DIGITAL ARTERITIS IN RHEUMATOID DISEASE*

BY

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Three broad types of vasculitis have been described in rheumatoid arthritis:

- (1) Subacute lesions of small vessels found in muscles by Sokoloff, Wilens, and Bunim (1951), and in the heart, muscles, and nerve sheaths by Cruickshank (1954).
- (2) Cases of rheumatoid arthritis complicated by severe widespread necrotizing arteritis affecting large vessels and indistinguishable from polyarteritis nodosa (Aronoff, Johnson, and Dworkin, 1960; Ball, 1954; Graef, Hickey, and Altmann, 1949; Levin, Rivo, Scott, Figueroa, Fred, and Barrett, 1953; Nyström, 1953; Ogryzlo, 1953; Schmid, Cooper, Ziff, and McEwen, 1961).
- (3) Bywaters (1957) has described patients with digital arterial narrowing due to obliterative endarteritis with evidence of visceral arterial involvement in two autopsied patients. This last type appears morphologically distinct from polyarteritis nodosa, consisting of a bland, sometimes mucoid, intimal proliferation. However, in each of the two autopsied cases described, there were in addition certain arteries with evidence of acute inflammatory disease, past or present, as well as a few arteries with secondary thrombosis and recanalization.

The relation between these three types of lesion is still far from clear.

Virtama (1959) carried out *post-mortem* brachial arteriography in cases of rheumatoid arthritis, and concluded from this that the characteristic alterations were local obliteration and post-stenotic

dilation of the digital arteries with arterial dilation near bone erosions.

This paper describes a study of digital lesions in selected patients with rheumatoid arthritis, using the radiological technique of brachial arteriography and correlating this with clinical findings, reactive hyperaemia, and, in some instances, with autopsy or biopsy material.

Methods

To perform brachial arteriography 10 ml. 45 per cent. "Hypaque" are rapidly injected into the brachial artery at the elbow. In the first few patients a general anaesthetic was given and the flow of injected "Hypaque" was controlled by intermittent inflation of a blood-pressure cuff while the films of the hand were obtained (Lynn, Steiner, and Van Wyk, 1955). We now usually give only a local anaesthetic at the site of the injection and dispense with the cuff, taking a rapid series of films at roughly one-second intervals for 8 seconds. No complications have been encountered; there is sometimes mild pain in the hand during the procedure; in one patient there was painful spasm of the brachial and smaller arteries for about 2 minutes.

Reactive hyperaemia (Pickering, 1933) was observed in the warm exsanguinated limb after 4 minutes' ischaemia, delayed flushing in the various digits being timed.

Results

We have performed arteriograms in ten adult patients with rheumatoid arthritis in whom there was some reason to suspect the presence of digital arterial disease. In all ten the arteriogram was abnormal. We have also performed *post-mortem* arteriography and histology in a further case. Because of these abnormal films, arteriography was carried out in two other patients with early rheumatoid arthritis in whom there was no indication of

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arteritis, and in both of them the arteriogram was normal. Fig. 1 shows the film of one of these, a woman aged 45 who had had rheumatoid arthritis for one year. The features of this normal arteriogram are the smooth even calibre of the arteries and the presence of two palmar digital vessels in each digit, though the two vessels in any one digit are not always of equal size; in the thumb, index, and little fingers, the median artery is often predominant (Edwards, 1960). The small vessels of the pulps are equally well filled.

Fig. 1.—Normal brachial arteriogram in a patient with early rheumatoid arthritis.

Case Histories

Case 1, a man aged 57, had rheumatoid arthritis which started in 1956, and developed extensive nodule formation. In April, 1957, treatment was commenced with prednisolone 15 mg. daily. In September, 1958, he developed pain in the right leg and was found to have an absent right ankle jerk and impaired cutaneous sensation over the dorsum and outer aspect of the right foot. At the same time small ischaemic lesions first appeared on all the digits of both hands, coming subsequently in recurrent crops (Fig. 2). In April, 1959, the index and middle fingers of the right hand were noticed to be cool, and reactive hyperaemia was delayed in these two fingers

Fig. 2.—Case 1. Digital ischaemic lesions.

