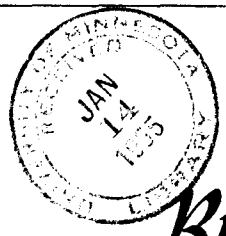

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Myocardial Proteins

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I. SOME EXPERIMENTAL OBSERVATIONS ON THE CONTRACTILE PROTEINS OF THE FAILING MYOCARDIUM*

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Knowledge of the properties of living muscle has increased greatly in recent years. Concommitantly, there has been an increase in knowledge of the chemistry of muscle and the chemistry of muscle contraction. Much of the latter followed the fundamental observation by Engelhardt and Lubimova in 1939 that myosin possessed adenosinetriphosphatase activity.[1] This provided a link for the first time between a contractile unit and what has been considered the major immediate source of energy for contraction. The fundamental studies of Szent-Gyorgyi and his co-workers clarified these relationships and Szent-Gyorgyi has aptly summarized them in the simple statement: "Contraction in muscle is essentially a reaction of actomyosin, adenosine-triphosphate (ATP), and ions."[2]

Actomyosin is a complex of two muscle proteins, F-actin and myosin. F-actin is a very long and asymmetrical fibrous protein, a linear polymer of g-actin. Myosin also is a fibrous protein, not as long as F-actin, with a molecular weight around 840,000. It possesses enzymatic activity (adenosinetriphosphatase) and is thought to anchor the long F-actin particles in place in the myofibril. On examining the fine structure of protein under the electron microscope, one sees a periodicity corresponding to the length of the long F-actin particles.

A number of theories concerning muscular contraction have been recently advanced.[3] Some of these approach this phenomenon from a purely physical standpoint. Others deal with it as a physico-chemical phenomenon. Basically these theories have the following in common: that muscular contraction is the result of the transformation of molecular units from a long to a short form. The unit which undergoes this transformation is the actomyosin complex which changes through interaction with ATP and ions. The evidence is based largely on observations made on muscle models. For example, in the glycerol-extracted skeletal muscle-bundle, a preparation in which the histologic structure of muscle is essentially preserved, the contractile process is no longer initiated through a physiological process of stimulation but through the action of ATP and electrolytes on actomyosin.[4] This has been confirmed for heart muscle preparations by Taeschler and Bing.[5] Also, certain very definite properties of actomyosin in solution such as its viscosity, its flow birefringence, and its sedimentation rate are altered conspicuously when ATP is added to the solution. These properties are ones that depend upon molecular size and shape. It is thus likely that interactions between ATP and actomyosin result in molecular transformations of the latter, possibly, as Szent-Gyorgyi believes an actual dissociation of actin and myosin and a further depolymerization of F-actin to its globular monomers.[4] These relationships are the subject of several excellent recent reviews.[4,6,7,8]

Much of our knowledge concerning heart failure has been acquired through study of the work of the heart as a pump. The discovery of the role of the kidney in the production of the edema of heart failure has also diverted attention from the myocardium

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itself. Important as these contributions are, they fail to give us direct information concerning the state of the heart muscle.

With these considerations in mind we sought to evaluate actomyosin of heart muscle in dogs with experimental valvular lesions and chronic heart failure as compared with that of normal control dogs. In this study actomyosin was extracted in a quantitatively reproducible manner, its characteristics noted, and direct comparisons were made between the two series.

Experimental Materials

Twelve adult mongrel dogs were included in the study. Six of these were random, unselected pound dogs, apparently normal, and these were used as controls. The remaining six constituted the experimental series. All of these had pulmonary stenosis and with one exception, tricuspid insufficiency produced surgically by methods described by Baronofsky and his coworkers.[2,3]

The post-operative history of these dogs and the autopsy findings are summarized in table 1. Characteristically, the dogs developed progressive ascites and in some cases anasarca which was first noticeable from three to four weeks post-operatively. The ascites became maximal 6 weeks to 3 months post-operatively and fluctuated and in some cases disappeared thereafter. Decreased appetite and progressive emaciation was generally noted in this later stage.

The conditions in these dogs which resemble those of congestive heart failure are:

(a) ascites and peripheral edema,
(b) high right atrial pressure,
(c) large, dilated heart on autopsy (the right atrium and right ventricle were most markedly dilated in these dogs), and
(d) congestion of the liver with increased liver weight and thickening of its capsule.

Anorexia and consequent emaciation is also associated with progressive myocardial failure.

Random control dogs and dogs of the experimental series were sacrificed and studied in alternate order. The dogs were sacrificed by anesthetizing them with sodium pentobartital intravenously, quickly opening the chest and excising the beating heart. The excised heart was immediately opened, its chambers washed free of blood with cold tap water and it was then quickly placed in a glass beaker surrounded by ice. After the epicardial fat and connective tissue and base of the great vessels were removed by dissection, the heart was weighed and then divided into the following parts: right ventricle, left ventricle, intraventricular septum, and atria. The tissue was kept in the refrigerator and that not used immediately was divided into small portions wrapped in Parafilm* and stored at -20°C. All extracting procedures on fresh tissue were carried out within three hours of obtaining the specimen.

Methods

Three samples of right ventricle and two of left ventricle were analyzed in each dog. In many cases results were re-checked using frozen muscle. The method of extraction of actomyosin is described in detail elsewhere.[9] It involves fine mincing and homogenization of the minced muscle with a Potter-Elvehjem tissue grinder and extraction with large volumes of Weber's solution (0.6 MKCl at pH of 8.7). Total tissue protein and non-collagen protein were determined on the homogenate, and total "soluble protein" and actomyosin on the Weber's extract by methods described elsewhere.[9,10] Actomyosin was separated from the Weber's extract by precipitation by dilution with water.[9] All procedures were carried out at 0 to 5°C.

