Changes in Metabolism.—Patients undergoing treatment were studied before and during administration of testosterone propionate. The tests were carried out by Professor N. F. MacLagan, and are summarized as follows:

1. The rise in blood calcium reported by previous workers has not been confirmed. The danger, therefore, of hypercalcaemia and renal failure emphasized by some as a sequence of androgens cannot be confirmed. In none of my patients was the blood calcium altered.
2. There was no alteration in the alkaline phosphatase and urinary creatinine.
3. There was no alteration in the basal metabolic rate.
4. There was a significant fall in the blood urea, plasma uric acid, and inorganic phosphorus concentration.
5. The 24-hourly urea excretion showed a marked fall to a figure not much greater than half the pre-treatment level.
6. The urinary 17-ketosteroids rose to an average level of 78 mg. per 24 hours. Most of this rise was accounted for by etiocholanolone and androsterone. The 17-ketosteroids dropped to the normal of 5 to 15 mg. very rapidly on cessation of daily injections—as a rule within two days. A few patients treated by oral administration of methyl testosterone showed similar changes except that the 17-ketosteroid excretion was not affected, as these compounds are not excreted as 17-ketosteroids. In no case was oral administration of methyl testosterone nearly as effective as the intramuscular injection of testosterone propionate. These findings confirm the well-known protein anabolic effects of androgens as illustrated by the fall in the blood urea concentration and urine-urea output. It is emphasized that there is no confirmation of a rise of blood calcium as reported by previous workers.

End-results.—Androgens are not in any sense a "cure" for cancer of the breast. In many cases, but not in all, they produce marked but temporary beneficial effects. After variable periods of time, from a few months to a few years, lesions recur and previously reossified skeletal metastases decalcify and new ones develop. Prolongation of life is obtained, but not all patients respond favourably, and some after an initial improvement appear actually to be made worse by treatment. The aggravating effects of androgens can be illustrated by the following two examples.

In one patient, in whom skeletal metastases were favourably influenced for a year, further administration of androgens was followed by replacement of the entire skeleton, with exception of the hands and feet, by mere shells of bony tissue filled with metastasis. There is little doubt that acceleration as well as regression of lesions can result from androgens.

Another example is that of a patient with a large metastasis in the right lobe of the liver; complete regression after testosterone administration was followed within six months by the development of an equally large metastasis in the left lobe of the liver which did not respond to further androgen therapy. Post-mortem examination showed that the right lobe of the liver had shrunk to a mere nodule of fibrous tissue smaller than the gall-bladder; in this case disappearance of hepatic metastasis was achieved only at the price of complete destruction of the right lobe of the liver.

Oestrogens

Regression of metastasis in the skin, healing of local ulceration of the primary growth, and favourable effects on pulmonary metastasis have followed oestrogen administration. The effect on skeletal metastasis is far less pronounced and far less frequent than that following androgens. It is now accepted that oestrogens should not be given to pre-menopausal patients, in whom aggravation of the disease may result from treatment. Uterine bleeding has followed oestrogen administration even in patients of 60 and 70 years of age—that is, 10 or 20 years after the menopause. Oestrogens have not proved of much value in carcinoma of the male breast, whereas castration often has marked favourable effects.

Conclusions

There is little doubt that the late stages of cancer of the breast can be influenced by sex hormones. Although mostly

favourable, these hormones do at times cause aggravation of the disease. The mode of action of the hormones is not understood—and this to-day is the most urgent problem. Hormones, like castration, produce their effect on the host and thus differ from all other methods of treatment of cancer which aim at a direct attack on the tumour. Hormones have thus opened up completely new avenues, and at present they are more important as research weapons than as therapeutic agents.

