The Relation of Fatness to Sterility.*

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That a relationship exists between obesity and sterility has been suspected since the time of Hippocrates, and it is quite evident from the modern literature that their association is more than a casual one. The problem, however, continually recurs as to whether obesity *per se* is a cause of sterility, or whether, when the two conditions are found together, they are not both the result of some primary and more widespread endocrine change.

In the present investigation, the object in view has been to examine the incidence of sterility in a series of obese married women and, if possible, to come to some conclusions as to the type of obesity with which it is associated, also to estimate the relative significance of the various factors concerned. For this purpose observations have been made on 198 cases of obesity in married women in whom, when sterility occurred, there was no apparent local pelvic cause. The cases were all of simple obesity, in the sense that there were no signs of gross endocrine disease, which would have enabled them to be classified under the recognized heading of myxœdema, simple goitre, dystrophia-adiposo genitalis, pituitary tumour and so on. Nevertheless, as the investigations will show, there can be little doubt that a certain proportion of these patients were suffering from minor degrees of endocrine dystrophy.

Historical.

The literature abounds with references to the association of obesity and sterility. Eden and Lockyer[1] (1917), for instance, point out that obesity is frequently mentioned as a cause of sterility. Child[2] (1922) stated that fat women are not as fertile as their thin sisters and quotes Oliver who says that he has frequently remarked

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that women who tend to lay on fat rapidly are apt to become barren. Crossen[3] (1923) mentions that sterility is present at times in patients inclined to stoutness, the condition developing when the patient accumulates fat and disappearing promptly on her reduction to normal weight. Graves[4] (1923) also considered that there was a relationship between adiposity and hypoplasia of the sex organs, and he quotes Müller's and Horrocks' opinion that over-nourished women show diminished fertility. He states, however, as his personal opinion that this cannot be a constant rule, because occasionally fat women show extreme fertility. In his opinion, when fat deposit and sterility are found in association, the fat is probably a manifestation of ovarian deficiency. McCann[5] (1925) considered that obesity developing subsequent to pregnancy was liable to be associated with absent or diminished fertility and he quotes the well-known fact that animals putting on flesh lose their value for stud purposes. Fairbairn[6] (1928) in his latest text-book of gynæcology and obstetrics remarks on the frequent association of sterility and obesity. Giles[7] (1928) in a paper on the diagnosis and treatment of sterility refers to errors of metabolism, such as obesity, as common causes of sterility. Cotte[8] (1928) in a full review of the subject of sterility states that obesity has been said to be the most frequent cause of this condition after cervical stenosis and salpingitis, but he thinks that when this statement was made conditions of ovarian and hypophyseal origin must have been included. He then points out that exogenous obesity does not appear to have a malign influence in this connexion. In Algeria and the East, for instance, exogenous obesity is common but the women are fertile.

On the other hand, Malcolm[9] (1925) has drawn attention to a custom in certain primitive races in Old Calabar, where all young girls of any social standing are subject to two or three years' fattening treatment, with the result that most of them prove to be completely sterile.

In 1929, Meaker[10] reviewed the metabolic aspect of sterility and concluded that extrinsic faults, e.g., lack of exercise and errors of diet, as opposed to those of intrinsic or endocrine origin result not infrequently in the familiar picture of obesity, amenorrhœa and sterility.

The various views expressed above would appear to be fairly representative of present day opinion, but when it comes to a question of facts, there is a comparative paucity of *data* to record. A series of cases of obesity and sterility was recorded by Kisch.[11] In this investigation, of 215 fat married women in his Marienbad clinic, 48, or 22.3 per cent were sterile. As Dietrich[12] points out in

a criticism of this paper, only cases of primary sterility were taken into consideration, and often the duration of sterility was comparatively short—a matter of some three years or so instead of the period of six years which he himself considers should be allowed. The results too are of relative value only for no details were given as to the cause of the obesity.

