been too short to get a full grasp of the technique, and in any case the number of those whom I have been able to meet personally is small compared with the many requests for further detailed information which keep coming in. I have tried to keep up with these demands by correspondence, but the volume of this work has become unmanageable and that is one excuse for writing this book.

In dealing with a disorder in which the patient must take an active part in the treatment, it is, I believe, essential that he or she have an understanding of what is being done and why. Only then can there be intelligent cooperation between physician and patient. In order to avoid writing two books, one for the physician and another for the patient – a prospect which would probably have resulted in no book at all – I have tried to meet the requirements of both in a single book. This is a rather difficult enterprise in which I may not have succeeded. The expert will grumble about long-windedness while the lay-reader may occasionally have to look up an unfamiliar word in the glossary provided for him.

To make the text more readable I shall be unashamedly authoritative and avoid all the hedging and tentativeness with which it is customary to express new scientific concepts grown out of clinical experience and not as yet confirmed by clear-cut laboratory experiments. Thus, when I make what reads like a factual statement, the professional reader may have to translate into: clinical experience seems to suggest that such and such an observation might be tentatively explained by such and such a working hypothesis, requiring a vast amount of further research before the hypothesis can be considered a valid theory. If we can from the outset establish this as a mutually accepted convention, I hope to avoid being accused of speculative exuberance.

THE NATURE OF OBESITY

Obesity a Disorder

As a basis for our discussion we postulate that obesity in all its many forms is due to an abnormal functioning of some part of the body and that every ounce of abnormally accumulated fat is always the result of the same disorder of certain regulatory mechanisms. Persons suffering from this particular disorder will get fat regardless of whether they eat excessively, normally or less than normal. A person who is free of the disorder will never get fat, even if he frequently overeats.

Those in whom the disorder is severe will accumulate fat very rapidly, those in whom it is moderate will gradually increase in weight and those in whom it is mild may be able to keep their excess weight stationary for long periods. In all these cases a loss of weight brought about by dieting, treatments with thyroid, appetite-reducing drugs, laxatives, violent exercise, massage, baths, etc., is only temporary and will be rapidly regained as soon as the reducing regimen is relaxed. The reason is simply that none of these measures corrects the basic disorder.

While there are great variations in the severity of obesity, we shall consider all the different forms in both sexes and at all ages as always being due to the same disorder. Variations in form would then be partly a matter of degree, partly an inherited bodily constitution and partly the result of a secondary involvement of endocrine glands such as the pituitary, the thyroid, the adrenals or the sex glands. On the other hand, we postulate that no deficiency of any of these glands can ever directly produce the common disorder known as obesity.

If this reasoning is correct, it follows that a treatment aimed at curing the disorder must be equally effective in both sexes, at all ages and in all forms of obesity. Unless this is so, we are entitled to harbor grave doubts as to whether a given treatment corrects the underlying disorder. Moreover, any claim that the disorder has been corrected must be substantiated by the ability of the patient to eat normally of any food he pleases without regaining abnormal fat after treatment. Only if these conditions are fulfilled can we legitimately speak of curing obesity rather than of reducing weight.

Our problem thus presents itself as an enquiry into the localization and the nature of the disorder which leads to obesity. The history of this enquiry is a long series of high hopes and bitter disappointments.

The History of Obesity

There was a time, not so long ago, when obesity was considered a sign of health and prosperity in man and of beauty, amorousness and fecundity in women. This attitude probably dates back to Neolithic times, about 8000 years ago; when for the first time in the history of culture, man began to own property, domestic animals, arable land, houses, pottery and metal tools. Before that, with the possible exception of some races such as the Hottentots, obesity was almost non-existent, as it still is in all wild animals and most primitive races.

Today obesity is extremely common among all civilized races, because a disposition to the disorder can be inherited. Wherever abnormal fat was regarded as an asset, sexual selection tended to propagate the trait. It is only in very recent times that manifest obesity has lost some of its allure, though the cult of the outsize bust – always a sign of latent obesity – shows that the trend still lingers on.

The Significance of Regular Meals

In the early Neolithic times another change took place which may well account for the fact that today nearly all inherited dispositions sooner or later develop into manifest obesity. This change was the institution of regular meals. In pre-Neolithic times, man ate only when he was hungry and on1y as much as he required to still the pangs of hunger. Moreover, much of his food was raw and all of it was unrefined. He roasted his meat, but he did not boil it, as he had no pots, and what little he may have grubbed from the Earth and picked from the trees, he ate as he went along.

The whole structure of man's omnivorous digestive tract is, like that of an ape, rat or pig, adjusted to the continual nibbling of tidbits. It is not suited to occasional gorging as is, for instance, the intestine of the carnivorous cat family. Thus the institution of regular meals, particularly of food rendered rapidly assimilable, placed a great burden on modern man's ability to cope with large quantities of food suddenly pouring into his system from the intestinal tract. 3
The institution of regular meals meant that man had to eat more than his body required at the moment of eating so as to tide him over until the next meal. Food rendered easily digestible suddenly flooded his body with nourishment of which he was in no need at the moment. Somehow, somewhere this surplus had to be stored.

Three Kinds of Fat
In the human body we can distinguish three kinds of fat. The first is the structural fat which fills the gaps between various organs, a sort of packing material. Structural fat also performs such important functions as bedding the kidneys in soft elastic tissue, protecting the coronary arteries and keeping the skin smooth and taut. It also provides the springy cushion of hard fat under the bones of the feet, without which we would be unable to walk.

The second type of fat is a normal reserve of fuel upon which the body can freely draw when the nutritional income from the intestinal tract is insufficient to meet the demand. Such normal reserves are localized all over the body. Fat is a substance which packs the highest caloric value into the smallest space so that normal reserves of fuel for muscular activity and the maintenance of body temperature can be most economically stored in this form. Both these types of fat, structural and reserve, are normal, and even if the body stocks them to capacity this can never be called obesity.
But there is a third type of fat which is entirely abnormal. It is the accumulation of such fat, and of such fat only, from which the overweight patient suffers. This abnormal fat is also a potential reserve of fuel, but unlike the normal reserves it is not available to the body in a nutritional emergency. It is, so to speak, locked away in a fixed deposit and is not kept in a current account[2], as are the normal reserves.

When an obese patient tries to reduce by starving himself, he will first lose his normal fat reserves. When these are exhausted he begins to burn up structural fat, and only as a last resort will the body yield its abnormal reserves, though by that time the patient usually feels so weak and hungry that the diet is abandoned. It is just for this reason that obese patients complain that when they diet they lose the wrong fat. They feel famished and tired and their face becomes drawn and haggard, but their belly, hips, thighs and upper arms show little improvement. The fat they have come to detest stays on and the fat they need to cover their bones gets less and less. Their skin wrinkles and they look old and miserable. And that is one of the most frustrating and depressing experiences a human being can have.

Injustice to the Obese
When then obese patients are accused of cheating, gluttony, lack of will power, greed and sexual complexes, the strong become indignant and decide that modern medicine is a fraud and its representatives fools, while the weak just give up the struggle in despair. In either case the result is the same: a further gain in weight, resignation to an abominable fate and the resolution at least to live tolerably the short span allotted to them – a fig for doctors and insurance companies.
Obese patients only feel physically well as long as they are stationary or gaining weight. They may feel guilty, owing to the lethargy and indolence always associated with obesity. They may feel ashamed of what they have been led to believe is a lack of control. They may feel horrified by the appearance of their nude body and the tightness of their clothes. But they have a primitive feeling of animal content which turns to misery and suffering as soon as they make a resolute attempt to reduce. For this there are sound reasons.

In the first place, more caloric energy is required to keep a large body at a certain temperature than to heat a small body. Secondly the muscular effort of moving a heavy body is greater than in the case of a light body. The muscular effort consumes Calories which must be provided by food. Thus, all other factors being equal, a fat person requires more food than a lean one. One might therefore reason that if a fat person eats only the additional food his body requires he should be able to keep his weight stationary. Yet every physician who has studied obese patients under rigorously controlled conditions knows that this is not true.
Many obese patients actually gain weight on a diet which is calorically deficient for their basic needs. There must thus be some other mechanism at work.

