conditions which we believe to be influential in curing psoriasis. It is useful for the patient to receive a first course of treatment in a properly equipped hospital department so that the physician has more opportunity of observing and knowing his patient, and the patient is more efficiently trained in treatment.

Some of this treatment can be continued as maintenance and prophylactic therapy after psoriasis has cleared. Thus the daily bath with tar soap—even a tar bath occasionally—and regular care of the scalp, are desirable. The use of a pomade with mercury, salicylic acid, and tar on the scalp may be adopted as a routine toilet measure. A daily exposure to ultra-violet light is still more profitable, especially when taken after a bath or a tar bath. For this purpose the powerful hospital pattern of lamp is desirable, so that the treatment may take no more than one or two minutes, become part of the toilet routine, and not be a burden.

It is not suggested that this is the only local treatment for psoriasis, but in this climate it is rapidly effective in all but the occasional patient.

A Hospital Service for Psoriatic Patients

It is not unreasonable to suggest that a national health service should, under present circumstances, provide a service of treatment which should be made available for the patient for purposes of clearing attacks of psoriasis, if not for prophylaxis. There is a large psoriatic population in this country, numbering perhaps half a million, and the disability and discomfort involved must be considerable. Such a service should be the responsibility of regional hospital boards, who should provide, under the direction of their dermatologists, facilities for such treatment. It means the provision of baths and sunlight lamps, of nurses or orderlies trained to conduct treatment, and effective supervision; it means, further, a permanent staff, for the untrained nurse will not produce results.

Such a service would not only relieve a large number of patients of an intolerable burden, but would place under the constant eye of the dermatologists in the service a mass of clinical material—a circumstance that must favour an advance in our knowledge and encourage research. That fundamental research is necessary and essential to advancement in this field is not to be questioned. It is suggested that the expenditure of money on such services as have been outlined would be profitable to the community and is the proper function of a health service. It cannot be doubted that such a scheme in practice would inevitably stimulate useful research, both clinical and fundamental, and would provide the opportunities likely to make it purposeful and successful.

Summary

It is suggested that psoriasis is a constitutional disorder in which the fundamental maladjustment relates to climate.

There is a tendency for the disease to be inherited and so to be associated with some racial predisposition.

Though climatic influences are primary and fundamental, secondary influences, such as the infective, endocrine, and nervous, may assume major importance.

The significance of the epidermal character of the reaction is stressed, and fundamental research into the mechanism of the reaction is necessary.

Treatment can be rapidly effective in clearing the eruption, and a hospital service should be provided for the treatment of psoriasis under the health scheme.

Such a scheme, in addition to providing a necessary service to the community, would favour the pursuit of purposeful research, both clinical and fundamental.

REFERENCES

- Astbury, W. T. (1950). *Brit. J. Derm. Syph.*, **62**, 1.
Gans, O. (1952). *Arch. Derm. Syph.*, **Chicago**, **66**, 598.
Romanus, T. (1945). *Acta derm-venereol.*, *Stockh.*, **26**, Suppl. 12, 138.

URINARY STEROID EXCRETION IN RHEUMATOID ARTHRITIS

CHANGES IN KETONIC AND NON-KETONIC FRACTIONS DURING HORMONE THERAPY

BY

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In 1949 Hensch, Kendall, Slocumb, and Polley reported that cortisone (17-hydroxy-11-dehydrocorticosterone) or A.C.T.H. (adrenocorticotrophic hormone) would alleviate the symptoms of rheumatoid arthritis and of acute rheumatic fever. This observation stimulated inquiries into the role of the adrenal cortex and its hormones in normal metabolism and in relation to the aetiology of rheumatic conditions. Many workers have shown that A.C.T.H. is capable of stimulating the adrenal cortex to increased secretion, and that prolonged administration of A.C.T.H. or cortisone to man results in the development of the symptoms of Cushing's syndrome associated with increased excretion of steroids.

The present paper gives further details of the excretion of specific 17-ketosteroids following cortisone administration (Copeman *et al.*, 1952) and of work extended to include the administration of A.C.T.H. A new method of analysing the "non-ketonic" fraction has revealed changes following hormone therapy not previously reported.

