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Discussion

The crucial lesion in the coronary arteries in every case was atherosclerosis. The findings of the present study reveal that coronary atherosclerotic lesions are present in almost all the adult autopsies. All the subjects above age of 30 had some degree of atheroma. The youngest male to show atheroma in the coronaries was 22 years; the youngest female was 25 years. The adult cases hence can not be divided into subjects having and those not having atherosclerosis, only the disease could be further classified according to the degree of stenosis in each artery. This concurs with the findings of Strong & McGill.

Thrombosis complicating atheromatous lesion was found in only one case and no thrombus was found in the absence of atherosclerotic plaques. This does not support the suggestion of Gresham and Howard[30, 31] that coronary thrombosis can occur in the absence of atherosclerotic plaques.

Morris[32], comparing the prevalence of coronary atherosclerosis recorded in postmortem examination at the London Hospital in 1908-13 with the records for 1944-49, showed that over this period there had been some reduction in the amount of mural atheroma in the population, but in spite of this there had been a tenfold increase in deaths from ischaemic heart – disease. In other words, Morris showed that there is no direct relationship between the amount of atherosclerosis in a population and the incidence of ischaemic heart disease. Others have interpreted Morris's observations as implying that coronary thrombosis occurred apart from atheroma. This, however, is completely contrary to the facts as observed not only in the study of Crawford, Dexter, Teare[2] but in the general experience of pathologists working with human material. Thrombosis is never seen without mural disease. The rare instances in which coronary occlusion may be due to syphilis, embolism, thromboangeits, or dissecting aneurysm from such a small part of the great number of cases as to be insignificant.

This study also indicates a strong correlation between the arterial lesions of coronary atherosclerosis and ischemic heart disease in that both the incidence and severity of atherosclerotic lesions were greater in the hearts that showed ischemic changes. This agrees with Strong and McGill who opine that this correlation between coronary lesions and fatal ischemic heart disease suggest that the modern epidemic is based primarily on atherosclerotic lesions rather than a terminal episode such a thrombosis, and that the severity of coronary lesions is the determining factor in morbidity and mortality from ischemic heart disease.

Interpreting the results to indicate that the mural arterial lesions determine the risk of ischemic heart disease does not rule not thrombosis as a terminal occlusive episode in most cases; the question is whether the terminal episode of thrombosis is influenced primarily by coronary

atherosclerotic lesion or by some other independent factor, such as coagulability of the blood. The evidence in the literature suggests that coronary atherosclerosis directly predisposes to occlusion by thrombosis and thereby to ischemic heart disease.

Strong and McGill also found coronary artery lesions to be more in males than females. In the study the incidence and severity of atheromatous stenosis was higher in males. Only an occasional study like the one by Roberts, Moses and Wilkins[33] denies the existence of sex differences in coronary atherosclerosis. The group of subjects whose hearts revealed myocardial ischemic changes after staining was found to have a significantly higher mean age than the other group. Though the mean body weight and body mass index of the former group was higher than the latter group, these differences were not found to be significant. No significant association was seen with the sex of the subject and myocardial ischemia. Also it was seen that the subjects above the age of 50 years were 13 times move frequently affected by myocardial ischemia than those below 50 years.

Another objective of the study was to correlate myocardial ischemia detected with the Grade of stenosis in the coronaries. Though it was known that the incidence of ischemia increases with the severity of the stenosis, a simple quantification of the relation was attempted. This was done for each of the arterial segments studied and also for the maximum grade of stenosis anywhere in the arterial tree. It was seen that the incidence of myocardial ischemia increased with the increasing grade of stenosis.