*Obtained from Marathon Paper Co., Menaska, Wisconsin.

Dog. No.	1E	2E	3E	4E	5E	6E
Pre-operative Weight, Kg.	15.9	18.1	20.8	15.0	25.4	21.8
Weight, Kg. 3 months after operation	17.7	20.0	24.5	17.4	30.1	23.7
Ascites	Moderate, Persistent	Mild, disappeared later	Moderate disappeared after a few months	Gradually appeared & became marked	Marked	Marked at first, gradually disappeared after 3 months
Right atrial* pressure mm Hg Pre-operative	--	--	1.0	--	--	0
Right atrial pressure, mm Hg 3 months after operation	9.5	5.0	12.5	8.0	13.5	9.0
Time Sacrificed mos. after operation	9	3	17	3	3	5
Autopsy Findings	Liver enlarged & congested. Liver weight = 42 gm/Kg body weight. Heart markedly dilated Approx. 2 liters of ascitic fluid	Emaciated. Liver only slightly congested. Heart moderately dilated. No ascitic fluid or edema	Liver congested Liver weight = 39 gm/Kg body weight Heart moderately dilated. Approx. 50 cc. of ascitic fluid	Markedly emaciated. Liver congested Liver weight = 39 gm/Kg body weight. Heart dilated. Extreme ascites (3.4 liters) Hind leg edema.	Liver moderately congested. Liver weight = 18/gm kg body weight. Heart markedly dilated. Extreme ascites & edema.	Markedly emaciated Liver congested Liver weight = 47/gm/kg body weight. Heart flabby & dilated. No ascites or edema

Table 1: Dogs of experimental series.

* Catheterization of 25 normal dogs disclosed right atrial pressure of -4 to +4.5 mm Hg.

Liver weight in normal dogs in this laboratory is approximately 30 ± 5 gm/kg body weight.

Per cent water and per cent dry weight were determined by weighing a sample of tissue before and after drying to constant weight at 105°C (24 hours). Percent fat-free dry weight was determined by the method of Lowry and Hastings.[11]

Viscosity measurements were made using Ostwald viscosimeters.[9] Adenosinetriphosphatase activity was measured in two ways: (1) as rate of phosphorus liberation: (2) as rate of ATP hydrolysis by following the time course of viscosity change after the addition of ATP. These methods are described in another paper.[9]

Actomyosin was determined in two ways: (1) as protein precipitable from a Weber's extract by dilution with water; (2) by the drop in viscosity on addition of ATP to Weber's extract of muscle.[9] The change in viscosity bears a quantitative relationship to the concentration of actomyosin as can be seen by determining this on purified actomyosin from heart muscle.[9] The degree of change is very similar to that found by others on actomyosin from other sources.[6,12] Results obtained by either of these methods were reproducible within 5% on replicate analyses. The viscosimetric method gave somewhat higher results consistently.

Results

Results of analyses of dry weight and fat-free dry weight are listed in table 2, together with heart weight expressed in grams per kilogram body weight. The mean and standard deviation of the mean for each group were obtained by use of standard formulae and are listed. Student's t test was applied and the significance of the difference in the means of the two groups was judged by reference to a table of values for t.[13] In respect to these determinations the differences did not prove to be significant.

Results of determination of the protein composition of the right ventricle in each group are listed in table 3 and for the left ventricle in table 4. It is noted that the differences are minor except with respect to soluble protein and actomyosin in which case the values in the experimental dogs are consistently lower than the normals. This is true of the left ventricle as well as the right. That these differences are not related entirely to differences in water content is evidenced by the data in table 5 which indicate that the differences in actomyosin are significant when actomyosin is expressed in terms of percent total protein of the tissue. These differences are also noted when actomyosin is expressed as a percent of the total soluble protein.

Because of the findings on viscosimetric analysis, we studied the viscosity response to ATP of separated actomyosin from right and left ventricle. Viscosity number is a term which expresses viscosity per unit protein concentration and is determined by use of the formula: Viscosity number = $Z_\eta = \frac{2.303 \log \text{rel}\eta}{C}$ where $\text{rel}\eta$ is the observed viscosity and C is the protein in grams per liter. The viscosity data before and after the addition of ATP are listed for each dog of both series in table 6.

Portzehl and her co-workers[14] use what she terms "ATP sensitivity" to characterized actomyosin solutions with respect to their actin-myosin ratio. This function is defined as $\frac{Z_\eta - Z_{\eta ATP}}{Z_{\eta ATP}}$, where Z_η and $Z_{\eta ATP}$ are the viscosity numbers before and after the addition of ATP respectively. These values for each dog are also listed in table 6. They are consistently lower in the experimental group, indicating a decreased ratio of actin to myosin.[14,15] Extraction of actin appears to be related in some measure to the degree of fragmentation

