≥ 4000 IU/L was significantly higher than those with peak CPK < 4000 IU/L ($p < 0.01$), while no difference was observed in PaO_2 between 2 groups. Wall motion point score had significant correlation with AaDO_2 ($r = 0.55$, $p < 0.005$), but not with PaO_2 . Although AaDO_2 in non survivors was significantly higher than survivors ($p < 0.05$), there was no significant difference in PaO_2 . These data indicates; (1) AaDO_2 on admission in patients with acute myocardial infarction had no significant relation with hemodynamic parameters but it may possibly reflect infarct size. (2) AaDO_2 was better index than PaO_2 in predicting hospital mortality.

-40-

EARLY AND LATE ATRIOVENTRICULAR BLOCK (AVB) COMPLICATING ACUTE MYOCARDIAL INFARCTION (AMI) AND THEIR CLINICAL FEATURE

Takashi Tanaka, Piboon Laohathai, Yoshinori Kobayashi, Takashi Nitta, Yuzuru Matsuyama, Yoshihiko Seino, Keiji Tanaka, Takao Katoh, Teruo Takano and Hirokazu Hayakawa*

Coronary Care Unit and Department of Internal Medicine*, Nippon Medical School, Tokyo

To evaluate the relationship between the onset time and clinical feature of high degree AVB (AVB#) complicating AMI, the consecutive 348 patients (pts) with AMI were reviewed. The incidence of AVB# was 34.8% (45/130) in inferior infarction and 3.7% (8/218) in anterior infarction. For further examination, 88 pts with first acute inferior infarction were classified into three groups. The early block group (E) consisted of 25 pts who developed AVB# within 6 hours (hrs) of onset of AMI and the late block group (L) consisted of 18 pts who developed AVB# later than 6 hrs. Control group (C) consisted of 45 pts without AVB. AVB# in E appeared earlier (2.5 vs 57.3 hrs, $p < 0.001$), terminated sooner (14.2 vs 136.1 hrs, $p < 0.001$), and their duration was shorter (11.7 vs 78.8 hrs, $p < 0.001$) compared to those in L. The heart rate (HR) of escaped rhythm during complete block was lower in E (40.9 vs 52.8/min, $p < 0.01$) than in L. Rate of P wave was not different statistically in both groups. No significant differences were observed in age, sex, cardiac index, pulmonary capillary wedge pressure, ejection fraction, peak CK value and peak CK-MB value in three groups. Populations of Killip III or IV on admission and mortality rate were significantly higher in the group of patients with AVB# (E+L) compared with C, but both rates in E and L were similar to each other (36% vs 28%, 32% vs 33%).

In conclusion, i) incidence of AVB# with AMI was 34.6% in inferior infarction and 3.7% in anterior infarction, ii) AVB# appeared within 6 hrs of inferior infarction was characterized by earlier appearance, sooner termination, shorter duration and lower rate of escaped rhythm, iii) patients with AVB# complicating inferior infarction showed higher severity and mortality rate.

-41-

PATHOGENESIS OF VENTRICULAR ANEURYSM FORMATION IN ACUTE MYOCARDIAL INFARCTION

Yutaka HAMANO, Keiichiro SUZUKI, Hiromi RAKUGUI, Osamu KATO, Sugao FUKUI, Takazo MINAMINO, Fumiko WATANABE*, Masayuki MATSUMOTO**

Division of Cardiology, Sakurabashi Watanabe Hospital, Osaka. *Ohtemae Hospital. **Kanazawa Medical College, Department of Geriatrics.

We designed this clinical study to investigate the pathogenesis of ventricular aneurysm (AN) formation, so we undertook a two-part study.

MATERIALS AND METHODS: 97 patients (pts) with 1st transmural anterior myocardial infarction, admitted within 12 hrs after the onset, were studied. 2-D Echo were performed on the 1st, 2nd, 3rd, 7th, 14th, and the 28th day of illness. An echocardiographic AN was defined as a localized area of akinesia or dyskinesia that deformed the ventricular chamber during both diastole and systole.