Material

Urine samples (24-hour) were collected from normal persons (10 males and 10 pre-menopausal females) and from patients suffering from rheumatoid arthritis. The urine was preserved with chloroform, and, in the case of patients, collection was begun 12 days before treatment and continued throughout therapy. The urine was hydrolysed by boiling for 15 minutes with 3.3% hydrochloric acid and extracted with benzene. The neutral extract prepared by washing the benzene solution with 2N sodium hydroxide and water was fractionated by Girard T reagent into ketonic and non-ketonic steroids. Each of these fractions was divided into alcohols and non-alcohols (Dobriner, Lieberman, and Rhoads, 1948) as shown in Fig. 1.

FIG. 1.—Scheme of separation of neutral steroid extracts.

The non-ketonic non-alcoholic fraction has not been shown to contain steroids and has not been further treated. The remaining fractions, two ketonic and two non-ketonic (3α and 3β alcohols), were analysed by chromatographic methods. The ketonic fraction was followed by a modification of the Dingemans technique (Dingemans, Huis in't Veld, and de Laat, 1946); the non-ketonic fraction by conversion of the component alcohols to 3:5 dinitrobenzoates (Kellie, Smith, and Wade, 1953; Kellie and Wade, 1953).

Comparison of Steroid Excretion in Normals and Rheumatoid Arthritics

Comparison of the steroid excretion of normal and arthritic individuals is possible both in respect of the composition of the neutral fraction and in that of the resulting subfractions. Table I shows the results of fractionating

composition. The customary artifacts 3- β -chloro- Δ^5 -androstene-17-one and Δ^2 or Δ^3 -androstene-17-one were present in reduced amounts, as reflected in the total fraction.

Non-ketonic (3α) Alcohols.—Based upon the results of the chromatographic analysis of the alcohol esters, Fig. 2 shows a comparison of the output of non-ketonic alcohols in rheumatoid arthritics as compared with that of normal persons. Work on the identity of the component alcohols of this fraction has not progressed sufficiently to be able to refer to them by name, but only by class. Urinary non-ketonic alcohols, following esterification with 3:5-dinitrobenzoyl chloride, separate readily on alumina into a series of fractions, here referred to by numbers I, II, III, etc., corresponding to increasing polarity of the eluents. Tentatively it is thought that fraction I consists of monohydroxy alcohols; fraction II of dihydroxy alcohols—for example, pregnanediol; fraction III of trihydroxy alcohols. The identity of the late fractions is still a matter of conjecture, but even in the absence of precise information it is possible

TABLE I.—Fractionation of Urine Extracts—Normal and Rheumatoid Arthritic

Fraction	Weight (mg./24 Hours)					Colorimetric Assay (mg./24 Hours)				
	Normal Subjects		Rheumatoid Arthritic Cases			Normal Subjects		Rheumatoid Arthritic Cases		
	(M)	(F)	1 (F)	2 (M)	3 (F)	(M)	(F)	1 (F)	2 (M)	3 (F)
Total neutral	66.4	56.8	16.0	36.4	28.8	13.5	10.6	4.2	4.3	4.0*
Ketonic	27.6	24.5	5.1	9.9	5.7	12.3	10.1	2.3	2.8	2.4*
Ketonic alcohols	13.5	10.0	3.1	4.0	4.4	8.8	6.3	1.6	1.9	1.5*
Ketonic non-alcohol	8.7	9.2	1.5	5.5	1.5	2.8	2.5	0.4	0.6	0.6*
Non-ketonic	25.5	24.4	7.5	12.4	10.5	—	—	—	—	—
Non-ketonic alcohol	8.6	6.6	3.2	8.8	7.4	18.8	16.0	8.4	13.1	9.9†

* Expressed as mg. equivalents of dehydroisoandrosterone. † Expressed as mg. equivalents of cholesterol.

the urinary extracts of three cases of rheumatoid arthritis. The reduced excretion of both ketonic and non-ketonic material is revealed by comparison with the corresponding values for normal subjects.