Glandular Theories
At one time it was thought that this mechanism might be concerned with the sex glands. Such a connection was suggested by the fact that many juvenile obese patients show an under-development of the sex organs. The middle-age spread in men and the tendency of many women to put on weight in the menopause seemed to indicate a causal connection between diminishing sex function and overweight. Yet,

when highly active sex hormones became available, it was found that their administration had no effect whatsoever on obesity. The sex glands could therefore not be the seat of the disorder.

The Thyroid Gland

When it was discovered that the thyroid gland controls the rate at which body-fuel is consumed, it was thought that by administering thyroid gland to obese patients their abnormal fat deposits could be burned up more rapidly. This too proved to be entirely disappointing, because as we now know, these abnormal deposits take no part in the body's energy-turnover – they are inaccessibly locked away. Thyroid medication merely forces the body to consume its normal fat reserves, which are already depleted in obese patients, and then to break down structurally essential fat without touching the abnormal deposits. In this way a patient may be brought to the brink of starvation in spite of having a hundred pounds of fat to spare. Thus any weight loss brought about by thyroid medication is always at the expense of fat of which the body is in dire need.

While the majority of obese patients have a perfectly normal thyroid gland and some even have an overactive thyroid, one also occasionally sees a case with a real thyroid deficiency. In such cases, treatment with thyroid brings about a small loss of weight, but this is not due to the loss of any abnormal fat. It is entirely the result of the elimination of a mucoid substance, called myxedema, which the body accumulates when there is a marked primary thyroid deficiency. Moreover, patients suffering only from a severe lack of thyroid hormone never become obese in the true sense. Possibly also the observation that normal persons – though not the obese – lose weight rapidly when their thyroid becomes overactive may have contributed to the false notion that thyroid deficiency and obesity are connected. Much misunderstanding about the supposed role of the thyroid gland in obesity is still met with, and it is now really high time that thyroid preparations be once and for all struck off the list of remedies for obesity. This is particularly so because giving thyroid gland to an obese patient whose thyroid is either normal or overactive, besides being useless, is decidedly dangerous. 4

The Pituitary Gland

The next gland to be falsely incriminated was the anterior lobe of the pituitary, or hypophysis. This most important gland lies well protected in a bony capsule at the base of the skull. It has a vast number of functions in the body, among which is the regulation of all the other important endocrine glands. The fact that various signs of anterior pituitary deficiency are often associated with obesity raised the hope that the seat of the disorder might be in this gland. But although a large number of pituitary hormones have been isolated and many extracts of the gland prepared, not a single one or any combination of such factors proved to be of any value in the treatment of obesity. Quite recently, however, a fat-mobilizing factor has been found in pituitary glands, but it is still too early to say whether this factor is destined to play a role in the treatment of obesity.

The Adrenals

Recently, a long series of brilliant discoveries concerning the working of the adrenal or suprarenal glands, small bodies which sit atop the kidneys, have created tremendous interest. This interest also turned to the problem of obesity when it was discovered that a condition which in some respects resembles a severe case of obesity – the so called Cushing's Syndrome – was caused by a glandular new-growth of the adrenals or by their excessive stimulation with ACTH, which is the pituitary hormone governing the activity of the outer rind or cortex of the adrenals.

When we learned that an abnormal stimulation of the adrenal cortex could produce signs that resemble true obesity, this knowledge furnished no practical means of treating obesity by decreasing the activity of the adrenal cortex. There is no evidence to suggest that in obesity there is any excess of adrenocortical activity; in fact, all the evidence points to the contrary. There seems to be rather a lack of adrenocortical function and a decrease in the secretion of ACTH from the anterior pituitary lobe.[3]
So here again our search for the mechanism which produces obesity led us into a blind alley. Recently, many students of obesity have reverted to the nihilistic attitude that obesity is caused simply by overeating and that it can only be cured by under eating.

The Diencephalon or Hypothalamus

For those of us who refused to be discouraged there remained one slight hope. Buried deep down in the massive human brain there is a part which we have in common with all vertebrate animals the so-called diencephalon. It is a very primitive part of the brain and has in man been almost smothered by the huge masses of nervous tissue with which we think, reason and voluntarily move our body. The diencephalon is the part from which the central nervous system controls all the automatic animal functions of the body, such as breathing, the heart beat, digestion, sleep, sex, the urinary system, the autonomous or vegetative nervous system and via the pituitary the whole interplay of the endocrine glands.

It was therefore not unreasonable to suppose that the complex operation of storing and issuing fuel to the body might also be controlled by the diencephalon. It has long been known that the content of sugar –

another form of fuel – in the blood depends on a certain nervous center in the diencephalon. When this center is destroyed in laboratory animals, they develop a condition rather similar to human stable diabetes. It has also long been known that the destruction of another diencephalic center produces a voracious appetite and a rapid gain in weight in animals which never get fat spontaneously.

The Fat-bank

Assuming that in man such a center controlling the movement of fat does exist, its function would have to be much like that of a bank. When the body assimilates from the intestinal tract more fuel than it needs at the moment, this surplus is deposited in what may be compared with a current account. Out of this account it can always be withdrawn as required. All normal fat reserves are in such a current account, and it is probable that a diencephalic center manages the deposits and withdrawals.

When now, for reasons which will be discussed later, the deposits grow rapidly while small withdrawals become more frequent, a point may be reached which goes beyond the diencephalon's banking capacity. Just as a banker might suggest to a wealthy client that instead of accumulating a large and unmanageable current account he should invest his surplus capital, the body appears to establish a fixed deposit into which all surplus funds go but from which they can no longer be withdrawn by the procedure used in a current account. In this way the diencephalic —fat-bank‖ frees itself from all work which goes beyond its normal banking capacity. The onset of obesity dates from the moment the diencephalon adopts this labor-saving ruse. Once a fixed deposit has been established the normal fat reserves are held at a minimum, while every available surplus is locked away in the fixed deposit and is therefore taken out of normal circulation.

THREE BASIC CAUSES OF OBESITY

(1) The Inherited Factor

Assuming that there is a limit to the diencephalon's fat banking capacity, it follows that there are three basic ways in which obesity can become manifest. The first is that the fat-banking capacity is abnormally low from birth. Such a congenitally low diencephalic capacity would then represent the inherited factor in obesity. When this abnormal trait is markedly present, obesity will develop at an early age in spite of normal feeding; this could explain why among brothers and sisters eating the same food at the same table some become obese and others do not.

(2) Other Diencephalic Disorders

The second way in which obesity can become established is the lowering of a previously normal fat-banking capacity owing to some other diencephalic disorder. It seems to be a general rule that when one of the many diencephalic centers is particularly overtaxed; it tries to increase its capacity at the expense of other centers. 5

In the menopause and after castration the hormones previously produced in the sex-glands no longer circulate in the body. In the presence of normally functioning sex-glands their hormones act as a brake on the secretion of the sex-gland stimulating hormones of the anterior pituitary. When this brake is removed the anterior pituitary enormously increases its output of these sex-gland stimulating hormones, though they are now no longer effective. In the absence of any response from the non-functioning or missing sex glands, there is nothing to stop the anterior pituitary from producing more and more of these hormones. This situation causes an excessive strain on the diencephalic center which controls the function of the anterior pituitary. In order to cope with this additional burden the center appears to draw more and more energy away from other centers, such as those concerned with emotional stability, the blood circulation (hot flushes) and other autonomous nervous regulations, particularly also from the not so vitally important fat-bank.