In Dietrich's own investigations a slightly different standpoint was adopted. He found that of 294 cases of primary sterility, only seven, or 2.37 per cent were fat individuals in whom no other cause could be found for the sterility except obesity. In his seven cases the obesity was clearly of endogenous origin, and in several he suspected a thyroid factor. In fact, he thinks that exogenous obesity is probably not a cause of sterility.

Dietrich's figures as to the incidence of obesity in sterility agree closely with those of Krampf and Wiebe whom he quotes. The former in a series of 527 sterile women found that only 2.85 per cent, were obese, while the latter, among 141 cases of sterility, concluded that obesity was a factor in only 1.4 per cent.

Other investigators have discussed this problem of the relationship between sterility and obesity, notably Seitz and Winter, quoted by Dietrich. These authors, and also Wiebe and Krampf, quoted above, came to the conclusion that the most important factor was ovarian hypofunction to which they consider that the obesity is generally secondary. It should be mentioned here that in Dietrich's opinion the endogenous factor in such cases is probably either thyroid or hypophyseal (*formes frustes* of dystrophia adiposo-genitalis) and that as a result of disturbances of these glands, a secondary ovarian deficiency occurs which results in inhibition of menstruation and ovulation and so causes sterility by preventing conception. Other views as to the endocrine aspect of this question will be discussed in a subsequent section.

Brief reference must be made finally to work on the relationship between fatness and sterility based on animal experiments.

In 1920, Marshall and Peel[13] found that fatness resulting from overfeeding in animals might cause sterility directly. In 1922, Marshall[14] stated that in domestic animals intensive fattening might lead to a break in, or entire cessation of, the œstrus cycle and to degenerative changes in the ovaries. A further series of experiments has recently been carried out by Parkes and Drummond[15] in rats, but they were careful to see that an adequate supply of the recently discovered anti-sterility Vitamin E was present in their artificial fattening diets. As a result of these experiments they conclude that appreciable degrees of obesity are not necessarily incompatible with the normal functioning of the

reproductive organs, and that excessive fatness does not necessarily lead to sterility. In their opinion, fatness is more often a result than a cause of sterility. It is quite possible that the intentional addition of Vitamin E in Parkes and Drummond's series of experiments accounts for the discrepancy in these investigations.

THE PRESENT INVESTIGATION.

Classification and Age Incidence of Obesity.

The 198 cases of obesity in married women which form the *data* of the present investigation can be grouped on a chronological basis according to the age of onset of their obesity. If this is done, it is found that 137 cases in which the problem of primary sterility can be investigated fall into two main groups, (1) constitutional obesities, cases dating from birth and puberty in the large majority of which there is a family history of obesity, generally of the same type; (2) obesities developing in later life: in these the hereditary factor is often negligible, but exogenous and endogenous factors are pronounced and often combined.

In addition to these 137 cases there is a further group of 61 cases in which obesity first developed during or after childbirth. These have been included in order to investigate the problem of whether obesity developing in early pregnancies is often a cause of subsequent sterility.

Table I.
Classification of 198 cases.

	Dating to	No. of cases.
Group I. Constitutional obesities	Birth	57
	Puberty	37
Group II. Obesities of later life.	Marriage (immediately after)	26
	Miscellaneous: Infections such as Typhoid	17
Group III. Obesity developing during or after childbirth		61

As we have already pointed out in the opening section of this paper, all these cases fall under the heading of simple obesity, but though no examples of gross endocrine disease are included, a certain proportion of them have features suggesting minor grades of endocrine disturbance. It does not seem possible at the present moment to classify these patients into exogenous and endogenous groups. A certain number, mostly those in which obesity