Ketonic Alcohols.—Based upon the chromatographic analysis of the ketonic alcoholic fraction, Fig. 2 shows in graphical form a comparison of the excretion of individual

FIG. 2.—Excretion of steroids by normal persons and rheumatoid arthritics. 1, monols; II, diols; III, triols, etc. 1, " β " compounds; 2, androsterone; 3, aetiocholanolone; 4, 11-oxygenated steroids.

steroids in normals and untreated arthritic persons. In each of several cases of rheumatoid arthritis analysed, the percentage of ketonic alcohols excreted as androsterone was much lower than in normal subjects (average values: arthritics, 18.7%; normal subjects, 30%), whilst the percentage excreted as aetiocholanolone was generally higher (arthritics, 46%; normal subjects, 38%). As a consequence the aetiocholanolone/androsterone ratio was higher in rheumatoid arthritis. The significance of this finding, if any, is not known. Robinson and Goulden (1949) observed a similar trend in cases of prostatic cancer.

Ketonic Non-alcohols.—The analysis of this fraction in several rheumatoid cases showed no departure from normal

to see that most fractions seem to be involved in the reduced excretion. The dihydroxy-alcohol fraction, normally the largest fraction, is substantially reduced. The amount of non-ketonic (3β) alcohols excreted in normal and rheumatoid arthritic individuals was negligible.

Comparison of Excretion in Rheumatoid Arthritis Before and During A.C.T.H.

The subject, a female rheumatoid patient aged 49, received 560 mg. of A.C.T.H. over a period of fourteen days. The starting dose of 60 mg. on each of four days was progressively decreased until for the last four days a maintenance dose of 10 mg. was being given. This treatment caused a disappearance of clinical symptoms, and the 17-ketosteroid excretion rose from an average of 4.2 mg. a day to 10.8 mg. a day during treatment. The increase was maintained until the daily dose fell to 30 mg.; below this figure there was a steady decline in output. Table II gives the daily excretion of ketonic and non-ketonic material during treatment.

TABLE II.—Fractionation of Urine Extracts Before and During A.C.T.H. (Female, 49 Years)

Fraction	Weight (mg./24 Hours)		Colorimetric Assay (mg./24 Hours)	
	Before A.C.T.H.	During A.C.T.H.	Before A.C.T.H.	During A.C.T.H.
Total neutral	16.0	45.0	4.2	10.8*
Ketonic	5.1	21.6	2.3	10.3*
Ketonic alcohols	3.1	9.5	1.6	5.6*
Ketonic non-alcohols	1.5	4.3	0.4	1.2*
Non-ketonic	7.5	18.6	—	—
Non-ketonic alcohols	3.2	10.1	8.4	16.7†

* Expressed as mg. equivalents of dehydroisoandrosterone.

† Expressed as mg. equivalents of cholesterol.

The substantial increase in weight of material excreted daily following the administration of A.C.T.H. is reflected in all fractions. The increase in the ketonic fraction which has already been reported by many workers is shown to

FIG. 3.—Excretion of steroids before and during A.C.T.H. I, monols; II, diols; III, triols, etc. 1, "β" compounds; 2, androsterone; 3, aetiocholanolone; 4, 11-oxygenated steroids.

aetiocholanolone. The aetiocholanolone/androsterone ratio was fairly constant—2.6:1 before and 2.0:1 during treatment—and did not restore the excretion pattern to normal. On the dose levels quoted only a very slight increase occurred in the excretion of 11-oxygenated steroids.

Non-ketonic Alcohols.—This fraction increased in weight about threefold during treatment. Fig. 3 shows the change in composition. Here again no information is yet available regarding the particular compounds responsible in fractions II, III, and V for the increase during treatment.

