cise or emotion. In these circumstances the strain on the capillary walls will be increased and there will be *imminent danger of capillary dilatation and rupture.*

Clinically, there is reason why this assumption is justified. Clawson[7] studied the autopsies and clinical data in 928 fatal cases of coronary sclerosis, and found that thrombosis was more common in patients with persistent hypertension than in those without hypertension. Master, Dack and Jaffe[8] studied 500 cases of coronary occlusion, and found that persistent hypertension was present in more than half of the men and in four-fifths of the women. Aring and Merritt[9] investigated 96 cases of cerebral thrombosis (in which the common precipitating factor is also intimal hæmorrhage[3]), and found that in 85 per cent of the cases the systolic blood pressure was in excess of 140 mm. of mercury. On clinical grounds, therefore, it would appear that persistent hypertension is a common etiological factor in the production of *coronary thrombosis, a disease which* we know to be caused by intimal hæmorrhage.

I have maintained for some time that if persistent hypertension can cause increased intracapillary pressure and intimal hæmorrhage the same effect will be produced by the transient and sudden hypertension that results from physical exertion and emotional stress.[3b-d] Actually, this opinion agrees with present day medical teaching, as expressed by Blumer[10] in 1939—"that either emotion or unusual physical exertion may play the part of the exciting rôle in an attack of coronary thrombosis". This hypothesis is obviously of medicolegal importance, particularly in Workmen's Compensation Board cases.

A group of physicians and pathologists in New York have disagreed with this argument for a variety of reasons, most of which have been dealt with elsewhere.[3d,f] Recently, Master[8] has claimed that *in fatal cases of coronary occlusion* the incidence of intimal hæmorrhage is the same in patients who have never had hypertension as *in those who have had hypertension.* Likewise, Horn and Finkelstein,[4] while admitting that sudden rises in coronary artery pressure *may conceivably disrupt intimal capillaries,* believe that this mechanism is not significant as one should otherwise encounter a much greater incidence of *coronary occlusion in hypertensive* individuals. Each of these authors admits that intimal hæmorrhage from capillary rupture is the important precipitating cause of coronary thrombosis, and the following statistical evidence, disagreeing with their findings and opinions, should therefore be of interest.

The material consisted of 186 consecutive autopsies on patients over the age of 40 years in the Department of Pathology of the Ottawa Civic Hospital from May, 1938, to September, 1940. The degree of coronary sclerosis, the existence of hypertension, and the presence of intimal hæmorrhages of the coronary (or cerebral) arteries were determined in each case.

The degree of coronary sclerosis was estimated as follows: It was considered slight (+) when the intimal changes were few and scattered; as moderate (++) when the intimal surfaces were covered with many plaques but the process had produced little or no narrowing of the lumen; and as marked (+++) when there was definite narrowing of the major branches (Davis and Klainer[11]).

Two criteria were used for determining the existence of hypertension: (1) A history of high blood pressure in excess of 150 mm. of Hg., systolic, and 100 mm. of Hg., diastolic, and/or (2) cardiac hypertrophy in the absence of valve defects (except aortic insufficiency) or any other known cause of hypertrophy. Heart weights of 500 g. or more in males, and 450 g. or more in females were the minimum weights regarded as indicating that hypertension had existed. Hearts with weights slightly below these figures (450 to 499 g. in males and 400 to 449 g. in females) were probably also hypertensive hearts, but these have been separated from the main series when no blood pressure readings were available and have been classed as indefinite cases. These criteria are substantially those established by Clawson.[7]

The presence or absence of intimal hæmorrhages was determined by careful examination of the coronary arteries (and cerebral arteries in some cases). When calcification was marked, the arteries were decalcified before examination. Gross lesions suggestive of intimal hæmorrhages were subjected to microscopic examination in all cases, as it was found occasionally that the hæmorrhages could be simulated by dilated and engorged capillaries without extravasation of blood. When coronary thrombi were present, the occluded portions of the arteries were decalcified, and sectioned serially at intervals of 300 microns; the sections were then examined for the presence of intimal hæmorrhages.

The results of this study indicate clearly the effect of persistent hypertension on the production of intimal hæmorrhage. When the series of 186 cases is taken as a whole it is found that intimal hæmorrhages are more than five times as frequent in hypertensive (42 intimal hæmorrhages in 70 cases, or 60 per cent) as in non-hypertensive persons (13 intimal hæmorrhages in 116 cases, or 11 per cent). On breaking down the series into the various grades of coronary sclerosis a similar relative incidence is *maintained. One hundred and seven cases of* marked and moderate coronary sclerosis (the grades that have intimal capillaries) were found *in the series. Forty-one out of 57 cases of* hypertension with these grades of sclerosis showed intimal hæmorrhages (72 per cent), while only 13 out of 50 cases of non-hypertensives with these grades of sclerosis showed in-