The Effect of Varying Dietary Magnesium on the Electrocardiogram and Blood Electrolytes of Dogs.

ICHIO ONO

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This study is concerned with the effects of varying the dietary levels of magnesium on body weight, blood electrolytes and electrocardiogram in the dog. Animals were fed purified diets containing varying levels of magnesium and potassium salts. A lowered intake of dietary magnesium resulted in a significant decrease in the serum potassium levels with a consequent peaking of the T waves. When the serum magnesium levels decreased to 0.8 mg% or less these electrocardiographic changes usually appeared.

THE PURPOSE OF this study is to correlate changes observed in the electrocardiogram with varying concentrations of serum magnesium.

There has been much discussion and some disagreement in the articles concerning electrocardiographic changes for hypo- and hyperpotassemia.

The electrocardiographic changes of hypo- and hyperpotassemia may be related to the myocardial intracellular or extracellular potassium concentrations, serum potassium, the transmembranous gradient of potassium.

On the other hand, excellent reviews of magnesium metabolism and requirement have been reported by Wacker and Vallee and O’Dell. Magnesium deficiency results in calcification of the heart and kidney. In 1938, Moore reported that the principal pathologic alterations in calves fed diets low in magnesium consisted of a deposition of calcium in the yellow elastic fibers of the endocardium, of the aorta, jugular vein and large arteries. Also notable were degeneration and calcification of Purkinje fibers. Recent studies of Vitale and co-workers have demonstrated that magnesium deficiency enhances the lipid deposition within the left ventricular valves and aorta, magnesium deficiency produces profound changes in the myocardium and enhances cardiovascular sudanophilia, and produces of calcification of the elastica, media of the aorta, of coronary and other peripheral arteries, and of the inner portion of the myocardium.

The present study deals with the effects of varying the dietary levels of magnesium and potassium on body weight, blood electrolytes and electrocardiogram in the dog.

METHODS

Experiment I

Nine male and female mongrel dogs, 6 weeks old, weighing 3.5 Kg., were fed purified diet which consisted of the following in per cent: cellulofine 30; glucose, 64.20; casein, 20.0; salt mixture*
(low K+, Mg++) 2.51; choline, 0.2; Vitamin (dog), 0.239; corn oil, 9.0; cod liver oil, 1.0; MgO, 0.08 (48 mg% Mg+2); the author divided above into three 5 Kg portions:

Group I:
Add 8.84 g K2CO3 per 5 Kg (100 mg% K+)

Group II:
Add 17.68 g K2CO3 per 5 Kg (200 mg% K+)

Group III:
Add 34.36 g K2CO3 per 5 Kg (400 mg% K+)

The dogs were fed and watered ad libitum and housed in individual cages with screen bottom in a temperature controlled room (72°F).

*Composition of Salt Mixture

<table>
<thead>
<tr>
<th>Component</th>
<th>Amount (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaCl</td>
<td>292.5</td>
</tr>
<tr>
<td>Ca3HPO4</td>
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<tr>
<td>CaCO3</td>
<td>138.05</td>
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<tr>
<td>FeSO4.7H2O</td>
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<tr>
<td>MnSO4</td>
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</tr>
<tr>
<td>ZnCl2</td>
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</tr>
<tr>
<td>CuSO4.5H2O</td>
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<tr>
<td>CoCl2.6H2O</td>
<td>0.0476</td>
</tr>
<tr>
<td>KI</td>
<td>1.66</td>
</tr>
</tbody>
</table>

For 1 part in grams

Prior to being fed the experimental diet, all dogs were dewormed and vaccinated with distemper. Venous blood was drawn approximately every two weeks or one month. The serum was analysed for sodium and potassium by flame photometry, for magnesium according to the method of microestimation by D. Schschlcer,8 for calcium by titration with EDTA,9 for potassium content in skeletal muscle and myocardium by M. Nickerson’s method,97 for hemoglobin determination by oxyhemoglobin method, for red blood cell potassium concentration by the flame photometer of Atomic Baird Inc. proceeded as in the procedure for determining serum potassium and was calculated by means of the Rowell’s formula.50