developed in later life, gave what appeared to be an exogenous history, but the proportion was surprisingly small. In the majority no clear cut exogenous history could be obtained and the obesity was found to have developed immediately after marriage or after some operation or infectious illness. In a number the gain in weight and fat tissue was so excessive and so entirely out of proportion to any alteration in diet, or diminution in energy expenditure, that it seemed necessary to assume some endogenous change. In the majority of cases, no doubt, both exogenous and endogenous factors played a part. Nevertheless, it is so difficult to assess the relative importance of these factors in any given case that we feel doubtful if an attempt at a classification on such a basis would be justified. It is slightly different with the cases which we have included under the heading of constitutional obesity, when obesity is present from birth or develops at puberty with the onset of menstruation. In these patients there is nearly always a family history of obesity and in certain individuals who are born fat the condition would appear to be transmitted directly from mother to child. It has not been at all unusual to find a history of a mother developing obesity on the birth of the first, second, third or fourth child and the child itself showing the same condition. It is difficult, of course, to estimate the influence of exogenous factors, in the case of the mother, during pregnancy and lactation, but in the case of the child it seems more than probable that at this early age there must be some pecularity of metabolism leading to fat storage. We have, therefore, adopted the term "constitutional obesity" for cases of the hereditary type.

Fertility in Obesity.

Of the 137 cases of obesity comprising the birth, puberty, marriage and miscellaneous groups, 82, or 60 per cent bore children, while an additional eight cases or 6 per cent had miscarriages only. The details as to the numbers of successful pregnancies in the individual groups can be seen in Table II.

Table II.

		No. of	One	Two children or more.	per cent
Constitutional obesity.	Birth	57	12	27	69
	Puberty	37	14	16	81
Obesities of later life.	Marriage	26	4	4	31
	Miscellaneous	17	5	0	29

In the birth and puberty obesities it can be seen that fertility is well up to the normal, for, averaging the two groups, three quarters of the patients bore children. When, however, the obesity develops in later life, fertility is at a low level, only between one-quarter and one-third of the individuals being fertile.

Primary Sterility.

In this series of 137 obese married women, there are 47 cases of primary sterility, or 34 per cent. The age of marriage in 46 of these 47 cases was as follows:— Seven before the age of 20, twenty in the five-year period 20–24, sixteen in the five-year period 25–29. Of the four remaining cases, one was married at 31 and two at 32. It will be seen that none of these patients, when they were married, had reached the age of 35. Age, therefore, would not appear to be a factor of importance in their sterility. The duration of sterility in these 47 cases is shown in Table III. It will be observed that in two cases sterility was of one year's duration only, but as both patients came to hospital on account of this symptom, they have been included in the series.

Table III.

Duration of Sterility in 47 cases.

No. of years' sterility.	1 yr.	2 yrs.	3 yrs.	4 yrs.	5 yrs.	6 yrs.	7 yrs.	8 yrs.	9 yrs.	10 yrs.	11 yrs.	12 yrs. or more.
No. of cases	1	1	8	8	5	3	3	3	1	3	3	8

In Table IV will be found details of the numbers of cases of sterility in each of the individual groups of obesity, subdivided further according to the regularity or irregularity of menstruation. In the first column are the cases with regular menstruation; in the second those with primary menstrual irregularity in whom from puberty menstruation had never occurred at less than two, three or four monthly intervals; and in the third column those with secondary amenorrhœa. The latter includes both patients who, after a period of normal menstruation, developed complete amenorrhœa and others who developed a three or four monthly rhythm-secondary irregularity.

A brief glance at Table IV shows at once a striking difference in the incidence of primary sterility in the group of patients with regular menstruation and in those with one or other form of amenorrhœa, for in the former it is comparatively low and in the

latter high. A more detailed examination of the factors in these 47 individual cases of primary sterility reveals information of considerable interest. We shall now examine these groups in turn.

Group I. Obesity. Regular Menstruation. Of the seven cases of primary sterility associated with obesity dating from birth, three were found to be suffering from a well-marked glycosuria. On further investigation it was found that these cases presented a picture which one expects to find in a mild diabetic, but as a matter of fact they had no signs of diabetes at all except a glycosuria. There was no loss of weight or ketosis. This condition we have already referred to in a previous communication. It would appear to indicate a profound disturbance of carbohydrate metabolism, which was probably a factor of importance in these patients' sterility. All seven cases in this group had a strong family history of obesity, but in none of them was there any indication of endocrine disturbance.

