Myocardial Blood Flow and Oxygen Consumption during Postprandial Lipemia and Heparin-Induced Lipolysis

By TIMOTHY J. REGAN, M.D., KENAN BINAK, M.D., SEYMOUR GORDON, M.D., VALENTINO DEFAZIO, M.D., AND HARPER K. HELLEMS, M.D.

ALTHOUGH the status of regional vascular tone and total cardiac output have been established as important determinants of regional blood flow, the precise role of the physical state of circulating blood has not been extensively explored. A study by Kety\(^1\) in man has indicated that the increased corporcular mass of polycytemia vera can be associated with a reduced cerebral blood flow. The induction of plasma lactescence in man after high lipid ingestion may also result in alterations of regional blood flow. Erythrocyte aggregation with circulatory slowing has been observed as a consequence of this postprandial circumstance,\(^2\) and varied clinical data have been accumulated to assign to it a pathogenetic basis for cardiac ischemia.\(^2,3\) To assess the consequences of the postprandial lipemic state upon myocardial blood flow and oxygen consumption these parameters have been studied during maximal plasma lactescence, and contrasted with the findings during post-heparin clearing and with data obtained in a group of fasting control subjects.

Materials and Methods

Both the 15 control subjects studied after an overnight fast and the group of 14 studied during the postprandial state were selected during the end of the recovery phase of their illness just prior to hospital discharge. All were male and comparable in age distribution. Similar affilliations were present in both groups, usually benign, acute bacterial infections of the lung, skin, or kidney. Patients with probable hemodynamic or pathologic alterations of the cardiovascular system were excluded, as were additional subjects, who did not fulfill hemodynamic criteria for normalcy, in most instances ascribable to anxiety. These requirements included a pulse rate of 60 to 95, a total body oxygen consumption of less than 160 ml. per square meter, a systemic arterial pressure less than 140/90 mm. Hg, a systemic arterial mixed venous oxygen difference between 35 and 45 ml. per liter of blood, and a cardiac index between 2.5 and 4.5 liters per minute per square meter.

The group of 14 were fed 1.5 Gm. of lipid per Kg. in cream (36 per cent fat) about 3 hours before the catheters were placed in the coronary sinus and right atrium and a Courmand needle was inserted into the brachial artery. Lumen was maintained patent by slow saline infusions, the lipid-clearing agents, heparin and glucose, being avoided. Since employment of the Fick principle for blood-flow determinations requires a steady state, serial total oxygen consumptions were performed to assess the postprandial alterations in 5 patients. The peak increment of total oxygen consumption occurred at 2 to 2½ hours, and by 3 hours a level was reached slightly higher than the control value. There was for the group no difference between the 3-hour and 3½-hour oxygen consumptions, 143 ± 11.1 ml. and 142 ± 12 ml. per minute, respectively. At approximately 3 hours and 15 minutes post cibum, the first coronary blood flow was determined by the nitrous oxide desaturation technic,\(^4\) immediately followed by simultaneous sampling for blood oxygen and carbon dioxide from the brachial artery, coronary sinus, and right atrium. Concurrently the expired air was collected for Fick cardiac output determinations, and the brachial artery pressure was recorded.

To induce lactescence-clearing, 60 mg. of heparin were then injected intravenously. After an initial increment during the rapid phase of lipid hydrolysis, serial total oxygen consumption determinations were found to remain relatively steady. Coronary and systemic hemodynamics were restudied 45 minutes after heparin administration. The small difference in total oxygen consumptions, prior to and following this repeat nitrous oxide inhalation,
169 ± 11 ml. and 164 ± 9 ml. per minute, respectively, indicated that a relatively steady state had been achieved.

In the group of 14 patients evaluated in this manner, serial arterial samples for plasma turbidity measurement were secured from 2½ hours post cibum, at 15-minute intervals, until the experiment was concluded. These specimens were collected in chilled tubes, refrigerated, and read at 650 and 700 m\(\mu\) on the Junior Coleman spectrophotometer. These are the usual wavelengths employed for measuring turbidity and are far enough removed from the optimal wavelengths for plasma pigments, such as hemoglobin, to avoid interference. A comparison of the readings at both wavelengths showed the 700 m\(\mu\) value to be slightly lower, but the relationship of the serial turbidity values at each wavelength was the same. The readings reported are those obtained at 650 m\(\mu\).

