SERUM CHOLINESTERASE DEFICIENCY
II: PREGNANCY

BY

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SUMMARY

The conflicting evidence regarding changes in serum cholinesterase during pregnancy is briefly reviewed. A technique for the estimation of serum cholinesterase by continuous titration at constant pH is described, and the results of a study of the enzyme in 149 pregnant women are presented. Pregnant women had a diminution of serum cholinesterase activity compared with non-pregnant women of the child-bearing age group, and this diminution is statistically highly significant. The fall appeared to begin soon after 10 weeks gestation. The reduction of enzyme activity in patients with toxaemia of pregnancy was not significantly different from that in non-toxaemic cases. Reasons are presented for believing that neither haemodilution nor hepatic insufficiency can provide an adequate explanation for the changes described. The possible clinical importance of the changes is outlined.

That serum cholinesterase levels are influenced by a variety of disease states is now well established, although the reasons for the fall in the enzyme in certain conditions and its clinical significance are less clear. Changes in serum cholinesterase in physiological states are less well documented.

Tourtellotte and Odell (1950) found decreased serum cholinesterase activity in pregnant women. Pritchard (1955) showed that decreased cholinesterase activity existed mainly in the third trimester of pregnancy, and suggested that it was due to haemodilution. Wetstone and colleagues (1958) demonstrated decreased cholinesterase activity as early as the first trimester of pregnancy, there being no correlation with parity. They considered that haemodilution did not entirely explain this decrease, since even with normal haematocrit values the mean esterase activity was depressed; they pointed out the known anticholinesterase activity of oestrogen and the possibility of specific inhibition of hepatic function. Nutrition was considered not to be influential. Friedman, Lapan and Taylor (1961) also found decreased esterase activity in pregnancy, with minimum values before term, and suggested that the changes might be statistically more marked by using a technique involving titration, rather than (as they used) a colorimetric method with the results expressed in ΔpH units. Henriquet (1962), using a test-paper (Acholest) method of estimating serum cholinesterase found no change in activity in early pregnancy, and decreased activity immediately postpartum. He described a case of apnoea for 2 hours after the end of a Caesarean section during which 300 mg of suxamethonium had been given in a period of 45 minutes. There was marked diminution of cholinesterase activity. It was implied that the decreased cholinesterase was caused by the pregnancy, when in fact it may well have been due to the presence of an inherited atypical enzyme; and the prolonged apnoea may have been due to this or the development of end-plate resistance ("dual block") or both.

In 1963, Rimbach and Dacic, using the same technique as Henriquet, found no difference in cholinesterase activity between non-pregnant and pregnant women. Meade and Rosalki (1963), studying a variety of enzymes in normal pregnancy, estimated serum cholinesterase by the method of De la Huerga, Yesineck and Popper (1952). They found no significant alteration of cholinesterase activity at any stage in pregnancy. Recently, Schnider (1965) studied serum cholin-
esterase in 30 healthy obstetric patients during labour, and 1 and 3 days postpartum. Of these, 10 were also studied in late pregnancy. The results showed decreased activity in late pregnancy, a slight increase during labour, and a further fall in the last 3 days after delivery. Schnider suggests that the changes cannot be explained by haemodilution, and points out the conflicting evidence regarding altered hepatic function in pregnancy.

Cons and Glass (1963), working on mice, suggested that there may be differential alterations in the electrophoretic components of serum cholinesterase in pregnancy.

The evidence for reduced serum cholinesterase activity in pregnant women is thus already considerable. A number of workers have been unable to confirm this evidence, however, and much of the uncertainty seems to arise because of the wide variety of techniques employed; the statistical evaluation of the results may be more difficult when relatively insensitive methods are used or when small numbers of cases are studied. Furthermore, previous figures and case reports relating to the administration of suxamethonium in pregnancy, have not taken into account the possible presence of atypical enzyme, or the development of endplate resistance.

The techniques employed by previous workers have usually been a variation of the electrometric method of Michel (1949), a colorimetric method (De la Huerga, Yesineck and Popper, 1952), a photometric method (Caraway, 1956), or a test-paper (Acholest) method.