The so-called stable type of diabetes heavily involves the diencephalic blood sugar regulating center. The diencephalon tries to meet this abnormal load by switching energy destined for the fat bank over to the sugar-regulating center, with the result that the fat-banking capacity is reduced to the point at which it is forced to establish a fixed deposit and thus initiate the disorder we call obesity. In this case one would have to consider the diabetes the primary cause of the obesity, but it is also possible that the process is reversed in the sense that a deficient or overworked fat-center draws energy from the sugar-center, in which case the obesity would be the cause of that type of diabetes in which the pancreas is not primarily involved. Finally, it is conceivable that in Cushing's syndrome those symptoms which resemble obesity are entirely due to the withdrawal of energy from the diencephalic fat-bank in order to make it available to the highly disturbed center which governs the anterior pituitary adrenocortical system.

Whether obesity is caused by a marked inherited deficiency of the fat-center or by some entirely different diencephalic regulatory disorder, its insurgence obviously has nothing to do with overeating and in either case obesity is certain to develop regardless of dietary restrictions. In these cases any enforced food deficit is made up from essential fat reserves and normal structural fat, much to the disadvantage of the patient's general health.

3) The Exhaustion of the Fat-bank
But there is still a third way in which obesity can become established, and that is when a presumably normal fat-center is suddenly — the emphasis is on suddenly — called upon to deal with an enormous influx of food far in excess of momentary requirements. At first glance it does seem that here we have a straight-forward case of overeating being responsible for obesity, but on further analysis it soon becomes clear that the relation of cause and effect is not so simple. In the first place we are merely assuming that the capacity of the fat center is normal while it is possible and even probable that only persons who have some inherited trait in this direction can become obese merely by overeating.

Secondly, in many of these cases the amount of food eaten remains the same and it is only the consumption of fuel which is
suddenly decreased, as when an athlete is confined to bed for many weeks with a broken bone or when a man leading a highly active life is suddenly tied to his desk in an office and to television at home. Similarly, when a person, grown up in a cold climate, is transferred to a tropical country and continues to eat as before, he may develop obesity because in the heat far less fuel is required to maintain the normal body temperature.
When a person suffers a long period of privation, be it due to chronic illness, poverty, famine or the exigencies of war, his diencephalic regulations adjust themselves to some extent to the low food intake. When then suddenly these conditions change and he is free to eat all the food he wants, this is liable to overwhelm his fat-regulating center. During the last war[4] about 6000 grossly underfed Polish refugees who had spent harrowing years in Russia were transferred to a camp in India where they were well housed, given normal British army rations and some cash to buy a few extras. Within about three months, 85% were suffering from obesity.

In a person eating coarse and unrefined food, the digestion is slow and only a little nourishment at a time is assimilated from the intestinal tract. When such a person is suddenly able to obtain highly refined foods such as sugar, white flour, butter and oil these are so rapidly digested and assimilated that the rush of incoming fuel which occurs at every meal may eventually overpower the diecenphalic regulatory mechanisms and thus lead to obesity. This is commonly seen in the poor man who suddenly becomes rich enough to buy the more expensive refined foods, though his total caloric intake remains the same or is even less than before.

Psychological Aspects
Much has been written about the psychological aspects of obesity. Among its many functions the diencephalon is also the seat of our primitive animal instincts, and just as in an emergency it can switch energy from one center to another, so it seems to be able to transfer pressure from one instinct to another. Thus, a lonely and unhappy person deprived of all emotional comfort and of all instinct gratification except the stilling of hunger and thirst can use these as outlets for pent up instinct pressure and so develop obesity. Yet once that has happened, no amount of psychotherapy or analysis, happiness, company or the gratification of other instincts will correct the condition.

Compulsive Eating
No end of injustice is done to obese patients by accusing them of compulsive eating, which is a form of diverted sex gratification. Most obese patients do not suffer from compulsive eating; they suffer genuine hunger – real, gnawing, torturing hunger – which has nothing whatever to do with compulsive eating. Even their sudden desire for sweets is merely the result of the experience that sweets, pastries and alcohol will most rapidly of all foods allay the pangs of hunger. This has nothing to do with diverted instincts.
On the other hand, compulsive eating does occur in some obese patients, particularly in girls in their late teens or early twenties. Compulsive eating differs fundamentally from the obese patient's greater need for food. It comes on in attacks and is never associated with real hunger, a fact which is readily admitted by the patients. They only feel a feral desire to stuff. Two pounds of chocolates may be devoured in a few minutes; cold, greasy food from the refrigerator, stale bread, leftovers on stacked plates, almost anything edible is crammed down with terrifying speed and ferocity.
I have occasionally been able to watch such an attack without the patient's knowledge, and it is a frightening, ugly spectacle to behold, even if one does realize that mechanisms entirely beyond the patient's control are at work. A careful enquiry into what may have brought on such an attack almost invariably reveals that it is preceded by a strong unresolved sex-stimulation, the higher centers of the brain having blocked primitive diencephalic instinct gratification. The pressure is then let off through another primitive channel, which is oral gratification. In my 6 experience the only thing that will cure this condition is uninhibited sex, a therapeutic procedure which is hardly ever feasible, for if it were, the patient would have adopted it without professional prompting, nor would this in any way correct the associated obesity. It would only raise new and often greater problems if used as a therapeutic measure. Patients suffering from real compulsive eating are comparatively rare. In my practice they constitute about 1-2%. Treating them for obesity is a heartrending job. They do perfectly well between attacks, but a single bout occurring while under treatment may annul several weeks of therapy. Little wonder that such

patients become discouraged. In these cases I have found that psychotherapy may make the patient fully understand the mechanism, but it does nothing to stop it. Perhaps society's growing sexual permissiveness will make compulsive eating even rarer.
Whether a patient is really suffering from compulsive eating or not is hard to decide before treatment because many obese patients think that their desire for food — to them unmotivated — is due to compulsive eating, while all the time it is merely a greater need for food. The only way to find out is to treat such patients. Those that suffer from real compulsive eating continue to have such attacks, while those who are not compulsive eaters never get an attack during treatment.

Reluctance to Lose Weight
Some patients are deeply attached to their fat and cannot bear the thought of losing it. If they are intelligent, popular and successful in spite of their handicap, this is a source of pride. Some fat girls look upon their condition as a safeguard against erotic involvements, of which they are afraid. They work out a pattern of life in which their obesity plays a determining role and then become reluctant to upset this pattern and face a new kind of life which will be entirely different after their figure has become normal and often very attractive. They fear that people will like them – or be jealous – on account of their figure rather than be attracted by their intelligence or character only. Some have a feeling that reducing means giving up an almost cherished and intimate part of themselves. In many of these cases psychotherapy can be helpful, as it enables these patients to see the whole situation in the full light of consciousness. An affectionate attachment to abnormal fat is usually seen in patients who became obese in childhood, but this is not necessarily so.

In all other cases the best psychotherapy can do in the usual treatment of obesity is to render the burden of hunger and never-ending dietary restrictions slightly more tolerable. Patients who
have successfully established an erotic transfer to their psychiatrist are often better able to bear their suffering as a secret labor of love.

There are thus a large number of ways in which obesity can be initiated, though the disorder itself is always due to the same mechanism, an inadequacy of the diencephalic fat-center and the laying down of abnormally fixed fat deposits in abnormal places. This means that once obesity has become established, it can no more be cured by eliminating those factors which brought it on than a fire can be extinguished by removing the cause of the conflagration. Thus a discussion of the various ways in which obesity can become established is useful from a preventative point of view, but it has no bearing on the treatment of the established condition. The elimination of factors which are clearly hastening the course of the disorder may slow down its progress or even halt it, but they can never correct it.

Not by Weight alone…
Weight alone is not a satisfactory criterion by which to judge whether a person is suffering from the disorder we call obesity or not. Every physician is familiar with the sylphlike lady who enters the consulting room and declares emphatically that she is getting horribly fat and wishes to reduce. Many an honest and sympathetic physician at once concludes that he is dealing with a —nut.‖ If he is busy he will give her short shrift, but if he has time he will weigh her and show her tables to prove that she is actually underweight.