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PRURITUS VULVAE*

BY

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Pruritus vulvae is a symptom, often of obscure origin, which is so distressing and difficult to relieve that it constitutes a real clinical problem. Moreover, it is common—how common only those who keep the most accurate records will realize. In my experience it is mentioned as a complaint, although not necessarily the primary one, by 10% of all patients comprising private gynaecological practice. The incidence in hospital practice is not less, and may be more. Its causes are so varied as to bring it within the scope of nearly all who practise medicine, although the general practitioner, the dermatologist, and the gynaecologist are mostly concerned. Each of these, however, tends to see different aspects of the problem or to approach it from a different and prejudiced standpoint. It therefore seems important to make a plea for a strict definition of the term "pruritus."

When patients complain of "irritation" of the vulva it is all too easy to assume that by this they mean "itching," whereas often they mean pain, tenderness, or a burning sensation. Unless the discomfort arouses a desire to scratch, it should not be classified as pruritus vulvae. The importance of this distinction between itching and other sensations is that their aetiology is different, and a failure to recognize this in the past has given rise to a fictitious list of causes of pruritus vulvae. It is equally important to determine accurately the site of the pruritus: is it associated with pruritus elsewhere? Is it mainly vaginal, or vulval, or perianal? Pruritus vulvae and pruritus ani often have different causes.

Pruritus seems to be a very particular type of response, and is almost specific to certain forms of stimuli or to stimuli arising under special circumstances. The fact that so little is as yet known of the mechanism of the production of this sensation and of the nervous pathways by which the impulse is conveyed to the central nervous system

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accounts in no small way for the problem presented when the vulva is the site of the trouble. Although it has been suggested (Königstein, 1936) that autonomic nervous pathways may be concerned, the more general view, expounded in recent years by Walshe (1942) and Ganado (1945), is that the impulses interpreted as itching travel by the somatic pain fibres, and that pruritus is a variant of pain. It is often regarded as a minimal pain sensation, and this, it is argued, explains why a healing wound is first painful and later, as the impulse weakens, itchy.

Clinical observation, however, makes it difficult to accept this view. So far as the vulva is concerned the causes of pruritus are dissimilar from those of pain; a minor degree of pain or soreness does not become an itch, and no matter how intense an itch or how severe the lesion causing it the sensation does not change to pain and the patient continues to scratch. Ultimately the scratch lesions become painful, but even then the patient distinguishes between this sensation and the underlying pruritus. There is other evidence, too, of a more general character. For instance, Königstein (1936) states that in some cases of organic disease of the central nervous system it is possible to induce itching in areas which are insensitive to pain. Moreover, recent work by Rosenthal and Sonnenschein (1948) on the role of histamine as a chemical mediator for the production of cutaneous pain goes to show that a "subthreshold pain" is stinging, tingling, or prickling but never itching. A sensation of itching can arise from central as well as peripheral stimuli (MacCormac, Sandifer, and Jelliffe, 1946). To read, write, or think of pruritus, or to see another person scratching, is enough to start an itch. The state of the central nervous system is probably important, for the skin is constantly exposed to minor irritations, such as the touch of rough clothing, which might well be interpreted as itching. Ordinarily they pass unnoticed by consciousness, but may not do so under circumstances such as fatigue of the higher centres (Goldsmith, 1939).

Aetiology

The secret of the successful treatment of pruritus vulvae lies in determining the cause, and for this reason I propose to deal almost entirely with aetiological considerations. The field can be narrowed at once by the exclusion of those cases in which the vulva is affected only as part of a generalized pruritus, as in conditions such as lymphadenoma, jaundice, generalized urticaria, and uraemia. Even so, according to the traditional lists many possible causes remain. These lists, however, need drastic revision, and it is hoped to show that the factors concerned in causing real pruritus can be defined fairly clearly. Diseases of the anus and rectum, such as haemorrhoids and fissures, even if they cause pruritus ani, which is doubtful, rarely if ever account for pruritus vulvae. Threadworms and fungus infection are responsible for some cases of itching around the anus, but threadworms do not give rise to vulval itching. The same is true of urinary-tract lesions—cystitis, pyuria, a highly acid or an extremely alkaline urine, and even urinary incontinence. The last-named nearly always leads to excoriation and soreness of the vulva but not to pruritus. The only exception is glycosuria, which is a common and important causal factor. Pediculosis gives rise to pruritus pubis rather than to pruritus vulvae.