The development of plasma lactescence during alimentary lipemia has been alleged to be dependent upon triglyceride concentration. Several in vitro studies have noted a lack of correlation between these two entities. More recently, plasma turbidity has been exhibited in man after a lipic meal in the absence of significant alteration of serum triglyceride. For these reasons, the physicochemical change in blood evidenced as lactescence, rather than the concentration of lipids, has been correlated with the coronary hemodynamic data.

A valid use of the nitrous oxide method for analysis of regional blood flow in the circumstances of the study requires that the solubility of this gas in blood be unaltered by the quantity of lipids present in the lipemic state. That the solubility of nitrous oxide may not be affected by lipid is indicated by studies of its solubility in brain, where it was not significantly greater than in blood. The partition coefficient, according to Kety, would be expected to change significantly only with such an altered composition of brain or blood as to be incompatible with life.

During postprandial lipemia, if one assumes lipid concentration to be higher in blood than in heart, a possible increase in nitrous oxide blood solubility would diminish the partition coefficient and thus the low flows recorded should actually be still lower. That the nitrous oxide is not more soluble in the postprandial lipemic blood is indicated by the nitrous-oxide value of arterial samples after saturation achieved by 12 minutes' inhalation of this gas. The lactescent samples did not differ significantly from the post-heparin samples. Instead of the higher value to be expected if the gas were more soluble, the mean lactescent figure was 0.2 volumes per cent lower than the post-heparin value.

Although no difference in lactescence was found between arterial and coronary sinus blood (0.01 ± 0.03), the arterial lipid concentration was probably higher than venous, and raises the question of a higher nitrous oxide solubility in arterial blood. This would give falsely high nitrous oxide arterial concentrations and, in the desaturation method, a narrowing of the integrated arteriovenous difference. Thus, the low coronary flow level found would actually be higher than the true value unaffected by a spurious arteriovenous difference. These considerations, based upon a slightly higher concentration of lipid in the arterial blood, are seen to be of no importance, since the arterial samples saturated with nitrous oxide do not differ in concentration despite a nearly 3-fold difference in turbidity and a probable lipid difference amounting to several thousand milligrams. After heparin, a higher lipid concentration in the myocardium probably occurs and might increase the tissue/blood solubility. If the partition coefficient increases, the numerator of the nitrous oxide Fick equation would, unless corrected, be somewhat low. Consequently, the return of coronary blood flow from low to normal levels, as detailed in the results, would be qualitatively correct even if the real change were greater. Despite these considerations, a significant nitrous oxide solubility change appears unlikely.

Results

Considerable variation existed in the actual level of lactescence after a lipid meal. The seven of 14 patients in whom substantial lipemia occurred, with optical density readings above 0.3 unit, were selected for evaluation of the lipemic state and are termed the lipemic group. The individual and mean values obtained from 15 fasting controls are detailed in table 1. The coronary blood flow for this control group (83 ml. per 100 Gm. per minute) was significantly higher than the postprandial flow of 67 ml. in the lipemic group \((p = <0.001)\) outlined in table 2. As a consequence of this flow reduction, the consumption of oxygen by the heart was, at 7.02 ml. per 100 Gm. per minute, diminished more than 20 per cent in contrast to the control fasting group value of 9.00 ml. per 100 Gm. per minute \((p = <0.05)\). This deficit occurred in the absence of any increment of oxygen extraction, since the myocardial arteriovenous difference of oxygen in the lipemic group (10.54 volumes per cent) is no higher than the 11.02