(25 and 40 sec.). The differential agglutination titre (D.A.T.) was positive, 1 : 32. Brachial arteriography (Fig. 3) showed many arterial blocks, in both the hand and the fingers. None of the digital vessels was intact except the median artery of the thumb, and this was abnormally tortuous. Of the finger vessels, the most nearly normal was the radial artery of the ring finger, but even here there was interruption and collateral circulation at the level of the middle phalanx.

Fig. 3.—Case 1. Brachial arteriogram.

Case 2, a man aged 50, had rheumatoid arthritis which started in 1952. In 1958, while on salicylate treatment only, he first noticed transient blanching, on exposure to cold, of any of the middle three digits of either hand. In July, 1960, because of progressive arthritis, with nodule formation, treatment with prednisone 10 mg. daily was commenced. The D.A.T. was positive, 1 : 64. In October, the second, third and fourth digits of the left hand became permanently cold and rather blue; thrombotic lesions appeared at the nail-edge. Impaired sensation was now noticed over the dorsum of the left foot. Reactive hyperaemia was slightly delayed in the three involved fingers (3 sec. compared with 1 sec. in the other digits). An arteriogram showed a number of severe abnormalities including a stenosed segment of radial artery and occlusion of all the digital vessels except the ulnar digiti quinti (Fig. 4a) and those of the thumb, and only in these two digits was any pulp filling seen.

A biopsy was taken of the occluded radial digital artery of the middle finger (by Mr. S. Harrison). Proximally, at about the middle of the proximal phalanx, the vessel showed thickening of the intima with no inflammation of the intima or adventitia, the lumen being reduced to a slit. Section a few millimetres distal to this point revealed total occlusion with fresh fibrin thrombus and similar intimal thickening around the thrombus. Scanty neutrophil polymorphonuclear leucocytes and histiocytes were present at the periphery of the thrombus

and in the adventitia. Further distally again (Fig. 5) there was intimal thickening with organizing thrombus

Fig. 5.—Case 2. Digital artery. The elastica is interrupted with prominent capillaries in these areas. The intima is thickened with organizing thrombus in the reduced lumen. Elastic and van Gieson $\times 75$.

Fig. 4.—Details from arteriograms:

(a) Case 2. Little finger. (b) Case 4. Ring finger. (c) Case 6. Index finger. (d) Case 8. Middle finger. (e) Case 11. Ring finger.

within the reduced lumen. The elastica had undergone segmental interruption, and capillaries were prominent in the areas of elastic deficiency.

Case 3, a woman aged 55, had nodular rheumatoid arthritis which started in 1955. After various forms of therapy, treatment with prednisolone was started in 1956, the dosage subsequently ranging between 10 and 45 mg. daily, according to disease activity. The only clinical sign of digital arterial disease was the appearance in 1960 of nailfold thrombotic lesions in the index, middle, and ring fingers of each hand. Reactive hyperaemia was normal. The D.A.T. was positive, 1 : 1,024. Arteriography (Fig. 6a) showed a constant deformity at the radial end of the deep palmar arch; the radial pollicis was obliterated 2 mm. from its origin and there was segmental blocking of the ulnar pollicis. The radial indicis and ulnar digiti quinti were also incomplete and in each case a collateral artery was seen crossing the middle phalanx. Pulp filling was, however, present, though a little unequal.