The paucity of histological alterations in the heart muscles deprived of their blood supply for 6 hours or less has stimulated investigators to search for easily recognizable biochemical changes. These have been found to include depletion of glycogen, glucose, pyruvate and other labile substrates, a shift to anaerobic metabolism of pyruvate with accumulation of lactate and a drop in pH, consumption of ATP, AADP, CP and diphosphorylated intermediates of the glycolytic cycle Nachlas & Shinkta,. Enzymes begin to leave the area of infarction later, when they appear in the blood, are regarded as evidence of irreversible myocardial damage, Thus, following ligation of a coronary artery in the dog, there was an initial period of little or no decreasee in enzyme activity, lasting 40-70 minutes with GOT, 2 hours with LDH and 4 to 5 hours with SDH, the tissue level of all three enzymes fell rapidly thereafter until values of 30 to 50% of the normal value were reached 12 to 15 hours after ligation (Jennings R.B. 1957). Most of the foregoing work was carried out either by biochemical assays or histochemical studies, after ligation of a coronary artery, the specimen being taken from anticipated areas of infraction. Jennings and Wartmann stressed that difficulties might arise from such experimental studies because of lack of homogeneity in the infarct. The absence of gross changes for 12 to 24 hours after deprivation of arterial blood supply also render uncertain the accurate selection of blocks of ischaemic muscle for microscopic examination. For these reasons the techniques for gross identification of early myocardial infarction are of great value.

Of the two methods staining employed in the study the T.T.C. method is more rapid, and provided that hemoglobin diffuses out of the muscle during incubation, a good contrast is noted between the bright red formazan pigment deposited on the normal muscle and the pale pink color of the myocardial infarct. Occasionally however, in small areas of necrosis in aged specimens and when the hemoglobin does not diffuse out, the contrast obtained with the T.T.C. method is not good as desired. In the light of recent work showing that electron transfer to T.T.C. occurs after cytochrome oxidase, it seems likely that their method is dependent on the combined loss of substrate, co-enzymes, dehydrogenase and cytochrome oxidase from the heart muscle. The method using Nitro-BT as the hydrogen acceptor reflects the activities of many dehydrogenases oxidizing endogenous

substrates and transferring the liberated electrons either directly or through coenzymes and diaphorases to the tetrazolium salt. The blue formazan is produced in 10 to 15 minutes incubation period and provides good contrast between normal and ischaemic myocardium. Furthermore the increased sensitivity of this method furnishes more reliable results in small lesions, early infarcts and aged specimens.

While applying the enzyme reaction for the detection and demonstration of myocardial infarction is has been observed that a few precautions are necessary to avoid false results. Occasionally the commercial tetrazolium salt may be of an inferior quality, to overcome this; a fresh piece of muscle with known dehydrogenase activity should be used whenever an experiment is run with a new reagent. The T.T.C. is photosensitive therefore care should be covered by black paper over the container. The method can not be expected to give reliable results when applied to specimens stored from more than 8 hours at 25 to 37^0 C or for more than 48 hours at 4^0 C. A weaker reaction is sometimes seen normally in the bulbospiral muscle of the left ventricle, because of the fibres running parallel to the transverse cut of heart slices. This does not mean that these fibers are ischaemic, but rather that better exposure of sarcosomes to the reagent solution is obtained when cut at right angles. The pH of the incubation medium has been found to be a crucial factor in either giving a false positive of false negative result. If the pH is acidic there is hardly any deposition of the pigment on the surface of the slice and the pH is above 9 there is a nonspecific dense deposition of the dye in both the normal and infracted myocardium. Finally the incubation solution should be sufficient to cover the heart slices completely for a depth of at least 2 cms to avoid patchy staining of the upper surface compared to that on the undersurface.

Another issue to be considered is the detection of myocardial ischemia in relation to the period of survival and also whether these techniques which outline experimental myocardial infarcts, before the gross or microscopic changes become manifest, can be used to assist Pathologists in the recognition of early or small infarcts in the human subject. Neoral and associates applied the Tellurite reaction to the hearts of 20 patients with suspected or proven myocardial infarcts. The earliest infarct which they recognized was in a patient who died 7 hours after the onset of clinical symptoms. There were neither gross nor microscopic findings. Jestadt and Sandritter examined 112 hearts. In 10 cases with clinical symptoms 5 to 15 hours duration preceding death, the T.T.C. reaction demonstrated infarcts in the absence of both gross and microscopic changes. Thus they suggested that it was possible to recognize infarcts by this method as early as 4 hours after the onset of signs and symptoms. However, their findings are difficult to evaluate because cases such as the above of 5-15 hours were said to show no histological changes while cases of 10 minutes to 1 hour did show microscopic evidence of muscle necrosis. Nachlas and Shinkta surveyed 23 human hearts treated with Nitro BT, which included 7 hearts developed homogeneous staining reactions and also failed to show gross or microscopic evidence of recent muscle necrosis. The earliest infarct in humans by this method was of 8 hours duration. Ramkisoon surveyed 31 human hearts at autopsy employing the Nitro BT method. Of these, 12 Patients had ECG, laboratory and histological evidence of myocardial infarction, 15 patients had atherosclerotic heart disease but without clinical and histological evidence of myocardial infarction and 4 patients had other disease which were expected to produce secondary myocardial infarction during the terminal episode. The earliest infarct showing loss of dehydrogenase activity was 8 hours old. Fine surveyed 11 hearts at autopsy using the Nitro B.T. method to demonstrate the detectable loss of enzyme activity. In 6 cases the interval was 0 to 4 1/2 hours and 5 cases it was more, but less than 12 hours. All showed severe narrowing of a coronary artery but without gross or microscopic evidence of myocardial

