NEW TRENDS IN THE TREATMENT OF THROMBOANGEIOSIS (BUERGER'S DISEASE)

Moynihan Lecture delivered at the Royal College of Surgeons of England

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by

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INTO THE LIFE of every man there come crucial moments, and this, for me, is one of them. And it reminds me of another crucial moment which, at the time, was important too, but for a different reason. It was in October 1942, the day I was interviewed by the Royal Army Medical Corps board. I was asked: " What were you doing before the War?", and I answered, with an unmistakable accent: " Sergeant! " The reply was: " I see, you have already been in the Army! " I hope my pronunciation will not play the same trick on me this afternoon.

But today I am faced with another problem. Like all surgeons throughout the world, I have been taught to respect your venerable College, and if I accepted your invitation to lecture here, an honour as redoubtable as it is great, it is because I felt there were convincing reasons for doing so. First, the memory of Lord Moynihan, who foresaw and recommended international exchanges between surgeons; secondly, the confidence I have in old friends who are present; thirdly, my remembrance of my Master, Professor Leriche, who spoke to you just over 20 years ago on vascular diseases; and last, but not least, my devotion to your country. The years I spent with the Royal Army Medical Corps were decisive ones in my life, and if this meeting enables me to offer public thanks to the Medical Service that accepted me, it will not have been entirely in vain.

In this country, where so many physiologists and surgeons have contributed greatly to the understanding of vascular diseases, it might seem presumptuous to approach such a subject before the College. It is not my intention to force you to listen to an academic lecture on that branch of surgery: I only wish to draw your attention to a few clinical facts, and to give you an account of my reflections on Buerger's disease, which we prefer to call thromboangeiosis rather than thromboangiitis, the latter denomination implying an idea of inflammation, and that is not proven. Finally, we shall discuss our therapeutic attempts.

It is worth recalling that the first anatomical description was written in 1878, in Vienna, by Winiwarter, an assistant of Billroth. Subsequently, the Chair of Surgery at the University of Liège was held by Winiwarter

from 1890 to 1917. Our interest in this line has not decreased in the last ten years; 580 lumbar sympathectomies, 57 upper thoracic sympathectomies, 30 suprarenalectomies and more than 1,800 arteriographs have been performed in our Department.

Thirty years after Winiwarter, Leo Buerger (1908) published his first paper on the disease, which has since borne his name. These monographs were the cause of many erroneous interpretations, which prevail even at the present time. Contemporary surgery is still suffering from the traditional anatomy which lay so heavily upon it, up to 25 or 30 years ago; indeed it has taken a long time for surgery to become a more physiological discipline. Not only the first description of thromboangeiosis but most

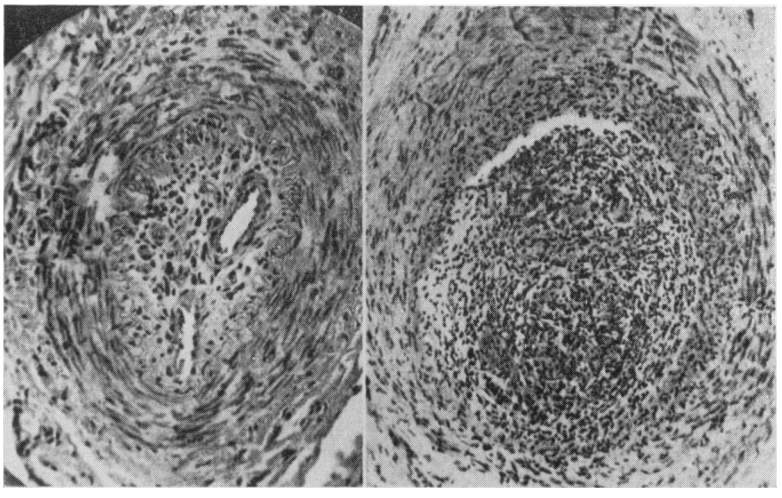

Fig. 1. Partial and ineffective recanalization through fibro-plastic block.

others also are based on studies of amputated limbs, the seat of year-long peripheral ulceration, and there lies the snag. The fact is that the criteria originally laid down to establish the diagnosis are wrong, because they were based on superimposed chronic infection.

In specimens taken in early cases and arteriectomies done at a distance from the extremity, we do not find the so-called " giant cells " and typical signs of inflammation which appear much later, after sepsis has been present for a long time. Professor Henry Dible has made similar observations.

The prominent feature is the presence of a thrombus in apparently healthy arteries. The clot is white in its peripheral segment, and red proximally; it has therefore crept up cephalad. It is only from the time the thrombus undergoes fibrinoplastic metaplasia that one can detect

the first reaction of the intima with stratified layers of thickening. After this, it becomes more difficult to distinguish what belongs to the clot or to the arterial wall. The media remain undisturbed, lipido-calcaneous deposits never appear. Some changes occur in the veins; much later does the pseudo-inflammatory process envelop the adventitia.

Recanalization takes place only in recent and short obliterations, otherwise repermeation does not intervene through a well-organized fibroplastic block (Fig. 1). Furthermore, at that stage it is histologically impossible to differentiate the picture of a Buerger thrombosis from a post-traumatic or post-embolic one. But the most striking fact is that the thrombus occurs in vessels which appear to be almost normal. In between the occluded segments, arteries may be perfectly healthy; lesions are

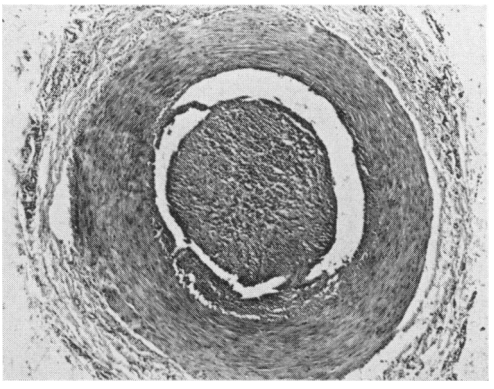

Fig. 2. Early case of thromboangeiosis (case R . . .). Obliteration of digital artery (four weeks old) and cellular organization of the thrombus.

patchy, sometimes scattered all over several extremities, with " skipped areas ", reminding one somehow of Crohn's disease. Moreover, in many places, one can see a shrinkage of small arteries below the obstacles (sometimes above it). This aspect of narrowing is well illustrated by arteriography; to us, it is the manifestation of spasm and of thrombosis.

Histology

As we have already pointed out, the study of sections of vessels from amputated limbs can be misleading, and research should bear on very early cases, and actually I cannot remember having seen one in surgical literature. However, I think we can show you one.

A man, 37 years old, very strongly built and in perfect health, was awakened one night by intense pain in the tip of his left third finger; within a few days, the pulp became cyanotic and a tiny patch of gangrene developed. He was given, unsuccessfully, vasodilatators by his general practitioner, and Novocaine block round the finger. He

kept on working as a garage mechanic. He died accidentally three weeks after the onset and, through the kindness of the Professor of Forensic Medicine, Paul Moureau, I was able to have the results of a very thorough post-mortem examination.

The digital artery of the finger involved showed cell proliferation coming from the intima, which was raised in places by new cells (Figs. 2 and 3), which have invaded the original thrombus (Fig. 4). There was no pathological change in the media. The disease seemed to start from the intima and progress towards the lumen of the vessel. A similar histological picture was found in vessels of the limbs, brain, bronchi and mesentery (the elastica was intact). In contradiction to this, in the aorta we found early signs of atheromatosis, quite different, with cellular changes and lipid deposits in the media.

What an extraordinary disease, when we think of that sturdy individual, apparently quite fit, while his visceral and limb arteries were marked by a generalized disorder progressing silently everywhere.

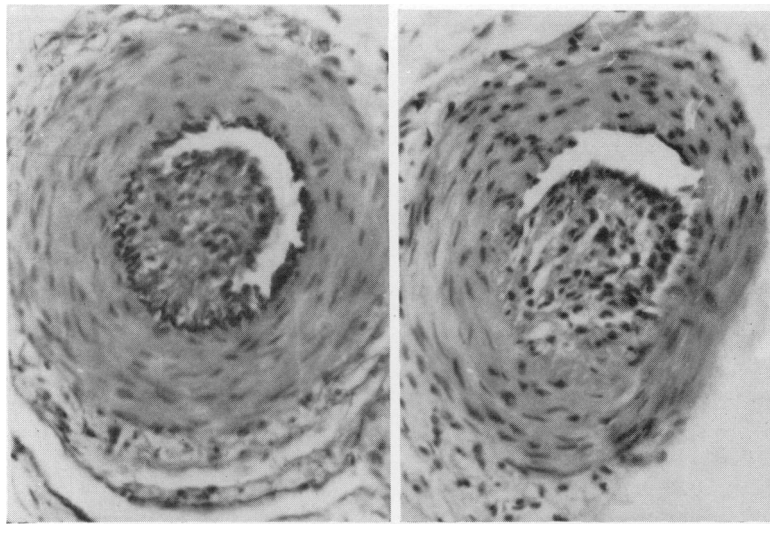

Fig. 3 (*a*) (Case R . . .) Cell proliferation coming from the intima. (*b*) (Case R . . .) Invasion of the lumen of the artery. The intima is raised by new cells.

In order to demonstrate how an artery can react under acute conditions, here is a rather disconcerting case.

A woman, 48 years old, who had always enjoyed good health, and had never suffered from any allergic disturbances or heart trouble, suddenly, while typing in her office, felt intense pain in all the fingers of her left hand. She was brought to us 65 hours later; she had no pyrexia, her blood was normal and no abnormality was detected in her heart: the classical picture of an embolus of the brachial artery. The fingers were white and cold (20° C.). There was complete palsy of the hand. Oscillometric readings diminished over the arm, and were absent in the forearm.