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Table 1

<table>
<thead>
<tr>
<th>Patient, sex</th>
<th>BEA Ml.</th>
<th>Coronary flow, ml./100 Gm./min.</th>
<th>Oxygen difference</th>
<th>Oxygen consumption, ml./100 Gm./min.</th>
<th>Cardiac index, L/min./M²</th>
<th>Pulse rate</th>
<th>Mean arterial pressure, mm. Hg</th>
<th>Left ventricular work index, Gm. M. beat/M²</th>
</tr>
</thead>
<tbody>
<tr>
<td>R. McS. 31, M.</td>
<td>1.84</td>
<td>84</td>
<td>10.73</td>
<td>9.00</td>
<td>3.26</td>
<td>75</td>
<td>81</td>
<td>47.9</td>
</tr>
<tr>
<td>F. E. 40, M.</td>
<td>1.69</td>
<td>80</td>
<td>12.20</td>
<td>9.80</td>
<td>4.20</td>
<td>87</td>
<td>99</td>
<td>64.6</td>
</tr>
<tr>
<td>W. C. 29, M.</td>
<td>1.63</td>
<td>99</td>
<td>8.60</td>
<td>8.50</td>
<td>4.01</td>
<td>87</td>
<td>92</td>
<td>57.5</td>
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<tr>
<td>L. M. 45, M.</td>
<td>1.73</td>
<td>70</td>
<td>11.05</td>
<td>7.70</td>
<td>3.21</td>
<td>79</td>
<td>77</td>
<td>54.4</td>
</tr>
<tr>
<td>C. A. 41, M.</td>
<td>1.78</td>
<td>83</td>
<td>11.99</td>
<td>10.00</td>
<td>3.22</td>
<td>94</td>
<td>102</td>
<td>47.0</td>
</tr>
<tr>
<td>R. W. 35, M.</td>
<td>1.74</td>
<td>73</td>
<td>12.99</td>
<td>9.40</td>
<td>2.76</td>
<td>74</td>
<td>86</td>
<td>43.5</td>
</tr>
<tr>
<td>S. A. 32, M.</td>
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<td>70</td>
<td>11.02</td>
<td>7.71</td>
<td>3.09</td>
<td>60</td>
<td>92</td>
<td>63.8</td>
</tr>
<tr>
<td>M. F. 40, M.</td>
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<td>96</td>
<td>11.22</td>
<td>10.77</td>
<td>3.09</td>
<td>74</td>
<td>95</td>
<td>54.7</td>
</tr>
<tr>
<td>R. F. 38, M.</td>
<td>1.73</td>
<td>92</td>
<td>11.99</td>
<td>11.00</td>
<td>3.52</td>
<td>74</td>
<td>94</td>
<td>60.0</td>
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<tr>
<td>J. L. 50, M.</td>
<td>1.80</td>
<td>76</td>
<td>10.99</td>
<td>8.28</td>
<td>3.46</td>
<td>70</td>
<td>88</td>
<td>59.2</td>
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<tr>
<td>W. A. 30, M.</td>
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<td>84</td>
<td>10.12</td>
<td>8.50</td>
<td>3.54</td>
<td>88</td>
<td>80</td>
<td>40.2</td>
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<tr>
<td>J. A. 29, M.</td>
<td>1.94</td>
<td>98</td>
<td>9.79</td>
<td>9.62</td>
<td>3.55</td>
<td>69</td>
<td>90</td>
<td>61.9</td>
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<td>F. C. 30, M.</td>
<td>1.72</td>
<td>77</td>
<td>10.75</td>
<td>7.76</td>
<td>3.26</td>
<td>68</td>
<td>92</td>
<td>59.3</td>
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<td>G. T. 37, M.</td>
<td>1.87</td>
<td>74</td>
<td>9.72</td>
<td>7.20</td>
<td>2.75</td>
<td>77</td>
<td>94</td>
<td>44.7</td>
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<td>A. K. 43, M.</td>
<td>1.83</td>
<td>87</td>
<td>11.07</td>
<td>9.64</td>
<td>3.33</td>
<td>83</td>
<td>98</td>
<td>54.0</td>
</tr>
<tr>
<td>Mean values</td>
<td>83</td>
<td>10.94</td>
<td>9.00</td>
<td>3.35</td>
<td>77</td>
<td>90</td>
<td>54.2</td>
<td></td>
</tr>
</tbody>
</table>

During the process of heparin-induced lipolysis, plasma lactic acid was reduced from a mean of 0.65 optical-density unit to 0.25 unit, 45 minutes after 60 mg. of intravenous heparin, when the repeat determination of coronary hemodynamics was performed. The initially low coronary flow during lactescence was in each instance restored toward normal during the clearing process from a mean of 67 ml. to 87 ml. per minute per 100 Gm. of left ventricle ($p = < 0.02$) (fig. 1). This change was paralleled by an augmented myocardial oxygen consumption from 7.02 to 9.57 ml. per 100 Gm. per minute ($p = < 0.05$). The

volumes per cent of the control fasting subjects. The relevant hemodynamic indices of pulse, mean arterial pressure, cardiac index, and stroke work index were not significantly different between the two groups. Whole blood viscosity was measured in 2 patients during lipemia* the values of 4.46 and 4.74 centipoise units, obtained on a rotating disk viscometer,† were within the limits found in fasting normal subjects.