I have never yet seen or heard of such a lady being convinced by either procedure. The reason is that in my experience the lady is nearly always right and the doctor wrong. When such a patient is carefully examined one finds many signs of potential obesity, which is just about to become manifest as overweight. The patient distinctly feels that something is wrong with her, that a subtle change is taking place in her body, and this alarms her.
There are a number of signs and symptoms which are characteristic of obesity. In manifest obesity many and often all these signs and symptoms are present. In latent or just beginning cases some are always found, and it should be a rule that if two or more of the bodily signs are present, the case must be regarded as one that needs immediate help.

Signs and symptoms of obesity
The bodily signs may be divided into such as have developed before puberty, indicating a strong inherited factor, and those which develop at the onset of manifest disorder. Early signs are a disproportionately large size of the two upper front teeth, the first incisor, or a dimple on both sides of the sacral bone just above the buttocks. When the arms are outstretched with the palms upward, the forearms appear sharply angled outward from the upper arms. The same applies to the lower extremities. The patient cannot bring his feet together without the knees overlapping; he is, in fact, knock-kneed.

The beginning accumulation of abnormal fat shows as a little pad just below the nape of the neck, colloquially known as the Duchess' Hump. There is a triangular fatty bulge in front of the armpit when the arm is held against the body. When the skin is stretched by fat rapidly accumulating under it, it may split in the lower layers. When large and fresh, such tears are purple, but later they are transformed into white

scar-tissue. Such striation, as it is called, commonly occurs on the abdomen of women during pregnancy, but in obesity it is frequently found on the breasts, the hips and occasionally on the shoulders. In many cases striation is so fine that the small white lines are only just visible. They are always a sure sign of obesity, and though this may be slight at the time of examination such patients can usually remember a period in their childhood when they were excessively chubby.
Another typical sign is a pad of fat on the insides of the knees, a spot where normal fat reserves are never stored. There may be a fold of skin over the pubic area and another fold may stretch round both sides of the chest, where a loose roll of fat can be picked up between two fingers. In the male an excessive accumulation of fat in the breasts is always indicative, while in the female the breast is usually, but not necessarily, large. Obviously excessive fat on the abdomen, the hips, thighs, upper arms, chin and shoulders are characteristic, and it is important to remember that any number of these signs may be present in persons whose weight is statistically normal; particularly if they are dieting on their own with iron determination. 7 Common clinical symptoms which are indicative only in their association and in the frame of the whole clinical picture are: frequent headaches, rheumatic pains without detectable bony abnormality; a feeling of laziness and lethargy, often both physical and mental and frequently associated with insomnia, the patients saying that all they want is to rest; the frightening feeling of being famished and sometimes weak with hunger two to three hours after a hearty meal and an irresistible yearning for sweets and starchy food which often overcomes the patient quite suddenly and is sometimes substituted by a desire for alcohol; constipation and a spastic or irritable colon are unusually common among the obese, and so are menstrual disorders.
Returning once more to our sylphlike lady, we can say that a combination of some of these symptoms with a few of the typical bodily signs is sufficient evidence to take her case seriously. A human figure, male or female, can only be judged in the nude; any opinion based on the dressed appearance can be quite fantastically wide off the mark, and I feel myself driven to the conclusion that apart from frankly psychotic patients such as cases of anorexia nervosa; a morbid weight fixation does not exist. I have yet to see a patient who continues to complain after the figure has been rendered normal by adequate treatment.

The Emaciated Lady

I remember the case of a lady who was escorted into my consulting room while I was telephoning. She sat down in front of my desk, and when I looked up to greet her I saw the typical picture of advanced emaciation. Her dry skin hung loosely over the bones of her face, her neck was scrawny and collarbones and ribs stuck out from deep hollows. I immediately thought of cancer and decided to which of my colleagues at the hospital I would refer her. Indeed, I felt a little annoyed that my assistant had not explained to her that her case did not fall under my specialty. In answer to my query as to what I could do for her, she replied that she wanted to reduce. I tried to hide my surprise, but she must have noted a fleeting expression, for she smiled and said —I know that you think I'm mad, but just wait.‖ With that she rose and came round to my side of the desk. Jutting out from a tiny waist she had enormous hips and thighs.
By using a technique which will presently be described, the abnormal fat on her hips was transferred to the rest of her body which had been emaciated by months of very severe dieting. At the end of a treatment lasting five weeks, she, a small woman, had lost 8 inches round her hips, while her face looked fresh and florid, the ribs were no longer visible and her weight was the same to the ounce as it had been at the first consultation.

Fat but not Obese

While a person who is statistically underweight may still be suffering from the disorder which causes obesity, it is also possible for a person to be statistically overweight without suffering from obesity. For such persons weight is no problem, as they can gain or lose at will and experience no difficulty in reducing their caloric intake. They are masters of their weight, which the obese are not. Moreover, their excess fat shows no preference for certain typical regions of the body, as does the fat in all cases of obesity. Thus, the decision whether a borderline case is really suffering from obesity or not cannot be made merely by consulting weight tables.

THE TREATMENT OF OBESITY

If obesity is always due to one very specific diencephalic deficiency, it follows that the only way to cure it is to correct this deficiency. At first this seemed an utterly hopeless undertaking. The greatest obstacle was that one could hardly hope to correct an inherited trait localized deep inside the brain, and while we did possess a number of drugs whose point of action was believed to be in the diencephalon, none of them had the slightest effect on the fat-center. There was not even a pointer showing a direction in which pharmacological research could move to find a drug that had such a specific action. The closest approach were the appetite-reducing drugs – the amphetamines—– but these cured nothing.

Mulling over this depressing situation, I remembered a rather curious observation made many years ago in India. At that time we knew very little about the function of the diencephalon, and my interest centered round the pituitary gland. Froehlich had described cases of extreme obesity and sexual underdevelopment

in youths suffering from a new growth of the anterior pituitary lobe, producing what then became known as Froehlich's disease. However, it was very soon discovered that the identical syndrome, though running a less fulminating course, was quite common in patients whose pituitary gland was perfectly normal. These are the so-called —fat boys‖ with long, slender hands, breasts any flat-chested maiden would be proud to posses, large hips, buttocks and thighs with striation, knock-knees and underdeveloped genitals, often with undescended testicles.
It also became known that in these cases the sex organs could he developed by giving the patients injections of a substance extracted from the urine of pregnant women, it having been shown that when this substance was injected into sexually immature rats it made them precociously mature. The amount of substance which produced this effect in one rat was called one International Unit, and the purified extract was accordingly called —Human Chorionic Gonadotrophin‖ whereby chorionic signifies that it is produced in the placenta and gonadotropin that its action is sex gland directed.

The usual way of treating —fat boys‖ with underdeveloped genitals is to inject several hundred International Units twice a week. Human Chorionic Gonadotrophin which we shall henceforth simply call HCG is expensive and as —fat boys‖ are fairly common among Indians I tried to establish the smallest effective dose. In the course of this study three interesting things emerged. The first was that when fresh pregnancy-urine from the female ward was given in quantities of about 300 cc. by retention enema, as good results could be obtained as by injecting the pure substance. The second was that small daily doses appeared to be just as effective as much larger ones given twice a week. Thirdly, and that is the observation that concerns us here, when such patients were given small daily doses they seemed to lose their ravenous appetite though they neither gained nor lost weight. Strangely enough however, their shape did change. Though they were not restricted in diet, there was a distinct decrease in the circumference of their hips.

Fat on the Move
Remembering this, it occurred to me that the change in shape could only be explained by a movement of fat away from abnormal deposits on the hips, and if that were so there was just a chance that while such fat was in transition it might be available to the body as fuel. This was easy to find out, as in that case, fat on the move would be able to replace food. It should then he possible to keep a —fat boy‖ on a severely 8 restricted diet without a feeling of hunger, in spite of a rapid loss of weight. When I tried this in typical cases of Froehlich's syndrome, I found that as long as such patients were given small daily doses of HCG they could comfortably go about their usual occupations on a diet of only 500 Calories daily and lose an average of about one pound per day. It was also perfectly evident that only abnormal fat was being consumed, as there were no signs of any depletion of normal fat. Their skin remained fresh and turgid, and gradually their figures became entirely normal, nor did the daily administration of HCG appear to have any side-effects other than beneficial.
From this point it was a small step to try the same method in all other forms of obesity. It took a few hundred cases to establish beyond reasonable doubt that the mechanism operates in exactly the same way and seemingly without exception in every case of obesity. I found that, though most patients were treated in the outpatients department, gross dietary errors rarely occurred. On the contrary, most patients complained that the two meals of 250 Calories each were more than they could manage, as they continually had a feeling of just having had a large meal.