At operation, the brachial artery was pulsating, and no embolus could be felt. The radial artery was exposed 10 cm. above the wrist; there was no pulsation. Arteriectomy over a length of 10 cm. was performed; there was a recent embolus obstructing the vessel, with blood just oozing from proximal and distal arterial ends. We finished with

a Smithwick operation (upper thoracic sympathectomy). The immediate result looked excellent: the pain had gone, the temperature of the fingers had risen to 31° C. Yet within three weeks, all fingers were affected by dry gangrene up to the first phalange. Amputation above the elbow was performed; there was a generalized thrombosis of veins and arteries. Healing *per primum* took place. Today, ten years later, she is very well, still working, but has had several benign attacks of phlebitis migrans, well localized, gradually disappearing after a few days. From the time of her menopause three years ago, she has had no more vascular trouble.

The pathological report (Professor Betz) (Figs. 5 and 6) stated: " Radial artery: fresh thrombosis; all layers involved by an inflammatory process. Granulomas erupting from the intima, very similar to the case discussed above. Culture: sterile."

We are at a loss how to classify this patient. It might be what is called " acute vegative arteritis ", although the patient never had any signs of infection, or a fulminating example of thromboangeiosis. Yet histological pictures can be usefully compared. It seems the disease starts

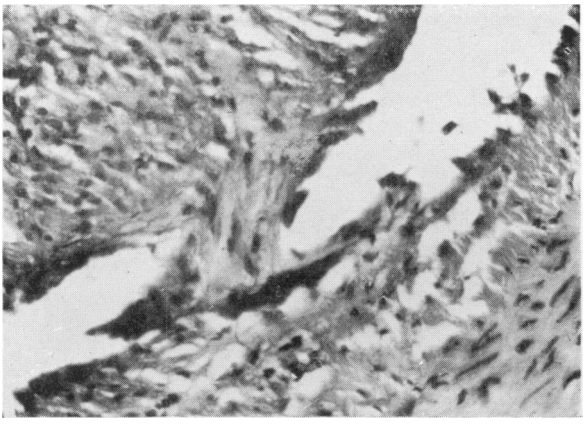

Fig. 4. (Case R . . .) High magnification of Figure 2 showing the track of penetration of intimal cells into the thrombus.

inside the intima but I cannot be positive about this. As Professor Dible wrote: " Something occurs which sets up thrombosis in vessels . . . which comes first, the vascular changes, or the thrombosis? " Telford and Stopford suggest that the primary lesion may be spastic occlusion of the vasa vasorum with consequent intimal alteration. I would be rather inclined to adopt that view, referring again to the first case shown, that of the man with a minute arterial thrombosis of *one* finger, and giving evidence of changes in the intima all over his body!

Localization

Usually, the disease originally affects the distal vessels of fingers and toes, radial and tibial arteries, although the popliteal artery is very often involved (Wertheimer and Sautot (1958)—50 per cent.). Upper limbs are

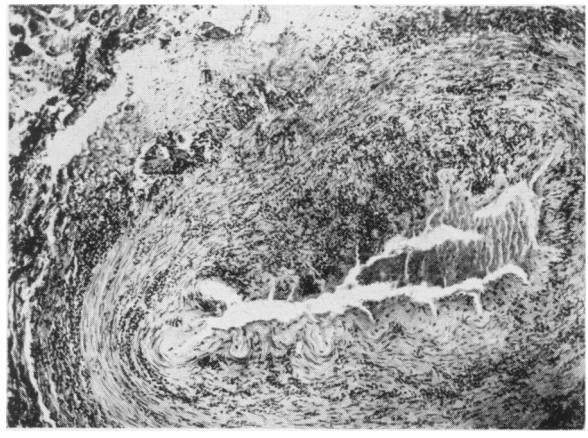

Fig. 5. (Case De P . . . woman, 48 years old—acute thrombosis of arteries and veins of upper extremity.) Radial artery, peri- and intramuscular inflammatory reaction, all layers are involved.

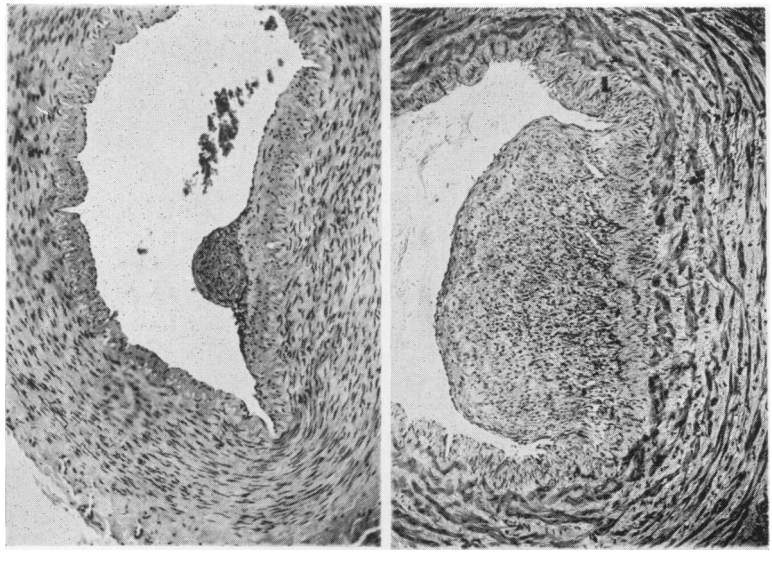

Fig. 6 (*a*) (Case De P . . .) Ulnar artery, granuloma erupting from the thickened intima (compare to Fig. 3 (*a*) and (*b*)). (*b*) (Case De P . . .) Venous thrombosis, same process as in arteries.

nearly always involved sooner or later; and we have seen obliteration of the radial and ulnar arteries years before the patients complained of their hands. Besides, more than 40–50 per cent. of the patients develop visceral and central thrombosis (brain, heart). As a rule, pelvic arteries remain undamaged.

Arteriography

Aortography: we have been using the fluorescein test: histamine wheals are raised on the popliteal fossa and on both tibial malleoli. 30 c.c. of sodium fluoresceinate solution are injected into the aorta; the normal average blood speed is 5–6 seconds for the knee, 10–12 seconds for the foot. It is the only way to reckon an adequate timing and to get a full injection of the foot; in some patients, one has to wait 23–30 seconds before taking the leg and foot X-ray.

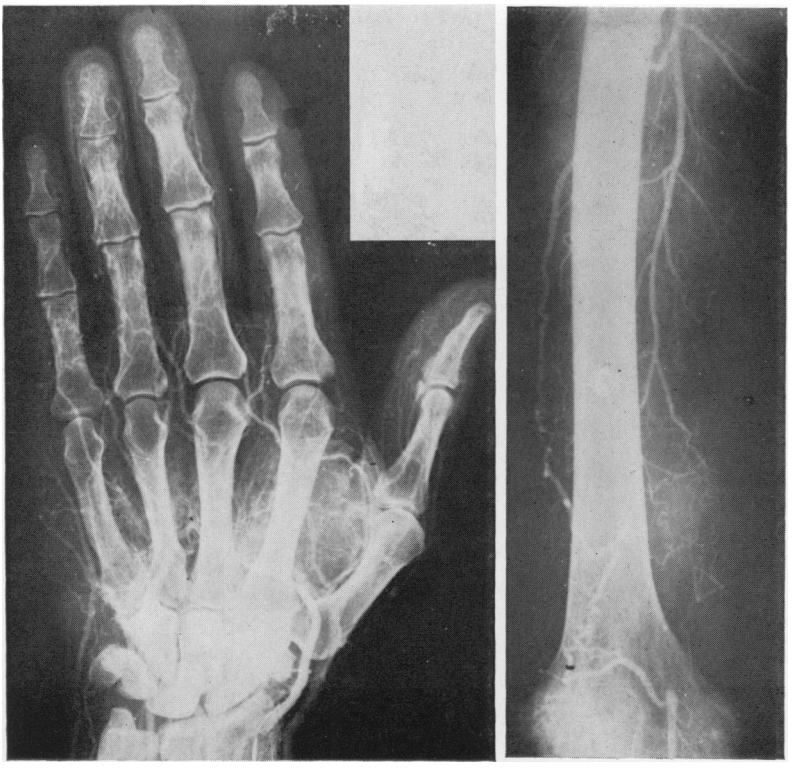

Fig. 7. (*a*) Buerger's disease. Gangrenous patches on tips of index and middle finger, whose digital arteries are distally obstructed. (*b*) Buerger's disease. Arteries are thin, fine-drawn, obliteration in Hunter's canal poor collaterally.

Arteriographic pictures are well-known: general shrinkage of peripheral vessels, thinning of the most distal part, patchy, erratic distribution of the obliterations more localized at the extremities to start with (Fig. 7 (*a*) and (*b*)). Collateral circulation varies according to the duration of the disease, tributaries look fine-drawn; in any case, collaterality is always less developed and less extensive than in atherosclerotic arteritis. Only arteriography can elicit cascade-like obliterations and the precise condition of small peripheral vessels.

Aetiology

Various aetiologies classically discussed—infection, tobacco, ergot, race —do not represent more than a historical interest.