The Michel method measures cholinesterase activity as ΔpH change per hour produced by the release of acetic acid from the action of cholinesterase on an acetylcholine substrate. The system incorporates a sodium barbitone buffer. Friedman, Lapan and Taylor, (1961) have pointed out the relative insensitivity of a method which expresses results as ΔpH change, and further objections to the technique have been raised by Dixon and Webb (1964). They suggest that since the pH of the reaction varies with time, the rate of the enzyme reaction will also vary because enzymes are sensitive to pH change. Furthermore, the rate of pH change depends not only on the rate of the reaction but also on the buffering power of the solution. This is considered by Dixon and Webb to be a serious objection, because proteins have a high buffering capacity, and in a series of experiments it is difficult to keep buffering capacity constant.

Colorimetric and photometric methods are open to the criticism that colour indicators may vary in absorbance in relation to time, pH, and protein content of the reaction mixture.

A simple paper-strip method of estimating serum cholinesterase has been described and its significance evaluated (Sailer and Braunsteiner, 1959). Filter paper is impregnated with a choline ester and bromothymol blue, and the addition of serum promotes a colour change. The time taken to produce a standard colour change is inversely proportional to the cholinesterase activity. The paper-strip (Acholest) method has been studied by Churchill-Davidson and Griffiths (1961), who, while claiming no great accuracy of the technique, suggested that it was clinically useful and fairly accurate except at very low esterase levels. Crowley and Lehmann (1962) stated that their experience with the technique was less favourable, and that of four cases of suxamethonium apnoea, all with low cholinesterase activity, only one would have been revealed by the paper-strip method.

The reliability of enzyme estimation has recently been improved by a technique in which the pH of the reaction is kept constant by frequent additions of alkali. The method has been outlined by Schwartz and Bodansky (1963), and its possible application to serum cholinesterase estimation has been suggested. Recording titrimeters, such as the Radiometer pH-stat and Titragraph have provided an automatic method of maintaining a constant pH, and allowing esterase activity to be followed as a straight-line graph indicating the amount of alkali added to a thermostatically controlled reaction vessel by a motor-driven micrometer syringe.

SERUM CHOLINESTERASE ESTIMATION

Estimations of serum cholinesterase activity have been made in 40 non-pregnant and 149 pregnant women. The technique has been standard throughout, and all estimations, with the exception of the first 20 in each group, were done personally by the author.
METHOD
Using a Radiometer pH-stat and Titragraph, set to maintain a constant pH of 7.4, the following mixture is added to the electrode cell:
- 5.95 ml normal saline;
- 0.25 ml acetylcholine bromide, the substrate consisting of 0.837 g acetylcholine bromide made up to 10 ml in distilled water;
- 0.05 ml serum.

Titration is automatic, consisting of additions of 0.02 N NaOH from a motor-driven micrometer syringe. The reaction is allowed to proceed for 10–15 minutes, when a straight-line graph is obtained. The acetylcholine bromide substrate is made up fresh before each set of readings, and the spontaneous hydrolysis rate of acetylcholine bromide (without serum) is estimated first. This value is deducted from the estimation performed with serum, and the final result is given in units (1 unit = 1 µl. 0.02 N NaOH per ml serum per hour).

Dibucaine number estimation
Dibucaine numbers were estimated in the 30 cases having a cholinesterase value of 130 units or less. The method was that described by Varley (1962), employing an Optica spectrophotometer.

RESULTS
Serum cholinesterase estimations were made on 149 pregnant women. Of these, 105 were essentially normal as regards progress in pregnancy; venous blood samples were taken at routine antenatal attendances. Thirty-three cases, mostly seen after admission to hospital, were diagnosed as having toxaemia of pregnancy, there being evidence of hypertension and albuminuria, with or without oedema. A further 11 cases had hypertension without albuminuria, and although some of these subsequently developed albuminuria, they have been classified with the non-toxaemic cases because of uncertainty of diagnosis in several cases. Estimations were performed within a few hours.
**TABLE I**

Distribution of serum cholinesterase (S.Ch.E.) in the pregnant and non-pregnant groups.

<table>
<thead>
<tr>
<th>S.Ch.E.</th>
<th>Pregnant No.</th>
<th>%</th>
<th>Non-pregnant No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 24</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25 - 49</td>
<td>1</td>
<td>0.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50 - 74</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>75 - 99</td>
<td>3</td>
<td>2.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 - 124</td>
<td>13</td>
<td>9.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>125 - 149</td>
<td>34</td>
<td>23.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>150 - 174</td>
<td>30</td>
<td>20.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>175 - 199</td>
<td>28</td>
<td>19.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>200 - 224</td>
<td>22</td>
<td>15.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>225 - 249</td>
<td>9</td>
<td>6.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>250 - 274</td>
<td>2</td>
<td>1.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>275 - 299</td>
<td>1</td>
<td>0.7</td>
<td></td>
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<tr>
<td>300 - 324</td>
<td>1</td>
<td>0.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>325 - 349</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>144</td>
<td>100.0</td>
<td></td>
<td>40</td>
</tr>
</tbody>
</table>

**TABLE II**

Mean cholinesterase values in the non-pregnant group, and in the pregnant group and main sub-groups.

