Experimental Studies on the Sugar Puncture. :
Part 2. On the Origin of the Hyperglycaemia
occurring after the Sugar Puncture

Mikio Miyake*
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Abstract

1. In the normal rabbits the intravenous application of adrenaline cause hyperglycaemia, but in rabbits which have been previously splanchniectomized on both sides, the adrenaline application always betrayed hyperglycaemia. 2. In the normal rabbits ergotoxine had no influence on the blood sugar content. Contrary to the normal animals, a rabbit which received previously an intravenous injection of ergotoxine by which the vasoconstriction of the blood vessels was made impossible, sugar puncture was not effected in the blood sugar content. 3. Contrary to the normal animals, in the rabbits which were treated previously with intravenous application of ergotoxine, the electrical stimulation of the basis of the brain did not cause hyperglycaemia. 4. By the perfusion of the liver of toad with Ringer solution, on adding of adrenaline in the perfusing liquid, discharge of the sugar in the perfusate was increased in percentage, but the absolute quantity was reduced on account of the reduction of the amount of perfusate. 5. The discharge of sugar from the perfusing liver of the toad, which was previously perfused sufficiently with ergotoxine-Ringer solution, on adding of adrenaline in the perfusing liquid showed no change compared with these without ergotoxine and adrenaline application. 6. The pique-hyperglycaemia may be assumed as adrenaline-hyperglycaemia. The origin of adrenaline-hyperglycaemia is rather mechanical, since the torrent of blood stream is increased by the strong contraction of blood vessels of skin and muscles than those of liver, washing out the reserved sugar in liver.

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Part 2. On the Origin of the Hyperglycaemia occurring after the Sugar Puncture

By

Mikio Miyake.

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Introduction

It is generally acknowledged that the hyperglycaemia caused by the sugar puncture are due to the transmission of the augmentatory impulses from the cells in the central nervous system to the adrenal glands and liver by means of splanchnic nerves. Further it is confirmed by the experiments of many investigators, so that if splanchnic nerves are previously cut off, the hyperglycaemia and glycosuria are markedly restrained.

Analogous results can be obtained when the central nervous system in a certain level is sectioned after the sugar puncture. For instance, Mellanby\(^2\) observed the delayed hyperglycaemia and glycosuria after the transverse section at the level of superior corpora quadrigemina in the course of sugar puncture; Bulatao and Cannon\(^3\) observed the similar effect if the cerebral cortex are removed; Beattie, Brown and Long\(^4\) saw also the similar results after the section of hypothalamus. It is generally believed that in the case of sugar puncture, the increased adrenaline discharge was effected by the excitation of the sympathetic nervous system. Adrenaline liberates sugar in the liver, which causes hyperglycaemia and glycosuria. From these facts, an attempt is made to prove that the effect of piqure is not other than adrenaline-hyperglycaemia-glycosuria. But Macleod or Evans\(^5\) noted that piqure or transverse section of the central nervous system causes hyperglycaemia even in a fasting animal, and that the total amount of such sugar produced, is by far greater in quantity than the quantity of the glycogen which was used up in the liver and muscle.
Regarding the sugar mobilising action of adrenaline, two means come into consideration; namely (1) adrenaline mobilises, acting chemically to a mother substance of sugar (probably glycogen), (2) owing to the raised blood pressure in abdominal arteries, increased perfusion of liver takes place and more sugar will be washed out. In order to decide which one of these assumptions is correct, the following experiments were carried out.

Methods of Experiments

Animals used for the experiments are male rabbits, about 2 kilograms or so of body-weight, and the toads chiefly male of about 150 - 250 gms. of body-weight.

The methods of experiments used on the rabbits are entirely the same with the previous report 1).

The perfusion of liver of the toad was made in situ. For this purpose the inflowing canula was inserted into the central cut end of abdominal vein, and the outflowing into the peripheral end of sinus venous of the heart. To prevent the outflowing of the perfusing blood, ligation of veins from the gall-bladder, lungs and vena cava posterior at the beneath of liver, vena bulbus cordis and portal vein before the perfusion took place. The pressure of the perfusing fluid was kept about 5 cms. water and almost constant by means of Mariott's flask. The perfusing speed was about 2 - 3 cc. per minute. Stock solution of ergotoxine (Merk) was dissolved in ratio 1 in 1,000 cc. of Ringer by adding a few cc. of N/100 H₃PO₄. The sugar content in the perfusate was estimated after Hagedorn-Jensen's method, just as noted in the first chapter 1).