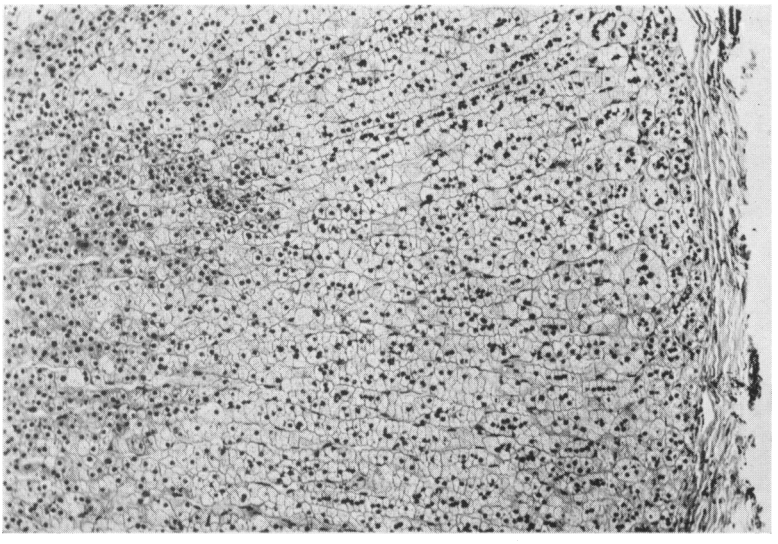

Fig. 8. Thromboangeiosis of suprarenal: enlargement of fasciculata, glomerulosa extremely reduced.

Apart from arteries and veins, pathologists have described other lesions in thromboangiitis; we may say we are not convinced by some papers describing degenerative changes in sympathetic ganglions.

But the study of the adrenals throws more light on the subject. Leriche (1946, 1949, 1954) and Fontaine (1950) had already emphasized that 75 per cent. of adrenals examined showed important changes in the cortex. Many other surgeons have made similar observations (Wertheimer (in 60 cases), 1958; Pettinari *et al.*, 1953, 1959 (61); Tingaud, 1952; and Carls (20), Ferrand and Elbaz, 1958, etc.).

I can produce the pathological reports of my 13 personal cases; they are exactly comparable to the French and Italian findings. Here is Professor Betz's report: " The study of the adrenals in 13 cases of Buerger's disease

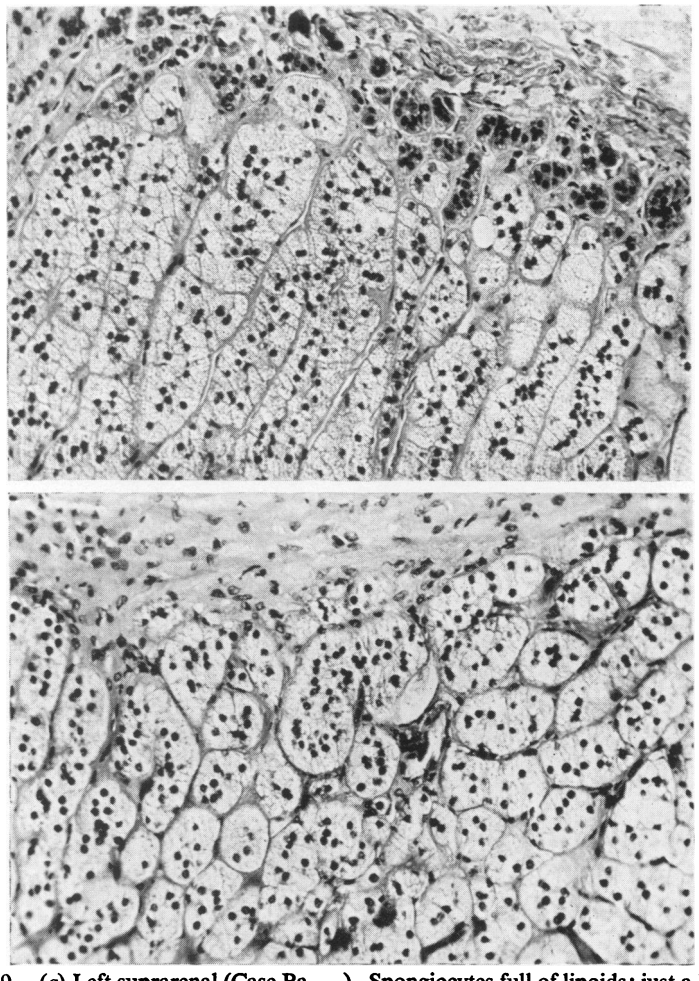

Fig. 9. (*a*) Left suprarenal (Case Ba . . .). Spongiocytes full of lipoids; just a layer of glomerulosa cells is still present. (*b*) (Case Ba . . . same as Figure 9 (*a*)). Right suprarenal nine months after left adrenalectomy. Glomerulosa has disappeared and there are no signs of compensatory hypertrophy.

reveals in all of them the same modifications of the cortex: hyperplasia of the fasciculata and wearing down of glomerulosa (Fig. 8). The fasciculata is noticeably enlarged and made of spongiocyte cells full of lipoids; in many cases pseudo-adenomatous formations can be found. The outer

part, the glomerulosa, is hardly discernible as such, the fasciculata contacting the capsule at various points. The reticularis (inner) does not show perceptible changes (Figs. 9 (*a*) and (*b*))."

No signs of compensatory hypertrophy were seen on the occasion of the second adrenalectomy (three to seven months elapsed between the two operations).

There is a general consensus of opinion that in thromboangeiosis the suprarenals show signs of dysfunction, even of overactivity (150 patients reported up to now).*

Physiopathology

I should like to state that I have the greatest respect for experimental surgery, but it must be said that in vascular pathology experiments on animals have sometimes led to certain blind alleys. The sympathetic system of the rabbit, or of the cat, is not the same as that of the dog, and in any case it could hardly be compared to that of a sick man. Moreover, it must be remembered first of all that these animals never develop spontaneous gangrene. Man is " an animal without fur or feathers " (Leriche), and has a vascular system which reacts in its own way, and quite differently in health and in disease. And yet human beings can be very different one from another; one reads in any text book: " cold, fear, mental excitation, etc., will result in vasoconstriction of the face and extremities and will be expressed in pallor ". This is partially true, but we know that many people will blush and get red all over under the same stress. Have we not met some good-looking girls whispering a lie, one becoming apparently bloodless, the other becoming bright red? Some young recruits on the battlefield are rooted to the very spot by diarrhoea, while their companions may be constipated.

These commonplace facts are not very academic, yet it is high time that they should be accounted for in the interpretation of man's diseases.

The whole problem of thromboangeiosis is dominated by two features: spasm and thrombosis.

Spasm is an undisputed observation. Very early in the disease, before the appearance of any obvious pathological signs, most patients show excessive reaction to cold, in fact a sort of Raynaud phenomenon; later, spasm attacks may be so severe that they simulate arterial embolism. Besides, aortography visualizes those areas of vasoconstriction below the obstruction (sometimes above). All the same, the returning pulse, the increase of local temperature following sympathectomy, arteriectomy (and

* Some authors have published comparable findings in adrenals of atherosclerotic arteritis patients; they assume this would be the clue to the fact that both diseases may have a common link in the adrenal disturbances; it is still an open question.

also adrenalectomy) plainly demonstrate the existence of spasm. The part played by the central and sympathetic nervous system and by hormones are classical knowledge, though very confused, but when it comes to analysing the local mechanism of spasm at artery level, arguments wax fierce. In this country, generally speaking, very few people believe in intra-mural centres in the artery; they would rather claim that the smooth muscle of the arterial wall just contracts according to the pressure gradient of the blood flow. I do not entirely share this view, and am more inclined to think there must be " some peripheral conducting mechanism ".

Let us go into more details on this point. We know that in man a cervical rib, a callus of the clavicle, the pressure of crutches or an iliac abscess can bring about downward vascular spasm. It has been shown by arteriography and relieved by surgery.

I remember a soldier, 28 years old, who had had a bullet through the thigh. I saw him seven months later complaining of coldness of the foot and intermittent claudication; he showed signs of severance of the internal saphenous nerve. Under local anaesthesia, we exposed the femoral artery; it was beating normally above an adhesion with a neuroma of the saphenous nerve; below, the artery was rigid, contracted. There were no traces of a proper arterial injury. Immediately after the neuroma coalescing with the adventitia was separated, the artery below started to beat normally. The patient was relieved of his femoral spasm, and twenty years later he is still leading a very active life; in fact, he still plays football. This is evidence that an artery with a healthy wall can go into spasm through local stimulation.

It is still more true if the artery wall is diseased. Thirty years ago, with the then so-called " dogs without arteries ", we demonstrated the spasmogenic influence of a ligature round an artery and the vasodilating effect of arteriectomy (with or without sympathectomy) on collateral circulation. Now, of course, arteriectomy has given way to grafting, although there are still some occasions when it is indicated. For instance, in Leriche's syndrome (bifurcation obliteration), when grafting appears unfeasible, excision of the bifurcation combined with bilateral lumbar sympathectomy can give good results.

A man, 28 years old, was treated during the Spanish war by ligature of the femoral artery in Hunter's canal. Six years later, intermittent claudication (X-ray: complete block at the lower third) was moderately relieved by a lumbar sympathectomy. Three years later he had pain and claudication, which became worse. The foot was cold, and oscillometric readings were almost absent. At the time, we did an arteriectomy of the obliterated segment (old ligature site) and within a few hours the foot became warm, and oscillometric readings reappeared. The patient made a full recovery after an arteriectomy done nine years after a ligature, and two years after an unsuccessful lumbar sympathectomy.