infarction. Enzyme alterations were seen in 10 cases and the detectable loss of enzyme activity was from 1½ hour onwards. Brody and associates studied 35 human hearts by the Nitro B.T. method at autopsy. They found that 21 patients showed findings of myocardial infarction both on gross inspection and after staining. In four of these cases the myocardial infarction was more extensive after staining than on gross inspection, 5 hearts showed myocardial infraction only after staining, which was not suspected on gross inspection. McVie studied 65 human hearts at autopsy. Out of these, 29 hearts were of patients who had died suddenly or within 12 hours of the onset of symptoms suggesting myocardial infarction. 10 cases out of these which had intervals shorter than 12 hours did not show apparent myocardial infarction on gross examination gave positive results after staining with Nitro B.T. The earliest infarct revealed by staining was 3½ hours duration. The mapping of the infarct was beautifully portrayed and provided simple correlation with the supply of an occluded coronary vessel. Andersen and Hansen in an effort to find out the value of Nitro B.T. studied 100 human hearts. In 15 of these cases the infarct was demonstrated only by the stain where there was no indication of infarction on gross inspection. The delineation of the unstained areas was unpredictable and affected almost any portion of the ventricular wall or the papillary muscle.

In the present study consisting of 110 hearts, that were examined for macroscopic enzymatic reactions employing the Nitro B.T. and the T.T.C. methods of staining, it was found that 17 hearts gave a positive test, showing infarcts in the myocardium. The earliest infarcts that was detected by both the methods adopted in this study was of 1 hour duration, which does not agree with the observation of Sandritter and Jestadt using the T.T.C. reaction and by Mc Vie by the Nitro B.T. method. It does not also coincide with the observations of Nachals and Shnikta and Ramkisson who could detect myocardial infarction using the Nitro B.T. method of staining only in cases where the patient had died 8 hours after the onset of symptoms suggestive of myocardial infarction. These observations were closest to those made by Fine, where he found loss of enzyme activity from 1½ hours after the onset of symptoms using the Nitro B.T. method. It coincides with the findings of Andersen and Hensen who detected localized subendocardial infarctions of 1 hour duration. They however reason that subendocardial infarctions presumably have a symptoms free period. It is also the hypothesis of Gregersen that all infarcts start in the subendocardial zone. We might deduce that perhaps had the subject survived for a longer period of time a transmural infarction might have been demonstrated due to the loss of enzyme activity. The earliest transmural infarction that has been demonstrated in this study is of 2 hours duration. This discrepancy in the time required for demonstration of loss of enzyme activity could also be due to unreliability of fixing the actual event of infarction to coincide with onset of pain or any other clinical symptoms.

The age of an infarct is notoriously difficult to establish in the human, as the onset of clinical symptoms, however dramatically abrupt, are often much later than the onset of the pathological lesion precipitated by a coronary occlusion. In animal experiments, a coronary vessel can be ligated at zero time and serial sacrifices made at different intervals to gain an accurate estimate of the age of the infarct. In the human, the time of chest pain and shock cannot be used in a similar fashion. When a victim of coronary disease dies say, eight hours after the onset of acute symptoms, though one might expect an early infarct to be visible on histological or histochemical examination, not frequently a well-demarcated yellow or tigroid area of necrosis is present; which must be several days old.

Regarding the site of the infract detected: The Right ventricle was the site, for 1 subendocardial infarct which is about 6 percent of all the cases, it agrees with the observations of Wartmann and Hellerstein[34] who found that the right ventricle was involved between 6 to 9 percent. All the other subendocardial infarcts were of the left ventricle.