Experiment II

The experiment was the continuation of experiment I, but dietary magnesium was changed approximately four months after experiment I. Magnesium in the form of magnesium oxide (MgO) was added to the basic diet at levels of 0.02% (12 mg% Mg+2), but dietary potassium was same as in experiment I and all dogs were divided into three groups. The animals were housed and fed as in experiment I. Venous blood was drawn approximately every one or two weeks. The serum potassium, sodium, magnesium, calcium levels, hemoglobin concentration, red blood cell potassium concentration were determined by the same methods as in experiment I. Electrocardiograms were taken by using Pavlov’s standing table while the dogs were awake without anesthesia. The limb lead, unipolar limb lead, right and left precordial leads (V3R and V4) were recorded in all animals. Various observations were made on the electrocardiogram including rate, P-R interval, Q-T interval, amplitude of QRS complexes, deviations of ST segment, amplitude of T and U waves, and the incidence of arrhythmias. The Q-T index was calculated by means of Bazett’s formula,10 the P-R interval was determined by means of the Ashman Hull’s table11 on lead II. T wave height were expressed as a percentage of the R-wave height in the chest lead. The material for this paper consisted of 241 electrocardiograms taken on 9 dogs. 9 dogs of this experiment were killed at 6 months.

RESULTS

Growth. There were no peculiar differentiation of the growth in the experiments of varying dietary magnesium.

Serum electrolytes.

Initially, serum potassium values were between 5.5 and 5.8 mEq/L, serum magnesium values were between 1.30 and 1.85 mg% in experiment I. Terminally, the average serum magnesium level of experiment I was 1.09 mg% and these values showed from 1.00 to 1.15 mg%.

However, when animals were changed to a very low magnesium diet (12 mg% Mg+++) on 4 months, terminally, serum magnesium levels showed the decrease from 0.5 to 0.9 mg% on 40 days (experiment II). On the other hand, these initial serum potassium levels were between 5.5 and 5.8 mEq/L in experiment I. After about 4 months on the low magnesium diet (48 mg% Mg++), the serum potassium of group II (dietary 48 mg% Mg++ and 200 mg% K+) decreased an average of 0.5 mEq/L, while group III (dietary 48 mg% Mg++ and 400 mg% K+) showed an increase of an average of 0.5 mEq/L and rose above 6.0 mEq/L. However, when dogs were changed to experiment II, terminally these values of group II and III decreased an average of 1.8 mEq/L, moreover there were no significant differentiation of terminal serum potassium

values between three groups. Although the values are not tabulated here, serum calcium and sodium did not appear to be affected by dietary magnesium levels. Table I illustrates the effect of low-magnesium diet on serum magnesium and potassium levels in experiment I and II.

Table II illustrates the results of variations of red blood cell potassium concentrations in experiment II and III. This experiment III was normal dietary magnesium (96.5 mg% Mg**+) and dietary potassium was same as in experiment II. Red blood cell potassium concentrations in experiment II were higher than that in experiment III and there were significant differentiation between both experiments. On the other hand, there were no difference on the hemoglobin and hematocrit.

**Electrocardiographic observations.**

There were the effect of varying dietary magnesium and potassium on electrocardiograms in the dogs (experiment I through II). T wave changes.

On 241 occasions in these experiments the serum potassium and magnesium levels were known and the T wave height was measurable in 8 leads of the electrocardiogram.

The lowered intake of dietary magnesium resulted in a significant decrease in the serum potassium and magnesium levels with consequent peaking of the T wave, especially in lead VR (Table I, III, Figs. 1 to 7). When the serum magnesium levels decreased to 0.8 mg% or less these peaked T waves

### Table I Potassium and Magnesium in Serum of Expt. I and II

<table>
<thead>
<tr>
<th>Date</th>
<th>Level</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>1/5</td>
<td>Mg</td>
<td>1.85</td>
<td>1.80</td>
<td>1.54</td>
</tr>
<tr>
<td></td>
<td>K</td>
<td>5.8</td>
<td>5.8</td>
<td>5.7</td>
</tr>
<tr>
<td>1/19</td>
<td>Mg</td>
<td>1.10</td>
<td>1.10</td>
<td>1.20</td>
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<tr>
<td></td>
<td>K</td>
<td>4.6</td>
<td>4.8</td>
<td>5.1</td>
</tr>
<tr>
<td>2/6</td>
<td>Mg</td>
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<td>1.10</td>
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<tr>
<td></td>
<td>K</td>
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<tr>
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<td>Mg</td>
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<td>1.18</td>
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<tr>
<td>3/8</td>
<td>Mg</td>
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<td>1.16</td>
<td>1.16</td>
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<td>4/5</td>
<td>Mg</td>
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<td>K</td>
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<td>K</td>
<td>4.1</td>
<td>4.2</td>
<td>4.0</td>
</tr>
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</table>