Comparison of Excretion in Rheumatoid Arthritis Before and During Cortisone

The patient, a woman aged 56, had a low initial excretion of 17-ketosteroids averaging 4 mg. a day over 11 pre-treatment days. Cortisone was given for a prolonged period, but urine collection was limited to the first 18 consecutive days of treatment, and during this period a total of 2.5 g. of cortisone was administered. The 17-ketosteroid excretion increased within 24 hours of starting treatment, but fluctuated greatly from day to day with no apparent relationship to the dose level. The average over a period of treatment was higher than pre-treatment level—5.7 mg. a day.

Fractionation of pre-treatment and treatment material showed that the small increase in total 17-ketosteroids was reflected in the ketonic alcohols. The weight of non-ketonic alcoholic material fell slightly (Table III).

TABLE III.—Fractionation of Urine Extracts Before and During Cortisone (Female, 56 Years)

Fraction	Weight (mg./24 Hours)		Colorimetric Assay (mg./24 Hours)	
	Before Cortisone	During Cortisone	Before Cortisone	During Cortisone
Total neutral	28.8	28.4	4.0	5.7*
Ketonic	5.7	8.0	2.4	4.0*
Ketonic alcohols	4.4	4.7	1.5	2.9*
Ketonic non-alcohols	1.5	2.6	0.6	0.7*
Non-ketonic	10.5	13.0	—	—
Non-ketonic alcohols	7.4	5.5	10.0	8.1†

* Expressed as mg. equivalents of dehydroisoandrosterone.

† Expressed as mg. equivalents of cholesterol.

Ketonic Alcohols.—Separation showed a slight fall in androsterone excretion, more than offset by a simultaneous rise in aetiocholanolone (Fig. 4). 11-Oxygenated steroids showed a noticeable increase, and from this fraction two

affect both ketonic alcohols and, to a less extent, ketonic non-alcohols. The increase in the non-ketonic alcohols is also dramatic, but has not previously been reported.

Ketonic Alcohols.

—Chromatography of this fraction following the administration of A.C.T.H. revealed no compounds not already present. The daily excretion figures shown graphically in Fig. 3, however, reveal that the increased excretion of total 17-ketosteroids is largely due to increased output of androsterone and

crystalline compounds were isolated and identified by infra-red spectroscopy. Of these two compounds, 11-keto-aetiocholanolone and 11-hydroxy-aetiocholanolone, only the former is a component of normal urine. Both could arise as endogenous compounds or as metabolic products of administered cortisone, and it is not possible to decide between these alternatives in the present instance.

Non-ketonic Alcohols.—Although the total non-ketonic alcohols

fell slightly during treatment, separation of the components (Fig. 4) showed that this was associated with a very small rise in fraction II (dihydroxy alcohols), offset by slight falls in most other fractions.

Comparison of A.C.T.H. and Cortisone Treatment in the Same Subject

The subject, a male patient aged 39 with rheumatoid arthritis, was studied by collecting urine samples throughout the following course of treatment:

- (a) Pre-treatment 11 days
- (b) A.C.T.H. 6 days, 100 mg. a day
- (c) A.C.T.H. 7 days, 300 mg. a day
- (d) Rest period 41 days
- (e) Cortisone 18 days, 100 mg. a day
- (f) Cortisone and methyl-testosterone 11 days, 100 mg. a day
- 25 mg. a day

The details of the 17-ketosteroid levels throughout treatment are summarized in Fig. 5. The first six days of A.C.T.H. treatment (100 mg. a day) led to no increase in 17-ketosteroid excretion, neither was there any significant clinical response. The dose was accordingly increased to 300 mg. a day, and was followed immediately by a rise in 17-ketosteroid output. The clinical response was such

FIG. 5.—Total 17-ketosteroid excretion before and during A.C.T.H.

that after seven days it was necessary to stop treatment as the patient showed marked diabetic symptoms. After a prolonged rest period the administration of cortisone caused a rise in 17-ketosteroid output. The fractionation of the material (excluding the first A.C.T.H. treatment) is shown in Table IV, and the excretion patterns of the ketonic alcohols and non-ketonic alcohols are illustrated in Fig. 6.