In order to show the relative importance of the alleged and real causes of pruritus vulvae, 254 consecutive private cases, in all of which pruritus was given as a symptom of varying degrees of priority, have been analysed. A large proportion of these received a reasonably full investigation,

especially the obscure cases, and in each one a determined attempt was made to find the cause. In 20 cases this was unsuccessful, sometimes because the patient refused investigation. The assessment of the cause also takes account of the response to treatment and the ultimate outcome. It should be emphasized that in order to simplify the picture all women who were pregnant were excluded. Moreover, the material is to some extent selected, all the cases having been first sifted by general practitioners; so the findings, at least as regards the emphasis on individual conditions, may well be somewhat different from those of the general physician or dermatologist.

Causes of Pruritus Vulvae in 254 Consecutive Cases (Pregnant Women Excluded)

	No. of Cases
Trichomonas vaginitis (not proved bacteriologically in 34, but clinically typical)	100
Chronic cervicitis, cervical erosion, and polypi	28
Trichomonas vaginitis probably present	9
Other factors present	4
Deficiency states	20
Vitamin B ₁₂ deficiency	3
Achlorhydria	13
Deficiency anaemia (iron or liver factors)	4
Psychogenic causes	18
Skin diseases not specific to vulva—psoriasis, intertrigo, intra-epidermal carcinoma, etc.	14
Prolapse	12
"Leucoplakia vulvae"	9
Glycosuria, diabetes mellitus	8
Fungus infection of the vagina and vulva	6
Senile vaginitis	6
Idiosyncrasy to chemicals (soap, "dettol," medicinal pessaries, contraceptives)	5
Allergic states	4
Post-menopausal atrophic vulvitis	4
Unknown	20

A few points which arise from consideration of this list call for special comment.

1. Vaginal Discharge

Contrary to the usual statements, discharges rarely make the vulva itch unless the infecting organism is either *Trichomonas vaginalis* or one of the fungus group, usually *Oidium albicans*. Gonorrhoea and pyogenic infection of the vagina or vulva cause pain and tenderness rather than pruritus. Trichomonas infection is the leading cause of pruritus vulvae, especially in the child-bearing period. It can occur after the menopause, but is then less common. The pruritus is felt within the introitus as well as outside, and is usually worse post-menstrually. It may be out of all proportion to the amount of the discharge, which may be so slight that the patient does not mention or notice it. Trichomonas infection can persist in a mild chronic form for years, and that despite intensive treatment. Apart from thrush, pruritus vulvae is occasionally the result of other forms of fungus infection of the vagina and vulva. This may be difficult to demonstrate, and sometimes a clue to the diagnosis is obtained only by finding evidence of epidermophytosis of the hands and feet. If pregnant women had been included in the analysis the number of cases of monilia infection would undoubtedly have been larger.

Chronic cervicitis, erosion, and cervical polypi are given as the significant finding in 28 cases, but on review a causal relationship to the pruritus is doubtful in most of them. In nine, for instance, the pruritus was almost certainly due to an associated unproved trichomonas or fungus vaginitis, and in at least six there is no evidence that the pruritus was better after radical treatment of the cervix. Indeed, considering how commonly cervical conditions cause discharge and how rarely they are associated with pruritus, it is open to question whether a discharge which is a true

leucorrhoea, or which is the result of cervical infection, ever causes pruritus vulvae.

2. Glycosuria

The fact that eight of this small series of previously selected cases proved to have glycosuria is significant. The diagnosis of diabetes may easily be missed if at the time of examination the patient has not eaten for several hours and the urine is temporarily free from sugar. If there is any doubt about the cause of pruritus the only safe procedure is a glucose-tolerance test. In glycosuria without diabetes the pruritus is nearly always the result of *Oidium albicans* infection, which thrives in the presence of a free supply of carbohydrate. The same is true of diabetes mellitus, but there is the added possibility of pruritus being the result of peripheral neuritis or of chemical changes in the skin. The vulva in these cases presents a typical appearance, the tissues being slightly swollen and oedematous with a characteristic dusky blue or purple coloration.