The cases which had ischemia could be divided into 2 groups. One group with high grades of arterial stenosis and the other with lower grades.

It was seen that those cases which revealed infarcts with grade 2 stenosis or less could be divided into two distinct sub-groups; one of age below 40 years (with marked hypotension due to hemorrhagic shock with or without hypertrophic hearts), and the other consisting of elderly subjects.

There were 3 such cases in the former sub-group: one was of a 40 year old male with grade 2 stenosis of the left main and grade 1 stenosis of others. A complicating infarct involving the anterior wall and subendocardium was seen in this case. The period of survival was 7 hours. In another case a transmural infarct of the anterior wall of the left ventricle was seen. Period of survival was 48 hours and the heart weighed 350 gms. The third case was of 36 years old male in whom period of survival was not known. A subendocardial infarct of the left ventricle was seen. In these cases ischemia was mainly due to the marked hypo perfusion of the myocardium. When the period of survival was more than 7 hours a transmural infarct had the scope to develop. The third case which had only a subendocardial infarct period of survival was perhaps not sufficient for the development of a complete transmural infarct.

The Second sub- group comprised of cases that were elderly and were in the terminal stages of head injury. In this scenario ischemia occurred with grade 2 stenosis or less. These hearts revealed only subendocardial infarcts though the period of survival was more than 8 hours in some of the cases. Therefore it may be presumed that there was a relative ischemia of the subendocardial myocardium to which these hearts were probably sensitive due to changes of senility.

In the group that had high grades of stenosis, some had complete occlusion of one or more segments of the arterial tree. This group mainly had transmural infarcts with or without subendocardial infarcts. There was only one case that of a 57 years old male, which revealed only subendocardial infarct, the period of survival in this case was not known. The query whether the subendocardial infarct might have progressed to a transmural infarct given a sufficient survival period can perhaps never be answered.

Considering the transmural infarcts 8 of the infarcts were located on the anterior wall, and 3 on the septal wall and 2 on lateral wall of the left ventricle. No such infarcts were revealed on the other areas of the myocardium though in most of the cases a high grade of stenosis was present in all the vessels. Most of the transmural infarct occurred on the anterior wall of the left ventricular although in one case there was complete occlusion of right coronary artery with grade 3 stenosis of the left coronaries. In one case transmural infarct was seen on the anterior and septal walls of left ventricle with only grade 2 stenosis of the vessels and the heart was found to be hypertrophic.

No simple correlation between the site of stenosis and the topography of the infarct could be found, which agreed with the findings of Wartmann and Souder[35], and also Andersen and Hansen who pointed out that the delineation of areas of infarction was unpredictable.

The high incidence of subendocardial infarcts about 55% of all infarcts was in concordance with the findings of Ramkisoon, Andersen and Hansen and McVie. There were 11 subendocardial infarctions both total and localized which gives a remarkably large incidence probably explained by the fact that most of the cases were elderly people suffering from severe coronary stenosis who were in shock or infection or terminal stages of head injuries. In only one case thrombosis of coronary arteries was demonstrated and in two cases coronary vessels presented mild atherosclerotic changes. In infarctions with transmural component thrombosis was found in one case only. This is in agreement with the fact that Ehrlich and Shinohara[36] in their study on multicentric type of infarctions, which mainly were located in the subendocardium, found coronary thrombosis in only 10 per cent. of cases. Sugiura[37] and colleagues in their eight cases of subendocardial infarctions found no coronary thrombosis but often severe arteriosclerotic changes. These cases were elderly people who apart from an often severe arteriosclerotic heart disease had shock as a probable contributory factor to the fresh infarctions.

In the 17 cases of fresh myocardial infarction the papillary muscle was involved in 4 cases (24%) in comparison Brand[38] and Colleagues found the papillary muscle to be involved in 33 percent and Heikkila[39] in 50 percent and Hansen and Andersen in 70%.