Experiment.

1. Influence of the intravenously injected adrenaline upon the blood sugar content of normal rabbits.

Just before the experiment, the stock solution of adrenaline chloride (Sankyo) was diluted 10 times with Ringer so as to make 0.01% concentration. This diluted solution was injected into the vein in proportion of 0.15 cc. per kilogram body weight. Sugar content

Table 1. Influence of the intravenously injected adrenaline on normal rabbits.
(The percentage of the blood sugar content.)

<table>
<thead>
<tr>
<th>No. of experiments</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before injection</td>
<td>0.112</td>
<td>0.090</td>
<td>0.092</td>
<td>0.091</td>
<td>0.094</td>
<td>0.096</td>
</tr>
</tbody>
</table>

http://escholarship.lib.okayama-u.ac.jp/amo/vol6/iss2/10
in the blood was estimated with the intervals of 30, 60 and 120 minutes after the injection. As Table 1 shows, the maximum of sugar content was found between 30 to 60 minutes after adrenaline injection and after 120 minutes it begins already to decrease. The maximum content of sugar was about 1.5 times of normal value (percentage of increase was about 50).

2. Influence of the intravenously injected adrenaline upon the blood sugar content of splanchnectomized rabbits.

Quantity of the adrenaline injected was the same ratio as in the normal animals. The sugar content of blood was estimated at 10, 30, 60 and 120 minutes intervals after the adrenaline injection.
Table 2. Influence of the intravenously injected adrenaline on the both sides of splanchniec tomized rabbits.
(The percentage of the blood sugar content shown below.)

<table>
<thead>
<tr>
<th>No. of experiments</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before splanchnectomy</td>
<td>0.094</td>
<td>0.101</td>
<td>0.096</td>
<td>0.090</td>
<td>0.095</td>
</tr>
<tr>
<td>Before adrenaline injection</td>
<td>0.095</td>
<td>0.105</td>
<td>0.098</td>
<td>0.092</td>
<td>0.098</td>
</tr>
<tr>
<td>After adrenaline injection (min.)</td>
<td>10</td>
<td>0.094</td>
<td>0.112</td>
<td>0.104</td>
<td>0.099</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>0.109</td>
<td>0.110</td>
<td>0.117</td>
<td>0.096</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>0.095</td>
<td>0.110</td>
<td>0.108</td>
<td>0.096</td>
</tr>
<tr>
<td></td>
<td>120</td>
<td>0.094</td>
<td>0.112</td>
<td>0.099</td>
<td>0.094</td>
</tr>
</tbody>
</table>

Fig. 2.

Except few cases, it was observed a slight increase of the blood sugar content, and in general showed no change of sugar content.

3. Influence of ergotoxine upon the blood sugar content of the normal rabbits.

For the purpose to paralyse the end of sympathetic, ergotoxine-Ringer solution was injected into the vein in the dose of 0.15, 0.1 or 0.05 mg per kilogram body weight. Ergotoxine alone, as following table shows, had no effect upon the blood sugar content.
Table 3. The blood sugar content of the ergotoxine injected rabbits (in %).

<table>
<thead>
<tr>
<th>No. of experiments</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before ergotoxine injection</td>
<td>0.088</td>
<td>0.092</td>
<td>0.097</td>
<td>0.093</td>
</tr>
<tr>
<td>30</td>
<td>0.090</td>
<td>0.090</td>
<td>0.096</td>
<td>0.092</td>
</tr>
<tr>
<td>60</td>
<td>0.090</td>
<td>0.101</td>
<td>0.098</td>
<td>0.096</td>
</tr>
<tr>
<td>120</td>
<td>0.092</td>
<td>0.104</td>
<td>0.102</td>
<td>0.099</td>
</tr>
<tr>
<td>180</td>
<td>0.090</td>
<td>0.099</td>
<td>0.098</td>
<td>0.096</td>
</tr>
<tr>
<td>Quantity of ergotoxine injected (mg. per kg.)</td>
<td>0.15</td>
<td>0.05</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 3.

4. Influence of ergotoxine upon the blood sugar content after the sugar puncture.

After the intravenous injection of ergotoxine (0.15, 0.10 or 0.05 mg per kilogram body weight), sugar puncture after Eckhard’s method was carried out.

Table 4. Influence of ergotoxine upon the sugar content of blood after the sugar puncture.