Case 4, a man aged 49, had had rheumatoid arthritis for 20 years. Rheumatoid nodules were present. The D.A.T. was positive, 1 : 256. Treatment with cortisone 87.5 mg. daily was started in May, 1957, and changed in May, 1958, to prednisolone 15 mg. daily. The only suggestion of vascular disease was his complaint (on direct questioning) that during the winter of 1959-60 the right foot was sometimes colder than the left. There were no symptoms referable to the hands, which, apart from the arthritis, were clinically normal, as was the reactive hyperaemia. There was, nevertheless, occlusion of the ulnar vessels of the ring and little fingers, and impaired pulp filling in the ring finger (Fig. 4b).

Case 5, a man aged 80, had rheumatoid arthritis which started about 1955. Treatment with prednisolone, 10 mg. daily, was started in 1957. In 1958, 2 months after withdrawal of the drug, there was an acute flare of the arthritis, accompanied by pericarditis; this was treated with cortisone 100 mg. daily, reverting to prednisolone 20 mg. daily in February, 1959. He

Fig. 6.—Parts of arteriograms.

(a) Case 3. Arrows indicate a deformity at the radial end of the deep palmar arch, obliteration of the radial pollicis artery 2 mm. from its origin and segmental blocking of the ulnar pollicis.

(b) Case 9. Obliteration of digital arteries and (arrowed) ulnar artery

subsequently developed subcutaneous nodules. The D.A.T. was positive, 1 : 256. In June, 1959, he complained of numbness of the feet which were cold with weak but not absent dorsalis pedis and posterior tibial pulses. Tactile and vibration sense was impaired in the feet and later there was loss of knee and ankle jerks. In August, 1960, an ischaemic lesion was noted on the right index finger. Sensory impairment developed in the fingers with purpura on the wrists. Reactive hyperaemia was normal. Arteriography showed severely impaired blood supply to all the digits except the little finger, and in that finger tortuosity and irregular calibre indicated arterial disease. A biopsy taken from the radial side of the index finger did not, unfortunately, include the main digital artery, but some small vessels showed collagenous intimal thickening, while others appeared normal.

Case 6, a woman aged 43, had had rheumatoid arthritis for one year only, which was remarkable for the striking involvement of the flexor tendons of the fingers, inflammation of which caused the skin to become tethered down, so as to present a picture not unlike scleroderma. The diagnosis of rheumatoid arthritis was, however, confirmed by clinical and radiological evidence of primary joint involvement and erosion of subchondral bone, and was supported by the fact that the patient's identical twin had typical sero-positive rheumatoid arthritis. The patient suffered with cold hands on exposure to cold, but in a hot environment they became pink and warm. Reactive hyperaemia was normal. In addition, she had a sensory neuritis involving the 4th and 5th fingers of the left hand, and also the trigeminal nerve on one side. Another unusual feature was the well-attested history of malignant hypertension with papilloedema 2 years previously, which resolved spontaneously. No nodules were observed in this patient. The D.A.T. was positive, 1 : 16. The arteriogram, performed before treatment with prednisolone was started, showed some of the digital arteries to be irregular and tortuous, with occlusion of the radial index and ulnar digiti quinti, and a collateral vessel crossing the second middle phalanx (Fig. 4c). Pulp filling was deficient.

Case 7, a man aged 51, had a 20-year history of uncomplicated rheumatoid arthritis. In September, 1960, he developed bilateral pleural effusions for which no cause was found other than the rheumatoid disease, together with patchy erythema on the trunk with areas of depigmentation and atrophy. The nature of the rash was uncertain, although it bore some resemblance to both lupus erythematosus and dermatomyositis. At the same time his hands became intermittently blue and cold, together with an exacerbation of his arthritis. Thrombotic fingernail lesions were now noted, but reactive hyperaemia was normal. There were no nodules. The D.A.T. was positive, 1 : 128. L.E. cells were not found. He had not received steroid treatment. The arteriogram showed digital artery obstruction with

inadequate pulp filling, and a collateral vessel crossing the middle phalanx of the index finger.

