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 studied the autopsies and clinical data in 228 fatal cases of coronary sclerosis, and found that thrombosis was more common in patients with persistent hypertension than in those without hypertension. Master, Dark and Jaffe studied 300 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 investigated 12 cases of cerebral thrombosis (in which the common precipitating factor is also intimal hemorrhage), 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 etiologic factor in the production of coronary thrombosis, a disease which we know to be caused by intimal hemorrhage.

I have maintained for some time that if persistent hypertension can cause increased intracapillary pressure and intimal hemorrhage the same effect will be produced by the transient and sudden hypertension that results from physical exertion and emotional stress. Actually, this opinion agrees with present day medical teaching, as expressed by Blumer in 1939—that either emotion or unusual physical exertion may play the part of the exciting role 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. Recently, Master has claimed that in fatal cases of coronary occlusion the incidence of intimal hemorrhage is the same in patients who have never had hypertension as in those who have had hypertension. Likewise, Horn and Pinkelstein, 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 hemorrhage 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 320 consecutive autopsies on patients over the age of 40 years in the Department of Pathology of the Ottawa Civic Hospital from May, 1933, to September, 1940. The degree of coronary sclerosis, the existence of hypertension, and the presence of intimal hemorrhages 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; moderate (-) when the intimal changes were numerous and scattered, and severe (+++) when the intimal changes were numerous and extensive. Arteries with intimal hemorrhages were classified as having severe coronary sclerosis.

Two criteria were used for determining the existence of hypertension: (1) A history of high blood pressure in excess of 160 mm. of Hg., systolic, in 160 mm. of Hg., diastolic, and/or (2) cardiac hypertrophy in the absence of Ewing's valve disease (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 450 g. in females) were probably hypertensive, but these have been excluded from the final series when no blood pressure readings were available and have been classified as indefinite cases. These criteria are substantially those established by Clawson.

The presence or absence of intimal hemorrhages was determined by careful section of the coronary arteries (and cerebral arteries in some cases). When collection was marked, the arteries were decaledified before examination. Granules suggestive of intimal hemorrhages were subjected to microscopic examination in all cases, as it was found occasionally that the hemorrhages could be stimulated by dilated and engorged capillaries, without any thrombi present in the coronary thrombi were present, the included portions of the arteries were decaledified, and sectioned serially at intervals of 300 microns; the sections were then examined for the presence of intimal hemorrhages.

The results of this study indicate clearly the effect of persistent hypertension on the production of intimal hemorrhage. When the series of 186 cases is taken as a whole it is found that intimal hemorrhages are more than five times as frequent in hypertensive (42 intimal hemorrhages in 70 cases, or 60 per cent) as in non-hypertensive persons (13 intimal hemorrhages 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 hemorrhages (72 per cent), while only 13 out of 50 cases of non-hypertensives with these grades of sclerosis showed in-