TABLE IV.

	I. Regular Menstruation.		II. Primary Irregularity.		III. Secondary Amenorrhœa or Irregularity.	
	No. of Cases	No. Sterile	No. of Cases	No. Sterile	No. of Cases	No. Sterile
Birth (57)	44	7 (16 per cent)	7	4 (57 per cent)	6	4 (67 per cent)
Puberty (37)	28	1 (3 per cent)	6	3 (50 per cent)	3	3 (100 per cent)
Marriage (26)	18	6 (33 per cent)	5	4 (80 per cent)	3	3 (100 per cent)
Miscellaneous (17)	11	6 (55 per cent)	5	5 (100 per cent)	1	1 (100 per cent)

A very different state of affairs, however, is found in the six cases of obesity following marriage, for here five of the patients presented features of hypothyroidism, both clinically and to metabolic tests. The other patient was suffering from Dercum's disease, which some think has a thyroid origin.

In the last and miscellaneous group of obesities developing in later life (6 cases) two of the patients were suffering from mild hypothyroidism; one case appeared to be frankly exogenous, as the condition developed when the patient changed her occupation and took control of a butcher's shop; one case developed obesity after an operation for appendicitis, and one after rheumatic fever with cardiac complications which necessitated a long period of rest in bed. In the sixth patient the onset of obesity was associated with the beginning of a series of narcoleptic attacks which persisted for 16 years, but no definite evidence of endocrine or other disease could be found.

To summarize :— The striking features in the cases of Group I with regular menstruation) is the rarity of sterility in the uncomplicated constitutional obesities (birth and puberty types) and the high incidence in the obesities developing in later life. It must be emphasized, however, that in the latter series there are seven and possibly eight instances of mild hypothyroidism. The regularity of menstruation in these individuals should be noted.

Group II. Obesity with primary menstrual irregularity. Of the seven cases of primary sterility associated with constitutional obesity under this heading, there was no evidence of endocrine disturbance, apart from obesity and menstrual irregularity, in six, but the other presented symptoms of mild hypothyroidism which appeared to have been present only for a few years. The majority of patients gave a strong family history of obesity and one was suffering from glycosuria without loss of weight. Her fasting blood sugar was 0.198 mg. per cent. The interesting feature of the nine cases of obesity developing in later life was the absence of evidence of endocrine disturbance apart from the menstrual irregularity in all except two patients, who were suffering from mild hypothyroidism of a few years standing. In all these cases the menstrual irregularity had been present for many years before the the onset of obesity. The factors which led to the latter were, in four cases, marriage and, in one case, an operation for appendicitis. In another case the factors appear to have been exogenous and one patient subsequentlydeveloped disseminated sclerosis.

The chief feature of the cases in Group II, therefore, is the apparent absence of endocrine disturbance, apart from the menstrual irregularity, except in three patients who had lately developed mild symptoms of hypothyroidism.

Group III. Obesity with secondary amenorrhœa or menstrual irregularity. The 11 cases of primary sterility associated with obesity under this heading may be considered together, for in none of them was there any evidence of endocrine disturbance apart from secondary amenorrhœa. The onset of the latter in three cases was attributable to illness, accident or change of climate, incidents in themselves which did not appear to be factors in these patients' subsequent sterility. In five other patients a history was obtained of amenorrhœa developing in the early months of marriage, and in each case this gave rise to a tentative diagnosis of pregnancy. Subsequent events, however, showed that in three cases this was the commencement of a period of complete amenorrhœa, while in the two others menstruation occurred afterwards at only three or four monthly intervals. The cause of the amenorrhœa in these patients is problematical.