TABLE IV.—*Fractionation of Urine Extracts—Treatment with A.C.T.H. and Cortisone (Male, 39 Years)*

Fraction	Colorimetric Assay (mg./24 Hours)				
	Pre-treatment	A.C.T.H. (300 mg./24 Hr.)	Rest Period	Cortisone	Cortisone and Methyltestosterone
Total neutral ..	4.3	11.2	4.6	7.4	7.2*
Ketonic ..	2.8	8.8	3.8	6.8	6.0*
Ketonic alcoholic ..	1.9	6.1	2.4	4.9	5.3*
Ketonic non-alcoholic ..	0.6	1.3	0.7	1.3	1.1*
Non-ketonic alcoholic ..	13.1	24.9	10.4	10.0	18.5†

* Expressed as mg. equivalents of dehydroisoandrosterone.

† Expressed as mg. equivalents of cholesterol.

FIG. 6.—Excretion of steroids during treatment with A.C.T.H. and cortisone. I, monols; II, diols; III, triols, etc. 1, "β" compounds; 2, androsterone; 3, aetiocholanolone; 4, 11-oxygenated steroids.

As in the previous case, whilst A.C.T.H. produced an increase in both ketonic and non-ketonic fractions, the administration of cortisone caused a rise in the former fraction but little change in non-ketonic alcohols. Although the amount of aetiocholanolone changed during each period, expressed as a percentage, it remained remarkably constant. Addition of methyltestosterone to the administered cortisone produced no marked change in the ketonic fraction but almost doubled the weight of non-ketonic alcohols.

Treatment with A.C.T.H., although at a high dosage level, produced very little increase in androsterone, in contrast to the previous case, whilst the increase in aetiocholanolone and in the 11-oxygenated steroids was quite marked. The pronounced changes in the non-ketonic pattern are most apparent in fractions II, III, and V. During the rest period between the two sets of treatment the excretion patterns returned substantially to normal.

Cortisone treatment also caused very little increase in androsterone. The rise in aetiocholanolone was less and that of 11-oxygenated steroids greater than during A.C.T.H. therapy. The non-ketonic alcohol pattern was unaltered during treatment with cortisone alone, but changed considerably when methyltestosterone was also given.

Discussion

Comparison of the steroid excretion of rheumatoid arthritis patients with that of normal individuals has shown that the excretion of 17-ketonic alcohols and of non-ketonic alcohols in this condition is low. The excretion of

androsterone, expressed as a percentage of total ketonic alcohols, was lower than in normals, whilst the percentage excreted as aetiocholanolone was generally higher. As a consequence the aetiocholanolone/androsterone ratio was higher in rheumatoid arthritis than in normal persons. This change has already been observed in at least one other pathological condition, and may have no specific bearing on steroid metabolism in rheumatoid arthritis.

Dobriner (1951a) reported the presence of 17-hydroxy-pregnanolone in the urine of four out of five cases of rheumatoid arthritis, although this compound is apparently absent from normal urine. It is not a 17-ketosteroid, and would not be revealed by the method used above unless present in excessive amounts, as, in the Zimmermann reaction, it gives approximately one-sixth of the colour intensity of a typical 17-ketone.

In the non-ketonic alcohols, fraction II, tentatively identified as the "dihydroxy alcohols," was decreased.

The changes in ketonic alcohols following A.C.T.H. and cortisone treatment have already been reported by others. A.C.T.H. caused a small increase in androsterone and 11-oxygenated steroids together with a substantial rise in aetiocholanolone excretion. After the administration of cortisone there was a greater increase in 11-oxygenated steroids and a substantial rise in aetiocholanolone. The latter finding is contrary to that of other workers, who found no increase in the excretion of androsterone and aetiocholanolone. Although Dobriner (1951b) has stated that the 11-oxygenated steroids do not contribute to the excretion of 11-deoxysteroids, the additional aetiocholanolone in these two cases must have come either from the administered cortisone or from some endogenous source.

