of ketone bodies and lactate. The modern spectrophotometric enzymic methods have greatly simplified the determination of ketone bodies[6] and lactate.[7] It should also be noted that the determination of lactate in serum may lead to spuriously high values due to lactic acid production during red cell glycolysis.—We are, etc.,

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Central Venous Pressure

SIR,—Under "New Appliances" (28 December, p. 825) there was a presentation of a central venous pressure (C.V.P.) self-levelling transducer (using an expensive, elaborate, and fairly complex piece of equipment for what should be a simple and routine measurement in seriously sick patients with fluid problems). The point of reference is the most complex part of C.V.P. measuring, most taking the centre of the chest (in three dimensions) so that a patient can be nursed in *any* position without having to be moved for C.V.P. measurements. This usually can be explained to intensive-ward staff without difficulty, and then very frequent measurements are "easy" so long as a free communication of the catheter is maintained and checked.

After spending many long hours with 3-ft. (90-cm.) or shorter bubble levels, broomsticks, and drip stands in comparing the C.V.P. catheter level with the right atrium (for example, centre of chest in three dimensions, antero-posterior, vertical, and horizontal) it is interesting to see such a simple principle become so complex.

It occurred to me that one could set up a loop of plastic tubing, one side running parallel to the C.V.P. on the measuring scale and the other mobile and near the patient. Enough water (even coloured to reduce confusion) is placed in this U-tube so that, when the tube is laid roughly vertical to the patient's side and the tube raised or lowered so that the patient's estimated reference point is matched by the fluid level, then the remote end will reflect the height of the centre of the chest. Several measurements—say three—should be done either by the same person or by several people and the mean taken. If more than half-hourly readings are needed, then the patient is probably sick enough to have a doctor with him during these periods, as in surgical, burns, trauma, and cardiac cases. The patient may be left to sleep except for taking the blood pressure and changing the posture. The main difficulty in using the skin at the sternal angle is returning the patient to a reference position—for example, on the flat or 45° on his back—to obtain an accurate reading. This may cause a patient to be continually disturbed in the name of B.P., C.V.P., and physical examination.—I am, etc.,

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Insulin Response

SIR,—The article by Dr. A. N. Rigas and others (5 October, p. 25) aroused much interest here. They suggest that chlorpropamide lowers the level of blood glucose necessary to elicit a given insulin secretion by the beta cells. A similar observation was made by Ping Chi Chiu *et al.*[1]

We have measured plasma tolbutamide, glucose, insulin, and non-esterified fatty acids in ten maturity-onset diabetics over 48-hour periods, both on a single, large dose of tolbutamide and on the same amount given in divided doses.[2] We noted a fluctuation in the blood sugar, which tolbutamide set at a lower mean level. The insulin response appeared to be dependent on the glucose and not the tolbutamide levels. We postulated that tolbutamide alters the "glucostat," which determines the insulin response.

It appears then that both tolbutamide and chlorpropamide may restore the abnormal insulin response of diabetics towards normality. Whether the primary adjustment is a reduction of glucose levels or enhanced insulin secretion with secondary lowering of glucose remains to be elucidated.—We are, etc.,

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Familial Non-Polypotic Carcinoma of Colon

SIR,—Carcinoma of the colon is well known to occur sooner rather than later in families with the multiple polyposis syndrome.[1] Solitary polypi which become malignant may also be an inherited condition.[2] Familial carcinoma of the colon apart from polyposis is rare. I should like to describe such a family in which the mother and four out of eight of the offspring have had histologically proved carcinoma of the colon and the father is reported to have died from carcinoma of the rectum. The details are shown in the Fig. and Table.

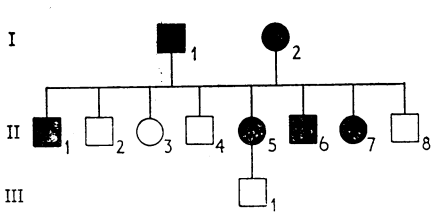

TABLE I

Family Details

I 1. Died at the age of 42 years, one year after surgery for reported carcinoma of the rectum.
2. Carcinoma of the sigmoid colon diagnosed at 75 years. Diverticular disease also present. No polypi. Histology: adenocarcinoma. Died six months later.