RESULTS: In the first examination we studied 57 pts treated conventionally. The incidence of AN was 36.8% (21/57). Single vessel disease was more highly in both AN(+) and AN(-), but the infarct-related segment in LAD was different; in AN(+) segment 7 was more highly (63.2%) on the other hand in AN(-) segment 6 was more highly (68.8%) ($p < 0.05$). The coronary artery narrowing was more severe in AN(+); 100% or 99% stenosis with delay was 68.4% in AN(+), but 32.4% in AN(-) ($p < 0.05$). All AN were detected within the 14th day of illness. Small AN were observed during the 1st day to the 7th day, and large AN were detected later. Thus small AN were detected relatively early phase of infarction, the mechanism was speculated that in all pts with small AN segment 7 in LAD was the infarct-related lesion and myocardial contractility on the base of left ventricle was not injured so the infarct area was protruded outward by the contraction of non-ischemic myocardium surrounding the infarct area.

In the 2nd study, We investigated 40 pts treated with PTCT. The incidence of AN was 15% (6/40), and especially in successful PTCT that was only 3% (1/33). In successful PTCT the time of recanalization ranged from 3hrs to 12 hrs, but in cases with late reperfusion as well as early reperfusion only few pt had AN. The degree of coronary artery narrowing in successful PTCT was significantly mild. So we may conclude that not only evolution of necrosis but also reserve of coronary blood flow during the phase of restoration of infarction closely related to the formation of ventricular AN.

-42-

(Paper appears on page 552)

-43-

CLINICAL CHARACTERISTICS OF ACUTE MYOCARDIAL INFARCTION IN PATIENTS WITH DIABETES MELLITUS

-WITH RESPECT TO THE INCIDENCE OF POWER FAILURE-

Shinichiro Ishikawa, Masayuki Taniguchi, Shunichi Fukuhara, Masako Toyama, Masahito Naito, Hideki Nagoshi, Masasada Honda

Department of Cardiology
The Second Tokyo National Hospital

There have been few reports as regards to whether Japanese diabetic patients (DM) with acute myocardial infarction (AMI) have higher incidence of power failure (PF) than non-diabetics (NDM). We analysed 107 consecutive cases of a first AMI, 29 DM and 78 NDM cases. DM cases were subdivided into diet therapy alone (D: n = 15) and drug therapy (M: n = 14). Location of AMI (anterior or inferior) was matched between DM and NDM. The incidences of PF (congestive heart failure and/or shock by MIRU criteria) were compared in these three subgroups.

The incidences of PF in each subgroup were as follows; NDM-33%, DM-48%, D-20%, M-79%. There was no statistically significant difference between NDM and DM (D+M), whereas the cases of M more frequently developed PF than NDM or D ($p < 0.01$).

Thus, we conclude that in this study, cases of M have a higher incidence of PF as a complication of AMI than NDM and D.

Chairman

44-49 N. Yanazaki, *Hamamatsu*
50-55 H. Kanaide, *Fukuoka*

-44- CHANGES AND IRREVERSIBILITY OF MITOCHONDRIA IN THE ACUTE ISCHEMIC MYOCARDIUM

Eiichi GESHI, Takashi KATAGIRI, Toru KITSU, Noburu KONNO, Toshikuni YANAGISHITA, Fumihiro TANNO, Mamoru MOCHIZUKI, Kazuhiko UMETSU, and Hirokazu NIITANI.

Third Department of Internal Medicine, Showa University School of Medicine, Tokyo.

We studied impairment of respiratory activity of mitochondria (Mt) in early ischemic myocardium dividing to subendo- (Endo) and sub-epicardium (Epi) in relation to cellular irreversibility by following reperfusion.

Acute myocardial ischemia was produced in 48 mongrel dogs by LAD occlusion for 15 to 60 min, and in one third of dogs reperfusion was performed for 60 min. Mt were prepared by modified method of Sordahl utilizing alkaline protease.