**Dietary Mg is changed (12 mg%) on 4/24**

<table>
<thead>
<tr>
<th>Date</th>
<th>Mg</th>
<th>K: Potassium mEq/L</th>
<th>Mg: Magnesium mg%</th>
</tr>
</thead>
<tbody>
<tr>
<td>5/11</td>
<td>0.92</td>
<td>0.92</td>
<td>0.99</td>
</tr>
<tr>
<td>5/25</td>
<td>0.62</td>
<td>0.62</td>
<td>0.98</td>
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<td>5/31</td>
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<td>0.63</td>
<td>0.66</td>
</tr>
<tr>
<td>6/7</td>
<td>0.70</td>
<td>0.85</td>
<td>0.98</td>
</tr>
<tr>
<td>6/14</td>
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<td>0.90</td>
</tr>
<tr>
<td>6/21</td>
<td>0.60</td>
<td>0.58</td>
<td>0.90</td>
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</table>

*Japanese Circulation Journal Vol. 26, August, 1962*
Table II Hemoglobin, Hematocrit and Red Blood Cell Potassium Concentration of Expt. II and III

<table>
<thead>
<tr>
<th>Date</th>
<th>Level</th>
<th>Expt. III</th>
<th>Expt. II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Group I</td>
<td>Group II</td>
</tr>
<tr>
<td>5/25</td>
<td></td>
<td>9</td>
<td>14.4</td>
</tr>
<tr>
<td>Ht</td>
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<td>9</td>
<td>14.6</td>
</tr>
<tr>
<td>Hb</td>
<td></td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>RBCK+</td>
<td></td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>5/31</td>
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<td>50</td>
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<td>14.6</td>
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<tr>
<td>Hb</td>
<td></td>
<td>48</td>
<td>49</td>
</tr>
<tr>
<td>RBCK+</td>
<td></td>
<td>48</td>
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</tr>
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<td>49</td>
<td>49</td>
</tr>
<tr>
<td>PBCK+</td>
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<td>49</td>
<td>49</td>
</tr>
</tbody>
</table>

Ht: Hematocrit %, Hb: Hemoglobin g/dl, RBCK: Red Blood Cell Potassium mEq/L

usually appeared.
Rarely, too, low amplitude of the T waves showed in animals having hypomagnesemia (serum magnesium level less than 0.09 mg%).

ST segment.

Depression of ST segment in limb lead or chest lead appeared in the dogs with a values for serum magnesium of less than 1.0 mg% (Table III).

P–R interval.

\[
\text{Fig. 1.}
\]

Note peaking of T waves in lead VL (No. 1 of Expt. II).

P–R interval almost remained in normal ranges. In a few cases, P–R interval was noted to prolong slightly as the serum potassium decrease (Table III).

Q–T interval.

The relationship of Q–T interval to concentration of serum potassium has been studied frequently by many investigators. Originally, the Q–T interval was thought to be prolonged by hypokalemia.\(^{12-15}\) In pre-
THE EFFECT OF VARYING DIETARY MAGNESIUM

![Fig. 3. Electrocardiograms of No. 9 (Expt. II). Peaked T waves are present in lead VL in tracing dated 4/21, 5/12 and in lead VR in tracing dated 6/2, 6/16.]

![Fig. 4. Note peaking of T waves in lead VR. (No. 5 of Expt. I, II.)]

![Fig. 5. Note peaking of T waves in lead VR (No. 2 of Expt. I, II).]

![Fig. 6. Electrocardiograms of No. 7 (Expt. I, II). In lead VR the T wave peaked.]

![Fig. 7. Electrocardiograms of No. 4 (Expt. II). Note peaking of T waves in leads II, III and AVF.]

esent study, however, Q–T interval almost remained in normal ranges.

Conduction defects and premature contractions.

In these experiments there were a low incidence of appreciably premature contractions accompanying abnormal serum magnesium levels. (Table III.)

Other observations

Within 3 to 7 weeks most of the low magnesium or magnesium-deficient dogs appeared some convulsion, by perirritability or hypersalivation when placed in the Pavlov's stand or the cage, in contrast with the fairly placid control animals (experiment III). Es-
especially, there was observed extreme hyperflexibility and hyperextensibility of the front paws.

**DISCUSSION**

There has been much discussion and some disagreement in the literature concerning electrocardiographic changes for varying concentrations of serum electrolytes, especially potassium. Sidney Ringer in 1883 first emphasized the importance of potassium and other ions in maintenance of normal cardiac functions. There are many reports of degeneration in heart muscle as a result of potassium depletion, but it is not clear even now whether the disturbed cardiac cellular function is dependent on myocardial depletion (heart muscle only) or total body depletion of potassium concentration. Many investigators have stated that the electrocardiogram is a reliable guide to potassium depletion, but some investigators have stated that the electrocardiogram cannot be relied on as a guide.