<table>
<thead>
<tr>
<th>No. of experiments</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Average</th>
<th>Sugar content</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before piqué</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>0.995</td>
</tr>
</tbody>
</table>
The results of the blood sugar estimation showed no difference from that of the normal rabbits, except that in case of 0.15 mg of ergotoxine injection which sometimes caused a very slight increase of blood sugar content.

5. Influence of ergotoxine upon the effect of electrical stimulation of the basis of the brain.

About 24 to 48 hours after the trepanation, 0.15, 0.10 or 0.05 mg of ergotoxine per kilogram of body weight was intravenously injected. The electrode has been pierced deep into the basis of brain in various directions, and stimulated electrically for 15 seconds. Normal blood sugar content as the control was determined before all the manipulations.

The electrical stimulation of the basis of brain after ergotoxine injection had no influence upon the blood sugar content.
Table 5. Influence of the electrical stimulation of the basis of the brain upon the blood sugar content after ergotoxine injection.

<table>
<thead>
<tr>
<th>No. of experiments</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before the electrical stimulation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sugar content</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(%)</td>
<td>0.108</td>
<td>0.098</td>
<td>0.103</td>
<td>0.104</td>
<td>0.103</td>
</tr>
<tr>
<td>After ergotoxine injection and electrical stimulation (min.)</td>
<td>30</td>
<td>0.104</td>
<td>0.098</td>
<td>0.110</td>
<td>0.115</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>0.107</td>
<td>0.100</td>
<td>0.113</td>
<td>0.124</td>
</tr>
<tr>
<td></td>
<td>120</td>
<td>0.112</td>
<td>0.101</td>
<td>0.101</td>
<td>0.103</td>
</tr>
<tr>
<td></td>
<td>180</td>
<td>0.117</td>
<td>0.101</td>
<td>0.099</td>
<td>0.100</td>
</tr>
<tr>
<td>Quantity of ergotoxine injected (mg. per kg.)</td>
<td>0.15</td>
<td>0.05</td>
<td>0.05</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 5.

6. Influence of adrenaline upon the sugar content of the perfusate of the toad's liver.

In these experiments the perfusate of the toad's liver became clear like water after 70 minutes perfusion with Ringer solution and free from albumine (sulfosalicylic acid test). After about 150 minutes from the beginning of perfusion, the sugar content of the perfusate became constant. Ringer solution with adrenaline was then perfused till the blood vessels of the liver contracted to some degrees. Perfusate was gathered and the sugar content was estimated.

The sugar content of the perfusate increased by about 42% after 15 minutes, 77% after 30 minutes from the beginning of adrenaline perfusion (1:10^8 adrenaline-Ringer solution in former case, 1:10^9 in
Table 6. The sugar content of the perfusate from the liver of toad's, which were perfused with adrenaline-Ringer.

<table>
<thead>
<tr>
<th></th>
<th>Time in min.</th>
<th>Conc. of adrenaline</th>
<th>Number of drops of perfusing liquid</th>
<th>Quantity of perfusing liquid in cc.</th>
<th>Sugar content (%)</th>
<th>Absolute quantity (mg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>25</td>
<td></td>
<td>0.028</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>22</td>
<td></td>
<td>0.025</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
<td>23</td>
<td>2.2</td>
<td>0.027</td>
<td>0.059</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>7</td>
<td>0.7</td>
<td>0.035</td>
<td>0.025</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>21</td>
<td></td>
<td>0.034</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>20</td>
<td></td>
<td>0.027</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
<td>20</td>
<td>2.0</td>
<td>0.027</td>
<td>0.054</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>11</td>
<td>1.1</td>
<td>0.047</td>
<td>0.052</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>23</td>
<td></td>
<td>0.037</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>22</td>
<td></td>
<td>0.032</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
<td>22</td>
<td>2.0</td>
<td>0.029</td>
<td>0.058</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>10</td>
<td>1.0</td>
<td>0.035</td>
<td>0.035</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>21</td>
<td></td>
<td>0.032</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>21</td>
<td></td>
<td>0.030</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
<td>20</td>
<td>2.0</td>
<td>0.029</td>
<td>0.058</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>9</td>
<td>0.9</td>
<td>0.041</td>
<td>0.037</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>26</td>
<td></td>
<td>0.035</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>25</td>
<td></td>
<td>0.031</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
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<td>2.5</td>
<td>0.030</td>
<td>0.075</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>16</td>
<td>1.6</td>
<td>0.044</td>
<td>0.070</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>9</td>
<td>0.9</td>
<td>0.058</td>
<td>0.052</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>33</td>
<td></td>
<td>0.043</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>30</td>
<td></td>
<td>0.037</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
<td>29</td>
<td>2.9</td>
<td>0.037</td>
<td>0.107</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>20</td>
<td>2.0</td>
<td>0.053</td>
<td>0.106</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td></td>
<td>12</td>
<td>1.2</td>
<td>0.051</td>
<td>0.061</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td></td>
<td>33</td>
<td></td>
<td>0.035</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td>30</td>
<td></td>
<td>0.030</td>
<td></td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1 : 10^n</td>
<td>29</td>
<td>2.9</td>
<td>0.030</td>
<td>0.069</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>15</td>
<td></td>
<td>20</td>
<td>2.0</td>
<td>0.043</td>
<td>0.052</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td></td>
<td>12</td>
<td>1.1</td>
<td>0.055</td>
<td>0.061</td>
</tr>
</tbody>
</table>
the latter). But the quantity of the perfusate diminished as a result of the contraction of the blood vessels, the absolute quantity of sugar which was poured out from the liver into the perfusate decreased correspondingly.