Pregnancy and Obesity
Once this trail was opened, further observations seemed to fall into line. It is, for instance, well known that during pregnancy an obese woman can very easily lose weight. She can drastically reduce her diet without feeling hunger or discomfort and lose weight without in any way harming the child in her womb. It is also surprising to what extent a woman can suffer from pregnancy-vomiting without coming to any real harm.
Pregnancy is an obese woman's one great chance to reduce her excess weight. That she so rarely makes use of this opportunity is due to the erroneous notion, usually fostered by her elder relations, that she now has —two mouths to feed‖ and must —keep up her strength for the coming event. All modern obstetricians know that this is nonsense and that the more superfluous fat is lost the less difficult will be the confinement, though some still hesitate to prescribe a diet sufficiently low in Calories to bring about a drastic reduction.

A woman may gain weight during pregnancy, but she never becomes obese in the strict sense of the word. Under the influence of the HCG which circulates in enormous quantities in her body during pregnancy, her diencephalic banking capacity seems to be unlimited, and abnormal fixed deposits are never formed. At confinement[5] she is suddenly deprived of HCG, and her diencephalic fat-center reverts to its normal capacity. It is only then that the abnormally accumulated fat is locked away again in a fixed deposit. From that moment on she is suffering from obesity and is subject to all its consequences. Pregnancy seems to be the only normal human condition in which the diencephalic fat-banking capacity is unlimited. It is only during pregnancy that fixed fat deposits can be transferred back into the normal current account and freely drawn upon to make up for any nutritional deficit. During pregnancy, every

ounce of reserve fat is placed at the disposal of the growing fetus. Were this not so, an obese woman, whose normal reserves are already depleted, would have the greatest difficulties in bringing her pregnancy to full term. There is considerable evidence to suggest that it is the HCG produced in large quantities in the placenta which brings about this diencephalic change.

Though we may be able to increase the dieneephalic fat banking capacity by injecting HCG, this does not in itself affect the weight, just as transferring monetary funds from a fixed deposit into a current account does not make a man any poorer; to become poorer it is also necessary that he freely spends the money which thus becomes available. In pregnancy the needs of the growing embryo take care of this to some extent, but in the treatment of obesity there is no embryo, and so a very severe dietary restriction must take its place for the duration of treatment.

Only when the fat which is in transit under the effect of HCG is actually consumed can more fat be withdrawn from the fixed deposits. In pregnancy it would be most undesirable if the fetus were offered ample food only when there is a high influx from the intestinal tract. Ideal nutritional conditions for the fetus can only be achieved when the mother's blood is continually saturated with food, regardless of whether she eats or not, as otherwise a period of starvation might hamper the steady growth of the embryo. It seems that HCG brings about this continual saturation of the blood, which is the reason why obese patients under treatment with HCG never feel hungry in spite of their drastically reduced food intake.

The Nature of Human Chorionic Gonadotropin

HCG is never found in the human body except during pregnancy and in those rare cases in which a residue of placental tissue continues to grow in the womb in what is known as a chorionic epithelioma. It is never found in the male. The human type of chorionic gonadotrophin is found only during the pregnancy of women and the great apes. It is produced in enormous quantities, so that during certain phases of her pregnancy a woman may excrete as much as one million International Units per day in her urine – enough to render a million infantile rats precociously mature. Other mammals make use of a different hormone, which can be extracted from their blood serum but not from their urine. Their placenta differs in this and other respects from that of man and the great apes. This animal chorionic gonadotrophin is much less rapidly broken down in the human body than HCG, and it is also less suitable for the treatment of obesity.

As often happens in medicine, much confusion has been caused by giving HCG its name before its true mode of action was understood. It has been explained that gonadotrophin literally means a sex-gland directed substance or hormone, and this is quite misleading. It dates from the early days when it was first found that HCG is able to render infantile sex glands mature, whereby it was entirely overlooked that it has no stimulating effect whatsoever on normally developed and normally functioning sex-glands. No amount of HCG is ever able to increase a normal sex function; it can only improve an abnormal one and in the young hasten the onset of puberty. However, this is no direct effect. HCG acts exclusively at a diencephalic level and there brings about a considerable increase in the functional capacity of all those centers which are working at maximum capacity.

The Real Gonadotrophins

Two hormones known in the female as follicle stimulating hormone (FSH) and corpus luteum stimulating hormone (LSH) are secreted by the anterior lobe of the pituitary gland. These hormones are real gonadotrophins because they directly govern the function of the ovaries. The anterior pituitary is in turn governed by the diencephalon, and so when there is an ovarian deficiency the diencephalic center concerned is hard put to correct matters by increasing the secretion from the anterior pituitary of FSH or LSH, as the case may be. When sexual deficiency is clinically present, this is a sign that the diencephalic center concerned is unable, in spite of maximal exertion, to cope with the demand for anterior pituitary stimulation.[6] When then the administration of HCG increases the functional capacity of the diencephalon, all demands can be fully satisfied and the sex deficiency is corrected. 9 That this is the true mechanism underlying the presumed gonadotrophic action of HCG is confirmed by the fact that when the pituitary gland of infantile rats is removed before they are given HCG, the latter has no effect on their sex-glands. HCG cannot therefore have a direct sex gland stimulating action like that of the anterior pituitary gonadotrophins, as FSH and LSH are justly called. The latter are entirely different substances from that which can be extracted from pregnancy urine and which, unfortunately, is called chorionic gonadotrophin. It would be no more clumsy, and certainly far more appropriate, if HCG were henceforth called chorionic diencephalotrophin.

HCG no Sex Hormone

It cannot he sufficiently emphasized that HCG is not sex-hormone, that its action is identical in men, women, children and in those cases in which the sex-glands no longer function owing to old age or their surgical removal. The only sexual change it can bring about after puberty is an improvement of a pre-existing deficiency, but never a stimulation beyond the normal. In an indirect way via the anterior pituitary, HCG regulates menstruation and facilitates conception, but it never virilizes a woman or

feminizes a man. It neither makes men grow breasts nor does it interfere with their virility, though where this was deficient it may improve it. It never makes women grow a beard or develop a gruff voice. I have stressed this point only for the sake of my lay readers, because, it is our daily experience that when patients hear the word hormone they immediately jump to the conclusion that this must have something to do with the sex- sphere. They are not accustomed as we are, to think thyroid, insulin, cortisone, adrenalin etc, as hormones.

Importance and Potency of HCG

Owing to the fact that HCG has no direct action on any endocrine gland, its enormous importance in pregnancy has been overlooked and its potency underestimated. Though a pregnant woman can produce as much as one million units per day, we find that the injection of only 125 units per day is ample to reduce weight at the rate of roughly one pound per day, even in a colossus weighing 400 pounds, when associated with a 500- Calorie diet. It is no exaggeration to say that the flooding of the female body with HCG is by far the most spectacular hormonal event in pregnancy. It has an enormous protective importance for mother and child, and I even go so far as to say that no woman, and certainly not an obese one, could carry her pregnancy to term without it.

If I can be forgiven for comparing my fellow-endocrinologists with wicked Godmothers, HCG has certainly been their Cinderella, and I can only romantically hope that its extraordinary effect on abnormal fat will prove to be its Fairy Godmother.

HCG has been known for over half a century. It is the substance which Aschheim and Zondek so brilliantly used to diagnose early pregnancy out of the urine. Apart from that, the only thing it did in the experimental laboratory was to produce precocious rats, and that was not particularly stimulating to further research at a time when much more thrilling endocrinological discoveries were pouring in from all sides, sweeping, HCG into the stiller back waters.