Corresponding to the changes in the ketonic alcohols the output of non-ketonic alcohols increased greatly after A.C.T.H. administration. The main increase was observed in the "dihydroxy alcohol" fraction, but simultaneous increases were observed in other, as yet unidentified, fractions. In this connexion it is interesting to note that Nathanson, Engel, and Kelley (1951), following A.C.T.H. administration to a patient with multiple myeloma, observed an increase in total steroid alcohols. Paschkis (1951) also reported an increase in pregnanediol output following the administration of A.C.T.H. to men.

The administration of cortisone to rheumatoid arthritis patients had very little, if any, effect on the output of non-ketonic alcohols. In one case of Addison's disease treated intramuscularly with cortisone, Engel (1950) observed an increase in total and "non-ketonic" alcohols. Figures for the latter were obtained as a difference between total steroid alcohols and 17-ketosteroid values, and are not synonymous with non-ketonic alcohols as determined above, as they include ketonic steroids other than those substituted in the 17 position.

The addition of methyltestosterone (25 mg. daily for 11 days) to the administered cortisone, while having little effect on the ketonic steroids, greatly changed the excretion pattern of non-ketonic steroids. As treatment with cortisone for 18 days had failed to affect this fraction in either of the two cases, it is probable that the changes in excretion were a result of the added methyltestosterone. These changes may give a clue to the metabolic fate of methyl testosterone, about which little is known at present.

Summary

Steroid excretion has been studied in rheumatoid arthritic patients before and during treatment with A.C.T.H. and cortisone.

Changes in the excretion of 17-ketosteroids, already reported by others, have been largely confirmed. In contrast to the findings of other workers, an increase in aetiocholanolone excretion was observed during cortisone administration.

A.C.T.H. increased the excretion of total non-ketonic steroids. The increase was most pronounced in the dihydroxy steroid fraction.

Cortisone had little effect on non-ketonic alcohol excretion.

REFERENCES

- Copeman, W. S. C., Savage, O., Bishop, P. M. F., Dodds, E. C., Kellie, A. E., Stewart, J. W., Glyn, J. H. H., Henly, A. A., and Tweed, J. M. (1952). *British Medical Journal*, 1, 397.
- Dingemans, E., Huis in't Veld, L. G., and de Laat, B. M. (1946). *J. clin. Endocr.*, 6, 535.
- Dobriner, K. (1951a). *Bull. rheum. Dis.*, 2, 5.
- (1951b). In *Steroids in Experimental and Clinical Practice*, edited by A. White. Churchill, London.
- Lieberman, S., and Rhoads, C. P. (1948). *J. biol. Chem.*, 172, 241.
- Engel, L. L. (1950). *Recent Progr. Hormone Res.*, 5, 335.
- Hench, P. S., Kendall, E. C., Slocumb, C. H., and Polley, H. F. (1949). *Proc. Mayo Clin.*, 24, 181.
- Kellie, A. E., Smith, E. R., and Wade, A. P. (1953). *Biochem. J.*, 53, 578.
- and Wade, A. P. (1953). *Ibid.*, 53, 582.
- Nathanson, I. T., Engel, L. L., and Kelley, R. M. (1951). *Proc. 2nd Clinical A.C.T.H. Conf.*, vol. 1. Churchill, London.
- Paschkis, K. E. (1951). *Ibid.*, vol. 1.
- Robinson, A. M., and Goulden, F. (1949). *Brit. J. Cancer*, 3, 62.

POST-GASTRECTOMY SYNDROME

DEVIATION OF THE AFFERENT LOOP FROM THE GASTRO-INTESTINAL ANASTOMOSIS

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Much has been written recently about the early post-gastrectomy syndrome. This is not surprising when we consider in what large numbers the Polya type of operation of partial gastrectomy is being undertaken to-day for gastroduodenal ulceration. For the moment a new disease is being created, commonly referred to as the "dumping" or post-gastrectomy syndrome, consisting of symptoms varying in character and severity. The milder cases improve with the passage of time, but the severe ones remain, and it is with these that we are concerned. What is the extent of the dumping syndrome, and what happens to these patients? One so frequently hears that the syndrome occurs in only a small percentage of cases which often right themselves.