The correlation of the electrocardiogram and serum potassium levels has not been perfect because the potassium content of myocardium may be low when the serum potassium level is high, and vice versa. The electrocardiographic changes may be related to the myocardial intracellular potassium, the serum potassium, the transmembranous gradient of potassium, the rate of transmembranous diffusion of potassium, however, it is not the purpose of this paper to present the details of these various theories.

Diet induced magnesium deficiency in male puppies slightly were resulted in lowered weight gain, but there were no important differentiating of the growth in present experiment of the lowered intake of dietary magnesium. The appearance of convulsion, hypersalivation or hyperirritability was showed within 3 to 7 weeks after feeding of low magnesium diet. Kruse et al were the first to describe the characteristic signs of magnesium deficiency in rats. These consist of peripheral vasodilatation and edema occurring after some days on a deficient diet. They observed hyperexcitability and convulsions after about 3 weeks. On the other hand, Watchorn & Mc Cance observed that hyperexcitability did not occur in larger rats. The serum magnesium levels of magnesium deficient dogs on a magnesium deficient diet

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(magnesium 48 mg%) dropped early until 1.0 mg% in the course of the experiment (experiment I), however, when dietary magnesium was changed (12 mg%) the serum magnesium levels all the more decreased to 0.8 mg% or less. As shown in table I, dogs fed the magnesium deficient diet (12 mg% Mg⁺⁺) with normal or high potassium diet (200 or 400 mg% K⁺) developed a lowered serum potassium concentration in contrast with control dogs which developed no such changes. J. Vitale and co-workers⁵ reported there was a significant decrease in the serum potassium concentration of the magnesium deficient dogs. Cotlove et al.⁶ have described a lesser degree of potassium depletion in rats subjected to milder and less prolonged magnesium deficiency. Mac Intyre and Davidson⁷ showed that magnesium deficiency in rats produced a secondary intracellular potassium deficiency of muscle from thigh and back, a slight decrease in the serum potassium, and a significant rise in the serum sodium. However, magnesium deficiency had no effect on serum sodium and calcium in the present study. Bartley, Davies & Krebs⁸ pointed out that mitochondria may be the basic units responsible for active transport in the cell and it is possible that the potassium deficit found in their experiments was due to an effect of magnesium deficiency on ion transport in vivo. Reynolds and co-workers⁹ reported that a number of abnormal magnesium determinations (both high and low) were no apparent electrocardiographic changes. Martt and associates¹⁰ found that no specific electrocardiographic alterations could be attributed to hypomagnesemia in their study. Grantham et al.¹¹ have been studied the effects of magnesium deficiency in the presence of normal calcium on the electrocardiogram in dogs depleted of magnesium by means of dialysis and it was found that reduction of external magnesium concentration to less than half of normal concentration failed to produce any electrocardiogram changes. In 1961, Vitale and co-workers¹² pointed out that peaking of the T waves and ST segment depression were observed in the dogs fed the magnesium deficient diet. I showed similar electrocardiographic changes of the peaked T waves and slight ST segment depression in case of hypomagnesemia in the present experiments. When the serum magnesium levels decreased to 0.8 mg% or less these electrocardiographic changes usually appeared. This peaked T wave often was observed in lead VR. For the differentiation of peaked T wave pattern between the hyperpotassemia and hypomagnesemia, the position of precordial leads are recommended, but there are some exceptions. A fairly close relationship between the height of T wave in the precordial leads and serum magnesium level is evident (Fig. 8). It will be seen that in those cases with unusually tall T waves (more than 40 % of R) the serum magnesium level was decreased in every instance except 3 cases. Talled and peaked T waves with a narrow base usually have been described as an important electrocardiographic change of hyperpotassemia. This peaking of T waves, however, occurred in dogs on the magnesium deficient diet, dogs with a decreased serum potassium concentration. Vitale and co-workers¹² suggested that the electrocardiographic changes seen in the magnesium deficient dogs may be the result of intracellular losses of potassium and perhaps, of magnesium as well.