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Mean</th>
<th>Variance</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-preg.</td>
<td>40</td>
<td>208.98</td>
<td>1511.6</td>
<td>38.9</td>
</tr>
<tr>
<td>1st preg.</td>
<td>90</td>
<td>173.18</td>
<td>1861.3</td>
<td>43.1</td>
</tr>
<tr>
<td>Multip.</td>
<td>54</td>
<td>167.69</td>
<td>1546.4</td>
<td>39.3</td>
</tr>
<tr>
<td>All preg.</td>
<td>144</td>
<td>171.12</td>
<td>1738.7</td>
<td>41.7</td>
</tr>
<tr>
<td>Non-tox.</td>
<td>111</td>
<td>172.50</td>
<td>1580.3</td>
<td>39.7</td>
</tr>
<tr>
<td>Toxaemic</td>
<td>33</td>
<td>166.30</td>
<td>3165.4</td>
<td>56.3</td>
</tr>
</tbody>
</table>

**FIG. 2**

Cholinesterase Activity (μL 0.02N NaOH per ml serum per hour)

Distribution of serum cholinesterase activity in pregnant women. The black area indicates the distribution of activity in toxaemic cases.
days of obtaining the blood samples, the sera being stored in a deep freeze at \(-20^\circ\text{C}\) until required.

For the purposes of comparing the pregnant and non-pregnant groups, 5 cases in the pregnant group who were found to have atypical enzyme associated with a low serum cholinesterase have been excluded from the statistical evaluations.

The distributions of serum cholinesterase values in the pregnant and non-pregnant groups are shown in table I. It is of interest to note that in the non-pregnant group 7.5 per cent of cases show activity of less than 150 units (the probable lower limit of normal by this technique), whereas in the pregnant group 35.4 per cent are below this figure. The differences in the distribution of the two groups will be seen in figure 1, which relates the frequency distribution (as a percentage of the total in each group) to the cholinesterase activity.

Table II shows the mean cholinesterase values of the major groups, and the variances and standard deviations. Tests of significance between the means of the non-pregnant and pregnant groups show that the difference is highly significant (\(P<0.001\)). The difference between means in the primiparae and multiparae is not significant (\(P>0.05\)). The study of serum cholinesterase values in albuminuric toxaemia shows a mean of 166.3 units, and a distribution pattern (fig. 2) which is similar to that of the entire group. However, the variance in the toxicaemic group is almost exactly double that of the non-toxicaemic group, and it is of interest that the highest and lowest values in the entire study were in toxicaemic patients.

In a small number of cases, repeat cholinesterase estimations were done at different stages of pregnancy and after delivery. In any individual, the cholinesterase values showed a progressive fall towards full term, with the exception of patients who developed severe pre-eclamptic toxemia. In such cases, especially if they were also post-mature, there was a tendency for the esterase to increase, and in one case the value increased from 123 units at 36 weeks, at which stage the patient had albuminuria without hypertension, to 250 units at 39 weeks, when the toxemia had become severe, and there was marked hypertension.

Dibucaine numbers were estimated in 30 cases, all with serum cholinesterase values of 130 or less. The results are shown in table III. Of these cases, 5 had intermediate dibucaine numbers, suggesting that they were heterozygous for the atypical enzyme. The subject with the lowest cholinesterase of the series (44 units) had a normal dibucaine number. At Caesarean section for foetal distress, this patient was apnoeic and in full surgical relaxation for 55 minutes after a 100-mg dose of suxamethonium followed by a further 25 mg. The duration of apnoea after the initial dose