CONTROL DOGS					EXPERIMENTAL DOGS				
Dog No.	Heart Wt. in Gm Per Kg. Body Wt.	Ventricle	Dry Weight in % of Total Wet Wt.	Fat-Free Dry Wt. in % of Wet Wt.	Dog No.	Heart Wt. in Gm Per Kg Body Wt	Ventricle	Dry Weight in % of Total Wet Wt.	Fat-Free Dry Wt. in % of Wet Wt.
1C	--	R	21.7	19.8	1E	--	R	22.1	17.1
		L	21.5	20.6			L	20.9	17.8
2C	8.0	R	23.6	20.8	2E	7.7	R	18.7	17.8
		L	22.4	21.9			L	20.0	19.4
3C	6.8	R	21.6	20.9	3E	8.9	R	22.4	18.8
		L	21.5	20.3			L	21.7	20.0
4C	5.2	R	24.7	19.1	4E	7.0	R	19.3	17.3
		L	23.8	20.6			L	20.1	19.1
5C	9.1	R	22.0	20.8	5E	7.6	R	20.5	19.3
		L	22.1	22.0			L	20.7	20.0
6C	8.2	R	22.2	18.3	6E	9.1	R	20.8	18.1
		L	21.3	19.1			L	20.2	19.3
Mean and Standard Deviation	7.4 ±1.4	R	22.6 ± 1.2	20.0 ± 1.1	Mean and Standard Deviation	7.8 ±1.0	R	20.6 ± 1.5	18.1 ± 0.9
		L	21.8 ± 0.5	20.8 ± 1.1			L	20.6 ± 0.6	19.3 ± 0.8

Table 2: Comparison of Control Dogs and Dogs of the Experimental Series: Total wet weight, dry weight and fat-free dry weight of heart ventricular muscle.

Dog No.	Total Protein Homogenate	Non-Collagen Protein	Collagen Protein	Non-Protein Nitrogen*	Total Protein Soluble Extract	Actomyosin (by Precipitation)	Actomyosin (by Viscosity Drop)
			CONTROL DOGS				
1C	14.1	12.5	1.2	3.0	11.1	5.6	6.4
2C	14.1	11.7	2.4	2.7	12.0	5.5	6.5
3C	14.5	13.0	1.4	3.0	11.4	6.0	6.0
4C	13.8	13.4	0.6	3.0	11.4	4.9	5.8
5C	15.6	13.2	2.3	3.5	12.2	5.8	6.8
6C	14.1	13.1	1.2	--	10.3	4.9	6.4
Mean & S.D.	14.4 ± 0.6	12.8 ± 0.6	1.5 ± 0.7	3.1	11.4 ± 0.7	5.4 ± 0.5	6.3 ± 0.2
			EXPERIMENTAL DOGS				
1E	13.4	11.9	2.2	2.6	9.0	3.6	3.3
2E	10.5	9.4	1.1	2.4	9.0	3.7	4.0
3E	14.4	13.3	1.1	2.1	9.6	4.2	4.1
4E	12.8	10.9	1.4	3.0	9.1	4.0	4.4
5E	12.4	11.2	1.2	--	9.5	4.2	4.3
6E	13.5	--	--	--	9.8	4.0	4.3
Mean & S.D.	12.8 ± 1.3	11.3 ± 1.4	1.4 ± 0.5	2.5	9.3 ± 0.3	3.95 ± 0.2	4.1 ± 0.4
t	2.53	2.32	0.34	--	6.94	6.74	10.0
Signif. at .01 level	(10) —	(9) —	(9) —		(10) +	(10) +	(10) +

*Non-protein nitrogen is expressed in mg. per gm. of whole wet tissue.

Table 3: Protein composition of Right Ventricular Muscle in Dogs with Experimental Heart Failure and Random Normal Control Dogs. Results are expressed, except as otherwise noted, in % wet weight of tissue.

Dog No.	Total Protein of Homogenate	Non-Collagen Protein	Collagen Protein	Non-Protein Nitrogen*	Total Protein Soluble Extract	Actomyosin (by Precipitation)	Actomyosin (by Viscosity Drop)
			CONTROL DOGS				
1C	16.5	15.8	0.8	--	13.1	5.8	7.0
2C	15.0	14.4	0.7	2.7	13.2	6.4	7.2
3C	14.9	13.2	1.8	3.2	11.8	5.9	7.2
4C	15.5	13.8	1.7	3.1	11.6	5.0	6.4
5C	15.9	13.4	2.7	3.8	12.5	6.1	7.3
6C	14.5	14.0	0.5	--	10.4	4.7	6.6
Mean & S.D.	15.4 ± 0.7	14.1 ± 0.9	1.4 ± 0.8	3.2	12.1 ± 1.1	5.65 ± 0.65	6.9 ± 0.4
			EXPERIMENTAL DOGS				
1E	14.35	11.6	2.8	2.2	9.0	3.5	4.1
2E	13.6	10.1	3.45	2.4	9.2	3.9	4.5
3E	15.7	14.4	1.3	2.3	10.65	4.9	5.5
4E	14.0	11.6	1.8	3.0	9.6	3.9	5.1
5E	14.8	12.65	2.0	--	10.3	5.3	5.2
6E	14.9	--	--	--	10.2	4.2	5.5
Mean & S.D.	14.55 ± 0.75	12.05 ± 1.25	2.25 ± 0.85	2.5	9.8 ± 0.65	4.3 ± 0.65	5.0 ± 0.35
t	1.89	3.12	1.94		4.44	3.54	9.36
Degrees of Freedom	10	9	9		10	10	10
Signif. at 0.01 level	—	—	—		+	+	+

*Non-Protein nitrogen is expressed in mg. per gram of whole wet tissue.

Table 4: Protein composition of Left Ventricular Muscle of Dogs with Experimental Heart Failure and Random Control Dogs. Figures are in terms of % wet weight of tissue.