There are several types of myocardial infarcts recognizable at autopsy:

A laminar infarct in which the subendocardial region of much – or even all – of the left ventricle is involved, sometimes extending through half or more of the thickness of the wall. This is due to a reduction in perfusion pressure to the inner zones, as all the coronary supply comes from the epicardial surface. Laminar infarcts are the result of generalized stenosis in the major branches of the coronary vessels, but there is usually a second factor, in that a drop in blood pressure or the oxygenation of the blood compromises the already poor supply so that the outer zones of the ventricular myocardium consume the available oxygen and nutrients, leaving little for the inner zone. A regional or focal infarct is more common in pure coronary artery disease, and is caused by a localized occlusion or severe stenosis in a coronary artery. These are true myocardial infarcts, as the definition of an infarct requires occlusion of the vascular supply, which strictly excludes some laminar infarcts when perfusion pressure or relative insufficiency is caused by hypertension, or aortic valve disease. The regional infarct is a topographically demarcated zone of muscle necrosis, the size and position depending on the site of vascular occlusion, though any collateral supply may modify these. Almost all infracts are in the left ventricle. Wartmann and Hellerstein found that between 6 and 9 percent involved the right ventricle and that 7 percent involved the atria, though Cushing's40 figure was 17 percent for atria. Most of these infarcts, however, were overlapping with infarcts in the left ventricle.

The relative immunity of the right ventricle and atria is presumably because of the relatively thin walls, which do not require so much blood; do not have the perfusion gradient of the thick left ventricle and which can more easily obtain oxygen and other constituents from the blood in the lumen of the ventricle. The presence of a conus artery, small branches to foci, such as to the sinus node and the presence of anastomotic channels between the left circumflex and right coronary arteries, must all contribute to the better survival of these areas. The site of left ventricular infarcts does not always correspond strictly to the site of the coronary occlusion of thrombus because of the presence of a collateral circulation.

It was also shown by Wartmann and Souders that many infarcts involve only certain layers of muscle, which cannot be supplied by a particular vessel. There is obviously no simple explanation for the topography of a myocardial infarct – this is further confirmed by the patchy nature of myocardial damage on microscopic study, as the alternate necrosis and survival of adjacent fibers or even segment of the same fiber cannot be explained solely by coronary perfusion.

The existence of hypertensive heart disease as a specific entity was strengthened by some investigations by the B.Knight[41, 42], in which it was shown that gross enzymes deficiencies exist in the inner part of the wall of the hypertrophied left ventricle. This lesion is not specific to hypertension, but is seen whenever the left ventricle is enlarged, notably in aortic valve disease. In severe instances, the defect is dehydrogenase activity extends from endocardium almost to the epicardium, there being only a narrow zone of normal activity. In some ways this defect is similar to the `laminar infarct' of coronary artery disease.

In pure hypertension and aortic valve disease, the cause is relative ischaemia of the inner zone of the ventricular wall caused by insufficiency of coronary blood supply compared with the demands of the thickened ventricular mass. The reason for the laminar distribution is the anatomy of the coronary supply, graphically described by Farrer-Brown[43, 44]. The various levels of strata of the ventricular wall have different arterial patterns. The subendocardial zone is supplied by arteries with many branching terminations that provide a profuse and concentrated blood supply. A second set of vessels passes straight through the ventricular wall without division all the coronary supply coming, of course, from the epicardial surface. This set serves the papillary muscles and trabeculae carnae, while a third group tends to run circumferentially parallel and below the epicardium, giving branches both outwards and inwards.

Farrer-Brown found that the termination of these vessels were of types, as they divided into arterioles and then capillaries. The direction of the tree-like branching in the subendocardial zone continued in the general line of the main artery. The area of myocardium supplied by these branches was small. This was in contrast to the pattern in the mid-zone of the wall, where the branches turned at an angle to the main artery and then divided to supply a much wider area of myocardium that was supplied by the terminal branch in the subendocardial zone. These anatomical differences suggest that the vessels in the mid-zone each have to supply a larger volume of muscle than in the other layers. This might well explain the greater vulnerability of this central zone to hypoxic changes, as indicated by reduced enzyme activity and the occurrence of laminar necrosis. In actual practice, the inner zone also suffers, apart from the immediate subendocardial layers, but the superficial subepicardial zone is almost always spared. The rationale of damage to the myocardium in hypertensive heart disease, as in Aortic stenosis, would appear to be a relative coronary insufficiency. In pure coronary artery disease, a part of a normal sized myocardial mass is rendered ischaemic by localized reduction of blood flow by a coronary stenosis or occlusion, the resulting necrosis being distributed in a focal pattern according to the location of the block.