### Table III

<table>
<thead>
<tr>
<th>S. Ch. E.</th>
<th>D. N.</th>
<th>S. Ch. E.</th>
<th>D. N.</th>
<th>S. Ch. E.</th>
<th>D. N.</th>
</tr>
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<tbody>
<tr>
<td>44</td>
<td>82</td>
<td>110</td>
<td>86</td>
<td>124</td>
<td>88</td>
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<tr>
<td>54</td>
<td>54</td>
<td>110</td>
<td>86</td>
<td>126</td>
<td>89</td>
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<tr>
<td>83</td>
<td>45</td>
<td>112</td>
<td>81</td>
<td>126</td>
<td>81</td>
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<td>81</td>
<td>86</td>
<td>118</td>
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<td>88</td>
<td>57</td>
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<td>130</td>
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<td>100</td>
<td>81</td>
<td>120</td>
<td>85</td>
<td>130</td>
<td>76</td>
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<td>90</td>
<td>54</td>
<td>122</td>
<td>81</td>
<td>130</td>
<td>77</td>
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<td>95</td>
<td>94</td>
<td>128</td>
<td>79</td>
<td>130</td>
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<tr>
<td>104</td>
<td>91</td>
<td>124</td>
<td>82</td>
<td>130</td>
<td>76</td>
</tr>
<tr>
<td>108</td>
<td>84</td>
<td>124</td>
<td>86</td>
<td>130</td>
<td>80</td>
</tr>
</tbody>
</table>

### Table IV

<table>
<thead>
<tr>
<th>Weeks of gestation</th>
<th>Number of cases</th>
<th>Mean S. Ch. E.</th>
<th>Standard Deviation</th>
<th>Standard error of mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 - 9</td>
<td>3</td>
<td>207.8</td>
<td>31.7</td>
<td>12.3</td>
</tr>
<tr>
<td>10 - 14</td>
<td>13</td>
<td>175.9</td>
<td>36.7</td>
<td>10.7</td>
</tr>
<tr>
<td>15 - 19</td>
<td>15</td>
<td>177.6</td>
<td>33.8</td>
<td>8.7</td>
</tr>
<tr>
<td>20 - 24</td>
<td>15</td>
<td>165.4</td>
<td>36.5</td>
<td>10.2</td>
</tr>
<tr>
<td>25 - 29</td>
<td>11</td>
<td>149.1</td>
<td>31.3</td>
<td>9.4</td>
</tr>
<tr>
<td>30 - 34</td>
<td>12</td>
<td>172.1</td>
<td>39.4</td>
<td>8.4</td>
</tr>
<tr>
<td>35 - 39</td>
<td>10</td>
<td>170.2</td>
<td>30.5</td>
<td>9.6</td>
</tr>
<tr>
<td>40-</td>
<td>10</td>
<td>176.3</td>
<td>64.3</td>
<td>16.1</td>
</tr>
</tbody>
</table>
was not precisely timed by the anaesthetist but was probably 30 to 35 minutes.

Table IV shows the relationship of mean serum cholinesterase values to periods of gestation. Although the numbers of cases in certain groups are insufficient to demonstrate statistically significant differences between means, there is some evidence that the fall in cholinesterase begins after the 10th week of gestation and that the maximum fall occurs at about mid-term of pregnancy.

DISCUSSION

This study gives evidence, to a high degree of statistical significance, that a fall in the concentration of serum cholinesterase occurs in pregnant women. The fall appears to begin after the 10th week of pregnancy. In albuminuric toxemia the mean cholinesterase activity is not significantly different from that of the whole group, although the extremes are greater.

The possible reasons for the fall in cholinesterase are difficult to assess. Pritchard (1955) suggested that the changes were primarily due to haemodilution. There is no doubt that in normal pregnancy there is an increase in plasma volume; Hytten and Paintin (1963), on the basis of plasma volume estimations in 39 pregnant women, concluded that there was little or no change before 10 weeks, although an increase was apparent by 12 weeks. In agreement with most workers, they found the increase in plasma volume to be maximal at about 34 weeks. Rominger (1964), using radio-iodine labelled albumin to measure plasma volume, found an increase in all trimesters, mostly occurring in the last 4-5 weeks. In mild toxemia, there was no difference as compared with normal pregnancy, but in severe toxemia and in eclampsia the plasma volume was decreased. Cope (1961) also found reduced plasma volume in toxemic pregnancies, and suggested that this was due to peripheral vasoconstriction. Hytten and Leitch (1964), reviewing the evidence for an increase in plasma volume in pregnancy, concluded that in healthy women in a normal first pregnancy the value increased from a non-pregnant level of about 2,600 ml by an average of 1,250 ml. This means that an average increase of 40–50 per cent occurs, which is considered to be "physiologically desirable".