Case 8, a man aged 54, had a 12-year history of rheumatoid arthritis and subsequent diffuse pulmonary fibrosis proceeding to "honeycomb lung". Rheumatoid nodules were present. The D.A.T. was positive, 1 : 256. Treatment with prednisolone 15 mg. daily was started in September, 1959, because of increasing dyspnoea, but the patient died in November, 1960, with pulmonary insufficiency. Digital arteritis was not diagnosed during life, and reactive hyperaemia had been normal, but arterial disease was shown by arteriography performed after death (Fig. 4d). (There was more small vessel filling than in arteriograms done during life, which was due to loss of arterial tone.) There was blockage of several large arteries. Histology at several levels of each main vessel from the 2nd and 3rd fingers showed fibro-elastic thickening of the intima and some reduplication of the elastica, which was incomplete (Fig. 7a and b, opposite).

There had been no clinical suggestion of mesenteric arteritis, but Fig. 7c shows a mesenteric artery where a similar picture is seen.

Case 9 illustrates that digital vascular disease may proceed to gangrene. This 53-year-old man's nodular rheumatoid arthritis started in 1949, and in 1957 he developed a peroneal and ulnar neuritis, together with a macular erythematous and purpuric rash and skin nodules, biopsy of which showed perivascular infiltration with eosinophils and polymorphs suggestive of polyarteritis nodosa. Treatment with corticosteroid hormones (oral hydrocortisone, triamcinolone, and later methyl prednisolone) was then started. The following year he had transient episcleritis. The D.A.T. was positive, 1 : 128. In 1960, the left index finger became cold and blue, and the tip became gangrenous (Fig. 8, opposite).

Reactive hyperaemia was also delayed in the 3rd and 4th digits (4 and 7 sec. compared with 2 sec. in other digits). Brachial arteriography showed obliteration of the ulnar artery and of all the digital vessels except those of the thumb (Fig. 6b). The gangrenous finger-tip was subsequently amputated: Fig. 9 (opposite) shows the histological appearance of a digital artery.

Certain features of this case, namely the skin lesions with a paucity of erosive changes radiologically, indicate that one is here on the difficult terrain between classical rheumatoid arthritis and polyarteritis nodosa.

Polyarteritis nodosa may involve the digits. Fig. 10a (overleaf) shows the *post-mortem* arteriogram of the foot of a woman with polyarteritis nodosa presenting with neuritis, who later developed mesenteric vascular occlusion, from which she died. She did not have rheumatoid arthritis. The arteriogram demonstrates blockage of several vessels. Histology of the digital toe vessels shows fibrinoid necrosis in the walls associated with inflammatory cells (Fig. 10b).

Fig. 7.—Case 8.

- (a) Digital artery. Fibro-elastic, concentric intimal thickening, with areas of reduplication of internal elastic lamina. Elastic and van Gieson $\times 66$.
- (b) Part of digital artery, showing segmental interruption of elastica. Elastic and van Gieson $\times 132$.
- (c) A mesenteric artery. Concentric intimal thickening with occasional interruptions of elastica. There is an abnormally thick adventitia. Elastic and van Gieson $\times 132$.

Fig. 8.—Case 9. Gangrenous left index finger.

Fig. 9.—Case 9. Longitudinal section of digital artery. The lumen is a central, longitudinal slit, with laminated collagen on either side of it, in which new fibro-muscular elements can be seen on the left-hand side of the photograph. The original internal elastica has been interrupted (white arrow), and two incomplete laminae are visible inside it, separated from each other by loose collagen. Note capillary (black arrow) within the thick, intimal collagen. Elastic and van Gieson $\times 132$.

Fig. 10.

- (a) Post-mortem arteriogram of the foot of a patient with polyarteritis nodosa, showing blockage of several vessels.
 (b) Branches of digital artery from the same case. There is thrombosis in the lumina, fibrinoid necrosis of the mediae, and inflammatory infiltrate in the adventitia.
 Haematoxylin and eosin. $\times 100$.