The chief characteristic then of the cases in Group III, as in Group II, is the absence of signs of endocrine disease but the presence of amenorrhœa or menstrual irregularity.

Obesity following Childbirth—Subsequent Fertility.

We have collected 61 cases of obesity, developing during or after childbirth in individuals who were previously normal or thin, for the purpose of this investigation. In only two of these cases was there any disturbance of menstruation after the onset of obesity. One patient developed secondary amenorrhœa and in the other a primary irregularity persisted. Neither patient became pregnant again.

The number of subsequent pregnancies in the other 59 cases with regular menstruation is recorded in Table V, which also indicates the original pregnancy during or after which obesity developed.

Table V.

Analysis of pregnancies in obesity following childbirth.
(59 cases with regular menstruation.)

Obesity following	No. of Cases	Subsequent pregnancies			
		None	Miscarriages only	One only	Two or more
1st pregnancy	40	13	4	8	15
2nd ,,	11	2	1	3	5
3rd ,,	3	0	1	1	1
4th ,,	5	0	0	2	3
Total	59	15	6	14	24 cases

Apart from the 13 patients out of a total of 40 who developed obesity after their first pregnancy, the figures in this Table do not require any further explanation, for the results are well within the normal limits. This figure, however, does require further analysis. Five of the 13 cases can be excluded, as the interval of time which had elapsed since the preceding pregnancy was under three years and the question of sterility had not arisen. In only five of the remaining cases was there a probability that secondary sterility was present after the onset of obesity. In only one patient out of 13 was there any evidence of endocrine disturbance, and she was suffering from mild hypothyroidism. This patient actually gained 56 pounds in weight in the twelve months following her original pregnancy. Metabolic investigations in the remaining 12 cases (B.M.R. 5 cases, Sugar Tolerance 7 cases) gave results within the normal limits in all. All these patients became exceedingly obese

after their first pregnancy, the average increase in weight being considerably more than 42 pounds per person.

It is perhaps surprising that there are in this group so few instances of hypothyroidism resulting from pregnancy and causing secondary sterility, for it is a condition which we have met with not infrequently. The explanation, however, is probably that in such cases obesity develops during pregnancy in association with a goitre and no cases of goitre have been included in this investigation.

The above observations can allow of one conclusion only. Simple obesity developing during or after pregnancy does not tend to produce secondary sterility.

Amenorrhœa and its Significance in Sterility.

The observations reported up to the present have emphasized the importance of amenorrhœa as a determining factor in the sterility of many of these cases. Its importance can be judged by the following statistics: In 101 cases with regular menstruation the incidence of sterility was 20 per cent only, and this includes the hypothyroid and glycosuria cases; but in 36 cases with amenorrhœa the incidence of sterility was 75 per cent.

As would be expected, complete secondary amenorrhœa would appear to be more frequently associated with sterility than primary menstrual irregularity, for in 11 cases of the former none became pregnant. On the other hand, of 23 cases of primary irregularity seven had pregnancies.

If amenorrhœa, such as we have described in these patients, may be regarded as an indication of ovarian hypofunction, our findings agree closely with those of Seitz, Winter, Wiebe and Krampf, who came to the conclusion that this was the most important factor in sterility associated with obesity. It must be pointed out, however, that few of our cases showed any other symptoms which could be attributed to ovarian hypofunction. In all, the development of the secondary sex characteristics was normal.

On the other hand, recent experimental work points to the possibility that the ovaries are not the sole regulators of menstruation and there is evidence to show that the anterior lobe of the pituitary body plays a part in this process. The possibility of a pituitary factor must not be overlooked in our patients and especially is this true in the type of case referred to as primary menstrual irregularity, the nature of which in itself suggests a developmental error. It must be admitted, however, that in the present state of our knowledge, and in the absence of a gross

pituitary lesion, it is difficult to know on what grounds a diagnosis of pituitary involvement in these cases would be justified. As far as one can tell none of our patients presented, apart from the menstrual disturbance, any abnormalities either of development or growth which would point to a disturbance of pituitary function.