*Through the courtesy of Dr. Perry C. Martineau, Department of Pathology, Wayne State University College of Medicine, Detroit, Mich.
†Brookfield Engineering Laboratory, Stoughton, Mass.
increment is largely ascribable to flow alteration as the oxygen extraction was relatively unchanged. The over-all response to heparin lipolysis entailed no significant rise in the cardiac index, pulse rate, mean arterial blood pressure, or stroke work index (table 2) that may have accounted for the observed facilitation of myocardial oxygen usage. Since this effect may rather have been achieved by a property of heparin unrelated to lipolysis, its activity in the absence of clearing effect has been analyzed in a group of 6 patients, 3 of whom were fasting. The remainder had low lactescence levels that were not altered by heparin. In this situation, heparin failed to increase the coronary blood flow (fig. 2). Instead, a slight decline in myocardial oxygen consumption was detected.

The relative state of the myocardial respiratory quotients compared to the simultaneous oxygen consumption is illustrated in figure 3. The low levels found in the fasting state are attributable to the dependence upon lipid as the major source of energy supply in this circumstance. In both the fasting normal group and in the postprandial lipemic group with a lower level of oxygen consumption, there was no difference in the comparative respiratory quotient. The unchanged respiratory quotient associated with heparin-induced lipolysis implies that accelerated fatty acid utilization has not yet occurred in this lipid-loading situation.

The data in the 7 patients in whom lactescence readings after lipid feeding were below 0.3 optical-density unit are given in table 3. The coronary blood flow and myocardial oxygen consumption did not differ significantly from the control group. In the 4 who received heparin, neither lactescence nor coronary dynamics underwent substantial change, additional characteristics that mark the difference between the fully developed lipemic state and the abortive form seen in this group.

| Table 2 |

| Hemodynamic Data in Seven Patients during the Lipemic State and after Heparin-Induced Lipolysis |

<table>
<thead>
<tr>
<th>Patient, sex</th>
<th>BSA M²</th>
<th>Optical-density units</th>
<th>Coronary blood flow ml./100 Gm./min.</th>
<th>Oxygen extraction difference %</th>
<th>Oxygen consumption difference %</th>
<th>Cardiac output difference %</th>
<th>Pulse rate</th>
<th>Mean arterial pressure mm. Hg.</th>
<th>Lactescence index</th>
<th>Left ventricular index</th>
<th>Arterial oxygen saturation %</th>
</tr>
</thead>
<tbody>
<tr>
<td>L. H.</td>
<td>1.83</td>
<td>.502*</td>
<td>61</td>
<td>10.39</td>
<td>6.34</td>
<td>3.48</td>
<td>73</td>
<td>51.9</td>
<td>92.5</td>
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<tr>
<td>24. M.</td>
<td>1.72</td>
<td>.070†</td>
<td>71</td>
<td>9.94</td>
<td>7.06</td>
<td>3.59</td>
<td>74</td>
<td>47.5</td>
<td>94.9</td>
<td>94.9</td>
<td>94.9</td>
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<tr>
<td>V. D.</td>
<td>.740*</td>
<td>72</td>
<td>11.51</td>
<td>8.50</td>
<td>5.33</td>
<td>4.21</td>
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<td>87</td>
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<td>93.0</td>
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<td>22. M.</td>
<td>.323†</td>
<td>.790*</td>
<td>74</td>
<td>10.09</td>
<td>7.47</td>
<td>2.54</td>
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<td>39</td>
<td>93.8</td>
<td>93.8</td>
<td>93.8</td>
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<td>35, M.</td>
<td>.840*</td>
<td>69</td>
<td>8.96</td>
<td>6.18</td>
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<td>7.48</td>
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<td>40</td>
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<td>93.8</td>
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<td>H. W.</td>
<td>1.77</td>
<td>.711†</td>
<td>82</td>
<td>10.89</td>
<td>8.93</td>
<td>3.14</td>
<td>78</td>
<td>100</td>
<td>97.7</td>
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<td>53, M.</td>
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<td>.110†</td>
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<td>3.35</td>
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<tr>
<td>W. B.</td>
<td>.550*</td>
<td>57</td>
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<td>7.03</td>
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<td>53</td>
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<td>44, M.</td>
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<td>.250†</td>
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<td>E. B.</td>
<td>.890*</td>
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<td>8.33</td>
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<td>102</td>
<td>96.7</td>
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<td>42. M.</td>
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<td>.958</td>
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<td>98.9</td>
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<td>Mean values</td>
<td>.648*</td>
<td>67</td>
<td>10.54</td>
<td>7.02</td>
<td>3.08</td>
<td>7.53</td>
<td>75</td>
<td>92</td>
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<td>p values</td>
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<td>&lt;0.01                 &gt;0.10</td>
<td>&lt;0.01</td>
<td>&gt;0.10</td>
<td>&lt;0.01</td>
<td>&gt;0.10</td>
<td>&gt;0.10</td>
<td>&gt;0.10</td>
<td>&gt;0.10</td>
<td>&gt;0.10</td>
<td></td>
</tr>
</tbody>
</table>