State 3 respiratory activity reduced significantly in Endo, as early as 30 min after occlusion, to 76% of that of non-ischemic myocardium, and in Epi at 60 min to 84%, and this activity was not recovered by reperfusion. Changes in respiratory control ratio were similar to those in state 3 respiratory activity. Complex I activity of sonicated Mt particles decreased at 30 min to 67% in Endo and at 60 min to 71% in Epi, and was not recovered by reperfusion. Complex II and IV activities did not change significantly in ischemia until 60 min. DNP-stimulated ATPase activity reduced to 79% in Endo at 15 min and to 70% in Epi at 30 min, but was recovered significantly by reperfusion until 30 min.

Appearance of amorphous density in Mt has been considered as a sign of irreversibility by ultrastructural studies. From results above, respiratory activity of Mt was impaired irreversibly in ischemia for 30 min in Endo and this spread to Epi later, and degradation of complex I is one of causes of myocardial irreversibility in early ischemia.

-45- THE CHANGES OF FREE RADICALS IN MYOCARDIAL MITOCHONDRIA DURING ISCHEMIA AND REPERFUSION

Hiroshi Watanabe, Masanori Kaneko, Tadashi Kamikawa, Akira Kobayashi, Noboru Yamazaki

The Third Department of Internal Medicine, Hamamatsu University School of Medicine, Hamamatsu, Japan

Free radicals have been implicated in many diseases. We investigated the changes of free radicals in myocardial mitochondria during ischemia and reperfusion by use of electron spin resonance (ESR) spectrometry. Anesthetized dogs were divided into 3 groups.

In group 1 and 2, the left anterior descending coronary artery (LAD) was occluded for 20 min and 40 min, respectively. In group 3, LAD was reperfused for 5 min following 20 min occlusion. In each group, myocardial mitochondria were prepared from non-ischemic, ischemic or reperfused areas. Free radicals were measured with an X-band JEOL FE2XG ESR spectrometer at -150°C . Results 1) ESR signals were detected at $g = 2.005$ in myocardial mitochondria. 2) Free radical levels in both ischemic and reperfused areas were significantly higher than those in non-ischemic areas. 3) In ischemic areas, free radical levels after 40 min of ischemia were significantly increased as compared with those after 20 min of ischemia. 4) Free radical levels in reperfused areas were significantly higher than those in ischemic areas. These results suggest that free radicals may be an important factor in the development of both ischemic and reperfusion injury.

-46- IDENTIFICATION AND QUANTIFICATION OF FREE RADICALS IN THE MYOCARDIUM WITH ESR SIGNALS

Hiroe K Nakazawa, Atsuko Okazaki, Haruka Okino, Takashi Masuda, Nobuo Aoki**, Hideaki Yoshino, Shingo Hori**, Masahiro Kono***

*Dept of Physiology 2, Tokai Univ, **Keio Univ., *** JEOL Ltd.

Electron Spin Resonance Spectroscopy (ESR) has been applied for the direct evaluation of free radicals in the myocardium, however, a standard technique for sampling and identification of each radical species has not been defined yet.

The purposes of this study are 1) to show time kinetics and temperature stability of radicals for the establishment of a sampling standard; 2) to identify the individual radical species; and 3) to quantify radicals in the ischemic and non-ischemic myocardium.

The measurements of ESR signals were performed at 123 K and samples from the myocardium & mitochondria were frozen at 77 K according to the time schedule. Each radical species were identified by subtraction method and quantified. There were two kinds of radicals in the myocardium, one was consistent with ubisemiquinone radical ($g=2.005$) and the other was defined as $\text{ROO}^{\bullet}$ ($g=2.006$). The time kinetic study disclosed that $\text{ROO}^{\bullet}$ was artificially generated when sampling was delayed for 20 sec and ubisemiquinone decreased rapidly with only 5 sec delay. On the contrary, there was only ubisemiquinone in mitochondria of which concentration was 5 times as much as in the myocardium. Thus, it was shown that we could quantify radicals directly in the myocardium if sampling and freezing were performed less than 1 sec. Radicals were quantified in the non-ischemic and ischemic myocardium after 20 min-LAD occlusion. Ubisemiquinone decreased from 2.3×10^{13} to 1.7×10^{13} spins/mg prot and $\text{ROO}^{\bullet}$ increased markedly in the ischemic myocardium.

-47- BASIC STUDIES ON THE PATHOGENESIS OF