Group		Total Protein Soluble Extract	Actomyosin (by Precipitation)	Actomyosin (by Viscosity Drop)
RIGHT VENTRICLE	Control	79.4 ± 4.4	37.9 ± 2.6	44.2 ± 1.95
	Experi-mental	73.75 ± 5.45	29.75 ± 2.65	32.15 ± 4.9
	t	1.96	5.39	5.59
	Degrees of Freedom	10	10	10
	Signif. at 0.01 level	—	+	+
LEFT VENTRICLE	Controls	78.55 ± 5.5	37.0 ± 4.2	45.2 ± 0.9
	Experi-mentals	68.7 ± 4.5	29.2 ± 3.45	34.9 ± 3.75
	t	3.39	3.23	6.52
	Degrees of Freedom	10	10	10
	Signif. at 0.01 level	+	+	+

Table 5: Total protein soluble in Weber's solution and Actomyosin Content expressed as percentage of total protein of tissue. Right and left ventricular muscle, experimental and control groups compared.

DOGS, CONTROL SERIES

	Ventricle	Dog No. 1	2	3	4	5	6	Mean	S.D.
Initial Viscosity Number ($Z\eta$)	R	.319	.314	.281	.286	.328	.319	.307	0.019
	L	.324	.312	.311	.326	.316	.336	.321	0.010
Viscosity Number Following ATP ($Z\eta_{ATP}$)	R	.173	.172	.158	.153	.179	.164	.166	0.010
	L	.167	.161	.167	.171	.162	.164	.165	0.003
ATP-sensitivity in % $\frac{Z\eta - Z\eta_{ATP} \times 100}{Z\eta_{ATP}}$	R	84.5	82.7	78.0	87.0	83.3	94.5	85.0	5.5
	L	94.0	93.8	86.3	90.6	95.0	104.8	94.1	6.2

DOGS, EXPERIMENTAL SERIES

	Ventricle	Dog No. 1	2	3	4	5	6	Mean	S.D.	t	Degrees of Freedom	Significance at 0.01 level
Initial Viscosity Number ($Z\eta$)	R	.232	.279	.224	.288	.254	.262	.257	0.025	7.6	10	+
	L	.254	.299	.276	.280	.263	.300	.279	0.024	4.8	10	+
Viscosity Number Following ATP ($Z\eta_{ATP}$)	R	.144	.161	.136	.172	.156	.150	.153	0.013	4.0	10	+
	L	.159	.168	.165	.167	.154	.162	.163	0.005	1.2	10	-
ATP-sensitivity in % $\frac{Z\eta - Z\eta_{ATP} \times 100}{Z\eta_{ATP}}$	R	61.0	73.2	64.7	67.4	62.8	74.6	67.3	5.5	5.5	10	+
	L	59.7	78.0	67.2	67.6	70.8	85.0	71.4	8.9	8.6	10	+

Table 6: Viscosity Data for Actomyosins Separated from Normal Control Dog ventricular muscle and that of dogs with experimental heart failure.

and dispersion of muscle tissue in the extraction medium.[16] In order to investigate the effect of such factors on the character of the actomyosin extracted, experiments were conducted in which the muscle tissue was homogenized for varying periods of time. Lengthening the period of homogenization beyond that used in these extractions did not increase the "ATP sensitivity" of actomyosin of either control or experimental dog hearts. No actin could be extracted from the residue of either control or experimental hearts. This suggests that the tissue was sufficiently comminuted for maximum extraction of actin and that the extraction process itself was not responsible for the differences in viscosity response or "ATP sensitivity" between the two groups.

Adenosinetriphosphatase activity of both the crude Weber's extract of the muscle and of actomyosin separated from it was determined and results are listed in table 7. The differences between the two groups studied are not remarkable, except in the primary soluble (Weber's) extract in relation to concentration of actomyosin (determined viscosimetrically). In this case, there is an increased ATP-ase activity per unit of actomyosin in the experimental group.

Sedimentation Studies

Since the differences in viscosity and "ATP sensitivity" between the actomyosins separated from heart muscle of the two groups suggest differences in the character of actomyosin, these were studied further in the ultracentrifuge. The actomyosin studied was separated from crude Weber's (primary soluble) extract and four determinations were made on three of the control dogs and four on three experimental dogs. A Spinco analytical ultracentrifuge, Model E, was used with temperatures varying between 16° and 22°C and speeds ranging from 35,600 to 59,780 r.p.m. (92,000 to 259,500 g. average). Protein concentration varied between 1 and 5 mg. per ml.

Figure 1 illustrates a typical pattern obtained on actomyosin from normal dog heart. This actomyosin sediments at a rapid rate and gives a uniform peak, somewhat asymmetrical in outline. The sedimentation rate is markedly dependent on protein concentration in the range of concentration studied. The $S_{20W\ C=0}$ was calculated as ranging between 90 and 280 which compares with that determined by Portzehl et al for actomyosin.[14] The shape of the sedimentation curve when plotted against protein concentration also resembled that of actomyosin measured by Portzehl.

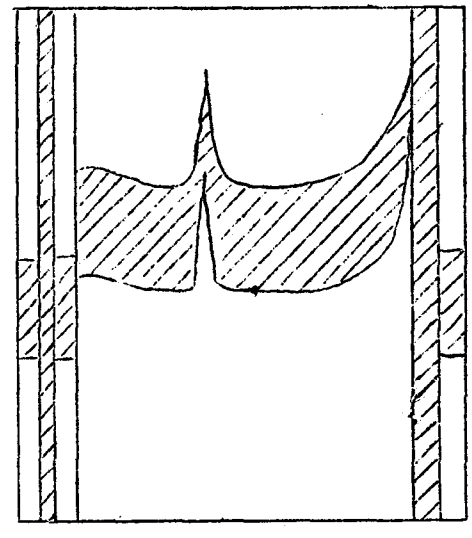

Figure 1.

Sedimentation pattern, cardiac actomyosin, control dog. 35,600 r.p.m., 34½ min., 2.46 mg. protein/ml.