The last two cases of rheumatoid disease also show diffuse arteritis and have striking clinical similarities.

Case 10, a man aged 48, had rheumatoid arthritis which started in 1953. In October, 1958, treatment with prednisolone 10 mg. daily was started, and in 1959 the patient developed sensory and motor peripheral neuritis of all limbs, episcleritis, and ulceration of the legs. Rheumatoid nodules were present. The D.A.T. was positive, 1 : 128. A single nail-edge thrombotic lesion was now seen, and reactive hyperaemia was delayed in several fingers up to a maximum of 20 sec., though the skin of the hands and fingers was warm and dry. The arteriogram (Fig. 11) showed considerable variation in arterial calibre and blockage of all the digital arteries, except the ulnar vessels of the thumb, middle, and ring fingers. Pulp filling was present in these three digits only. This patient subsequently died after two laparotomies for mesenteric arteritis, and at autopsy a recent myocardial and several recent renal infarcts were also present. Unfortunately digital vessels were not obtainable for histology. Fig. 12 (*a, b*, opposite) shows two mesenteric arteries, one with a bland intimal thickening, the other with fibrinoid necrosis of part of the wall associated with oedema and intense inflammatory reaction, the characteristic picture of polyarteritis nodosa. Both types of lesion were also seen in the adrenal, testis and coronary vessels.

Fig. 11.—Case 10. Brachial arteriogram.

Fig. 12.—Case 10.

- (a) Mesenteric artery, showing concentric intimal thickening, with interruption of internal elasticae Elastic and van Gieson $\times 31$.
 (b) Mesenteric artery, showing fibrinoid necrosis and inflammatory cells. Haematoxylin and eosin. $\times 50$.

Case 11, a man aged 59, had rheumatoid arthritis which began in 1951. In October, 1956, treatment was started with cortisone 75 mg. daily. In January, 1960, he complained of paraesthesiae in the right leg, and, in February, of impaired sensation in both feet, followed by bilateral foot drop. He was now found to be diabetic, with glycosuria and a fasting blood sugar of 229 mg. per cent., controlled by an 800-calorie diet and Lente insulin 20 units daily. Examination at this time showed severe generalized rheumatoid arthritis with subcutaneous nodules, peripheral neuritis involving the feet and hands, and haemorrhagic vesicles over the lower legs, but no digital vascular lesions. The D.A.T. was positive, 1:1,024. Reactive hyperaemia was slightly delayed in the fourth finger of the left hand only (4 sec., all other digits being 2 sec.).

Arteriography demonstrated some pulp filling in all digits, but the vessels showed abnormal variation in calibre and tortuosity, and there was some blockage in the radial artery of the index finger and both arteries of the ring finger (Fig. 4e); 10 days after this film was taken this patient also had mesenteric infarction necessitating excision of gangrenous intestine, which he fortunately survived. Fig. 13 shows the histology of a mesenteric artery.

Calf-muscle biopsy showed some foci of muscle

Fig. 13.—Case 11. Small mesenteric artery. The lumen is reduced, a second elastica is incompletely formed inside thickened intimal collagen, and the original elastica is interrupted. Inflammatory cells are largely in the adventitia. Elastic and van Gieson $\times 31$.

necrosis and collagenous intimal thickening in small arteries, with interruption of the internal elastic lamina.

Discussion

In this series of brachial arteriograms, no characteristic anatomical pattern of rheumatoid arteritis

has emerged. Any artery in the hand or digits may be involved, though the blood supply of the thumb appears to be less vulnerable than that of the fingers. The radiological lesions are irregularity, narrowing, or obliteration of the arterial lumen, sometimes with opening of collateral vessels. We have not observed post-stenotic dilation nor arterial dilation near bone erosions as reported by Virtama (1959), but his procedure of *post-mortem* arteriography, with magnification of the abundant filling obtained in small vessels, allowed more observation of detail than is possible with arteriograms performed during life.