* Lipemic state.
† Lipemic state compared to fasting controls of table 1.
‡ Lipemic state compared to values in same patient after heparin.
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Discussion

The data from this study indicate that the development of a sufficient degree of plasma lactescence during the course of alimentary lipemia will limit oxygen delivery to the overtly normal myocardium. Definite evidence bearing on the basis for this concentration-dependent phenomenon in the resting state is lacking. Preliminary information indicates, however, that the enhanced circulatory requirements of the exercised lipemic patient are indeed restricted even at low lactescence values. Failure of the anticipated increments of oxygen extraction to compensate for the reduction of myocardial blood flow may be attributable to the phenomenon of erythrocyte aggregation, observed in the conjunctival vessels of man. A firm causal relationship in the myocardium itself remains to be established.

Since metabolic events within the myocardium may exert a controlling influence upon the rate of coronary blood flow, it is conceivable that the depressed myocardial oxygen consumption during lipemia in the absence of significant hemodynamic alterations is related to a shift in substrate utilized for energy production. Other data indicate that the relative usage of carbohydrate and fat by the heart seems reasonably well reflected in the simultaneous respiratory quotient. The stability of this parameter under the three conditions studied implies that, even if the heart is extracting lipid in response to high extrinsic concentrations, significant utilization has not yet occurred in accord with the storage con-
It is equally difficult to assign a deleterious role to the lipemic state in the heart with coronary artery disease, even with the assumption of a similar effect on oxygen uptake as observed in the normal. For it would then seem necessary to postulate a differential effect upon oxygen delivery with the establishment of an oxygen gradient within the myocardium. Certainly the sparseness of clinical reports to date, intimating an association of lacescence and angina pectoris, suggests that such a relationship may be found in but a minority of patients. Nevertheless, it is conceivable that in the development of coronary thrombosis the lipemic state may have pathogenetic significance related to the induction of circulatory slowing and accelerated coagulation activity\(^{17}\) within an atherosclerotic vessel.

Employing the usual oxygen energy equivalent,\(^ {18}\) one may consider an enhanced mechanical efficiency of the heart to exist in the lipemic state. An accurate estimation of efficiency is not obtainable, however, without knowledge of the relative status of oxygen usage during the different phases of the cardiac cycle. Conceivably, the oxygen utilized and energy liberated during the part of the cycle concerned with external cardiac work may be unaltered.\(^ {18}\)

Although the usual mechanism for accomplishing a decline in regional blood flow in the absence of systemic hemodynamic changes is through enhanced arteriolar resistance, the observation of abnormal erythrocyte aggregations in human conjunctival vessels during lipemia\(^ {2}\) implies that the modification in flow may be achieved more by an increase in viscosity than in vasomotor tone. While viscosity has been demonstrated to rise in certain animal studies, the alterations associated with circulatory slowing may occur in the absence of a measurable increase in blood viscosity,\(^ {19}\) which conforms to the findings in 2 subjects of the postprandial lipemic group studied by the shear-viscosity method. The methodologic difficulties of in vitro viscosimetry have been emphasized by the disappearance of erythrocyte aggregation in freshly drawn blood.\(^ {20}\)
Even more perplexing is the problem of duplicating the geometry of the components of the vascular system and their varied effects upon cellular deformation. Whatever the actual status of blood viscosity, this factor alone would not account for the observed decrement in cardiac oxygen consumption. A substantial restriction of blood flow to the brain occurs in erythremia associated with enhanced viscosity, yet cerebral oxygen consumption is maintained by virtue of a larger oxygen extraction. Thus, the lack of anticipated increment in oxygen extraction during lipemia suggests an impaired blood-tissue oxygen transport, perhaps related to the abnormal aggregation of red cells observed by others.