3. Psychogenic Causes

A close relationship between the mind and the skin and the part played by psychological upsets in causing skin diseases are recognized by all. Many psychiatrists emphasize the sexual basis of mental disturbances, so it is little wonder that the skin of the vulva can become the site for manifestations of psychoneuroses. Some hold that nearly all cases of pruritus vulvae have a psychogenic basis, but there is a danger of the importance of this factor being exaggerated. In general, mental and emotional upsets should be accepted as causing pruritus vulvae only when every other possibility has been excluded. When they are merely contributory factors, as they often are, they usually cease to operate when other causes are removed. Among the common psychogenic causes may be mentioned guilt over masturbation, cancerophobia, fear of venereal disease, marital disharmony with an underlying desire to avoid coitus, or a scratch habit.

4. Prolapse

Prolapse either of the uterus or of the vagina seems to be a rare but none the less definite cause of pruritus. Considering how common is prolapse, however, and how infrequently pruritus is a symptom, it is natural to doubt the association, yet a few cases seem to be cured completely by a repair operation. However, the possibility that the cure is due to something incidental to the operation, such as sterilization of the vagina, rest in bed, or a psychological effect, cannot be excluded.

5. Allergy and Drug Sensitivity

Skin sensitivity to various chemical constituents of toilet preparations occasionally cause pruritus vulvae. Sometimes acetarsol, silver picrate, and other drugs used in the treatment of trichomonas vaginitis are the cause of pruritus, or lead to its continuance after the original causal infection is under control.

Apart from urticaria associated with the same condition elsewhere it is doubtful how far allergy is responsible for pruritus vulvae. The possibility has been much discussed and investigated in recent years, and is the basis of empirical treatment with ergotamine and antihistamine drugs. Allergic reaction to the patient's own sex hormones has been blamed. Skin-sensitivity tests either with endocrine preparations or with other possible antigens are rarely helpful, however, and in my experience antihistamine drugs generally give unsatisfactory results.

6. Leucoplakia Vulvae

Our knowledge of the pathogenesis of leucoplakia vulvae and kraurosis vulvae is as confused and insecure as it was forty years ago, when Berkeley and Bonney (1909) attempted to define them. There is a renewed tendency to return to the older view that kraurosis is the ultimate atrophic phase of leucoplakia. If kraurosis is to be regarded as a condition of extreme atrophy affecting the introitus only and often associated with senile vaginitis, this can hardly be correct. Some writers even distinguish between leucoplakia and leucoplakic vulvitis, and Hunt (1943) regards leucoplakia as a form of lichen planus. The variation in views, few of them supported by convincing evidence, is borne out by Savill (1940) and by Miller and others (1947), together with the discussion which followed this latter paper.

In my experience kraurosis vulvae as originally described is very rare and does not cause pruritus but rather pain and dyspareunia; this feature, which was emphasized long ago by Berkeley and Bonney (1909), tends to be overlooked. Nevertheless senile vaginitis, possibly associated with vulval atrophy, is occasionally associated with pruritus, which responds to oestrogen therapy. Leucoplakia, at least in its hypertrophic phase, is not difficult to recognize. It affects the outer aspects of the labia and may spread to the perineum and around the anus, and is undoubtedly precancerous. It occurs at any age and does not bear any relation to ovarian function, so it is not improved by oestrogen therapy. There is good reason to believe that it may not be a specific entity but merely one type of skin reaction to many possible stimuli: prolonged scratching alone can produce somewhat similar skin changes. It is sometimes the result of vitamin A deficiency, and is also seen occasionally in various types of anaemia and in diabetes. One of the most typical cases of "leucoplakia" I have ever seen was in a doctor's wife who suffered from vitamin B deficiency as a result of a diet which consisted almost entirely of a few water-biscuits a day. The administration of riboflavin quickly restored the vulval skin to normal. The nine cases of leucoplakia shown in the list were "idiopathic" in the sense that an underlying cause was not demonstrated; there were five other cases in which the leucoplakic skin changes were considered secondary to other causes, and they appear under other headings.