The clinical features of our cases are summarized in the Table. The significance of these features is limited because it was in fact the presence of some of them which led to arteriograms being done. We seem to have encountered a preponderance of males (9 to 2) with digital arteritis. All the patients were over 40 years of age. The differential agglutination test was positive in all, sometimes strongly so, and nine of the eleven had subcutaneous nodules. L.E. cells were looked for in all the patients, but were not found. Ischaemic digital lesions were seen in only seven of the eleven cases and similarly only

five of the eleven had abnormal reactive hyperaemia: one of the most impressive findings of this study has been the demonstration of digital arteritis in some patients with clinically normal hands and normal reactive hyperaemia. Peripheral neuritis was found in seven cases, associated in all but one instance with ischaemic digital skin lesions. There was evidence of visceral involvement in seven cases, though in some instances the connexion with rheumatoid arthritis is questionable.

Most of the patients had received corticosteroids because of severe rheumatoid arthritis, but not Cases 6 and 7: in Case 2 the symptoms of digital ischaemia preceded steroid treatment. We cannot contribute to the problem of corticosteroid therapy as a possible aetiological factor in the development of arteritis except to confirm previous findings (Bywaters, 1957) that arteritis may appear in the absence of steroid treatment.

The histological appearances of the digital arteries suggest an intermittent, progressive collagen increase in the intima, with reduction of lumen, sometimes followed by total occlusion due to thrombosis. No acute arteritis was seen in any of these digital arteries, and although there was elastic

TABLE
CLINICAL FEATURES OF ELEVEN CASES OF RHEUMATOID ARTHRITIS WITH DIGITAL ARTERITIS
SHOWN BY ARTERIOGRAPHY

Case	Sex	Age (yrs)	Differential Agglutination Titre	Sub-cutaneous Nodules	Digital Lesions	Reactive Hyperaemia	Peripheral Neuritis	Visceral Involvement	Steroids (yrs)
1	M	57	1 : 16	+	+	Abnormal	+	0	2
2	M	50	1 : 64	+	+	Abnormal	-	0	4 mths*
3	F	55	1 : 1,024	+	+	Normal	0	0	4
4	M	49	1 : 256	-	0	Normal	0	0	3
5	M	70	1 : 256	+	+	Normal	-	Pericarditis	2
6	F	43	1 : 16	0	0	Normal	-	Previous hypertension with papilloedema	0
7	M	51	1 : 128	0	+	Normal	0	Pleural effusions	0
8	M	54	1 : 1,024	-	0	Normal	0	Pulmonary fibrosis Mesenteric arteritis	1
9	M	53	1 : 128	+	Gangrene	Abnormal	-	Rash Episcleritis	3
10	M	48	1 : 128	+	+	Abnormal	-	Leg ulcers Episcleritis Mesenteric arteritis Myocardial infarcts Renal infarcts	1½
11	M	59	1 : 1,024	+	0	Abnormal	+	Leg ulcers Mesenteric arteritis Muscle arteritis	3

* But Raynaud's phenomenon antedating steroid therapy.

interruption, this did not usually cover a large segment of the vessel. However, as pointed out formerly (Bywaters, 1957: Case 2), there may be signs of previous adventitial reaction consisting of circumferential deposits of haemosiderin almost all round the external elastic lamina together with the presence of capillaries within the media. Furthermore, the visceral arteries may show, in the same person, a bland obliterative endarteritis at one place, but occasionally acute inflammatory arthritis elsewhere (Cases 10 and 11). It is difficult to believe that there is not some connexion between these two types of arteritic change, and it seems possible that an acute arteritis of the polyarteritis nodosa type present at one point in the vessel might well lead distally to an obliterative type of change, and perhaps also to thrombosis, both at the site of acute disease or in the secondarily occluded vessel. In Case 2 thrombosis appears to have started peripherally, as shown by multiple sections beyond the obliterated segment. Histological changes along the course of a diseased vessel have yet to be studied in detail; the topographical and temporal relation of the acute arteritis undoubtedly present in some of these patients to the much more commonly observed intimal thickening has yet to be worked out.

