sarcoma cells were beginning to undergo pycnosis. Typical blood granulocytes were in the extensive necrotic regions, but they were most abundant in zones surrounding the necrotic regions and were also present in spaces between morphologically normal appearing sarcoma cells bordering the necrotic regions. In these necrotic regions could also be observed the irregular, hematoxylin stained material described for the necrotic regions of untreated tumors. The blood granulocytes were present in quantities throughout the degenerative changes of the irradiated tumors.

Tests to exclude the possibility that the leucocytic response was due to possible infection of the irradiated tumors were made by bacteriological agar slants. These were uniformly sterile. Moreover, the tissue cultures, in which bacterial colonies develop rapidly and very early, were sterile during their maintenance for over a week. The cultures closely resembled those of fragments of starch-injected tumors in which sterile precautions were rigorously maintained.

These findings indicate that irradiation of Sarcoma 180 induces changes which call forth an accumulation of blood granulocytes in the tumor. The question is still open whether the accumulation is the cause of or subsequent to the necrotic changes. The fact remains that granulocytic response and necrosis accompany one another in the irradiated tumors while they do not in the spontaneous necrotic changes of non-radiated tumors of Sarcoma 180.

9354 P

Rôle of Arteries in Peripheral Resistance of Hypertension.

MYRON PRINZMETAL AND ENID TRIBE OPPENHEIMER. (Introduced by B. S. Oppenheimer.)

From the Medical Service of the Mount Sinai Hospital, New York City.

It seems established that arterial hypertension must be due to an increased peripheral resistance, but which part of the vascular bed is concerned in this increase is still somewhat uncertain. Evidence on the whole supports the view that it is due to arteriolar constriction, but some workers believe that the larger arteries also become narrowed in hypertensive conditions.[1]

Our observations represent an attempt to ascertain whether or not the larger arteries are involved in the production of the increased

[1] Weiss, S., Haynes, F. W., and Shore, R., *Am. Heart J.*, 1936, **11**, 402.

resistance of chronic hypertension. It is known that normally the blood pressure in the arterial bed falls gradually from large to small arteries, and that the greatest fall occurs in the region of the arterioles. If the arteriolar resistance should increase, the arterial blood pressure would be increased throughout, in small as well as large arteries, so that the pressure gradient from large to small arteries would either remain substantially unchanged, or become less. If an increased resistance should occur in the arteries themselves, the pressure gradient would tend to become steeper, since the pressure would be raised in the largest arteries, but would be reduced in the smallest.

We therefore measured the blood pressure in a large and a small artery of subjects with hypertension, and compared the figures with those obtained from individuals having normal systolic pressures. Blood pressure was determined in the brachial artery by the usual auscultatory method, and in the digital artery by Gaertner's method,[2] whereby, after rendering the finger bloodless, the pressure is noted at which the finger again becomes suffused with blood. This pressure is assumed to be the systolic pressure in the digital artery.

TABLE I.
Average Brachial-Digital Gradients.

Type of Case	No. of Cases	Brachial Syst. Press.	Digital Syst. Press.	Brachial-Digital Gradient in mm.
Normal	25	124	95	29
Hypertensive				
(a) Benign H.T.	32	184	152	32
(b) Secondary H.T. (renal)	10	187	156	31
(c) Malignant H.T.	9	210	186	24
Averages for all Hypertensives	51	188	158	30
Hypotensive	14	98	71	27

From Table I it will be seen that the average brachial to digital pressure gradient in cases of hypertension shows no significant difference from that in subjects with normal or with low blood pressure. The most marked variation is perhaps a slight *reduction* of gradient in the cases of malignant hypertension.

It would seem, therefore, that there is certainly no significant increase in the brachial-digital pressure gradient in chronic hypertension, suggesting that the increased resistance occurs, not in the larger arteries, but mainly in vessels smaller than the digital artery.

This contention is further supported by observations made on a patient with paroxysmal hypertension due to pheochromocytoma. During the hypertensive attacks, easily induced by moderate exer-

2 Formijine, P., *Am. Heart J.*, 1934, **10**, 1.

cise, large amounts of epinephrin were demonstrated in the blood[3] by the Pissemski method.*[4] This condition was of particular interest to us, since epinephrin is known to cause constriction of arteries as well as of arterioles. Table II shows that during the hypertensive attacks the brachial systolic pressure rose to 280 mm., while the digital pressure, on the contrary, showed a slight fall, resulting in a steep brachial-digital gradient of 150 mm., as contrasted with the 30 mm. gradient of chronic hypertension. The marked contrast between this case, where arterial spasm is admittedly present, and the cases of chronic hypertension suggests that in the latter condition, where there is no increased gradient, there is no constriction of the larger arteries.

TABLE II.

Type of Case	No. of Cases	Brachial Syst. Press.	Digital Syst. Press.	Brachial-Digital Gradient
Pheochromocytoma				
(a) Resting	1	184	141	43
(b) During attack		280	130	150
(c) After removal of tumor		133	90	43
Obliterative Vascular Disease				
Thromboangiitis oblit.	2	112	43	70
Sclerodactylia	1	107	27	80
Thrombosis of brach. art.	1	104	29	75

In 4 cases also of obliterative vascular disease (Table II), we found that the brachial-digital fall of pressure was much greater than in normal subjects. These cases indicate that obstruction of the arteries, whether due to spasm or to organic changes, causes an increased gradient in the fall of pressure between brachial and digital arteries.

We therefore conclude that in chronic hypertension the increased resistance occurs in vessels smaller than the digital artery.

[3] Beer, E., King, F. H., and Prinzmetal, M. To be published shortly.

* In this method the fluid to be tested is perfused through the vessels of a denervated rabbit ear. In our case the patient's blood plasma caused vasoconstriction, which was inhibited by ergotoxin.

[4] Pissemski, A., *Pflüger's Arch.*, 1914, **156**, 430.