The actomyosin from the experimental dog hearts in each instance showed the sedimentation pattern illustrated in figure 2. Two distinct peaks are seen. The more rapid of these has the dependence on protein concentration of actomyosin. The second peak is much slower and of variable amplitude being most marked in dog 1E. The rate of movement of this peak was not markedly dependent on protein concentration having an $S_{20W\ C=0}$ of 7 to 8 which is

Series	Extract	Ventricle	PER GRAM WET TISSUE: P liberated in mM x 10^{-3} per min	PER GRAM WET TISSUE: ATP Hydrolyzed in mM x 10^{-3} per min	PER GRAM TOTAL PROTEIN OF TISSUE: P liberated in mM x 10^{-3} per min	PER GRAM TOTAL PROTEIN OF TISSUE: ATP Hydrolyzed in mM x 10^{-3} per min	PER GRAM ACTOMYOSIN: P liberated in mM x 10^{-3} per min	PER GRAM ACTOMYOSIN: ATP Hydrolyzed in mM x 10^{-3} per min
Control Dogs	Primary soluble extract	R	1.96 ± .12	0.84 ± .07	13.7 ± 1.2	5.8 ±0.6	31.6 ± 3.2	13.3 ± 1.7
		L	2.11 ± .29	0.85 ± .08	13.7 ± 2.1	5.6 ±0.8	30.1 ± 4.4	12.3 ± 1.6
	Actomyosin, separated	R	3.66 ± .79	3.22 ± .45	26.0 ± 6.0	22.7 ± 2.3	71.8 ±13.9	60.8 ± 9.5
		L	3.40 ± .37	3.13 ± .37	22.4 ± 3.1	20.6 ± 2.9	60.7 ± 5.9	55.4 ± 7.5
Experimental Dogs	Primary soluble extract	R	1.68 ± .20	0.74 ± .07	13.3 ± 0.8	5.8 ±0.7	41.5 ± 6.1	19.0 ± 4.0
		L	1.70 ± .28	0.89 ± .06	12.2 ± 1.8	6.2 ±0.3	34.7 ± 3.3	16.8 ± 1.0
	Actomyosin, separated	R	2.56 ± .36	2.04 ± .58	19.5 ± 2.2	14.8 ± 4.4	93.4 ±22.2	67.4 ±21.5
		L	3.04 ± .37	2.76 ± .35	21.5 ± 3.6	18.8 ± 2.8	90.3 ±25.7	72.5 ± 9.7
t	Primary soluble extract	R	2.87	2.22	0.68	0	3.54	4.16
		L	3.27	0.82	1.35	1.49	2.06	4.90
	Actomyosin Separated	R	3.09	4.24	2.49	3.51	2.04	0.67
		L	1.02	1.51	0.47	0.97	2.81	3.19
Significance at 0.01 level	Primary soluble extract	R	0	0	0	0	+	+
		L	+	0	0	0	0	+
	Actomyosin separated	R	0	+	0	+	0	0
		L	0	0	0	0	0	0

Table 7: Adenosinetriphosphatase activity of heart ventricular muscle actomyosin. Results expressed for two methods, one of which measures rate of inorganic phosphorus liberation and the other, rate of hydrolysis of ATP

the rate of uncombined myosin.[14,17]

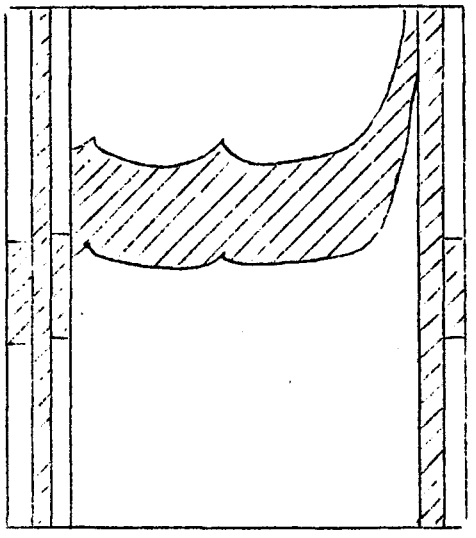

Figure 2.

Sedimentation pattern, cardiac actomyosin, experimental dog. 35,600 r.p.m., 26 3/4 min., 1.79 mg protein/ml.

Discussion

These observations indicate that the actomyosin of the failing myocardium differs from that of normal heart muscle and that this difference may be due to the presence in the former of significant amounts of uncombined myosin. The decreased concentration of actomyosin, the lowered viscosity response to ATP-ase activity per unit of actomyosin, and the sedimentation pattern all favor this interpretation. All of the hearts of the dogs with chronic heart failure were dilated, especially the right ventricles. All had high right atrial pressures which suggest increased diastolic volume. Bing and Taeschler have recently shown that a condition resembling acute failure in work output is produced in washed heart muscle strips by overstretching the fiber by suspending a heavy weight to the muscle strip.[18] In the Starling heart-lung preparation, acute failure also followed increased diastolic volume and overstretching the muscle.[19] Others have observed that a muscle bundle stretched and held at constant length under increased tension loses its intrinsic double refraction in time.[8,20] The intrinsic birefringence is a function of the conformity of actomyosin and reflects the same molecular or micellar characteristics as viscosity and sedimentation rate: i.e., its length and axial asymmetry. Conclusions drawn from this study must be tentative since the evidence is entirely indirect, but the hypothesis is attractive that the changes in actomyosin of the failing heart as noted in this study are related in some way to the physical changes in the heart itself, namely, increased diastolic volume, increased tension, and overstretching of the muscle syncytium. It is possible that actomyosin of the heart muscle, which seems normally arrayed in a state in which actin and myosin are very closely associated, may be partially depolymerized under the influence of stretch beyond certain limits and increased resting tension, conditions which exist in the dilated failing heart.