How these arteritic lesions are produced is unknown, but a direct rôle of rheumatoid factor is suggested by the recent demonstration of Hess and Ziff (1960) of its binding to blood elements and vessel walls. The significance of arteritis, and the extent to which it is an integral part of the rheumatoid inflammatory process, are still quite speculative.

Summary

Brachial arteriography has been used to investigate the digital circulation in eleven patients with rheumatoid arthritis, in all of whom (except one in whom arteriography was performed at autopsy) there was some reason to suspect the presence of digital arterial disease. In all there was a varying degree of vascular occlusion or distortion, and histological examination has shown a non-specific arterial lesion with intimal thickening. Visceral arteritis was sometimes present with lesions indistinguishable from either acute or healed polyarteritis nodosa. In two patients with early rheumatoid arthritis, and no signs of peripheral vascular disease, arteriography was normal.

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DISCUSSION

DR. J. J. BUNIM (*Bethesda, Md*): I thoroughly enjoyed this paper and cannot add anything substantial to it. It is very interesting that all of the eleven patients had a positive sheep cell agglutination test and that nine of them had nodules. The association of the rheumatoid factor with vasculitis has been observed previously and is well confirmed by this study. I think it is very important to distinguish between digital arteritis and polyarteritis nodosa. I realize that it was impossible to do so in this study, but nevertheless we see patients with these thrombotic digital lesions, who are immediately suspected to have polyarteritis nodosa. A grave prognosis is given and steroid therapy instituted, but it is found that these patients do not really have polyarteritis nodosa, because the course of the disease is rather benign and they do much better than those who have a thrombosis as part of polyarteritis nodosa. I should like to congratulate Dr. Scott and his group for such a constructive study and hope to emulate them by adopting these studies as soon as I return home.

PROF. J. H. KELLGREN (*Manchester*): I should like to congratulate Dr. Scott on the beautiful illustrations. I have one relevant observation: there are patients who have arteritis of this type, but no arthritis or joint disease, who yet have positive rheumatoid factor tests. We had the opportunity in Manchester of studying over twenty patients of this type, mostly middle-aged women and a few men, who presented with acute secondary Raynaud's phenomenon. There was no clinical or radiological evidence of joint disease, but nevertheless over 50 per cent. had positive tests. This raises the question whether there is not a rheumatoid type of arterial disease which may or may not be associated with joint disease, and also the question of the prevalence of positive tests and rheumatoid arthritis in the general population. The correlation of the factor with joint disease in the

population as a whole is relatively low, but we do not know to what extent other pathological processes are associated with this factor.

DR. BASIL STRICKLAND (*London*): As a guest radiologist I really have no right to speak, but should like to add my congratulations to the radiologists. I think one may assume that arteriograms in such advanced cases will show changes, whatever the diagnosis may be. Arteriograms of really early cases with such good quality pictures might be much more informative.

The second point is rather technical. I was very interested to hear that these arteriograms were carried out with a local anaesthetic. I would prefer to do them under a general anaesthetic. I think that if you inject the brachial artery under local anaesthesia and later on give a general anaesthetic you get a different picture. It is often difficult to determine the degree of spasm under local anaesthesia with this particular artery, but with general anaesthesia that spasm can often be obviated and more definite conclusions drawn.

DR. STEINER: I will confine myself to answering Dr. Strickland's question. I have been interested in this matter for some time. We originally investigated Raynaud's disease, and in true cases of Raynaud's phenomenon we found that a general anaesthetic is undoubtedly essential. These patients are of course sensitive to puncture, so that the artery goes into spasm, and, as Dr. Scott said, the first few patients were studied under general anaesthesia. However, we later found we could manage with a local, and this has been so much simpler with "Hypaque", which is less toxic than the original dye used. In one case of this series there was considerable arterial spasm following injection, and two other cases of Raynaud's disease not reported also went into spasm.