![Fig. 8. Relation of the serum magnesium level to the height of the T wave in chest leads in exp. I and II. The two vertical lines indicate the approximate normal range of T wave height expressed as a percentage of the R wave height in the same lead.](image)

In 1939 Barkes and associates¹³ reported that acidosis was found to increase the T wave amplitude, Weaver and Burchell¹⁴
reported that in hypokalemic patients the acid-base balance was disturbed, showed tall T waves. Serum pH, phosphate and carbon dioxide were not studied in the present experiments, however, potassium levels in red blood cell in hypomagnesemia of magnesium deficient diet dogs were higher than that of the control animals (experiment III). Mac Intyre et al. found that potassium content of skeletal muscle fell to 80% of the initial content after 64 days on magnesium deficient diet in rats, but they did not observe potassium content of myocardium. In contrast, in the present experiment, potassium content of heart muscle was not peculiarly or sometimes showed slightly higher than that of the control animals. (Table IV). In view of these facts, I suppose that perhaps there is an relative increase of intracellular potassium concentration in myocardium. Table III illustrates the comparison of electrocardiographic changes at varied serum magnesium concentrations.

<table>
<thead>
<tr>
<th>dog No.</th>
<th>muscle K⁺ concentration (mEq/Kg. wet wt.)</th>
<th>skeletal muscle</th>
<th>myocardium</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>83.2</td>
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<td></td>
</tr>
<tr>
<td>2</td>
<td>86.4</td>
<td>85.6</td>
<td></td>
</tr>
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</tr>
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<td>9</td>
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</tr>
<tr>
<td>control</td>
<td>110.0±2.0</td>
<td>85.1±1.6</td>
<td></td>
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</table>

**Summary**

This research is concerned with the effects of varying the dietary levels of magnesium on body weight, blood electrolytes, electrocardiogram in the dog. Dogs were fed purified diets containing varying levels of magnesium and potassium salts.

1) A lowered intake of dietary magnesium resulted in a significant decrease in the serum potassium levels with a consequent peaking of the T waves (especially in lead VR), slight depression of the ST segment. When the serum magnesium levels decreased to 0.8 mg% or less these electrocardiographic changes usually appeared.

2) For the differentiation of peaked T wave pattern between the hyperpotassemia and hypomagnesemia, the position of precordial leads are recommend, but there were some exceptions.

3) Arrhythmias sometimes appeared in hypomagnesemia.

4) Potassium levels in red blood cell, there were some differentiations between hypomagnesemia (experiment II) and hypopotassium (experiment III).

5) Within 3 to 7 weeks the magnesium deficient dogs were noted some convulsion, hypersalivation, and hyperirritability.

6) The potassium content of skeletal muscle fell to 86% of the controls, however, the potassium content of myocardium approximately showed slight increase in contrast with control dogs.

The author gratefully acknowledges their indebtedness to Dr. M. Hegsted and Dr. B. Lown, Department of Nutrition, Harvard University of Public Health, for kind guidance in conducting this study. The author is also greatly indebted to Dr. J. Di Giorgio for the technical aid.

**REFERENCES**


ショック 重症感染症 危急時に

速効性・水溶性強力合成副腎皮質ホルモン

デカドロン注射液
（デキサメサゾン＝21－塩酸エステル）

特長
○卓越せる効力を有します。
極めて強力な抗菌性、抗アレルギー性、抗リウマチ性を有し、コートゾンの35倍、ハイドロコーチゾンの28倍、ブレドニゾン及びブレドニゾロンの7－10倍の効力を示すだけでなく、副作用のほとんどが投与後最初の合成副腎皮質ホルモン、デカドロンの水性注射剤です。

○唯一の水溶性製剤で、極めて速効性です。
完全な水溶液となっており、極めて速効性で発作、危急時にも救命的効果を示します。又昏睡、悪心、嘔吐、麻酔等で経口投与が不可能の場合にも使用出来る唯一の全身性非経口投与製剤です。

○いろいろな用途に使用出来ます。
筋注はもちより静注、静脉点滴、関節腔内注、帯組織内注、結膜下注、皮内注更に稀釈点眼や吸入にも使用出来、広範囲の疾患にもっとも効果的な方法で投与出来ます。

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熱ペン書き心電計です
○周波数特性が特に優れ微少
波形をも写真式同様に描記
できます
○電源電圧変動に対しても極
めて安定です

トランジスタ化
直記式心電計
S T－1968 A形
○トランジスタを主体とした
感熱紙使用によるもので非
常に省形軽量(6.5kg)且つ
コンパクトにできているた
め従用としても特別に便利
○消費電力が極めて少なくて
すむことは申すまでもあり
ません

集団検診用心電計
S T－1819形

三要素心電計
S T－1574 B形

Toshiba

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