Experimentally, it has been shown that chemical obliteration of arteries can bring about distal changes in the intima, eventually going into thrombosis in the vessel below. In their memorable experiments Reilly and Laplan have beautifully illustrated the same conception. By injecting strictly underneath the intima some diphtheria endotoxin, they were able to create all histological phases of vascular disease: spasm, intimal proliferation, thrombosis, very similar to thromboangeiosis; the previous

painting of the artery with phenol would prevent the whole process. Quite recently, S. M. Hilton published a very good paper in the *Journal of Physiology* (1959) on similar effects of direct application of various drugs on the conduction along arteries.

In true Raynaud's disease of long duration, one can see digital blocks on arterioles which have suffered from repeated attacks of spasm. The whole process develops as if repeated spasm could lead to thrombosis which is the ultimate end of the disease. If this was not the appropriate explanation, how could we make out these cascade-like thromboses, interspersed with normal segments, and when blood is quite normal?

Does vasoconstriction of vasa vasorum alter the intima and then allow platelets to conglomerate to it, thus causing coagulation? It is very plausible, and, in my opinion, it is the best working hypothesis. And when we talk of circulation, we do not confine ourselves to the artery alone, but we also think of the satellite vein and nerve. It is just another case of the eternal triangle!

Treatment

We do not have great faith either in intravenous injections of saline or of typhoid vaccine, or in a diet deprived of rye bread. As regards anti-coagulant treatment, strongly recommended by distinguished doctors, we can only say that we have no experience of it. Nevertheless, results are conflicting, and to me it seems very difficult to keep patients for years at an appropriate level of hypocoagulation. Furthermore, if we are to believe that the trigger lesions lie in the intima, one wonders how such a specific blood medication could work.

Sympathectomy

The primary aim of the treatment has been to prevent gangrene, or at least to retard its appearance for some time. It was known that sympathetic tone being at its highest at the extremities, sympathectomy would have the maximum effect over these distal parts, and consequently sympathectomies were done many years ago. Increase of blood flow and of local temperature were recorded after these operations, but it became obvious that after a few weeks they tended to diminish, peripheral blood flow and temperature having a progressive tendency to fall to various levels. There is no doubt that it is the English school which has carried out the most thorough investigations on the physiology of the sympathetic system, and it is beyond the scope of this lecture to discuss it.

I wish only to make a few clinical comments. It is generally contended that post-sympathectomy vasodilatation is limited to the skin of the foot and of the hand and is, as I have said, of rather short duration. I agree partially with that view, and I think there is some answer in the arterial

tree and in the muscles of the limb concerned. Ruberti and co-workers (1960) and Scarabelli and co-workers (1959) have published very accurate recordings supporting that view (even years after operation).

With regard to duration of results of sympathectomy, there is something more to say. We do know, of course, that many sympathectomized patients have recurrences of vasoconstriction attacks, of sweating, and so on, but, oddly enough, not all. You certainly know as much as I do of patients operated on, years ago, who still have warm, non-sweating extremities (10, 15, 30 years). To me, these so-called " exceptions " have a great importance; they signify that under unknown individual (hormonal?) conditions the effects of sympathectomy may last for a very long time. The more physicists provide us with sensitive electronic gadgets, the more we find recordings of fairly valuable blood-flow years after sympathectomy. There are many anomalies in the observation of sympathectomized cases. For instance, you have certainly observed patients exhibiting signs of returning sympathetic activity and still doing well; we also know that failure of the pulse to return does not preclude a good clinical result. As an hypothesis, one could assume that in some cases, after sympathectomy, the arterio-venous shunts (glomi, Sucquet's canals) remain permanently dilated, allowing a relatively adequate blood supply, with hands cold and blue.

Whatever the scholastic arguments may be on regeneration, sprouting, reorganization of the interrupted sympathetic, what are the results of sympathectomy in thromboangeiosis? About 60 per cent. of patients draw benefit from the operation: ulcerations heal, pain is lessened; in rare cases, intermittent claudication is improved, but after a variable length of time, patients will come back with recurrences and aggravation of all their symptoms. That was bound to happen; with sympathectomy, we have attacked only one aspect of the disease, spasm, but nothing has been done against thrombosis.

When there is no return flow of the blood in the main arterial tree below an established thrombosis, no surgery can save the limb. That very much disregarded notion is the reason of many failures of surgery in thromboangeiosis.

It would be ideal to restore the circulation with grafts, but that wonderful technique is not applicable to our problem. Thromboangeiosis is a disease in which the successive outbreaks occlude the artery at multiple levels with prevalence in the extremities, so that local surgical approach is impossible.

For many years, however, surgeons have been trying to find another way to fight the disease at its roots. A new field was opened by Oppel, when in 1921 he performed the first adrenalectomy, assuming (wrongly) that

adrenaline was the cause of thromboangeiosis. In 1925, Leriche and his school followed suit, as did the Italians; in 1947, Leriche wrote a paper on 98 adrenalectomies combined with lumbar sympathectomy and contra-lateral-splanchnicectomy.

At the European Congress of Vascular Surgery (1952), Fontaine and Dos Santos stated that adrenalectomy (combined with sympathectomy) was an operation of value, which apparently seemed to slow down or even check the pace of the disease.

Out of 66 patients thus treated, they registered 74 per cent. of good results (compared to 53 per cent. for patients treated by sympathectomy alone); Wertheimer (57 cases) and Pettinari and his colleagues (61 cases) had produced similar results. Even if that period of surgery was purely empirical, it nevertheless raised interest, and undoubtedly patients did improve. With that technique, effects of sympathectomy appear reinforced and prolonged; recurrences are less frequent and take place later. At that time we had already stressed a most intriguing fact: some patients *not* improved by sympathectomy were much relieved by a later unilateral adrenalectomy. You may object—" that is grossly unscientific "—and I would agree with you; yet they just happen to be human facts.

In the meantime, research on the physiopathology of the suprarenals was being carried out in many places, and resulted in new facts.

Very early on, we realized that repeated adrenaline injections could not induce changes in vessels. Later, a few others (Naggi and Parodi, Froelich, Cavalli, Lucinesco, Pico and Scartozzi) attempted the same thing with successive grafts of adrenal in the rabbit. But their results remain disputable; they produced variant images of thickening of the intima and scattered thrombosis.

Selye and co-workers, in 1943, published a series of thrilling experiments on many animals (chicken, rat, dog, monkey, guinea-pig). With a prolonged course of injections of desoxycorticosterone (DOCA), combined with either a salty or salt-free diet, they created in arteries many surprising changes; some are similar to periarteritis nodosa or arteritis obliterans and also to thromboangeiosis.

In many cases, the similitude is absolutely striking, including final images of granulomas and thrombosis. But perhaps the most interesting part of Selye's work is the following: the concomitant administration of glycocorticoid prevents any deleterious effects of DOCA on arteries; it stops the formation of experimental arteritis. Even if the interpretations of the microscopical sections can be argued, there is a logical conclusion to be drawn: the mineralocorticoids can cause deep vascular alterations. Physicians had already noted vascular disturbances, sometimes serious, in patients treated by ACTH or DOCA.

That was roughly what we knew of the possible relation between adrenals and vascular disease until about 1945. Since then, biochemists have been hard at work and the matter of adrenal activity has become a large province of physiology (more than 30 cortico-steroids have been described so far).

The big impact was caused by the synthesis of cortico-steroids, thus making possible bilateral adrenalectomy.

This technique, at first applied to Cushing's disease, adreno-genital syndrome and metastasis, was later tried in vascular diseases. It appears that in some cases of malignant hypertension (Green *et al.*, 1947), and in

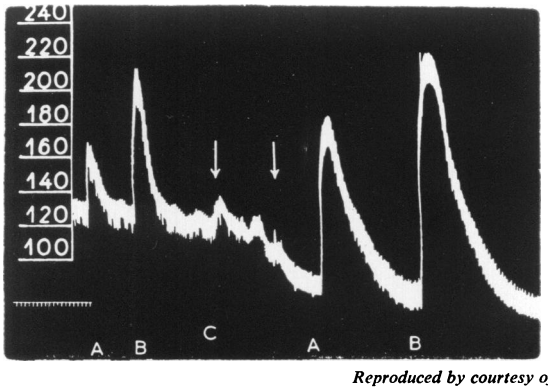

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Fig. 10. (Cat.) Dial Ciba intraperitoneal anaesthesia. Bilateral cervical vagotomy. Tracheostomy and intubation. Time 5 seconds. Diagram of carotid pressure.

in A: Injection of levogyre Adrenaline (2.5 mg./Kg.).
in B: Injection of levogyre Adrenaline (5 mg./Kg.).
in C: in between the two arrows, injection of 10 mg./Kg. of hydrocortisone (alcoholic solution).

The hypertensive reactions due to adrenaline are increased in peak and duration by the injection of hydrocortisone. (The drop of the blood pressure observed after hydrocortisone injection is an effect of the ethylic alcohol, which by itself has no sensibilizing property.)

degenerative arteritis of diabetes (Kimmelstiel-Wilson syndrome) (Wortham and Headstream, seven cases), there is overactivity of the adrenal cortex: in both conditions, good results of adrenalectomy have been recorded. Naturally, the idea of bilateral adrenalectomy in thromboangeiosis seems even more imperative.

We have already discussed earlier the anatomical, clinical and experimental reasons for suspecting the adrenal of being one of the causes of the disease, but let us have a look at the latest advances biochemistry has made along that line.