7. Influence of adrenaline upon the sugar content of the perfusate of the toad’s liver which was treated previously with ergotoxine.

The toad’s liver, which was treated previously with ergotoxine and was made impossible to contract as a result of paralysis of sympathetic nerve-endings of the blood vessels, was perfused with adrenaline-Ringer. The concentration of ergotoxine was 1:10⁴. After 30 minutes of perfusion with the solution, the perfusing liquid was changed to 1:10⁶ adrenaline containing solution, and then estimation of sugar content was followed in different time intervals.

The sugar content of the perfusate did not decrease by adrenaline if the liver was treated previously with ergotoxine. And in these cases, as the blood vessels of the liver do not contract and the quantity of the perfusing liquid does not diminish, so that the absolute quantity of poured out sugar neither decreased nor increased. In addition to the above mentioned facts, it must be mentioned especially that ergotoxine in the perfusing liquid did not relax the
Table 7a. The sugar content of the perfusate from the liver of toad's which were perfused with ergotoxine and adrenaline.

<table>
<thead>
<tr>
<th></th>
<th>Time in min.</th>
<th>Concentration of ergotoxine solution</th>
<th>Concentration of adrenaline</th>
<th>Number of drops of the perfusing liquid</th>
<th>Sugar content (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ringer</td>
<td>90</td>
<td>1 : 10⁴</td>
<td>1 : 10⁶</td>
<td>26</td>
<td>0.021</td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td></td>
<td>25</td>
<td>0.020</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td></td>
<td></td>
<td>24</td>
<td>0.020</td>
</tr>
<tr>
<td>Ergotoxine</td>
<td>30</td>
<td></td>
<td></td>
<td>23</td>
<td>0.020</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>10</td>
<td></td>
<td></td>
<td>21</td>
<td>0.025</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td></td>
<td></td>
<td>22</td>
<td>0.021</td>
</tr>
<tr>
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<td>1 : 10⁶</td>
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<td>0.025</td>
</tr>
<tr>
<td></td>
<td>120</td>
<td></td>
<td></td>
<td>30</td>
<td>0.024</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td></td>
<td></td>
<td>28</td>
<td>0.023</td>
</tr>
<tr>
<td>Ergotoxine</td>
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<td></td>
<td></td>
<td>27</td>
<td>0.024</td>
</tr>
<tr>
<td>Adrenaline</td>
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<td></td>
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<td>30</td>
<td></td>
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<td>29</td>
<td>0.025</td>
</tr>
<tr>
<td>Ringer</td>
<td>90</td>
<td>1 : 10⁴</td>
<td>1 : 10⁶</td>
<td>29</td>
<td>0.021</td>
</tr>
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<td></td>
<td>120</td>
<td></td>
<td></td>
<td>28</td>
<td>0.020</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td></td>
<td></td>
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<td>0.020</td>
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<tr>
<td>Ergotoxine</td>
<td>30</td>
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<td>26</td>
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</tr>
<tr>
<td></td>
<td>30</td>
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Experimental Studies on the Sugar Puncture. Part 2.

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<th>Time in min.</th>
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<th>Concentration of adrenaline</th>
<th>Number of drops of the perfusing liquid</th>
<th>Sugar content (%)</th>
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**Fig. 7 a.**

Blood vessels of the liver, so the absolute quantity of poured out sugar did not increase.