If you suspect vascular spasm of the Raynaud type you should give a general anaesthetic. However, in cases of rheumatoid arthritis local anaesthesia will suffice.

In cases of vascular spasm the whole arterial tree is involved and the appearance of local organic blocking and collateral circulation which was seen in these cases cannot be observed.

PROF. BYWATERS: I am glad to endorse Dr. Bunim's comment about polyarteritis nodosa. Obviously, classical polyarteritis is a different sort of picture. What has worried me since our first publication in 1957, is that these patients with rheumatoid obliterating endarteritis sometimes show a fairly large vessel with acute inflammation. We do not yet know the spatial relationship along the length of the artery. Is there acute inflammatory disease with secondary bland obliterating endarteritis further up the vessel, or is the acute lesion secondary in some way to the obliterative lesion? I think clinically we can recognize classical polyarteritis nodosa, but we still know little about the incidence of isolated and healed arteritis and much more information is wanted about *formes frustes* of polyarteritis, such as

are found without clinical signs after appendectomy or amputation of the cervix. The Rose-Waaler test may be of some help in differentiating classical polyarteritis and the obliterating arteritis of rheumatoid arthritis.

DR. SCOTT: With regard to Dr. Strickland's first point, it was in fact because of abnormal arteriograms in the patients who showed some indication of arterial disease that we investigated two further cases with early rheumatoid arthritis, in both of whom the arteriogram turned out to be normal. Such information as we have, therefore, suggests that arteritic changes tend to be late. We do not think that vascular spasm is an important factor in the abnormal films, because the picture is usually one of rather abrupt blockage often with collateral circulation, and histology has shown the presence of organic occlusion.

Digital arteriography may be of practical value in the individual case. For example, the gangrenous index finger illustrated (Case 9) has since been amputated, and on clinical grounds the elective site of amputation made was through the second metacarpal bone, allowing apposition of the thumb and middle finger. When arteriography showed that the blood supply of all the other fingers was so bad, however, it was decided to amputate through the proximal interphalangeal joint, leaving as much as possible of the index finger in case the other fingers should be lost.

Artérite digitale dans la maladie rhumatismale

RÉSUMÉ

On étudia au moyen de l'artériographie brachiale la circulation digitale chez onze malades atteints d'arthrite rhumatismale; chez tous ces malades (sauf un, chez qui l'artériographie fut faite à l'autopsie) il y avait une raison pour soupçonner l'existence d'une affection artérielle des doigts. Chez tous on trouva des occlusions et des déformations vasculaires d'intensité variable et l'examen histologique révéla des lésions artérielles non spécifiques avec un épaississement de l'intime. L'artérite viscérale fut quelquefois présente et les lésions observées ne se distinguaient pas de celles qu'on trouve dans la polyartérite noueuse aiguë ou guérie. Chez deux malades avec une arthrite rhumatismale précoce et sans signes de maladie vasculaire périphérique, l'artériographie fut normale.

Arteritis digital en enfermedad reumatoide

SUMARIO

Se investigó por medio de arteriografía braquial la circulación digital de once enfermos con artritis reumatoide; en todos ellos (excepto uno, en quien la arteriografía se practicó en autopsia) existía alguna razón para sospechar la existencia de enfermedad arterial de los dedos. En todos existía oclusión y deformación vascular en grado variable, y exámenes histológicos mostraron la presencia de una lesión arterial no específica con engrosamiento de la intima. En algunos casos se observó la presencia de arteritis visceral con lesiones indiferenciables de las halladas en poliarteritis nodosa, aguda o curada. En dos enfermos con artritis reumatoide incipiente, y sin signos de enfermedad vascular periférica, la arteriografía fué normal.