Many dosages of steroids have been published in relation to man's diseases; not very many can be retained; either the methods used have proved to be deceptive, or samples have been limited to urine (we know this is quite inadequate), or the chemical work has simply not been done properly. I am no biochemist but, watching the work done in my hospital by a team of highly specialized colleagues, I realized the pitfalls of the techniques, and the skill required to reach valid conclusions. Therefore, I think we have to be extremely careful when evaluating steroid dosages in thromboangeiosis.

Some steroids, for instance, are quickly metabolized and, so far, their precise tracing is very difficult. In the past few years, several papers have been published, giving reliable figures tending to demonstrate an increase

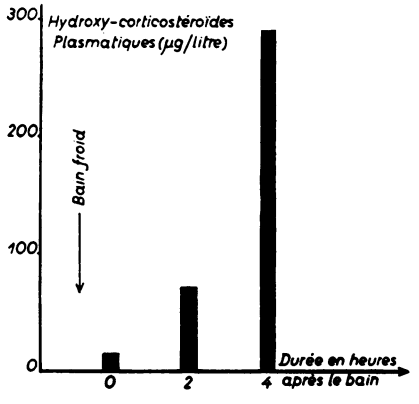


Fig. 11. Rate of 17-hydroxycorticosteroids in a patient suffering from cold hypersensitivity. The left limb was immersed in an icecold bath, at 0 time. Strong increase of the plasma content in 17-hydroxycorticosteroids following liberation of endogenous histamine induced by the cold bath. (Average rate of 17-hydroxycorticosteroids : 5 mg. per cent.)

of some steroids and catecholamines in thromboangeiosis. You will see in a moment my own figures which are reliable, I hope, though I insist it is still a very intricate problem.

Yet physiologists have confirmed, beyond doubt, functional interrelation between the cortex and the medulla: (*a*) adrenaline stimulates the secretion of corticosteroids, (*b*) hydrocortisone increases the peripheral effects of adrenaline (Van Cauwenberghe *et al.*, 1959). This somehow throws the limelight, although in an indirect way, on the action of adrenaline on vessels (Fig. 10).

I can show you a very different condition in which vasospastic attacks are also accompanied by a tremendous increase of the plasmatic 17-hydroxycorticosteroids; among stress-inducing agents, cold is classically

admitted as being one of the most potent stimulants of cortical activity. Here is the case of a woman, 35 years old, sent to me as a Raynaud's disease; as a matter of fact, it was a true case of hypersensibility to cold: immersion of one forearm in ice-cold water brings about all signs of hyperhistaminaemia: redness of the face, tachycardia, hypotension, headache, etc.: gastric pH rises from 1 to 5, histaminaemia from 17 mg. per cent. to 25 mg. per cent., and plasmatic 17-hydroxycorticosteroids jump from 30 to 300 mg. (Fig. 11).

According to what we have seen from the last arguments discussed, it seemed logical to suppress at one stroke the source of mineralo-corticoids, the adrenaline, nor-adrenaline and their noxious vasoconstrictive effects in a disease of which the dominant feature is, most emphatically, spasm.

There must be an intermittent functional origin to explain the acute outbreaks and remissions of the disease, in spite of grossly established obliterations, and thus one is inclined to relate them to a hormonal cause.

There is another point. As we said, it is admitted that sympathectomy has an indication in thromboangeiosis. It is also believed that sympathectomy hypersensitizes vessels to adrenaline (and nor-adrenaline), thereby doing more harm than good. I will, therefore, find more reasons for cutting off the supply of these noxious hormones. We somehow had a hint of this whilst observing the surprising effect of unilateral adrenalectomy after failure of sympathectomy. For the first time, we could hope to treat more than the secondary signs, and to get closer to the essence of the disease.

Was it ethical to suggest bilateral adrenalectomy in thromboangeiotic patients?

To start with, I would like to remind you that the whole world followed Huggins after he published his findings on 18 cases of total adrenalectomy in 1952. By now, we know of about 61 cases of bilateral adrenalectomies, total or sub-total, in thromboangeiosis—enough apparently to take a decision.

Before answering this question about total adrenalectomy, I would like to quote Telford (1937): " In a disease so variable in its rate of progress and its severity as thromboangeiosis, it is very difficult to judge the efficacy of any one treatment ". This is correct, but concerns a certain number of patients in whom the disease is of slow evolution, quiescent; but on the other hand, we know of many patients in whom the course of the disease is fast, implacable, leading to death through successive outbreaks of gangrene.

Therefore, it is our duty to do our utmost to help these patients, namely, we should try everything that is sensible and innocuous, even if we do not

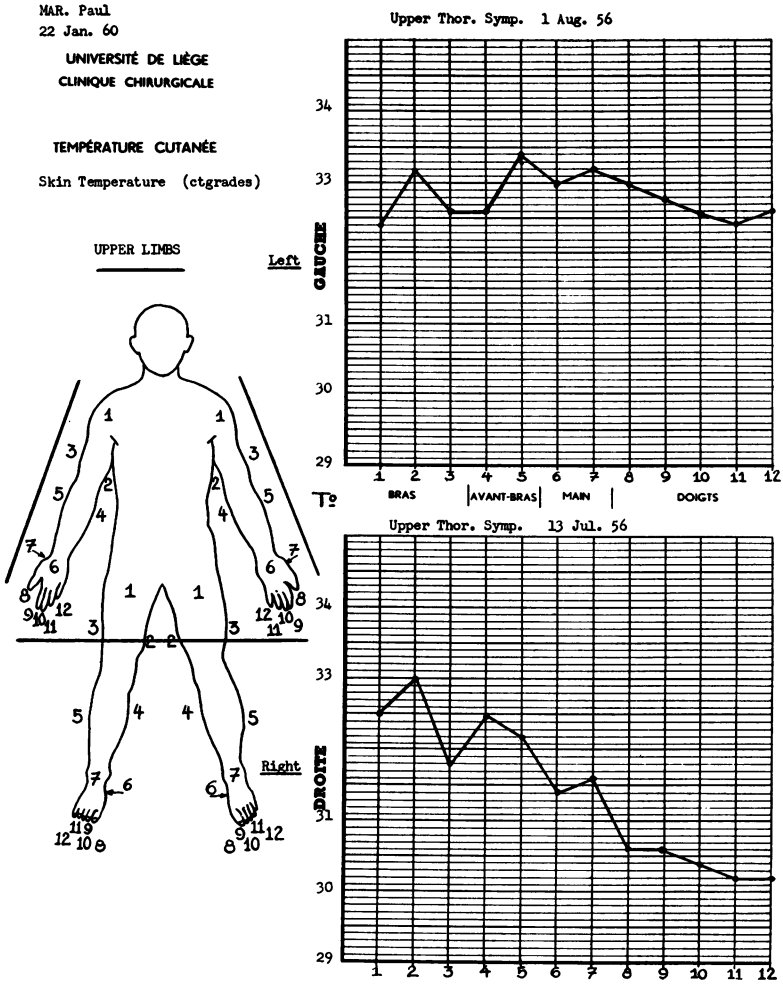

MAR. Paul
22 Jan. 60
UNIVERSITÉ DE LIÈGE
CLINIQUE CHIRURGICALE
TEMPÉRATURE CUTANÉE
Skin Temperature (ctgrades)
UPPER LIMBS
Left
Right
Upper Thor. Symp. 1 Aug. 56
GAUCHE
Upper Thor. Symp. 13 Jul. 56
DROITE
T°
BRAS
AVANT-BRAS
MAIN
DOIGTS

Fig. 12.

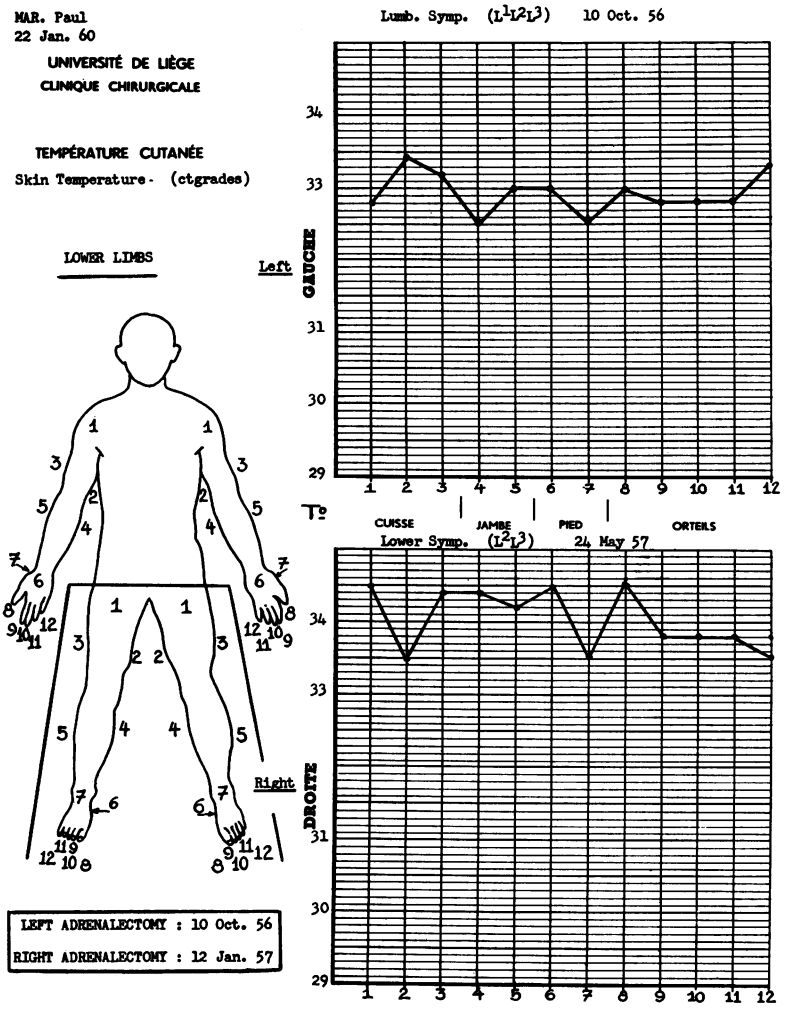

MAR. Paul
22 Jan. 60
UNIVERSITÉ DE LIÈGE
CLINIQUE CHIRURGICALE
TEMPÉRATURE CUTANÉE
Skin Temperature - (ctgrades)
LOWER LIMBS
Left
Right
LEFT ADRENALECTOMY : 10 Oct. 56
RIGHT ADRENALECTOMY : 12 Jan. 57
Lumb. Symp. (L1L2L3) 10 Oct. 56
GAUCHE
Lower Symp. (L2L3) 24 May 57
DROITE
CUISSE
JAMBE
PIED
ORTEILS
To

Fig. 13.

fully understand the whys and wherefores. Leriche wrote one day: " Any new operation which establishes a new order of things, arouses controversy which seeks the support of figures . . . that is the ransom of progress; even if these objections are stimulating, they are only verbal, non-constructive criticism."