Even in case of the perfusion of strong solution of ergotoxine (1 : 5,000) and adrenaline (1 : 10⁴), the results were the same as those obtained with weak solution.
Table 7 b.

<table>
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<th>Concentration of adrenaline</th>
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Fig. 7 b.

Discussion.

Since Fröhlich and Pollak discovered the sugar mobilisation with adrenaline, the steady progress has been made on the problem. If
adrenaline is added to the perfusing liquid of frog's liver, it was affirmed by many authors that it causes sugar discharge in high degree. In this investigation it was also confirmed that adrenaline causes the increase of the percentage of sugar in the perfusing fluid of the liver of the toad. And in these cases, if splanchnic nerves of both sides are cut off, the sugar discharge is almost completely restrained.

In order to paralyse vasoconstrictor, ergotoxine was injected into the auricle vein of rabbit. It was often said that ergotoxine restrains adrenaline hyperglycaemia and also the hyperglycaemia is of central origin. But according to Kimura’s experiment on rabbits showed that hypodermical injection of ergotoxine causes a slight increase of the blood sugar, while intravenous injection decrease in the beginning and then increase or from the beginning the increase of sugar content was noted; Takehira is in the opinion that the increase of blood sugar with adrenaline is merely washing out of sugar; Nakamura experimented also adrenaline hyperglycaemia under ergotoxine and found that a temporary hyperglycaemia and hypoglycaemia afterwards. In my experiment in rabbits, ergotoxine alone has no influence upon the blood sugar content in the dosis of 0.05 mg to 0.15 mg per kilogram body weight. Ergotoxine inhibits the increase of the blood sugar content by the sugar puncture or by the electrical stimulation of the basis of the brain. These facts indicate that the hyperglycaemia after the sugar puncture or the electrical stimulation of the basis of the brain will be suppressed by ergotoxine. The splanchnectomy on both sides also suppresses the hyperglycaemia by equal manipulation. The splanchnectomy naturally cut off the transmission of nervous stimuli to the blood vessels, so that the effect of stimulation of the brain upon blood vessel is made impossible. Ergotoxine injection paralyses vasoconstrictor nerve and therefore any action upon the blood vessel from nerve becomes impossible.

Supposing if sugar mobilisation is due to the chemical actions of adrenaline, the sugar content of the perfusate must be nearly proportional to the amount of sugar in the perfusate. In this investigation, it was found that the perfused liver diminishes sugar content in perfusate owing to the contraction of blood vessels. If the nervous action on blood vessels is paralysed by ergotoxine, this change of sugar content is abolished. These facts show that adrenaline has no chemical action on sugar mobilisation from glycogen. On the contrary, sugar is washed out from liver by the torrent of blood, which will be increased by the contraction of the systemic arteries through adrenaline.
Summary.

1. In the normal rabbits the intravenous application of adrenaline causes hyperglycaemia, but in rabbits which have been previously splanchnicectomized on both sides, the adrenaline application always betrayed hyperglycaemia.

2. In the normal rabbits ergotoxine had no influence on the blood sugar content. Contrary to the normal animals, a rabbit which received previously an intravenous injection of ergotoxine by which the vasoconstriction of the blood vessels was made impossible, sugar puncture was not effected in the blood sugar content.

3. Contrary to the normal animals, in the rabbits which were treated previously with intravenous application of ergotoxine, the electrical stimulation of the basis of the brain did not cause hyperglycaemia.

4. By the perfusion of the liver of toad with Ringer solution, on adding of adrenaline in the perfusing liquid, discharge of the sugar in the perfusate was increased in percentage, but the absolute quantity was reduced on account of the reduction of the amount of perfusate.

5. The discharge of sugar from the perfusing liver of the toad, which was previously perfused sufficiently with ergotoxine-Ringer solution, on adding of adrenaline in the perfusing liquid showed no change compared with these without ergotoxine and adrenaline application.

6. The piqûre-hyperglycaemia may be assumed as adrenaline-hyperglycaemia. The origin of adrenaline-hyperglycaemia is rather mechanical, since the torrent of blood stream is increased by the strong contraction of blood vessels of skin and muscles than those of liver, washing out the reserved sugar in liver.

In conclusion, I wish to express my sincere thanks to Prof. Dr. S. Oinuma for his kind advice and suggestions during this investigation.

Bibliography.