One of the most obscure types of pruritus is that in which the irritation is localized to a very discrete area of thickened pale skin usually situated on one labium majus. It may be mistaken for leucoplakia, although hyperkeratinization and cracking are not pronounced features. It responds badly to treatment—even to excision—and is probably a form of localized neurodermatitis with lichenification.

7. Deficiency States

One of the greatest advances of recent years is the recognition that various deficiency states can cause pruritus vulvae, and the development and details of this knowledge are set out in papers by Swift (1932, 1936), Wright (1944), Stannus (1944), Hughes (1946), and Whitfield (1947). These authors are concerned mainly with the effect of vitamin A and various members of the vitamin B₂ complex. Other agents may operate, however, and among these may be mentioned iron, liver factors, and possibly other vitamins. Pruritus vulvae is therefore occasionally a manifestation of hypochromic or hyperchromic anaemia; it then responds quite dramatically to the specific treatment of these conditions. The lesions produced in the vulva vary from slight

reddening or pallor and oedema to typical leucoplakia. As might be expected, other evidences of deficiency states, such as glossitis, cheilosis, and angular stomatitis, are often found in these cases. It is of interest to note that the scrotum, which is homologous with the labia majora, is also the seat of "dermatitis" in ariboflavinosis (Frankland, 1948).

If obvious local causes such as trichomonas infection are excluded, deficiency states can be shown to be responsible for approximately 20% of the cases of pruritus vulvae. In this country they rarely arise as a result of gross dietetic errors, but such should be looked for, especially in elderly women living alone. More often they are due to some disturbance of the alimentary tract which interferes with biosynthesis or with absorption. The most important factor in this respect, and one whose significance was first pointed out by Swift (1932, 1936), is achlorhydria, which is presumed to act by interfering with the absorption of vitamin A and iron.

Achlorhydria

During the last ten years the investigation of cases of obscure and intractable pruritus seen in private or hospital practice has included a fractional test meal, and the details of 46 women (including the 13 mentioned in the private series) with histamine-fast achlorhydria are available. The mere finding of achlorhydria, which is common in women of advancing years, does not of course prove that it was the cause of pruritus, but the response to treatment suggests that in the majority it was. In four of the cases the skin lesion was typical leucoplakia; in others there was pallor or reddening of the labia majora with secondary changes due to scratching. Although the vulva was the main site of the pruritus the perianal skin was also involved in some cases. A significant degree of hypochromic anaemia was often present. All these patients were given acid. hydrochlor. dil. by mouth, and iron preparations when necessary, but no other general or local treatment, and the immediate results were as follows: cured, 34; slight relief, 1; unknown, 4; no response, 7.

Leucoplakia was present in two of the seven failures, and excision was required; in another case monilia infection explained the itching. It seems reasonable to conclude that in some of these seven and in the one with only partial relief the achlorhydria was probably merely coincidental, because the usual response to treatment is striking, even dramatic. Women who have had intolerable itching for years get complete relief within one to two weeks. One constant feature, however, is that an associated pruritus ani is not relieved to the same extent.