Complicating Disorders

Some complicating disorders are often associated with obesity, and these we must briefly discuss. The most important associated disorders and the ones in which obesity seems to play a precipitating or at least an aggravating role are the following: the stable type of diabetes, gout, rheumatism and arthritis, high blood pressure and hardening of the arteries, coronary disease and cerebral hemorrhage.

Apart from the fact that they are often – though not necessarily – associated with obesity, these disorders have two things in common. In all of them, modern research is becoming more and more inclined to believe that diencephalic regulations play a dominant role in their causation. The other common factor is that they either improve or do not occur during pregnancy. In the latter respect they are joined by many other disorders not necessarily associated with obesity. Such disorders are, for instance, colitis, duodenal or gastric ulcers, certain allergies, psoriasis, loss of hair, brittle fingernails, migraine, etc.

If HCG + diet does in the obese bring about those diencephalic changes which are characteristic of pregnancy, one would expect to see an improvement in all these conditions comparable to that seen in real pregnancy. The administration of HCG does in fact do this in a remarkable way.

Diabetes

In an obese patient suffering from a fairly advanced case of stable diabetes of many years duration in which the blood sugar may range from 3-400 mg%, it is often possible to stop all antidiabetic medication after the first few days of treatment. The blood sugar continues to drop from day to day and often reaches normal values in 2-3 weeks. As in pregnancy, this phenomenon is not observed in the brittle type of diabetes, and as some cases that are predominantly stable may have a small brittle factor in their clinical makeup, all obese diabetics have to be kept under a very careful and expert watch.

A brittle case of diabetes is primarily due to the inability of the pancreas to produce sufficient insulin, while in the stable type, diencephalic regulations seem to be of greater importance. That is possibly the reason why the stable form responds so well to the HCG method of treating obesity, whereas the brittle type does not. Obese patients are generally suffering from the stable type, but a stable type may gradually change into a brittle one, which is usually associated with a loss of weight. Thus, when an obese diabetic finds that he is losing weight without diet or treatment, he should at once have his diabetes expertly attended to. There is some evidence to suggest that the change from stable to brittle is more liable to occur in patients who are taking insulin for their stable diabetes.

Rheumatism

All rheumatic pains, even those associated with demonstrable bony lesions, improve subjectively within a few days of treatment, and often require neither cortisone nor salicylates. Again this is a well known phenomenon in pregnancy, and while under treatment with HCG + diet the effect is no less dramatic. As it does after pregnancy, the pain of deformed joints returns after treatment, but smaller doses of pain-relieving drugs seem able to control it satisfactorily after weight reduction. In any case, the HCG method makes it possible in obese arthritic patients to interrupt prolonged cortisone treatment without a recurrence of pain. This in itself is most welcome, but there is the added 10 advantage that the treatment stimulates the secretion of ACTH in a physiological manner and that this regenerates the adrenal cortex, which is apt to suffer under prolonged cortisone treatment.

Cholesterol
The exact extent to which the blood cholesterol is involved in hardening of the arteries, high blood pressure and coronary disease is not as yet known, but it is now widely admitted that the blood cholesterol level is governed by diencephalic mechanisms. The behavior of circulating cholesterol is therefore of particular interest during the treatment of obesity with HCG. Cholesterol circulates in two forms, which we call free and esterified. Normally these fractions are present in a proportion of about 25% free to 75% esterified cholesterol, and it is the latter fraction which damages the walls of the arteries. In pregnancy this proportion is reversed and it may he taken for granted that arteriosclerosis never gets worse during pregnancy for this very reason.
To my knowledge, the only other condition in which the proportion of free to esterified cholesterol is reversed is during the treatment of obesity with HCG + diet, when exactly the same phenomenon takes place. This seems an important indication ofhow closely a patient under HCG treatment resembles a pregnant woman in diencephalic behavior.
When the total amount of circulating cholesterol is normal before treatment, this absolute amount is neither significantly increased nor decreased. But when an obese patient with an abnormally high cholesterol and already showing signs of arteriosclerosis is treated with HCG, his blood pressure drops and his coronary circulation seems to improve, and yet his total blood cholesterol may soar to heights never before reached.

At first this greatly alarmed us. But then we saw that the patients came to no harm even if treatment was continued and we found in follow-up examinations undertaken some months after treatment that the cholesterol was much better than it had been before treatment. As the increase is mostly in the form of the not dangerous free cholesterol, we gradually came to welcome the phenomenon. Today we believe that the rise is entirely due to the liberation of recent cholesterol deposits that have not yet undergone calcification in the arterial wall and therefore highly beneficial.

Gout
An identical behavior is found in the blood uric acid level of patients suffering from gout. Predictably such patients get an acute and often severe attack after the first few days of HCG treatment but then remain entirely free of pain, in spite of the fact that their blood uric acid often shows a marked increase which may persist for several months after treatment. Those patients who have regained their normal weight remain free of symptoms regardless of what they eat, while those that require a second course of treatment get another attack of gout as soon as the second course is initiated. We do not yet know what diencephalic mechanisms are involved in gout; possibly emotional factors play a role, and it is worth remembering that the disease does not occur in women of childbearing age. We now give 2 tablets daily of ZYLORIC to all patients who give a history of gout and have a high blood uric acid level. In this way we can completely avoid attacks during treatment.

Blood Pressure
Patients who have brought themselves to the brink of malnutrition by exaggerated dieting, laxatives etc, often have an abnormally low blood pressure. In these cases the blood pressure rises to normal values at the beginning of treatment and then very gradually drops, as it always does in patients with a normal blood pressure. Normal values are always regained a few days after the treatment is over. Of this lowering of the blood pressure during treatment the patients are not aware. When the blood pressure is abnormally high, and provided there are no detectable renal lesions, the pressure drops, as it usually does in pregnancy. The drop is often very rapid, so rapid in fact that it sometimes is advisable to slow down the process with pressure sustaining medication until the circulation has had a few days time to adjust itself to the new situation. On the other hand, among the thousands of cases treated, we have never seen any untoward incident which could be attributed to the rather sudden drop in high blood pressure.
When a woman suffering from high blood pressure becomes pregnant her blood pressure very soon drops, but after her confinement it may gradually rise back to its former level. Similarly, a high blood pressure present before HCG treatment tends to rise again after the treatment is over, though this is not always the case. But the former high levels are rarely reached, and we have gathered the impression that such relapses respond better to orthodox drugs such as Reserpine than before treatment.

Peptic Ulcers
In our cases of obesity with gastric or duodenal ulcers we have noticed a surprising subjective improvement in spite of a diet which would generally be considered most inappropriate for an ulcer patient. Here, too, there is a similarity with pregnancy, in which peptic ulcers hardly ever occur. However we have seen two cases with a previous history of several hemorrhages in which a bleeding occurred within 2 weeks of the end of treatment.

Psoriasis, Fingernails, Hair, Varicose Ulcers
As in pregnancy, psoriasis greatly improves during treatment but may relapse when the treatment is over. Most patients spontaneously report a marked improvement in the condition of brittle fingernails. The loss

of hair not infrequently associated with obesity is temporarily arrested, though in very rare cases an increased loss of hair has been reported. I remember a case in which a patient developed a patchy baldness – so called alopecia areata – after a severe emotional shock, just before she was about to start an HCG treatment. Our dermatologist diagnosed the case as a particularly severe one, predicting that all the hair would be lost. He counseled against the reducing treatment, but in view of my previous experience and as the patient was very anxious not to postpone reducing, I discussed the matter with the dermatologist and it was agreed that, having fully acquainted the patient with the situation, the treatment should be started. During the treatment, which lasted four weeks, the further development of the bald patches was almost, if not quite, arrested; however, within a week of having finished the course of HCG, all the remaining hair fell out as predicted by the dermatologist. The interesting point is that the treatment was able to postpone this result but not to prevent it. The patient has now grown a new shock of hair of which she is justly proud.
In obese patients with large varicose ulcers we were surprised to find that these ulcers heal rapidly under treatment with HCG. We have since treated non obese patients suffering from varicose ulcers with daily injections of HCG on normal diet with equally good results. 11

The "Pregnant" Male
When a male patient hears that he is about to be put into a condition which in some respects resembles pregnancy, he is usually shocked and horrified. The physician must therefore carefully explain that this does not mean that he will be feminized and that HCG in no way interferes with his sex. He must be made to understand that in the interest of the propagation of the species nature provides for a perfect functioning of the regulatory headquarters in the diencephalon during pregnancy and that we are merely using this natural safeguard as a means of correcting the diencephalic disorder which is responsible for his overweight.