Seitz, Winter, Wiebe and Krampf, quoted above, suggested that the adiposity in their patients was usually a secondary result of ovarian hypofunction. That this cannot be true of all our cases is obvious from a study of our original classification, for a certain proportion of them, 13 out of 36 with amenorrhœa, had been obese from birth. In others, however, obesity either developed concurrently with menstruation or menstrual irregularity had been present for many years. One cannot deny the undoubted fact that ovarian hypofunction, such as is encountered after castration and at the climacteric, and probably in certain cases of amenorrhœa, is often associated with a tendency to grow fat. It may have been a contributory factor in the obesity of some of our patients, but obviously only in a small percentage.

Another interesting problem is as to whether obesity *per se* is a contributory cause of amenorrhœa. Can errors of diet, for instance, which cause obesity also produce amenorrhœa? There is also the question as to whether amenorrhœa is more likely to occur in fat women than in those of normal proportions or thin. Experimental results are as yet inconclusive as to the former problem, while as to latter the only figures which we have available, though perhaps they are not strictly comparable with the present ones, suggest that amenorrhœa is no more common in fat women than thin. During the same period in which these 36 obese cases were sent to us for investigation, we also examined from the same sources 25 cases of primary menstrual irregularity and secondary amenorrhœa in normal or thin married women. Although the latter were not quite so numerous, the figures do not suggest that amenorrhœa is much more common in obesity. This group of normal and thin cases stresses again the importance of amenorrhœa in sterility, for of nine cases of primary irregularity eight were sterile, while of 16 cases of secondary amenorrhœa none became pregnant after the condition developed.

Hypothyroidism.

In this investigation we have referred from time to time to cases of obesity and sterility of thyrogenic origin. They make up 23 per cent of the 47 cases of obesity and sterility considered. The majority were found in the groups of obesity with regular men-

struation developing after marriage or in later life; in fact, in these types of obesity signs of mild hypothyroidism were common.

That hypothyroidism is a not infrequent cause of sterility has long been recognized. Eden and Lockyer,[1] Cotte,[8] Blair Bell,[16] Vignes and Cornil[18] have all referred to it, while Dietrich,[12] quoted above, concluded that all his seven cases of endogenous obesity with sterility were thyrogenic in origin.

That sterility is common in gross hypothyroidism can be seen from a study of a series of myxœdema of our own. Out of a total of 62 cases of myxœdema, there were 16 only who developed this condition during the probable child-bearing period. Few of these 16 were younger than 35 years of age when symptoms appeared. As fertility is known to decrease considerably in the normal person after this age, due allowance must be made for this in considering the following figures. Of these 16 cases, 10 were subsequently sterile, while eight pregnancies occurred in the other six patients. Of these eight pregnancies, however, seven only went to term, a miscarriage usually occurring in the early stages of the pregnancy. A further point of interest in these 16 cases is that menstruation remained regular after the onset of the disease in all, though five patients suffered from menorrhagia. These figures show that sterility is common and a full time pregnancy rare in the presence of gross hypothyroidism.

We must refer again to the condition of the menses in hypothyroidism. Both in the above series of myxœdematous patients and in the cases of obesity with hypothyroidism reported in this paper, menstruation was invariably regular, though the losses were often excessive. In the few obese patients who presented menstrual irregularity, the latter appeared to have been the primary condition and to have been present for many years before the onset of hypothyroidism. Vignes and Cornil,[18] who have studied this question, consider that there are two types of thyrogenic sterility, one with menorrhagia which they suggest renders it difficult for the ovum to be satisfactorily embedded, and one with amenorrhœa in which sterility is the result of atrophy of the genital tract. It is our impression that in the large majority of married patients in whom hypothyroidism and amenorrhœa are associated, the latter is the primary condition and due to other causes, while hypothyroidism is generally a sequel. The information we[19] obtained from a study of the menstrual histories of 300 cases of thyroid disease is strongly in favour of this view and we see no reason to alter it from observations on the present cases. Moreover, we think that it is owing to the continuance of regular menstruation that treatment in thyrogenic sterility is often successful, while in those associated with

amenorrhœa the results in our hands, with thyroid extract at any rate, have been almost entirely negative. We are at the moment treating a series of cases of the latter type with ovarian hormone, but it is too early yet to form any opinion as to its efficacy. The results of treatment of a group of cases of thyrogenic origin will now be considered.