In the last few years, our plan has been as follows: the first step, at the very first sign of the disease, is to try to convince the patient that he has to go through a series of operations, even if he thinks he is cured after two or three stages.

Technique

The full programme is: bilateral subtotal adrenalectomy (leaving $\frac{1}{2}$ gr. = $7\frac{1}{2}$ gr.) of the right one, with extensive sympathectomy of four limbs.

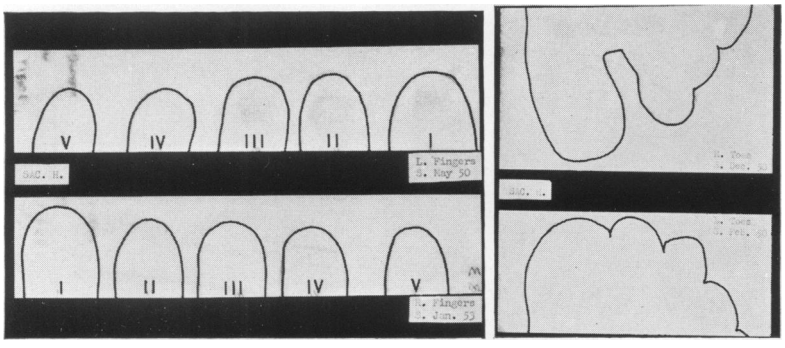


Fig. 14. (*a*) Ninhydrine test (Patient Sa . . ., No. 2, see Table I). Monolateral adrenalectomy. Absence of sudation in upper limbs seven years after upper thoracic sympathectomy. (*b*) (Patient Sa . . ., No. 2.) Absence of sudation in lower limbs 10 years after lumbar sympathectomy. Temperature of toes and fingers 32° C.

As a rule, when the patient is in pain, we start by doing a sympathectomy on the side involved, i.e. left lumbar sympathectomy L_1, L_2, L_3, combined with a left total adrenalectomy (retroperitoneal approach); then, according to the prevalent symptoms, right lumbar sympathectomy (L_2, L_3, left upper thoracic sympathectomy, posterior approach), right sub-total adrenalectomy plus right upper thoracic sympathectomy. We usually perform the last stage this way: the right thorax is opened between the 7th and 9th ribs, a right upper sympathectomy is done intrapleurally; then we open the diaphragm parallel to the rib to find the suprarenal beautifully exposed from above. This technique enables us to dissect the gland very gently, without any pressure or pull; this is how we make sure of the amount of gland (6–8 gr.) we wish to leave behind with an undamaged

blood supply. For the posterior approach for upper thoracic sympathectomy (extra-pleural), we use the sitting position. The patient being on a neuro-surgical table, the process is very easy, and he hardly bleeds at all.

We think it is advisable to group operations over a rather short period (eight to 10 months). When one waits too long between operations, results are less spectacular.

You will, not without reason, raise the objection: " If you are consistent with yourself, you should do a total adrenalectomy and not leave a source of harmful hormones behind!" I think I can answer that: (*a*) on ethical

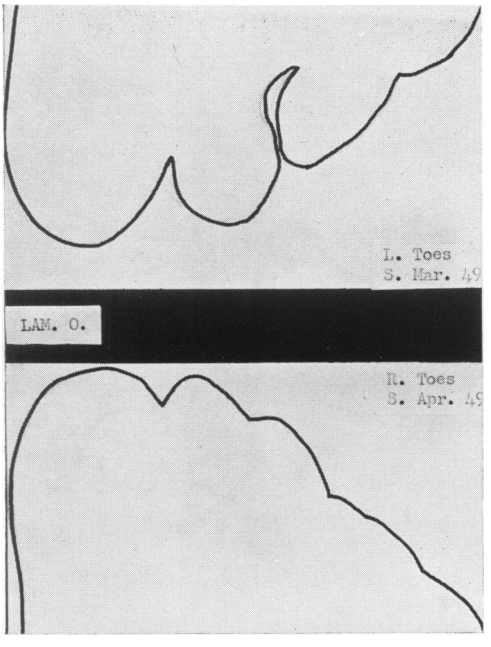


Fig. 15. Ninhydrine test (Patient Lam . . ., No. 4). Bilateral subtotal adrenalectomy. No sudation 11 years after lumbar sympathectomy.

grounds, we do not dare impose on young individuals such a drastic operation. As Ferrand has said, " most of them have to remain ' outpatients ' of the hospital for ever ".

(*b*) If the remnant of the gland left behind is just enough to take care of ordinary daily needs, it cannot be considered as an appreciable source of noxious secretions. As a matter of fact, all our patients are on the verge of Addisonism, as proved in our charts. In case of minor stress (overwork or a mild attack of influenza), they have to compensate with 5 or 10 mg. of hydrocortisone.

TABLE I

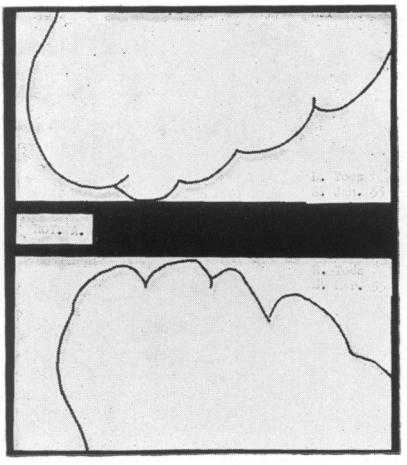
Names	*Age*	*Sympathectomies*								*Adrenalectomies*				*Cortisone Daily Intake*
		Upper				*Lumbar*								
		L		*R*		*L*		*R*		*L*		*R*		
1. Deb.	32					XI	49	X	49	XI	49			
2. Sac.	40	V	50	I	53	II	50	XII	50	II	50			
3. Pid.	43					III	56			III	56			
4. Lam.	46	V	59	VIII	59	III	49	IV	49	V	59	VIII	59	Hyd. Cort. 10 mg.
5. Rot.	31					I	55	III	55	I	55	III	55	0 occ.
6. Pro.	37					IV	56	IX	55	IV	56	X	57	0
7. Mar.	29	VIII	56	VII	56	X	56	V	57	X	56	I	57	0 occ.
8. Thi.	36	I	60	V	59	I	57	XII	56	I	57	V	59	0 occ.
9. Hol.	34	X	58	I	60	XII	56	III	58	XII	56	II	57	0
10. Bat.	44	XI	57	I	58	VII	58			VII	58	X	58	25 mg.
11. Moe.	34					I	58	XII	57	I	58	III	58	0
12. Ste.	42			XII	59	IV	58	X	59	VIII	58	XII	59	0
13. Lem.	26	III	59	IV	59		49		49	III	59	IV	59	Hyd. Cort. 15 mg.

TABLE II

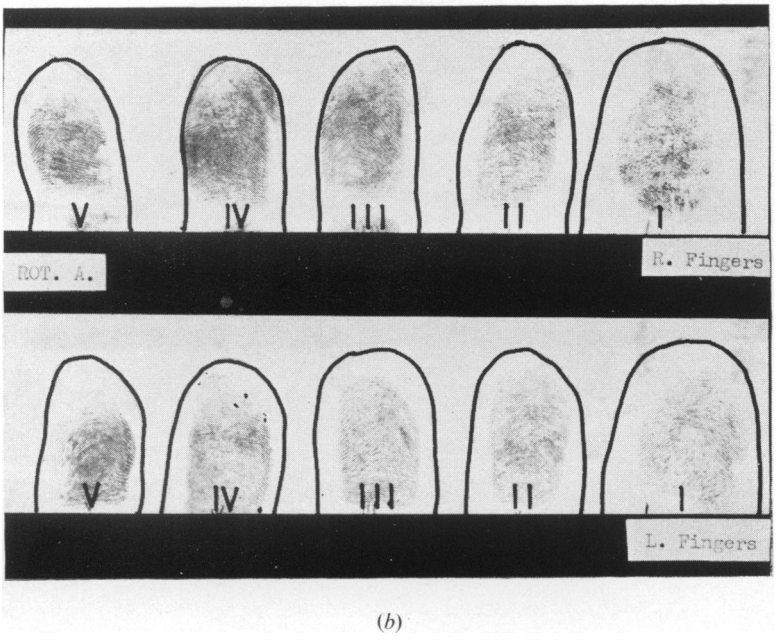
GANGRENES AND ULCERATIONS
(on admission)

		Upper Limb	*Lower Limb*	*Present result*
1.	Deb.		ulceration first R. toe	healed (still smoking)
2.	Sac.		ulceration third L. toe	healed
			gangrene second L. toe	amputation (second toe)
3.	Pid.		gangrene first L. toe	amputation (first toe) (still smoking)
4.	Lam.		gangrene third L. toe	amputation (third toe)
5.	Rot.		gangrenous patches on several L. toes	healed
			gangrenous patch first R. toe	healed
7.	Mar.	gangrene second R. finger ulceration third L. finger	gangrenous patches on several toes of both feet	healed
9.	Hol.		gangrenous patches on first L. toe, fifth L. toe	healed (still smoking)
10.	Bat.	gangrene on tips of second/third L. fingers; fourth/fifth R. fingers		healed with pulpar atrophy
11.	Moe.		ulceration first R. L. toe	healed
13.	Lem.		gangrene first L. toe, fifth R. toe	amputation left foot healed temporarily amputation right leg

N.B. Seven patients suffered from acrospasms of the upper limbs.