Summary

Extracts of protein of heart muscle of dogs in chronic heart failure show certain distinct differences from those obtained from normal dogs. These relate most conspicuously to the contractile protein, actomyosin. This protein appears to be present in decreased concentration in the failing heart, and, in addition, shows decreased viscosity response to ATP and a change in the sedimentation pattern. These characteristics are ones which reflect molecular conformity and arrangement and suggest that actomyosin is partially depolymerized in the heart muscle in chronic failure. Corrolaries exist between these findings and those of others on overstretched muscle suggesting the possibility that the changed state of actomyosin in this study of the failing myocardium is related in some way to increased diastolic volume of the heart, increased resting tension, and overstretching of

muscle.

Acknowledgment

The authors acknowledge with deep appreciation the counsel of Drs. M. B. Visscher, David Glick, and G. T. Evans. The stimulation and assistance of the latter was responsible for this study's initiation and is especially noted. Miss Marlene Johnson gave valuable technical assistance. The assistance of Mr. Adrian Lawler in the ultracentrifuge studies is emphasized.

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II. MEDICAL SCHOOL NEWS

Coming Events

Jan. 31 - Feb. 2	Continuation Course in Ophthalmology for Specialists
February 3	Special Lecture: "The Cell and Its Parts; Genetic Re-Combination Mechanism;" Dr. Joshua Lederberg, University of Wisconsin; 8:00 p.m.
February 3 - 5	Continuation Course in Otolaryngology for Specialists
February 7 - 12	Continuation Course in Neurology for General Physicians and Specialists
February 9	J. B. Johnston Lectureship: "Cerebral Circulation and Oxygen Consumption;" Dr. Seymour S. Kety, National Institute of Neurological Diseases and Blindness; National Institute of Health, U. S. Public Health Service, Bethesda, Maryland; Mayo Auditorium; 8:15 p.m.
February 14 - 16	Continuation Course in Internal Medicine for Internists
February 17 - 19	Continuation Course in Cancer Detection for General Physicians

* * *

Continuation Course

The University of Minnesota announces a continuation course in Neurology which will be presented at the Center for Continuation Study from February 7 to 12, 1955. The course, which will be of interest both to physicians in general practice and to those with a special interest in neurology, will stress diagnosis and management of the most frequently-met neurological disorders. Guest speakers will include Dr. G. Milton Shy, Clinical Director, National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda; Dr. Howard D. Fabing, President, American Academy of Neurology, Cincinnati; and Dr. Edward B. Schlesinger, Clinical Assistant Professor of Neurosurgery, Columbia University College of Physicians and Surgeons, New York City. The annual Johnston Lecture will be presented by Dr. Seymour S. Kety, also of the National Institute of Neurological Diseases and Blindness. The course will be presented under the joint direction of Dr. A. B. Baker, Professor and Head, Division of Neurology, and Dr. William T. Peyton, Director, Division of Neurosurgery, University of Minnesota Medical School.

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III.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

January 17 - 22, 1955

Monday, January 17

Medical School and University Hospitals

9:00 - 9:50	Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.
9:00 - 10:50	Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.
10:00 - 12:00	Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.
11:30 - 12:30	Physical Medicine Seminar; Vocational Rehabilitation; Robert Walker; Heart Hospital Auditorium.
11:30 -	Tumor Conference; Doctors Hitchcock, Zimmermann, and Stenstrom; Todd Amphitheater, U. H.
12:15 -	Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.
12:30 - 1:30	Physiology Seminar; Excitability Studies in the Cerebral Cortex; Berry Campbell; 214 Millard Hall.
1:00 - 2:00	Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.
1:30 - 2:30	Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.
1:30 - 3:30	Dermatology Hospital Rounds; H. E. Michelson and Staff; Dermatology-Histopathology Room, C-394 Mayo Memorial.
4:00 - 6:00	Anesthesiology Conference; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.
4:30 -	Public Health Seminar; A Type 3 Poliomyelitis Outbreak on St. Paul Island; Carl Eklund; 15 Owre Hall.
4:30 -	Pediatric-Medicine Infectious Disease Rounds; Station 33, U. H.
5:00 - 6:00	Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss and Staff; Eustis Amphitheater.

Ancker Hospital

8:00 - 9:00	Pediatrics Contagion Rounds; Richard Lein; Contagion 5.
8:30 - 10:30	Medical and Surgical Chest Conference; Dr. Gehlen and Staff; Auditorium.
9:30 - 12:00	Visiting Staff Rounds.
10:00 - 12:00	Surgery Grand Ward Rounds; Begin Floor E4.
11:00 - 12:00	Pediatric Rounds; Harry Orme; Contagion 1.
12:30 - 2:30	Surgery Out-Patient Clinic; Room 8.
2:00 - 3:00	Routine EKG Interpretation; Dr. Sommers and House Staff; Medical Record Library.
2:30 - 3:00	Discussion of Problem Case; Auditorium.

Monday, January 17 (Cont.)

Ancker Hospital (Cont.)

3:00 - 4:00 Surgery Journal Club; Classroom.

3:00 - 4:00 Lectures on Electrocardiography; Ben Sommers; Auditorium.

4:00 - 5:00 Medical Clerk Journal Club; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Richard Raile; Station K.

10:30 - 12:00 Medicine Rounds; Thomas Lowry; Station F.

11:00 - Orthopedic and Fracture Rounds; Drs. John Moe and Arthur Zierold; Station B.