The argument that the fall in cholinesterase in pregnancy is due to haemodilution may equally well be applied to the changes in albumin concentration. MacGillivray and Tovey (1957) showed that in normal pregnancy, plasma albumin fell steadily until the 7th month, then remained unaltered for the remainder of pregnancy. In toxemia, they found a continuing fall in total protein and albumin during the last trimester. Brewer (1962) also found an aggravation of hypoalbuminaemia in severe toxemia associated with a contracted blood volume, and he warned against the danger of inducing a shock state in such cases by the administration of diuretics. Brewer (1963) recommended the infusion of albumin in cases of severe toxemia. He further stated that, although the cause of low plasma albumin in toxemia was not known, it was most likely related to impaired hepatic synthesis of albumin, toxemia of pregnancy being primarily a metabolic hepatic disease brought on by malnutrition. It has been suggested that a strong correlation exists between malnutrition and the severity of toxemia (Maqueo, Ayala and Cervantes, 1964).

The fall in plasma albumin in pregnancy is proportionately greater than that of total protein, particularly in toxemia (De Alvarez and Afonso, 1964). Furthermore, a rise occurs in \( \alpha_1 \), \( \alpha_2 \), and \( \beta \)-globulin and fibrinogen (Hytten and Leitch, 1964), which is not consistent with suggestions of haemodilution, unless there is a marked increase in the formation of these fractions. Hytten and Leitch state that "one thing is certain: the fall of plasma protein is not due to 'dilution' as the plasma volume expands. Albumin is the only fraction which is significantly 'diluted' and its concentration appears to fall abruptly at a time in pregnancy when plasma volume is just beginning to rise."

There is considerable evidence that the hepatic productions of albumin and cholinesterase are closely related (Faber, 1943; Stefanelli, 1961; Montgomery, 1963), and one might therefore expect changes in albumin levels to be paralleled by changes in serum cholinesterase in pregnancy. Since the changes in serum cholinesterase reported in this study appear to agree with those previously described for albumin, it is reasonable to conclude that haemodilution is not a major factor affecting cholinesterase levels during pregnancy.

The fact that there is no rise in cholinesterase
in pregnancy in response to albumin loss, as occurs in the nephrotic syndrome, suggests either that the fall may in fact be "physiological", or that there is impairment of hepatic response. If such impairment does exist, it would appear not to be aggravated by toxaemia. It has been suggested that serum cholinesterase activity is a fairly sensitive index of hepatic dysfunction (Khan, 1962). Routine liver function tests in toxaemia and eclampsia show no abnormality, but serum glutamic-pyruvic transaminase is a more specific and sensitive index of hepatocellular injury and has been shown to be increased in relation to the severity of toxaemia (Dass and Bhagwanani, 1964). It would thus seem that any liver dysfunction during pregnancy is not sufficiently severe to contribute to the reduction of serum cholinesterase.

Malnutrition, as previously stated, may cause low cholinesterase activity, but it would seem unlikely that in a high proportion of normal pregnancies this is of sufficient severity to account for the enzyme changes.

The extent and significance of the anticholinesterase effect of oestrogen have not yet been adequately assessed. Whatever the explanation of decreased cholinesterase activity in pregnancy sera may be, and whatever its physiological significance may be, it would seem that pregnant women constitute a population with moderate and in a few cases severe diminution of this enzyme, without other evidence of severe metabolic or toxic upsets. In this latter respect they differ from patients with severe burns.

Henriquet (1962) has remarked upon the greater sensitivity to suxamethonium of patients at the end of pregnancy, and has suggested lower cholinesterase as the explanation. This neuromuscular sensitivity, except in a few cases, can only be of a relatively minor order. Foldes (1960) pointed out the rough correlation between the level of serum cholinesterase and the duration of suxamethonium, but suggested that the correlation may be closer in subjects with abnormally low cholinesterase levels.

Foldes (1951) had earlier stated that the autonomic effects of paralyzing doses of suxamethonium were negligible. However, Phillips (1954) showed varying degrees of bradycardia, ventricular extrasystoles and nodal rhythm in association with suxamethonium, and suggested caution in the administration of the drug to patients with disorders of cardiac conduction. Since then, numerous reports and reviews have indicated that suxamethonium, particularly by intermittent injections, is associated with a high incidence of bradycardia and arrhythmias (Johnstone, 1955; Leigh et al., 1957; Martin, 1958; Andreucci, 1962) and although atropine provides some protection, its effectiveness varies greatly according to dose and route of administration. Some protection appears to be afforded by the prior administration of thiopentone (Williams et al., 1961; McIntyre, 1962) or by deep anaesthesia (Graythorne, Turndorf and Dripps, 1960).