(*a*)

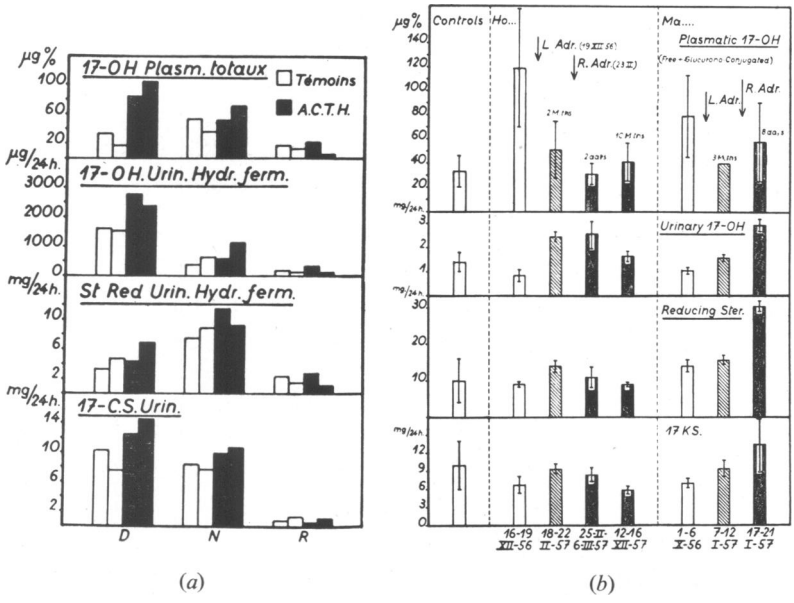


(*b*)

Fig. 16. (*a*) (Patient No. 5, Ro . . .) Bilateral subtotal adrenalectomy. No sudation of the toes five years after lumbar sympathectomy. (*b*) (Patient No. 5) Hypersudation of fingers (non-sympathectomized).

(*c*) One would say that the taking of one or two tablets of cortisone is not a real inconvenience; still, it is better to do without it. Only very occasionally do three patients out of ten have to take a minimal dose; only their follow-up will allow us to decide.

We have collected in recent literature 61 cases of thromboangeiosis treated by bilateral adrenalectomy (total or sub-total) combined with a variable percentage of sympathectomies. Results are generally good in selected cases operated on early enough.



(*a*) (*b*)

Fig. 17. (*a*) Variation of plasma and urine rates of corticosteroids during the two control days and during administration of ACTH (25 mg. in intravenous perfusion during six hours two consecutive days) in a control and in two patients with suprarenal deficiency. (*b*) Average rate of total plasma 17-hydroxycorticosteroids (free + glucurono-bound), of 17-hydroxycorticosteroids, of reductor steroids and of the urine 17-ketosteroids in controls and in patients with Buerger's disease. White columns correspond to average rates previous to any operation; hatched columns correspond to average variable delays after second suprarenalectomy.

Looking carefully through them, one can see that most failures occur in too advanced cases, almost ready for amputation; all operative deaths occurred in total bilateral adrenalectomies.

Table I shows the full list of the 13 patients operated on (39 sympathetic operations, 23 adrenalectomies); all are well proven cases of Buerger's disease; all are still alive. Actually, patient No. 13 should be considered apart. We saw him 10 years after the first outbreak of gangrene. Three years after amputation of the foot, there was no return of the blood in the main vessels below the obliterations. As a matter of fact, he should

not have been operated on at all, but he insisted on something being done to relieve the torture. Since then he has had two more amputations, but the pain has lessened a good deal and he does not receive morphia any more.

The first group consists of unilateral adrenalectomies.

No. 1. When admitted 11 years ago had pre-gangrene of the right big toe, and he had been sleeping with his feet hanging down for six months previous to the operation.

No. 2. Treated ten years ago as an emergency case screaming with pain in the left hand. There was ulceration of the third left toe.

No. 3. A Jewish doctor, treated for years for confirmed gangrene of the big left toe, although I believe he should be operated on for the right foot, at least.

These three patients are doing well, and do not wish for a further operation.

All the others are bilateral adrenalectomies (sub-total, 6/7 gr. of right gland remaining): seven patients out of nine, under normal circumstances, do not take any more cortisone; No. 4 will be taken off it this month. No. 10 is the only patient who will still receive cortisone, for steroid dosage has shown that the remannt of gland very likely does not secrete at all.

No. 9. Was a very indocile patient. When we managed to see him again 10 months after the second adrenalectomy, he was taking erratic doses of cortisone, and had developed tuberculosis, which responded well to treatment. He was able to undergo a lumbar and two upper thoracic sympathectomies a few months later. Now, he seems cured.

On admission, all patients but one had ulcerations somewhere. All had vascular trouble in the lower limbs, seven out of 12 had signs of involvement of the upper extremities (Table II).

Table III shows the surprising effects of the second adrenalectomy on the pain, within a few hours after operation. Eight patients out of 10 spontaneously said the pain had gone. Another inexplicable fact is that

TABLE III

Names		*Modifications following the Second Adrenalectomy*
4.	Lam.	Terebrating rest pain has disappeared in both feet.
5.	Rot.	Pain and cramps in left and right legs gone.
6.	Pro.	Disappearance of pain in left foot.
7.	Mar.	Disappearance of pain in right lower limb of six months' duration (non-sympathectomized side).
8.	Thi.	Disappearance of pain and swelling in the left upper limb (non-sympathectomized).
9.	Hol.	Complete disappearance of pain in the lower limbs, though the right lower limb was not sympathectomized.
10.	Bat.	Feels very depressed (due to total adrenalectomy).
11.	Moe.	No change.
12.	Ste.	No more pain in the right leg. No more cramps in the left leg.
13.	Lem.	Total suppression of pain in both lower limbs.
SUMMARY		Out of 10 cases—eight patients felt a definite improvement after the second adrenalectomy. Five patients (Ste., Lam., Hol., Pro., Lem.) had pain in sympathectomized limbs which was relieved by adrenalectomy.

the same improvement was observed in limbs not relieved by a previous sympathectomy!

TABLE IV

			Sweat Test (Feb. 1960) (Ninhydrine reaction) Upper Limbs				Temperature (Feb. 1960) (Centigrade) (1st toe — 2nd finger) Lower Limbs			
			L	*Temp.*	*R*	*Temp.*	*L*	*Temp.*	*R*	*Temp.*
MONOLATERAL	1.	Deb.	+	28.2	+	24.4	– S. Nov. 49	29.3	– S. Oct. 49	29.9
	2.	Sac.	+ S. May 50	32.2	– S. Jan. 53	31.1	± S. Feb. 50	32	± S. Dec. 50	33.4
	3.	Pid.	+	31.6	+	31.6	– S. Mar. 56	5th toe 29.8	+	26.4
	4.	Lam.	+ S. May 59	27	± S. Aug. 59	27.8	– S. Mar. 49	29.3	– S. Apr. 49	30
	5.	Rot.	+ +	30	+ +	30	– S. Jan. 55	24.8	– S. Mar. 55	19.4
BILATERAL	7.	Mar.	– S. Aug. 56	32.8	– S. Jul. 56	30.6	– S. Oct. 56	33	– S. May 57	34.6
	8.	Thi.	– S. Sep. 59	30	– S. May 59	28.5	– S. Jan. 57	31.5	– S. Dec. 56	32.2
	9.	Hol.	± S. Jan. 60	34	– S. Oct. 58	33	– S. Dec. 56	32.6	– S. Mar. 58	32.6
	10.	Bat.	– S. Nov. 57	32.6	– S. Jan. 58	28.4	– S. Jul. 58	33.8	+ +	23.6
	11.	Moe.			+	30.2	– S. Jan. 58	32.8	– S. Dec. 57	33.2
	12.	Ste.	+	31.2	– S. Dec. 59	34.3	– S. Apr. 58	34	– S. Dec. 59	34.4
	13.	Lem.	– S. Mar. 59	29.6	– S. Apr. 58	29.2				

S. : Sympathectomized

++ : heavy sweating
+ : sweating

± : traces
– : absence of sudation

Table IV demonstrates the sweating test recorded quite recently; it shows very clearly that in our series the effects of sympathectomy are amazingly prolonged: seven patients still have a temperature of 30° C. (normal) or more at all four extremities (Figs. 12 and 13). Of 37 sympathectomized limbs, only one sweats almost normally, and five exhibit traces of sudation. I earnestly think that these observations will call for further comments on the efficacy of sympathectomy and perhaps too