The prevalence of this phenomenon of reduced myocardial blood flow and oxygen consumption following the rather unphysiologic circumstance of a predominantly fat meal appears to depend upon an abnormal response to mixed feeding. Even though modest amounts of available carbohydrate appear to minimize the degree of alimentary lipemia in normal subjects, it remains conceivable that the metabolic processes associated with coronary atherosclerosis may involve an aberrant lipid transport mechanism so that substantial lipemia ensues in a nutritional circumstance in which this would be normally improbable.

Summary

The role of the physical state of plasma as a determinant of oxygen availability to the myocardium has been investigated during the course of alimentary lipemia. After the development of substantial plasma lactescence, the coronary blood flow (nitrous oxide method) and myocardial oxygen consumption were assessed in 7 normal human subjects and repeated after heparin-induced lipolysis. The lipemic state was further contrasted with a control fasting group, comparable in age and sex.

The mean coronary blood flow for 15 fasting controls was 83 ml. per 100 Gm. of left ventricle per minute with a myocardial oxygen extraction of 11.04 volumes per cent, and a myocardial oxygen consumption of 9.0 ml. per 100 Gm. of left ventricle per minute. By contrast, the mean coronary blood flow during maximal lipemia in the 7 subjects fed cream was 20 per cent below normal, with a value of 67 ml. per 100 Gm. per minute (p < 0.01). As the extraction of oxygen was not significantly affected, the calculated myocardial oxygen consumption was proportionately reduced to 7.02 ml. per 100 Gm. per minute (p < 0.01). A failure of the anticipated oxygen extraction increment in the face of coronary blood flow reduction suggests an impediment of blood-tissue oxygen transport during lipemia.

After the administration of 60 mg. of heparin to the 7 lipemic subjects, a 65 per cent decline in plasma lactescence was observed by 45 minutes, when the coronary blood flow and myocardial oxygen consumption were elevated to 87 ml. per 100 Gm. per minute (p < 0.05) respectively. Thus, the reduced coronary flow and myocardial oxygen consumption were restored in each instance to normal levels during the process of plasma clearing. There were no associated systemic hemodynamic changes to account for such in-
crements. These heparin effects appear dependent on the lipemia-clearing property, for no alteration in coronary dynamics was found in 6 additional patients in whom this activity was not manifest after the same heparin dosage.

The residual lactescence after post-heparin lipolysis was associated with no significant deviation of coronary dynamics from the normal. That a concentration-dependent phenomenon is operative, was confirmed in a separate group of patients in whom low lactescence values developed in the course of alimentary lipemia without affecting myocardial oxygen consumption.

The relevance of the lipemic state, per se, to the pathophysiology of myocardial ischemia appears to depend upon the establishment of an oxygen gradient within the myocardium, presumably through altered pressure-flow relationships produced by lipemic blood within a pathologic vessel.

### Acknowledgment

The authors wish to acknowledge the technical assistance of Miss Virginia Everett and Miss Margaret Reese and the secretarial services of Mrs. Shirley Loy.

### References


HEPARIN-INDUCED LIPOLYSIS


The study of nature will ever yield us fresh matter of entertainment, and we have great reason to bless God for the faculties and abilities he has given us, and the strong desire he has implanted in our minds, to search into and contemplate his works, in which the farther we go, the more we see the signatures of his wisdom and power, everything pleases and instructs us, because in everything we see a wise design. And the farther researches we make into this admirable scene of things, the more beauty and harmony we see in them: and the stronger and clearer convictions they give us, of the being, power and wisdom of the divine Architect, who has made all things to concur with a wonderful conformity, in carrying on, by various and innumerable combinations of matter, such a circulation of causes and effects, as was necessary to the great ends of nature.—Stephen Hales, B.D., F.R.S. Haemastatics. Ed. 3, Dedication, p. vi.
Myocardial Blood Flow and Oxygen Consumption during Postprandial Lipemia and Heparin-Induced Lipolysis
TIMOTHY J. REGAN, KENAN BINAK, SEYMOUR GORDON, VALENTINO DEFAZIO and HARPER K. HELLEMS

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