More severe arrhythmias following suxamethonium have been reported in digitalized patients (Dowdy and Fabian, 1963). Periods of asystole for up to 16 seconds are not uncommon (Martin, 1958; Bullough, 1959), and the severity of the effect appears to be related to the dose of suxamethonium (Galindo and Davis, 1962), and in carefully controlled experiments has been found to be proportional to the duration of apnoea (Graf, Ström and Wählin, 1963). It may be deduced from these facts that subjects with diminished cholinesterase activity are possibly more prone to develop severe arrhythmias after suxamethonium. It is probable that patients with a moderate acquired deficiency of cholinesterase, especially if it is associated with systemic or cardiac disease, are more liable to arrhythmias than patients with inherited cholinesterase deficiency, because in the latter the marked prolongation of apnoea after a first administration of suxamethonium is likely to discourage the administration of repeated doses. Presumably any arrhythmias in the presence of low cholinesterase in otherwise healthy patients go largely undetected and cause such patients no harm. The tendency in current anaesthetic practice to omit atropine premedication may make the detection of suxamethonium-induced arrhythmias more widespread, and the suggestion that intermittent suxamethonium is the most suitable relaxant technique for obstetric operations (Hodges and Tunstall, 1961; Tunstall, 1963) makes this field promising for the investigation of arrhythmias in the presence of diminished serum cholinesterase activity. This is the subject of a further study.
ACKNOWLEDGEMENTS
I wish to record my indebtedness to Professor S. C. Fraser, Department of Chemical Pathology, for permission to use the facilities of his department, and to Dr. G. P. Fraser for instruction in the use of the Radiometer pH-stat and Titragraph, and for allowing me to use a number of his serum cholinesterase estimations in the non-pregnant and pregnant groups. Thanks are due to the obstetricians of the Aberdeen Maternity Hospital and of the Obstetric Medicine Research Unit for providing access to their patients, and to the technical staff of the Obstetric Medicine Research Unit, Aberdeen Maternity Hospital, particularly Mr. G. A. Myers, who performed the statistical evaluations, and to thank Dr. W. N. Rollason, Department of Anaesthetics, for his advice regarding this work.

REFERENCES


LA DEFICIENCE EN CHOLINESTERASE SERIQUE

II: GROSSESE

SOMMAIRE

On passe brièvement en revue les faits contradictoires concernant les modifications de la cholinestérase sérique pendant la grossesse. On décrit une technique pour connaître la cholinestérase sérique par titrage continu à pH constant, et on présente les résultats d'une étude de l'enzyme chez 149 femmes. Les femmes enceintes ont une diminution de l'activité cholinestérase en comparaison avec les femmes non enceintes de l'âge de celles qui attendent un enfant, et cette diminution est statistiquement très significative. La chute semble commencer peu après 10 semaines de gestation. La réduction de l'activité de l'enzyme chez les malades présentant une toxémie gravidique n'a pas été très différente de celle des cas non toxémiques. On présente les raisons de croire que ni l'hémodilution ni l'insuffisance hépatique ne peuvent fournir une explication convenable des modifications décrites. L'importance clinique possible des modifications est soulignée.

CHOLINESTERASEMANGEL IM SERUM

II: SCHWANGERSCHAFT

ZUSAMMENFASSUNG

Die einander widersprechenden Ansichten über die Veränderungen der Serumcholinesterase während der Schwangerschaft werden kurz besprochen. Es wird eine Methode zur Bestimmung der Serumcholinesterase mit kontinuierlicher Titration bei konstantem pH beschrieben und die Ergebnisse einer Untersuchung über dieses Enzym bei 149 Schwangeren vorgelegt. Im Vergleich zu nicht schwangeren Frauen im gebärfähigen Alter fand sich bei den schwangeren Frauen eine Abschwächung der Serumcholinesteraseaktivität und diese Abschwächung ist statistisch sehr signifikant. Der Abfall scheint kurz nach der zehnten Schwangerschaftswoche einzusetzen. Die Verringерung der Enzymaktivität bei Patientinnen mit Schwangerschaftstoxikose war nicht signifikant verschieden im Vergleich zu den nichttoxischen Fällen. Es werden Gründe für die Annahme vorgelegt, daßweder die Blutverdünnung noch die Leberinsuffizienz die beschriebenen Veränderungen ausreichend erklären. Auf die mögliche klinische Bedeutung der Veränderungen wird hingewiesen.