11:00 - Pediatric Seminar; Erling Platou; Classroom, Station M.

12:30 - Surgery Grand Rounds; Dr. Zierold, Station E.

1:30 - 2:30 Tuberculosis Conference; J. A. Myers; Station M.

2:00 - Pediatric Rounds; Stations I and J.

Veterans Administration Hospital

9:30 - Infectious Disease Rounds; Drs. Hall, Zinnemann, and J. Brown.

1:30 - Cardiac Conference; Drs. Smith, Berman, Hoseth, Simonson, Tamlyn, and Farquhar; Conference Room, Bldg. I; Rounds immediately following conference.

Tuesday, January 18

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, Irvine McQuarrie and Staffs; Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 104 Jackson Hall.

12:30 - Physiology Seminar 210; Transport; Selected Topics in Permeability; Nathan Lifson; 129 Millard Hall.

12:30 - Physiological Chemistry Seminar; Transport of Cations Across Membranes; L. J. Greenberg; 214 Millard Hall.

12:30 - Bacteriology and Immunology Seminar; 1050 Mayo Memorial.

12:30 - Anatomy Seminar; Mechanics of Locomotion as Applied to the Rehabilitation of the Physically Disabled; Sayed K. Hamed; 226 Jackson Hall.

3:30 - General Physiology Seminar; 323 Zoology Building.

3:30 - Pediatric Seminar; Accidents in Childhood; Harry Orme; 1450 Mayo Memorial.

4:00 - 5:00 Pediatric Rounds on Wards; Irvine McQuarrie and Staff; U. H.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by University Hospitals Staff; Eustis Amphitheater, U. H.

Tuesday, January 18 (Cont.)

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Dale Cumming; Contagion 1.

9:00 - 10:30 Visiting Staff Rounds.

9:00 - 12:00 Practical Diagnostic Clinic; Harry Orme; Out-Patient Department.

11:00 - 12:00 Medical X-ray Conference; J. R. Aurelius; Auditorium.

2:30 - 4:00 Routine EKG Interpretations; Resident Staff.

4:00 - 5:00 Medical-Pathological Conference; W. F. Mazzitello, Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds Elizabeth Lowry; Station J.

9:30 - 10:30 Obstetrics and Gynecology Staff Rounds; William P. Sadler and Staff; 301 Harrington Hall.

10:00 - Psychiatry Grand Rounds; R. W. Anderson, Station H.

11:30 - 12:30 Neurology-Neurosurgery Conference; Classroom, Station M.

12:30 - 2:30 Dermatology Rounds on Clinic; Carl W. Laymon and Staff.

12:30 - ECG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Coe, and Lipschultz; Classroom.

3:30 - Pediatric-Psychiatry Rounds; Jack Wallinga; Station I.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Surgical Conference Room, Bldg. 43.

8:30 - Hematology Rounds; Drs. Hagen and Wexler.

8:30 - Surgery Journal Club; Conference Room, Bldg. I.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery-Tumor Conference; D. Ferguson and J. Jorgens.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

4:00 - Thoracic Surgical Problems; Conference Room, Bldg. I.

5:00 - Fluid Balance Conference; Conference Room, Bldg. I.

5:30 - Physiology Seminar; Surgical Conference Room, Bldg. 43.

Wednesday, January 19

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler, Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery-Pediatrics Conference; Todd Amphitheater, U. H.

Wednesday, January 19 (Cont.)

Medical School and University Hospitals (Cont.)

12:30 - Physiology Seminar 212; Selected Topics in Respiration: Respiratory and Circulatory Effects of Hypothermia; E. B. Brown; 129 Millard Hall.

12:30 - 1:20 Radio-Isotope Seminar; Betatron Room in Cobalt Underground Section, U. H.

1:00 - 2:00 Dermatology Clinical Seminar; F. W. Lynch; 300 North Clinic.

1:30 - 3:00 Pediatric Allergy Clinic; Albert V. Stoesser and Lloyd Nelson; W-211, U. H.

3:30 - 4:30 Dermatology-Pharmacology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:30 - 5:50 Dermatology-Infectious Disease Seminar; 3rd Floor, Conference Room, Heart Hospital.

5:00 - 6:00 Residents Lectures; Retrospectroscope; Leo G. Rigler; Todd Amphitheater, U. H.

5:00 - 5:50 Urological-Pathological Conference; C. D. Creevy and Staff; A503, Mayo Memorial.

5:10 - 6:10 Endocrine Seminar; 271 Lyon Laboratories.

5:30 - 7:30 Dermatology Journal Club and Discussion Group; Hospital Dining Room.

7:30 - 9:30 Dermatology Seminar; Review of Interesting Slides of the Week; Robert W. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; J. Noble; Auditorium.

11:00 - 12:00 Pediatric and Contagion Rounds; Harry Orme; Contagion 1.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

3:30 - 4:30 Pediatric Surgery Conference; Harry Orme and I. D. Baronofsky; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Henry Staub; Station 1.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

12:00 - Surgery-Physiology Conference; Arthur Zierold, E. B. Brown; Classroom.

12:15 - Pediatrics Staff Meeting; Classroom, Station I.

1:30 - Pediatric House Staff Seminar; Erling Platou; Station I.

1:30 - Pediatric Rounds; Erling Platou; Classroom, Station I.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Surgical Conference Room, Bldg. 43.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Zieve, Ferguson, Brakel, Swenson, Nesbitt, and Sadoff.

Wednesday, January 19 (Cont.)

Veterans Administration Hospital (Cont.)

10:30 - Psychosomatic Conference; C. K. Aldrich; 7th Floor, Bldg. 43.