Custer, Butt, and Waugh (1946) thought that all but the mildest cases lasted indefinitely, and that the symptoms could be so severe and disabling as to warrant the use of the term "gastric cripple." Capper and Butler (1951), in examining the results of 558 Polya type of gastrectomies, found that 79 cases showed the early syndrome, and that 61 of these had symptoms lasting from two to eleven years. Goligher and Riley (1952) reported that six months after operation dumping occurred in 75% of their patients, which was severe in 12.5%, and that eighteen months after operation 47.5% still had dumping and 10% severe dumping. Others maintain that the percentage is not so high, but we suggest that a personal study of each post-gastrectomy patient will reveal some evidence of the syndrome in a large proportion of cases. The severe cases, by reason of their regular attendance at a follow-up clinic, can be most disconcerting and not at all helpful to other patients. When we consider the scale upon

which the operation for peptic ulcer is being performed, the number of these unfortunate people must be large. Moreover, the operation is undertaken for men usually at the height of their wage-earning capacity and with a family to support. If, as a result of the dumping syndrome, they are unable to return to their pre-operation occupation, then the operation of gastrectomy cannot be regarded as a success.

This paper—a preliminary report—deals with the early post-gastrectomy syndrome following the Polya type of gastrectomy for duodenal ulcer. Our experience has been an average one, and, confronted by the problem of what to do for a small number of severe "dumpers," we decided to follow the procedure of Illingworth (quoted by Muir, 1949), who treated three cases successfully by lateral anastomosis of the efferent and afferent loops of the jejunum. Our patients readily accepted the opportunity of being rid of their miserable condition.

Lateral Anastomosis

In our series of antecolic Polya gastrectomies with a Hofmeister valve seven cases of persistent severe "dumping" occurred, which was very distressing both to the patients and to ourselves. These patients (five male, two female) were all submitted to an anastomosis between the efferent and afferent loops of the jejunum. Six had a gastrectomy for duodenal ulcer and one for a gastric ulcer. Their symptoms can be grouped conveniently under four headings:

1. *Vomiting Bile.*—All patients complained of this distressing symptom. Vomiting varied from one to three times a day, after food, and in amounts from 5 to 20 oz. (140 to 570 ml.) at a time.
2. *Vasomotor Symptoms.*—Four patients complained of severe sweating and dizziness after food, and had to lie down until the attack had passed off.
3. *Loss of Weight.*—Six cases lost amounts varying from 7 to 35 lb. (2.7 to 15.9 kg.).
4. *Vomiting Food.*—Three cases vomited food by itself or mixed with bile.

The onset of symptoms in all cases occurred within four weeks of discharge from hospital. All seven patients were emphatic that their symptoms were worse after the midday meal—that is, the big meal of the day.

Investigation of Cases.—A barium meal and a fractional test meal were carried out in all cases. The x-ray film showed afferent-loop filling and the fractional test meal a complete achlorhydria. Haematological investigations were all within normal limits.

Interval Between Operations.—The average interval was 15 months, the longest being two years and the shortest five months.

It is now about three years since these cases had their second operation, and their present condition can most conveniently be studied under the same headings as before:

1. *Vomiting Bile.*—This has been completely abolished in five cases. Two cases admit to an occasional vomit of bile every two or three weeks, but are greatly improved.
2. *Vasomotor Symptoms.*—These persisted in three out of the four cases affected, the fourth being completely free from symptoms. One case, in which the symptoms persisted, obtained relief by leaving the table and lying down.
3. *Loss of Weight.*—In five of the cases there was no gain in weight after entero-enterostomy; the other two were back to their pre-gastrectomy level and had gained 14 and 28 lb. (6.4 and 12.7 kg.).
4. *Vomiting Food.*—This was abolished in the three cases affected.

Six of the patients submitted to the operation were very satisfied with the result. One patient has undergone a further operation (see below). It would seem that the post-cibal vomiting was the most distressing symptom, as these patients were very glad to have this abolished and felt very much better in spite of the persisting vasomotor symptoms. Our conclusion from this short series was that,