Results of Treatment of Thyrogenic Obesity and Sterility.

Successfully treated cases of thyrogenic obesity and sterility have been reported from time to time, notably by Nassauer and Santa Maria y Marron (quoted by Cotte[8]) Vignes[17] and Couvelaire.[20] Among the present series of thyrogenic cases of primary sterility associated with obesity, we can report six cases in which pregnancy followed treatment with thyroid extract. Some measure of dietetic restriction was also enforced in these cases. The results of pregnancy in four of these individuals was eminently satisfactory, a full time healthy child being born, but in the other two, pregnancy was not successfully completed; in one instance a miscarriage occurred at three months and in the other the child died just before term during a Cæsarean section. The details of these six cases are given in Table VI.

The period of sterility in these patients had been considerable, varying between three and seven years. In three thyrogenic obesity developed after marriage, while in one the condition had been present from the age of 18, but in all four patients menstruation had been regular throughout. In the other two cases, a primary menstrual irregularity had been present from puberty, but symptoms of hypothyroidism did not develop until many years later. Two years was the maximum duration of treatment in any of these cases before pregnancy occurred.

In addition to the above we are also reporting successful results in 10 cases of secondary sterility, the details of which are given in Table VII. All were treated with thyroid extract in addition to dietetic measures, but only cases 1 and 5 appeared clinically to be hypothyroidic. The remainder were cases of simple obesity, mostly of the constitutional type. Although, as we have pointed out, primary sterility is rare in constitutional obesity, secondary sterility developing after one or two pregnancies does not seem to be so unusual in these women in later years.

A study of these successfully treated cases of primary and secondary sterility of thyrogenic origin lends support to the views of others that certain cases of sterility associated with obesity can be successfully treated with thyroid extract. With regard to the

TABLE VI.

Primary Sterility.

No.	Name	Age	Onset of Obesity.	Onset of Hypothyroidism	Duration of Sterility	Menstruation	Duration of treatment before Pregnancy	Result
1.	Mrs. O.	36	Marriage	Marriage	7 years	Reg. 2/28	Over 12/12	F.T.N.
2.	Mrs. H.	29	Marriage	Since 22	6 years	Reg. 5/6/52	2 years	Misc. at 3/12
3.	Mrs. L.	29	Puberty	Since 22	4 years	P.I. 3/12-4/12	12/12	F.T.N.
4.	Mrs. R.	36	Puberty	Since marriage	6 years	P.I. 3/12-4/12	?	FT died (Cæs)
5.	Mrs. L.	28	Marriage	6/12	3 year	Reg. 4/28	2/12	F.T.N.
6.	Mrs. P.	31	Since 18	Since 18.	5½ years.	Reg. 3/30.	7/12	F.T.N.

TABLE VII.