In order to determine the ultimate effect all of the 34 "cured" cases have been followed up. The findings are as follows:

Permanent cure for 1 to 8 years	25 cases
Died from intercurrent disease	1 case
Not traced	2 cases
Relapsed and did not respond to further treatment	6 cases

Some of those permanently cured report that they need to continue taking hydrochloric acid, otherwise the itching returns at once; this is what one might expect. Others, however, after regular treatment for a few months have been able to discontinue taking the acid for long intervals—even years—without a return of pruritus. This is an extraordinary phenomenon which is not explained by present knowledge but is confirmed by Hyams and Bloom (1947). Also interesting are the six women who were initially cured but did not get relief from the same treatment when symptoms recurred. One of these was ultimately

cured by an operation for prolapse; another can be kept comfortable only by periodic blood transfusions. In her case hypochromic anaemia persists or recurs despite the administration of hydrochloric acid and iron.

Opinions differ about the relative importance of achlorhydria as a cause of pruritus vulvae. Swift (1936) originally stated that it is responsible for *all* cases of leucoplakia and kraurosis; Haultain (1948) says that it is present in 30% of cases of leucoplakia; while Hyams and Bloom (1947) put the figure at 60%. Miller *et al.* (1947), however, deny it any significant place in the aetiology of leucoplakia vulvae. In my experience it accounts for approximately 20% of cases of "pruritus" for which there is no obvious local cause, and is present in about one-third of women who have what I regard as real leucoplakic changes in the vulval skin. In these the vulval lesion is not always reversed by the administration of hydrochloric acid. Moreover, and despite the statement of Hyams and Bloom (1947) to the contrary, hypochlorhydria is rarely, if ever a significant finding in pruritus vulvae, and the acid deficiency must be complete if it is to operate. The fact that leucoplakia in association with achlorhydria is often not cured by administering acid raises the question whether it represents a skin reaction which has progressed so far as to be irreversible. Such a concept is in keeping with the observations of Stannus (1944) to the effect that in ariboflavinosis the essential pathology is capillary damage, and that the vascular disturbance may lead ultimately to permanent skin changes.

Even when dietetic errors and alimentary upsets are excluded there still remains the possibility of a local tissue deficiency to account for some cases of pruritus vulvae. This might occur, for instance, if the blood supply is inadequate; and the question then arises whether surface ischaemia in leucoplakia is the cause or the result of poor nutrition to that area. Deficiency states, vascular disturbances, and pruritus have some close association. A vascular upset is often the basis for itching, as, for example, in urticaria and in varicose veins of the legs, the latter being accompanied by faulty nutrition to the skin. It is tempting to suggest that venous stasis is the significant lesion when pruritus vulvae seems to be caused by prolapse. The view that some local circulatory factor may cause pruritus vulvae is also supported by the fact that when the vulva is excised for leucoplakia healthy skin brought from other sites to cover the area often undergoes leucoplakic changes.

Even when the supply of essential factors is normal the local tissues may be unable to utilize them, and in this connexion it is interesting to note that it has been suggested that diabetes mellitus can produce pruritus by interfering with the utilization of vitamins by the skin or by promoting their rapid excretion. Carrying the argument further, it may be pointed out that when senile states cause pruritus vulvae the nutritive factor which is absent is oestrogen.

The only types of pruritus which are difficult to account for on the basis of a general or local nutritional deficiency are those due to trichomonas and fungus infection. It may be postulated, however, that, since these infections are so specific in their pruritus-producing character, perhaps the organisms concerned have a special metabolism which deprives the host tissues of some vital factor. They or their metabolic products may act as antivitamin or toxamins; this suggestion is purely hypothetical, but its validity deserves serious inquiry.

One of the difficulties in accepting the concept of poor nutrition of the tissues as the basis of pruritus vulvae is

that, with the exception of those cases which show a positive therapeutic test, there is little concrete evidence to support it. In some of our cases Professor R. A. Morton kindly carried out estimations of the amount of carotene and vitamin A in the plasma, and in none was any significant decrease demonstrated. This agrees with the findings of Porter and Godding (1945) in relation to various skin disorders, and with those of Miller and others (1947) and Hyams and Bloom (1947) in respect of leucoplakia vulvae. The last-named workers were also unable to demonstrate any biochemical evidence of vitamin C and vitamin B₂ deficiency. The negative findings in regard to vitamin A, however, are not necessarily conclusive, because it is now known that the plasma content of this vitamin remains normal for very long periods after it has been completely withdrawn from the diet (Glover, Goodwin, and Morton, 1947) and is not easily altered by administering vitamin A. Moreover, local deficiency states, if present, cannot be demonstrated by examination of the blood and urine.