TECHNIQUE

Warnings
I must warn the lay reader that what follows is mainly for the treating physician and most certainly not a do-it-yourself primer. Many of the expressions used mean something entirely different to a qualified doctor than that which their common use implies, and only a physician can correctly interpret the symptoms which may arise during treatment. Any patient who thinks he can reduce by taking a few —shots‖ and eating less is not only sure to be disappointed but may be heading for serious trouble. The benefit the patient can derive from reading this part of the book is a fuller realization of how very important it is for him to follow to the letter his physician's instructions.

In treating obesity with the HCG + diet method we are handling what is perhaps the most complex organ in the human body. The diencephalon's functional equilibrium is delicately poised, so that whatever happens in one part has repercussions in others. In obesity this balance is out of kilter and can only be restored if the technique I am about to describe is followed implicitly. Even seemingly insignificant deviations, particularly those that at first sight seem to be an improvement, are very liable to produce most disappointing results and even annul the effect completely. For instance, if the diet is increased from 500 to 600 or 700 Calories, the loss of weight is quite unsatisfactory. If the daily dose of HCG is raised to 200 or more units daily its action often appears to be reversed, possibly because larger doses evoke diencephalic counter-regulations. On the other hand, the diencephalon is an extremely robust organ in spite of its unbelievable intricacy. From an evolutionary point of view it is one of the oldest organs in our body and its evolutionary history dates back more than 500 million years. This has tendered it extraordinarily adaptable to all natural exigencies, and that is one of the main reasons why the human species was able to evolve. What its evolution did not prepare it for were the conditions to which human culture and civilization now expose it.

History taking
When a patient first presents himself for treatment, we take a general history and note the time when the first signs of overweight were observed. We try to establish the highest weight the patient has ever had in his life (obviously excluding pregnancy), when this was, and what measures have hitherto been taken in an effort to reduce.
It has been our experience that those patients who have been taking thyroid preparations for long periods have a slightly lower average loss of weight under treatment with HCG than those who have never taken thyroid. This is even so in those patients who have been taking thyroid because they had an abnormally low basal metabolic rate. In many of these cases the low BMR is not due to any intrinsic deficiency of the thyroid gland, but rather to a lack of diencephalic stimulation of the thyroid gland via the anterior pituitary lobe. We never allow thyroid to be taken during treatment, and yet a BMR which was very low before treatment is usually found to be normal after a week or two of HCG + diet. Needless to say, this does not apply to those cases in which a thyroid deficiency has been produced by the surgical removal of a part of an overactive gland. It is also most important to ascertain whether the patient has taken diuretics (water eliminating pills) as this also decreases the weight loss under the HCG regimen.

Returning to our procedure, we next ask the patient a few questions to which he is held to reply simply with —yes‖ or —no‖. These questions are: Do you suffer from headaches? rheumatic pains? menstrual disorders? constipation? breathlessness or exertion? swollen ankles? Do you consider yourself greedy? Do you feel the need to eat snacks between meals?
The patient then strips and is weighed and measured. The normal weight for his height, age, skeletal and muscular build is established from tables of statistical averages, whereby in women it is often necessary to make an allowance for particularly large and heavy breasts. The degree of overweight is then calculated, and from this the duration of treatment can be roughly assessed on the basis of an average loss of weight of a little less than a pound, say 300-400 grams-per injection, per day. It is a particularly interesting feature of the HCG treatment that in reasonably cooperative patients this figure is remarkably constant, regardless of sex, age and degree of overweight.

The Duration of Treatment
Patients who need to lose 15 pounds (7 kg.) or less require 26 days treatment with 23 daily injections. The extra three days are needed because all patients must continue the 500-Calorie diet for three days after the last injection. This is a very essential part of the treatment, because if they start eating normally as long as there is even a trace of HCG in their body they put on weight alarmingly at the end of the treatment. After three days when all the HCG has been eliminated this does not happen, because the blood is then no longer saturated with food and can thus accommodate an extra influx from the intestines without increasing its volume by retaining water.
We never give a treatment lasting less than 26 days, even in patients needing to lose only 5 pounds. It seems that even in the mildest cases of obesity the diencephalon requires about three weeks rest from the maximal exertion to which it has been previously subjected in order to regain fully its normal fat-banking capacity. Clinically this expresses itself, in the fact that, when in these mild cases, treatment is stopped as soon as the weight is normal, which may be achieved in a week, it is much more easily regained than after a full course of 23 injections.

As soon as such patients have lost all their abnormal superfluous fat, they at once begin to feel ravenously hungry in spite of continued injections. This is because HCG only puts abnormal fat into circulation and cannot, in the doses used, liberate normal fat deposits; indeed, it seems to prevent their consumption. As soon as their statistically normal weight is reached, these patients are put on 800-1000 Calories for the rest of the treatment. 12 The diet is arranged in such a way that the weight remains perfectly stationary and is thus continued for three days after the 23rd injection. Only then are the patients free to eat anything they please except sugar and starches for the next three weeks.

Such early cases are common among actresses, models, and persons who are tired of obesity, having seen its ravages in other members of their family. Film actresses frequently explain that they must weigh less than normal. With this request we flatly refuse to comply, first, because we undertake to cure a disorder, not to create a new one, and second, because it is in the nature of the HCG method that it is self limiting. It becomes completely ineffective as soon as all abnormal fat is consumed. Actresses with a slight tendency to obesity, having tried all manner of reducing methods, invariably come to the conclusion that their figure is satisfactory only when they are underweight, simply because none of these methods remove their superfluous fat deposits. When they see that under HCG their figure improves out of all proportion to the amount of weight lost, they are nearly always content to remain within their normal weight-range.

When a patient has more than 15 pounds to lose the treatment takes longer but the maximum we give in a single course is 40 injections, nor do we as a rule allow patients to lose more than 34 lbs. (15 Kg.) at a time. The treatment is stopped when either 34 lbs. have been lost or 40 injections have been given. The only exception we make is in the case of grotesquely obese patients who may be allowed to lose an additional 5-6 lbs. if this occurs before the 40 injections are up.

Immunity to HCG
The reason for limiting a course to 40 injections is that by then some patients may begin to show signs of HCG immunity. Though this phenomenon is well known, we cannot as yet define the underlying mechanism. Maybe after a certain length of time the body learns to break down and eliminate HCG very rapidly, or possibly prolonged treatment leads to some sort of counter-regulation which annuls the diencephalic effect.
After 40 daily injections it takes about six weeks before this so called immunity is lost and HCG again becomes fully effective.

Usually after about 40 injections patients may feel the onset of immunity as hunger which was previously absent. In those comparatively rare cases in which signs of immunity develop before the full course of 40 injections has been completed-say at the 35th injection- treatment must be stopped at once, because if it is continued the patients begin to look weary and drawn, feel weak and hungry and any further loss of

weight achieved is then always at the expense of normal fat. This is not only undesirable, but normal fat is also instantly regained as soon as the patient is returned to a free diet.
Patients who need only 23 injections may be injected daily, including Sundays, as they never develop immunity. In those that take 40 injections the onset of immunity can be delayed if they are given only six injections a week, leaving out Sundays or any other day they choose, provided that it is always the same day. On the days on which they do not receive the injections they usually feel a slight sensation of hunger. At first we thought that this might be purely psychological, but we found that when normal saline is injected without the patient's knowledge the same phenomenon occurs.