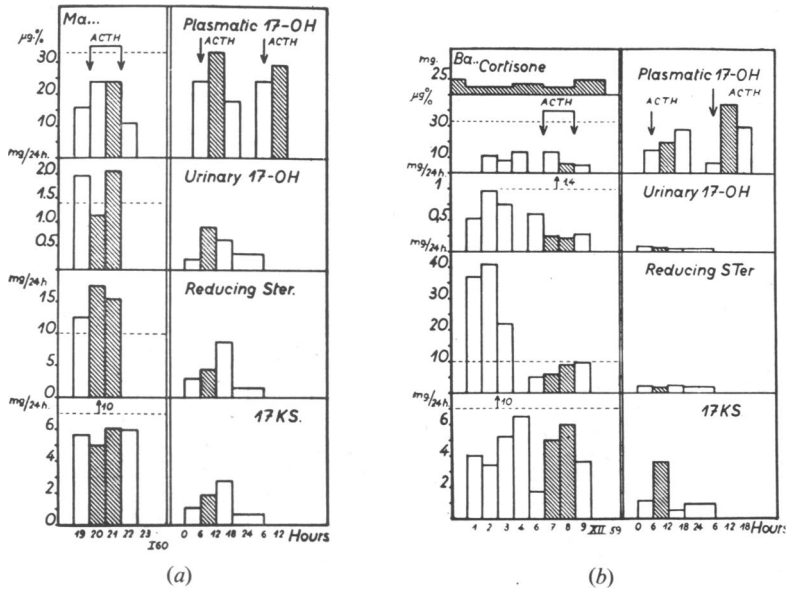


Fig. 18. (*a*) (Refers to case No. 7, see Table I.) Corticosteroids output three years after second subtotal adrenalectomy. Similar results have been found in all patients treated by the technique described. As in preceding figures, the left part of the slide corresponds to dosages done during the 48-hour test intravenous perfusion of ACTH two consecutive days, with collection of urines over 24 hours, and blood sampling every morning on fasting patients. White columns correspond to periods before and after ACTH perfusions and hatched columns to the two days during perfusion. On the right part of the illustration are shown the results of six-hour tests, and the urines collected every six hours before and during the eighteen hours following ACTH perfusion (white column), and during this perfusion (hatched column). Blood samplings are done immediately before the perfusion and six hours after (white columns), as well as at the end of the perfusion. The dotted line corresponds to the average rate observed in the controls. Attention is drawn in this figure to the increase of corticoiduria on the second day of the 48-hour test and to the transitory increase of corticoidaemia during the six-hour test. (*b*) (Patient No. 10, see Table I.) In this case, cortisone could not be taken off because of an *involuntary total adrenalectomy*. Daily dose administered is shown on the top left. Administration of ACTH induces no significant increase of corticoiduria or of basal corticoidaemia during the 48-hour test. During the six-hour test, no significant increase of urinary steroids is seen. After second ACTH perfusion, there is a very slight increase of corticoidaemia.

TABLE V

		Names	Weight (Kgs.) Before operation		Weight (Kgs.) Present		Cortisone Daily intake	Profession	Working capacity
MONOLATERAL	1.	Deb.			65.5	(Feb. 60)		Decorator	full
	2.	Sac.	78	(Jan. 53)	81.5	(Jan. 60)		Carpenter	full
	3.	Pid.	75 ±	(Dec. 56)	74.5	(Feb. 60)		Doctor	full
BILATERAL	4.	Lam.	63	(Apr. 49)	81	(Aug. 59)	Hydrocort. 10 mg.	Post clerk	full
	5.	Rot.	55	(Jun. 55)	65	(Jan. 60)	none (occasionally Hydrocort. 10 mg.)	Workman (scrap iron)	full
	6.	Pro.	60	(Nov. 57)	66	(Apr. 58)	none	Shop assistant	full
	7.	Mar.	59	(Jul. 57)	62	(Jan. 60)	none (occasionally Hydrocort. 10 mg.)	Test driver	full
	8.	Thi.	85	(Jan. 57)	98	(Jan. 60)	none (occasionally Hydrocort. 10 mg.)	Workman (electric soldering)	full
	9.	Hol.	72	(Dec. 57)	84	(Jan. 60)	none	Workman	
	10.	Bat.	72	(Jan. 58)	69	(Nov. 59)	Cortisone 25 mg.	Forester	reduced
	11.	Moe.	62.5	(Jan. 57)	70	(Feb. 60)	none	Bookbinder	full
	12.	Ste.	80	(Apr. 58)	83.5	(Feb. 60)	none	Workman	reduced

on the means of improving it with adrenalectomies (Figs. 14 (*a*) and (*b*), 15, 16). One patient, No. 10, had lost weight (7 lb.); he is the one who had an involuntary total adrenalectomy. All the others had put on weight (10 to 20 lb.). All patients, except No. 2, have resumed their previous occupations (Table V).

Conclusions (of steroid dosage) (Fig. 17 (*a*)).

1. In cases in which we are able to have analyses done before adrenalectomies, there is a tremendous increase of 17-OH steroids (three to four times more than normal); unfortunately the figures are too few in number to be statistically valid (Fig. 17 (*b*)).

2. In all cases tested after sub-total adrenalectomies, 17-OH steroids and 17-ketosteroids have been found permanently well under the mean concentration (Fig. 18 (*a*)).

3. After adrenalectomies, repeated and prolonged dosages have shown that ACTH stimulation cannot increase 17-OH steroids and 17-ketosteroids up to a subnormal level. This is evidence that the secretory potentialities of the minute fragment left behind (½ gr.) is really minimal, just enough to maintain the patient's balance, and it is very likely innocuous (Fig. 18 (*b*)).

4. Even after a few years' follow-up, we have registered neither chemical nor clinical signs of compensatory hypersecretion of the residual fragment of the adrenal.

Results

None of the 12 patients considered has developed new outbreaks of gangrene at all; except for two toes ready to separate on admission (1950–1956), no amputation was required. Acrospasms have disappeared, pain has gone, ulcerations healed within a few weeks. The 12 of them had intermittent claudication before operation; now, five are still complaining of it when they walk briskly; seven can walk almost normally. All have put on weight; nine have returned to their previous occupations; three are still smoking. Only one mentioned " dry intercourse ". They did not have any sympathetic neuralgia.

Conclusions

As for many other diseases, we are still at a loss to understand the causes of thromboangeiosis. Apparently, the summing up of pathological, experimental and clinical facts seems to purport that adrenals are one of the links in the pathogenesis of Buerger's disease. So far, bilateral adrenalectomy (even sub-total), combined with extensive sympathectomy, appears to have fully relieved patients for some time.

I want to make it clear that the follow-up has been short, and that these results may be challenged. We know that our point of view may be shaken by further observation; yet this is a dreadful disease, and it is worth while repeating experiments.

I would like to thank you once again for the great honour you have done me, and for the patience and attention with which you have listened to my talk.

ACKNOWLEDGMENTS

I wish to acknowledge with thanks the personal reports and valuable comments I received from Aimes (Montpellier), Delannoy (Lille), Ferrand (Alger), Fontaine (Strasbourg), Govaerts (Bruxelles), Kunlin (Paris), Pettinari (Padova), Tinguad (Bordeaux) and Wertheimer (Lyon).

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Finally, special appreciation is extended to my assistant, L. van de Berg, for the great care he took following up the patients.

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HONOURS CONFERRED ON FELLOWS AND MEMBERS

IN THE RECENT New Year Honours List the following Fellows and Members were graciously honoured by Her Majesty The Queen.

DENIS JOHN BROWNE, F.R.C.S.	K.C.V.O.
CHRISTOPHER HOWARD ANDREWES, F.R.S., M.D., F.R.C.P., M.R.C.S.	Knight Bachelor
BRIAN WELLINGHAM WINDEYER, F.R.C.S., F.R.C.S. Ed., F.F.R.	,, ,,
JAMES CECIL HOGG, F.R.C.S.	C.V.O.
FRANK LEO HUTTER, F.R.C.S.	C.B.E. (Milit.)
RONALD BRAMBLE GREEN, F.R.C.S.	C.B.E. (Civil)
CHARLES HORNER GREEN MACAFEE, F.R.C.S.	,, ,,
HARVIE KENNARD SNELL, M.D., M.R.C.S.	,, ,,
TERENCE GEORGE WARD, M.B.E., F.D.S.R.C.S.	,, ,,
ARTHUR WARRINER WILLIAMS, M.D., F.R.C.P., M.R.C.S.	,, ,,
ARTHUR GORDON WATERMAN BRANCH, M.D., M.R.C.S.	O.B.E. (Civil)
RONALD GEORGE GIBSON, M.R.C.S.	,, ,,
RONALD BRODIE HEISCH, M.D., M.R.C.S.	,, ,,
JOHN LEWIN, F.R.C.S., J.P.	,, ,,
KEITH DIGBY YOUNG, M.R.C.S.	,, ,,
THOMAS TURNER, V.R.D., M.R.C.S.	M.B.E. (Civil)

APPOINTMENT OF FELLOWS AND MEMBERS TO CONSULTANT POSTS

R. B. CHATTOPADYAYA, F.R.C.S.	Reader in Surgery, Calcutta Medical College.
D. P. GREAVES, F.R.C.S.	Consultant Ophthalmologist, Moorfields Eye Hospital.
R. J. H. SMITH, F.R.C.S.	Consultant Ophthalmologist, Moorfields Eye Hospital.
P. D. TREVOR-ROPER, F.R.C.S.	Consultant Ophthalmologist, Moorfields Eye Hospital.