Management and Treatment

From the foregoing it is clear that the most important part of the management of pruritus vulvae is a thorough investigation to find the cause. This necessitates taking a very careful history and making a full physical examination, and the minimum requirements as regards special tests are: (a) repeated examination of fresh vaginal swabs for trichomonas and *Oidium albicans* and other fungi; (b) fractional test meal; (c) full examination of the blood; (d) glucose-tolerance test; and (e) biopsy of vulval skin in selected cases. Unless a cause can be found treatment is mostly unsatisfactory.

Antipruritic ointments are of little use except as temporary measures and perhaps to break the scratch habit. The injection of local analgesics gives disappointing results, and the division of all cutaneous nerves is usually short-lived in its effect. The place of radiotherapy in the treatment of pruritus vulvae is very strictly limited and should be used only as a last resort and after very careful consideration. Although it may give relief by a mechanism unknown, the pruritus, if caused by a persisting factor, usually returns. Moreover, x rays and radium are apt to produce permanent skin damage, which can itself be the cause of intractable pruritus. Oestrogen therapy is useless except when there is clear evidence of failure of ovarian function, and it may even be dangerous in precancerous lesions such as leucoplakia.

Leucoplakia appears to be helped sometimes by emollients (including cod-liver oil) and also by the local application of heat as suggested by Savill (1940), the idea being to improve blood supply. Often, however, nothing short of vulvectomy is of real value, and this should not be deferred too long, because of the possibility of malignant disease. Excision is of limited value except for clearly demarcated lesions such as leucoplakia, and even then there is a tendency for the condition to recur in the surrounding areas. Unless there is evidence of achlorhydria, gross dietetic errors, or other lesions characteristic of a deficiency state, the empirical administration of vitamin preparations even in massive doses is rarely helpful, despite a few enthusiastic reports to the contrary. Moreover, if, as postulated above, any deficiency state in these cases is of local origin it is unreasonable to expect a response from such medication.

Throughout this paper the emphasis has been on the obscure and difficult cases of pruritus vulvae, and the same is true in regard to treatment. Lest it convey too

gloomy a view of the general picture it is perhaps fitting to conclude by emphasizing that even in the present state of knowledge a definite organic cause amenable to treatment can be found in not fewer than four out of every five women who complain of pruritus vulvae, and in not more than 5% is it impossible to give real relief by one means or another.

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RELIEF OF POST-OPERATIVE PAIN AND ITS INFLUENCE ON VITAL CAPACITY

BY

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It has been recognized for many years that post-operative pulmonary complications occur much more frequently after surgical operations involving the upper abdomen than after those on other parts of the body (King, 1933; Campbell and Gordon, 1942). It is indeed significant that we are now more likely to hear the expression "post-operative chest" than the more old-fashioned "post-anaesthetic pneumonia."

In Table I is shown the incidence of post-operative pulmonary complications in a series of 5,869 anaesthetics. These were all personally administered and examined post-operatively by me, thus introducing a reasonably constant factor and ensuring that approximately the same minimum of abnormal physical signs in a chest qualified it for the label "pulmonary complications."

TABLE I.—Post-operative Pulmonary Complications

Site of Operation	No. of Cases	Pulmonary Complications
Upper abdomen ..	331	63 (19%)
Lower abdomen ..	1,334	145 (10.9%)
Outside abdomen*	4,204	29 (0.7%)
Total ..	5,869	

* Does not include thoracic surgery.

Why this high incidence of pulmonary complications following abdominal operations and especially those on the upper abdomen? Langton Hewer (1948) lists several main causes of post-operative lung complications, including "impaired respiratory movements after operation and