II 1. Mucoid adenocarcinoma of hepatic flexure removed at 43 years in 1951. No polypi. Barium enema 15 years later: normal.
2. Aged 56 years. Symptom free. Barium enema: diverticular disease. No polypi.
3. Aged 54 years. Symptom free. Barium enema: diverticular disease. No polypi.
4. Aged 52 years. Symptom free. Refused investigation.
5. (Propositus) Aged 51 years. Annular adenocarcinoma ascending colon. Two simply polyps in adjoining mucus membrane.
6. Mucoid adenocarcinoma of transverse colon removed at 25 years. One simple polyp in adjoining mucus membrane. Died two years later.
7. Relatively undifferentiated carcinoma of ascending colon at 42 years. No polypi. Has had further surgery for local recurrence.
8. Aged 33 years. Symptom free. Normal barium enema.

III 1. Aged 31 years. Symptom free. Normal barium enema.

The relationship, if any, between adenomatous polyps of the colon and subsequent cancer of that viscus has been the subject of much debate and controversy. Majority opinion now favours the view that, unlike either papillary adenomas or papillomas, true malignant change in adenomas is extremely rare. When one considers that between 10% and 50% of people over 30 years of age have polyps in their colon, this seems reasonable.[3] Moreover, in a follow up survey of 4,000 patients over a period of five years there was no difference in the incidence of carcinoma between those with and those without polyps.[4] No matter what one's view concerning this relationship, in the family described here only two of the ten colons examined macroscopically and histologically contained polypi, and it therefore seems reasonable not to consider them aetiologically related.

Similar families have infrequently been described. Mathis[5] reported a Swiss family in which 11 out of 24 members in three generations had carcinoma of the colon. In his family again only two were noted to have polypi, and, as in this study, they numbered but two and one in the members affected. Kluge[1] described seven cases in four generations without evidence of polypi. The families discussed by Peltokallio and Peltokallio[6] are not so impressive, as only two members are involved in two of their families, and one of the cases quoted had long-standing early-onset ulcerative colitis. No such families seem to have been described previously in this country. While not strictly accurate or sufficiently exclusive, a term such as familial non-polypotic carcinoma of the colon is suggested as a way of describing such families.

Although a case can be made for prophylactic colectomy in the, as yet, unaffected members of this family, it is proposed to follow them on an outpatient basis with barium enemas yearly, or immediately should symptoms occur.

I would like to thank Dr. M. G. FitzGerald, under whose care the propositus was admitted, for his help and advice, and Dr. H. Thompson for reviewing the histology.

—I am, etc.,

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Scabies in Negroes

SIR,—I was interested to read of the impression gained from Dr. F. A. Ive's experience in West Africa and that scabies was a rare disease in Africans (14 December, p. 706). It is certainly not rare among the urban Africans in Rhodesia and it has also been reported from Tanzania by Dr. R. W. Smith (15 July 1967, p. 174) that in Central Tanganyika scabies is a serious problem and in some villages almost 100% of children over 6 months old are affected.

In Salisbury we have found it necessary, as a public health department, to set up a scabies clinic in the Harari township, and the monthly total of patients treated at this clinic in the past three years has varied between 1,000 and 5,000. Bearing in mind that the total African population of the township is reputedly 236,000, this represents a fairly high endemic rate of scabies. Recurrences are frequent, particularly among single men and families in the older types of accommodation sharing ablution facilities.

While we do not examine every instance by scrapings, many of these have been confirmed microscopically and respond to the usual treatment (benzyl benzoate or monosulfiram). Some of our patients report that a similar type of skin condition has been occurring in their home reserves, so it is clear that scabies is not limited to Africans living in the township.—I am, etc.,

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SIR,—Dr. F. A. Ive (14 December, p. 706) has kindly invited comment on some inconsistent features of scabies as between negro and white patients. Clinical observation over 20 years in Pretoria has taught us that Dr. Ive's puzzles are not imaginary.

In the white population the regular reservoirs of scabies were for many years the country boarding schools, inferior boarding houses, and similar crowded residences. Importation of scabies into "nice" families would come from teenage children home for the holidays from these boarding schools, or from the vicissitudes of travel. When a baby or a toddler developed scabies it was never acquired from a Bantu nanny but usually from a young and helpful white friend or relative. Furthermore, scabies in whites has been extremely scarce for the last few years. Recovery of the acarus has been very easily accomplished in white patients at all times.