12:30 - Medical Journal Club; Doctors' Dining Room.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

1:30 - 3:00 Metabolic Disease Conference; Drs. Flink and Williams.

3:30 - Urology Pathology Slide Conference; Dr. Gleason; Conference Room, Bldg. I.

7:00 - Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, January 20

Medical School and University Hospitals

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Room 3.148 Mayo Memorial.

11:00 - 12:00 Cancer Clinic; K. Stenstrom, A. Kremen, and B. Zimmermann; Todd Amphitheater, U. H.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Anesthesiology Seminar; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.

5:00 - 6:00 Radiology Seminar; Trip to Colombia, South America; Harold O. Peterson; Eustis Amphitheater, U. H.

7:30 - 9:30 Physiology 211 Seminar; Selected Topics in Heart and Circulation: Hemodynamics; M. B. Visscher and Robert Evans; 271 Lyon Laboratories.

Ancker Hospital

8:00 - 9:00 Pediatric Clinical Staff Conference; Contagion Classroom.

9:00 - 10:00 Pediatric Contagion Rounds; Alexander Stewart; Contagion 5.

9:30 - 10:30 Medical Grand Rounds; Auditorium; Visiting Staff Rounds immediately following Grand Rounds.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

11:00 - 12:00 Pediatric X-ray Conference; Auditorium.

2:00 - 3:00 Routine ECG Interpretation; Ben Sommers; Medical Record Library.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

9:30 - Pediatric Contagion Rounds; R. B. Raile; Station K.

10:00 - Psychiatry Grand Rounds; R. W. Anderson and Staff; Station H.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

12:30 - 2:30 Dermatology Rounds and Clinic; Carl W. Laymon and Staff.

1:00 - Fracture X-ray Conference; Drs. Zierold and Moe; Classroom.

1:00 - House Staff Conference; Station I.

Thursday, January 20 (Cont.)

Veterans Administration Hospital

8:00 - Experimental Surgery Laboratory Meeting; Conference Room, Bldg. I.

8:30 - Hematology Rounds; Drs. Hagen and Doe.

9:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

9:00 - Surgery Ward Rounds; D. Ferguson and Staff; Ward 11.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

1:00 - Infectious Disease Conference; Conference Room, Bldg. I. (Rounds immediately following conference.)

4:00 - 5:00 Medical-Surgical Conference; Medical Conference Room, Bldg. I.

Friday, January 21

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

11:00 - 12:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

11:45 - 12:50 University of Minnesota Hospitals Medical Staff Meeting; Factitial Proctitis: A Preliminary Report; B. J. Kaplan; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

1:30 - 2:30 Dermatology Grand Rounds; Presentation of Cases from Grouped Hospitals (University, Ancker, General and Veterans) and Private Offices; H. E. Michelson and Staff; Eustis Amphitheater, U. H.

2:30 - 4:00 Dermatology Hospital Rounds; H. E. Michelson and Staff; Begin at Dermatological Histopathology Room, C-394 Mayo Memorial.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Dermatology-Physiology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:00 - 5:00 Physiology Seminar 213; Selected Topics in Advanced Neurophysiology: Role of the Vestibular Apparatus and the Cerebellum in the Extra-pyramidal Motor Activity; Werner Koella; 129 Millard Hall.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hanson and Staff; E-534; U. H.

5:00 - Urological Seminar and X-ray Conference; A503, Mayo Memorial.

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Charles Steinberg; Contagion 1.

11:00 - 12:00 Contagion Rounds; Harry Orme; Contagion 5.

10:30 - 11:30 Pediatric Contagion Rounds; Richard Smith; Contagion 1.

2:00 - 3:00 Routine EKG Interpretation; Resident Staff.

3:00 - 4:00 Medical-Surgical-Pathological Conference; Auditorium.

Friday, January 21 (Cont.)

Ancker Hospital (Cont.)

4:00 - 5:00 Medical Journal Club; Conference Room, E5.

4:00 - 5:00 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Elizabeth Lowry; Station J.

10:00 - Otolaryngology Conference; Robert A. Priest; Large Classroom.

10:30 - Pediatric Surgical Conference; Tague Chisholm and B. Spencer; Classroom,. Station I.

12:00 - Surgery-Pathology Conference; Drs. Zierold and Coe; Classroom.

1:00 - 3:00 Clinical-Medical Conference; Thomas Lowry; Classroom, Station M.

1:30 - Pediatric Contagion Rounds; L. Wannamaker; Station K.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

11:00 - 12:30 Psychiatry Case Conference; Werner Simon; Psychiatry Department, VA Hospital Annex.

12:30 - Urology X-ray Conference; X-ray Department.

1:00 - CPC Conference; Conference Room, Bldg. I.

2:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, January 22

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 9:30 Pediatric Grand Rounds; Eustis Amphitheater, U. H.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Rounds; J. L. McKelvey and Staff; Station 44, U.H.

10:00 - 12:00 Otolaryngology Seminar on Current Literature; L. R. Boies and Staff; Todd Memorial Room, A-675, Mayo Memorial.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

9:30 - 11:00 Medicine Grand Ward Rounds; W. F. Mazzitello.

11:00 - 12:00 Medical Clerk Case Conference; W. F. Mazzitello.

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

9:00 - Psychiatry Grand Rounds; R. W. Anderson; Station H.

Saturday, January 22 (Cont.)

Minneapolis General Hospital (Cont.)

9:30 - Pediatric Rounds on all Stations; R. B. Raile.

11:00 - 12:00 Medical X-ray Conference; O. Lipschultz, Thomas Lowry and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Medical X-ray Conference; Conference Room, Bldg. I.