Menstruation
During menstruation no injections are given, but the diet is continued and causes no hardship; yet as soon as the menstruation is over, the patients become extremely hungry unless the injections are resumed at once. It is very impressive to see the suffering of a woman who has continued her diet for a day or two beyond the end of the period without coming for her injection and then to hear the next day that all hunger ceased within a few hours after the injection and to see her once again content, florid and cheerful. While on the question of menstruation it must he added that in teenaged girls the period may in some rare cases be delayed and exceptionally stop altogether. If then later this is artificially induced some weight may be regained.

Further Courses
Patients requiring the loss of more than 34 lbs. must have a second or even more courses. A second course can be started after an interval of not less than six weeks, though the pause can be more than six weeks. When a third, fourth or even fifth course is necessary, the interval between courses should be made progressively longer. Between a second and third course eight weeks should elapse, between a third and fourth course twelve weeks, between a fourth and fifth course twenty weeks and between a fifth and sixth course six months. In this way it is possible to bring about a weight reduction of 100 lbs. and more if required without the least hardship to the patient.
In general, men do slightly better than women and often reach a somewhat higher average daily loss. Very advanced cases do a little better than early ones, but it is a remarkable fact that this difference is only just statistically significant.

Conditions that must be accepted before treatment
On the basis of these data the probable duration of treatment can he calculated with considerable accuracy, and this is explained to the patient. It is made clear to him that during the course of treatment he must attend the clinic daily to be weighed, injected and generally checked. All patients that live in Rome or have resident friends or relations with whom they can stay are treated as out-patients, but patients coming from abroad must stay in the hospital, as no hotel or restaurant can be relied upon to prepare the diet with sufficient accuracy. These patients have their meals, sleep, and attend the clinic in the hospital, but are otherwise free to spend their time as they please in the city and its surroundings sightseeing, bathing or theater-going.
It is also made clear that between courses the patient gets no treatment and is free to eat anything he pleases except starches and sugar during the first 3 weeks. It is impressed upon him that he will have to follow the prescribed diet to the letter and that after the first three days this will cost him no effort, as he will feel no hunger and may indeed have difficulty in getting down the 500 Calories which he will be given. If these conditions are not acceptable the case is refused, as any compromise or half measure is bound to prove utterly disappointing to patient and physician alike and is a waste of time and energy.
Though a patient can only consider himself really cured when he has been reduced to his statistically normal weight, we do not insist that he commit himself to that extent. Even a partial loss of overweight is highly beneficial, and it is our experience that once a patient has completed a first course he is so enthusiastic about the ease with which the – to him surprising – results are achieved that he almost invariably comes 13 back for more. There certainly can be no doubt that in my clinic more time is spent on damping over-enthusiasm than on insisting that the rules of the treatment be observed.

Examining the patient
Only when agreement is reached on the points so far discussed do we proceed with the examination of the patient. A note is made of the size of the first upper incisor, of a pad of fat on the nape of the neck, at the axilla and on the inside of the knees. The presence of striation, a suprapubic fold, a thoracic fold, angulation of elbow and knee joint, breast-development in men and women, edema of the ankles and the state of genital development in the male are noted.
Wherever this seems indicated we X-ray the sella turcica, as the bony capsule which contains the pituitary gland is called, measure the basal metabolic rate, X-ray the chest and take an electrocardiogram. We do a blood-count and a sedimentation rate and estimate uric acid, cholesterol, iodine and sugar in the fasting blood.

Gain before Loss

Patients whose general condition is low, owing to excessive previous dieting, must eat to capacity for about one week before starting treatment, regardless of how much weight they may gain in the process. One cannot keep a patient comfortably on 500 Calories unless his normal fat reserves are reasonably well stocked. It is for this reason also that every case, even those that are actually gaining must eat to capacity of the most fattening food they can get down until they have had the third injection. It is a fundamental mistake to put a patient on 500 Calories as soon as the injections are started, as it seems to take about three injections before abnormally deposited fat begins to circulate and thus become available. We distinguish between the first three injections, which we call —non-effective‖ as far as the loss of weight is concerned, and the subsequent injections given while the patient is dieting, which we call —effective‖. The average loss of weight is calculated on the number of effective injections and from the weight reached on the day of the third injection which may be well above what it was two days earlier when the first injection was given.

Most patients who have been struggling with diets for years and know how rapidly they gain if they let themselves go are very hard to convince of the absolute necessity of gorging for at least two days, and yet this must he insisted upon categorically if the further course of treatment is to run smoothly. Those patients who have to be put on forced feeding for a week before starting the injections usually gain weight rapidly – four to six pounds in 24 hours is not unusual – but after a day or two this rapid gain generally levels off. In any case, the whole gain is usually lost in the first 48 hours of dieting. It is necessary to proceed in this manner because the gain re-stocks the depleted normal reserves, whereas the subsequent loss is from the abnormal deposits only.

Patients in a satisfactory general condition and those who have not just previously restricted their diet start forced feeding on the day of the first injection. Some patents say that they can no longer overeat because their stomach has shrunk after years of restrictions. While we know that no stomach ever shrinks, we compromise by insisting that they eat frequently of highly concentrated foods such as milk chocolate, pastries with whipped cream sugar, fried meats (particularly pork), eggs and bacon, mayonnaise, bread with thick butter and jam, etc. The time and trouble spent on pressing this point upon incredulous or reluctant patients is always amply rewarded afterwards by the complete absence of those difficulties which patients who have disregarded these instructions are liable to experience.

During the two days of forced feeding from the first to the third injection – many patients are surprised that contrary to their previous experience they do not gain weight and some even lose. The explanation is that in these cases there is a compensatory flow of urine, which drains excessive water from the body. To some extent this seems to be a direct action of HCG, but it may also be due to a higher protein intake, as we know that a protein-deficient diet makes the body retain water.

Starting treatment

In menstruating women, the best time to start treatment is immediately after a period. Treatment may also be started later, but it is advisable to have at least ten days in hand before the onset of the next period. Similarly, the end of a course of HCG should never be made to coincide with menstruation. If things should happen to work out that way, it is better to give the last injection three days before the expected date of the menses so that a normal diet can he resumed at onset. Alternatively, at least three injections should be given after the period, followed by the usual three days of dieting. This rule need not be observed in such patients who have reached their normal weight before the end of treatment and are already on a higher caloric diet.

Patients who require more than the minimum of 23 injections and who therefore skip one day a week in order to postpone immunity to HCG cannot have their third injections on the day before the interval. Thus if it is decided to skip Sundays, the treatment can be started on any day of the week except Thursdays. Supposing they start on Thursday, they will have their third injection on Saturday, which is also the day on which they start their 500 Calorie diet. They would then have no injection on the second day of dieting; this exposes them to an unnecessary hardship, as without the injection they will feel particularly hungry. Of course, the difficulty can be overcome by exceptionally injecting them on the first Sunday. If this day falls between the first and second or between the second and third injection, we usually prefer to give the patient the extra day of forced feeding, which the majority rapturously enjoy. 14

The Diet

The 500 Calorie diet is explained on the day of the second injection to those patients who will be preparing their own food, and it is most important that the person who will actually cook is present – the wife, the mother or the cook, as the case may be. Here in Italy patients are given the following diet sheet.

Breakfast: Tea or coffee in any quantity without sugar. Only one tablespoonful of milk allowed in 24 hours. Saccharin or Stevia may be used.

Lunch:

1. 100 grams of veal, beef, chicken breast, fresh white fish, lobster, crab, or shrimp. All visible fat must be carefully removed before cooking, and the meat must be weighed raw. It must be boiled or grilled without additional fat. Salmon, eel, tuna, herring, dried or pickled fish are not allowed. The chicken breast must be removed from the bird.
2. One type of vegetable only to be chosen from the following: spinach, chard, chicory, beet-greens, green salad, tomatoes, celery, fennel, onions, red radishes, cucumbers, asparagus, cabbage.
3. One breadstick (grissino) or one Melba toast.
4. An apple, orange, or a handful of strawberries or one-half grapefruit.

Dinner :
The same four choices as lunch (above.)