In the Bantu population we were wont to diagnose scabies uncritically in the early 1950s in about 5% to 6% of outpatients. After a few years we started to wonder if we had been dreaming. Follicular rashes there still were, but convincing scabies vanished. In the late 1950s the writer often challenged the registrars to produce an acarus from the negro skin, and none was forthcoming. An occasional Indian patient back from India, who had acquired scabies over there or on the boat, made us realize that a brown skin was no bar to finding the acarus. Clarke's Nigerian figures of 25% scabies in Lagos, published in 1959,[1] were almost unbelievable, since I had by then become convinced that scabies with us was confined to the white population. When Dr. Roger Harman visited us in 1962, after his stay in Ibadan, we were eager to ask him how many acari he had seen recovered from patients. Because of his short stay there he gave a guarded answer, and could not solve the mystery. Then from about 1965 onward a slow trickle of Bantu patients with readily demonstrable scabies started coming our way. The flow gained in strength over the ensuing three years, until it became torrential by the middle of 1968. At this time the skin outpatients' department had doubled its patient turnover, 50% of the cases being scabies. The epidemic was then taken in hand by the local health department and the numbers are declining steadily.

From these experiences, hitherto unreported, we must agree fully with Dr. Ive that clinical statistics for scabies in dark skins are liable to both clinical error and genuine fluctuation. Moreover, it seems now quite probable that an epidemic affecting one population group need by no means spill over into another living in the same locality—as seen by our experience in Pretoria and his in Durham. Let me assure any doubters that if our acarus of scabies is fastidious, the local *Treponema pallidum* is not.—I am, etc.,

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REFERENCE

1 Clarke, G. H. V., *Skin Diseases in the African*, 1959, p. 1. London.

SIR,—Dr. F. A. Ive in his letter (14 December, p. 706) said that he doubted very much whether scabies was a common disease in Africa. We, situated on the edge of the Kalahari desert, are in the midst of yet another major outbreak among the Setswana tribe. At a rough estimate we have treated 2,600 cases in the last six months, and at our daily outlying clinics have been treating as many as 30 cases of scabies at one session.

We find that the isolated case and the inpatient respond extremely well to Ascabiol (benzyl benzoate), provided this is used correctly. The outpatient receives his first bath and an application of benzyl benzoate in the hospital, and the mother is asked to watch and learn how to do both. His clothes are also washed. However, with such a widespread epidemic he is likely to be reinfected, and we, being two doctors to 350 inpatients and serving outpatients and district clinics over an area of 5,000 square miles, are unable to supply the mass treatment that would obviously be preferable. We have, it is true, never isolated the acarus, not having the facilities, but the lesions are very typical, being small white papules always in the interdigital webs and very commonly spreading from the wrists and axillae to cover the trunk and lower limbs, and occasionally, although very rarely, the face. They always respond to correct treatment with Ascabiol. They are very irritating to the patient, especially at night, and they commonly become secondarily infected in these people, who live in extreme poverty.

I also quote Michael Gelfand in the *Sick African*[1] (without which, together with Hamilton Bailey's *Emergency Surgery*,[2] no doctor should contemplate leaving London airport), where he says, "Scabies is encountered throughout the natives of Africa"—however, perhaps he has never been to Nigeria ? (14 December, p. 706.)—I am, etc.,

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1 Gelfand, M., *Sick African*, 3rd ed., 1957. Cape Town.
2 *Hamilton Bailey's Emergency Surgery*, 1967, edited by T. J. McNair, 8th ed. Bristol.

SIR,—I was interested in Dr. F. A. Ive's letter (14 December, p. 706) regarding the rarity of the demonstration of the acarus of scabies in Africa. Some points occur to me.

There is, firstly, the changing pattern of disease, which is often quite rapid in a developing country. In 1962 I pointed out that the incidence of scabies had apparently dropped from 25% to 11% in the decade ending in 1960.[1] The enormous sales of such toilet articles as monosulfiram soap must have been having some effect!

Secondly, as regards the differential diagnosis from onchocerciasis. Undoubtedly the latter can mimic scabies, and this is the origin of the French term *gale filarienne*. Although they are sometimes similar, it is usually easy to differentiate the two. The distribution confined to one segment of the body and residence in an endemic area are perhaps the most important clues.

Thirdly, it is certainly true that most practitioners in Africa (as probably elsewhere) do not confirm the diagnosis of scabies by finding the acarus. With vast hordes of patients there is simply not the time, and the demonstration would only probably be attempted for teaching purposes.—I am, etc.,

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REFERENCE

1 Clarke, G. H. V., *Brit. J. Derm.*, 1962, **74**, 123.

Amputations for Ischaemia

SIR,—Your leading article on amputation for ischaemia (11 January, p. 69) belittles the role of the team of doctors and physiotherapists whose attention "boosts the patient's morale if it does nothing else." May I suggest that such a team can be of greater importance than you indicate ? In these elderly patients rapid mobilization and early