No.	Name	Age	Onset of Obesity	Previous Children	Duration of Sterility	Menstr.	Duration of treatment before Pregnancy	Result	Remarks
1.	Mrs. B.	35	Puberty	1 at 32	3 years	Reg. 4/28	4/12	FT.N.	Wt. 15/3. B.M.R.-16.2 per cent.
2.	Mrs. D.	33	Birth	1 at 29	3½ years	Reg. 5/27	3/12	FT.N.	Wt. 14/. Clinically Hypothyroid Sug. tol. +
3.	Mrs. S.	32	Puberty	M at 30	2 years	Reg. 3/26	6/12	FT.N.	Clinically thyr. deficient
4.	Mrs. R.	34	At 31	2 at 26-28	6 years	Reg.	2 yrs.	FT.N.	Hypothyroid B.M.R.-40.8 per cent
5.	Mrs. R.	40	Some yrs	3 children	Some yrs.	Reg. (Menorrhagia)	?	FT.N.	Hypothyroid B.M.R.-37 per cent
6.	Mrs. L.	34	Puberty	2 at 22-23	11 years	Reg. 5/28	3/12	FT.N.	Wt. 18/5.
7.	Mrs. B.	28	Birth	2 at 24-25	3 years	P.I. 3/12 etc	14/12	FT.N.	——
8.	Mrs. C.	37	Birth	1 at 26	11 years	Reg.	6/12	FT.N.	Fat glycosuric type. Wt. 16/4.
9.	Mrs. R.	29	Marriage	M at 26	3 years	Reg. 5/26	2 yrs.	FT.N.	Recd. corpus luteum. also.
10.	Mrs. B.	39	At 38	1 at 37	2 years	Reg. 2/26.	4/12	FT.N.	——

*FT.N. = Full Time Normal pregnancy. B.M.R. = Basal Metabolic Rate.

cases of primary sterility, successful results have been obtained only in individuals showing well-marked hypothyroid symptoms, but in those of secondary sterility there would appear to be cases of simple obesity without clearly defined hypothyroid symptoms which are remarkably tolerant to, and can be successfully treated by, the administration of thyroid extract.

Discussion.

Up to the present two divergent views have been held as to the relationship between obesity and sterility; first that of Eden and Lockyer, Child, Oliver, Crossen, Müller and Horrocks, McCann, Giles, Malcolm, Meaker and Kisch that obesity is a casual factor in sterility; and secondly, that of Graves, Cotte, Dietrich, Seitz, Winter, Wiebe and Krampf that it is not, but that sterility in these patients is due to ovarian or thyroid hypofunction. Our observations support the second view.

Our 198 cases of obesity in married women fall into three main groups: (1) Those with a family tendency to obesity, i.e. hereditary or constitutional obesity, who are either fat from birth (28 per cent) or who develop the condition in early life especially about the time of puberty (18.7 per cent). (2) Those who become fat in later life at times other than childbirth (21.6 per cent). (3) Those in whom obesity dates to childbirth (30.9 per cent).

In the first group, the hereditary form, primary sterility was rare, but when it was found it was associated with amenorrhœa. Our statistics, however, do not suggest that amenorrhœa is any more common in those who are constitutionally fat than in those of normal build. In a certain proportion of these constitutional cases in later life there was a tendency to develop secondary sterility with hypothyroidism.

In the second group of cases in which obesity developed in later life at times other than pregnancy, sterility was common. This was especially true of those patients who became obese immediately after marriage. In the majority there was amenorrhœa or mild hypothyroidism. In the latter menstruation remained regular throughout.

In the third group of cases in which obesity dated from childbirth, subsequent sterility was rare. This appears to be a form of simple obesity which is seldom associated with amenorrhœa or hypothyroidism. There is consequently no clinical support for the view that obesity developing subsequently to pregnancy is liable to be associated with absent or diminished fertility.

Conclusion.

Obesity *per se* is not a cause of sterility. When the two conditions occur together, sterility is usually the result of amenorrhœa, or occasionally of hypothyroidism. A feature of the hypothyroid cases is the maintenance of regular menstruation. The prognosis of sterility associated with obesity depends on the cause. Those cases with amenorrhœa are the most intractable, and those with hypothyroidism the most amenable to treatment. A number of successfully treated cases have been described.

In conclusion, we wish to express our thanks to Professor Hugh Maclean, the Director of the Medical Unit, St. Thomas's Hospital, to the members of the Staff of St. Thomas's Hospital and to the Medical Research Council for the facilities they have given us for carrying out this investigation.

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