In hypertensive heart disease, normal coronary arteries do supply a much larger muscle mass, so that there is the same imbalance between supply and demand. As there is no focal blockage, however, the resulting lesion is diffuse and the areas that suffer most are those in the mid-and inner zones, probably because of the microanatomy of the vessels described above. The same type of lesion may be seen in any severe sustained hypotensive episode of extra cardiac cause, though the early onset of death in many of these will preclude the development of morphological evidence of laminar myocardial damage.

In this study there were 4 hearts out of 110 studied which weighed more than 450 gms of which 3 hearts weighed 500 gms. Ischemic changes were not detected on staining in 2 of these hearts which had only grade I or grade II stenosis of the vessels examined, however the period of survival of both these subjects who died in road traffic accidents was not known so comment can be made regarding the absence of ischemic changes which might not have had the time to develop. The other 2 hearts revealed infarct areas, one of them had a transmural infarct of the anterior wall and septal wall of left ventricle, this heart also had incompetence of the mitral valve. The other heart showed a total subendocardial infarct of the left ventricle. The generalization that hypertrophied hearts tend to develop ischemia only of the subendocardial zone has not been validated in this study.

Considering the sensitivity of the Nitro B.T. and the T.T.C. staining methods, compared to the findings in the gross naked eye examinations. It was found that there where 4 hearts where the fresh infarct could be seen on naked eye examination. Neoral et al, Nachals and Shnikta, Ramkisoon, Brody et al and Mc Vie who noted that in cases where the infarct was located on naked eye examination, the actual infarction after enzymatic staining turned out to be more extensive, covering a bigger area of he myocardium. This agrees with the present study which had 4 hearts where the fresh infarct could be seen on naked eye examination. In all 4 cases extension of the infraction was larger than had been suspected at the ordinary macroscopic evaluation. *The most important results were that in 13 cases staining revealed fresh myocardial infarction which had not been suspected at the ordinary macroscopic evaluation.* The number of unexpected fresh infarctions revealed by staining is in agreement with the findings of Kalderon[45] in his examination of 26 cases, in which he found seven cases with positive results. Among 31 patients Brody and associates found an unsuspected positive reaction with Nitro BT test in only one, and in four cases the fresh infarction was found to be more extensive than had been suspected at the ordinary macroscopic examination. In 17 cases the findings before and after Nitro BT test showed conformity. Ramkisoon also found unexpectedly positive reaction in 15 patients with known arteriosclerotic heart disease, but he did not give the percentage of the entire series. Further more he mentioned a group of four patients who might be expected terminally to develop relative myocardial ischemia, in whom he found “positive” reaction. However, this reaction subsided at prolonged incubation after addition of substrate.

Comparing the two methods of staining that were employed in this study it was found that the Nitro BT method was more sensitive and gave a better contrast between the normal and infracted myocardium than the T.T.C. method. The incubation period required for the Nitro B.T. method was less than that required for the T.T.C. method. The color of the dye deposited in the Nitro B.T. being deep purple, it was considerably easy to demarcate the non-stained pale pink infracted muscle from the stained healthy myocardium whereas in the case of T.T.C., the deposited dye being bright red, it took considerable effort to localize the non stained pink myocardium from the stained normal myocardium especially when the lesions were small in size. The observation was comparable to those noted by Nacklas and Shnikta who found that the Nitro B.T. method was more sensitive and reliable providing good contrast between normal and infracted myocardium.

Considering the validity of the enzymatic staining reaction in relation to the increasing postmortem interval, where the bodies were stored between 4^{o}C to 2^{o}C 91 to the 110 hearts subjected to the staining gave equal deposition of the dye, with same intensity and sensitivity from 4 hours to 60 hours of post-mortem interval. This observation is similar to that of Andersen and Hansen, who

were able to apply the method 72 hours after death on bodies kept at 4^{O}C, but is contrary to the observations of Morales and Fine, who could apply the method only till 36 hours, Nachlas and Shnikta and Ramkissoon, who were able to apply the methods only till 48 hours after death, bodies being kept at 4^{O}C. In two hearts of the 110 examined there was deposition of the dye by both Nitro B.T. and T.T.C. methods only with incubation up to 45 minutes. These two bodies had been examined 5 days after death the bodies being stored at 4 degree C. The results cannot be compared with previous work as none have mentioned such long intervals.

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