SERUM CHOLINESTERASE DEFICIENCY
I: DISEASE AND INHERITANCE

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SUMMARY

A review is presented of the literature regarding the nature, site of production, physiological function and pharmacological function of serum cholinesterase. A study of the literature indicates that the following conditions may be associated with deficiency of serum cholinesterase: liver disease; chronic anaemias; malignant states; cardiac failure; severe acute infections; malnutrition; tuberculosis; uraemia; collagen diseases; surgical shock; severe burns; blood dyscrasias; exposure to organophosphorus compounds; haemodialysis; therapeutic radiation; treatment with albumin, AB-132 and certain phenothiazine drugs. The literature relating to serum cholinesterase as a liver function test is reviewed, with reference to the changes in liver function after surgery, and in the assessment of chronic liver disease and venous shunt operations. The genetic aspects of serum cholinesterase deficiency are briefly reviewed.

Cholinesterases were classified by Mendel and Rudney (1943) into "true" cholinesterase and "pseudo" cholinesterase, the latter existing in glial tissue, heart muscle, intestine, skin, liver and plasma. There is evidence (Liddell, Newman and Brown, 1963) that cholinesterases found in serum and in such tissues as liver, brain, ileum and kidney are determined by the same structural gene and that the amino-acid sequence of the enzymes found in these various tissues is probably identical. In anaesthetic practice pseudocholinesterase has more recently been referred to as serum cholinesterase, the adopted biochemical name now being acylcholine acylhydrolase (Dixon and Webb, 1964).

The site of production of serum cholinesterase in the liver is now well established, and three acylcholine acylhydrolase fractions have been separated, by chromatography and gel filtration, in liver tissue (Svensmark, 1963). Fraction II was found to be identical with human serum cholinesterase with respect to enzymatic properties and electrophoretic mobility, while fractions Id and Ie, although identical with serum cholinesterase regarding enzymatic properties, differed in their molecular properties. Svensmark concludes that the findings are consistent with the assumption that serum cholinesterase is produced in the liver, with fractions Id and Ie as precursors, and fraction II as the final product.

Serum cholinesterase is a mucopolysaccharide with m.w. 165,000 (Henriquet, 1962). The main cholinesterase activity of plasma is associated with α-globulin (Kekwick, Mackay and Martin, 1953). Electrophoretically, activity is found mainly in the α_2-β-globulin area (Harris, Hopkinson and Robson, 1962), although about 5 per cent is associated with albumin. On two-dimensional electrophoresis, four zones of activity are discernible, and a fifth zone in some 5 per cent of individuals (Harris et al., 1963). The existence of this fifth component appears to be associated with increased cholinesterase activity.

The physiological function of serum cholinesterase remains uncertain, but the suggestion by Lehmann, Silk and Liddell (1961), that it may protect the organism against the effects of choline esters formed during metabolism, was supported by the proposal (Clitherow, Mitchard and Harper, 1963) that the principal biological function of the enzyme might be to hydrolyze butyrylcholine preferentially, in order to prevent its powerful nicotinic action when it is produced during fatty acid degeneration. This proposal is in turn supported by the observation of a seasonal variation

in serum cholinesterase in horses to parallel the seasonal variation in fat metabolism, both being maximal in late summer. Furthermore, Thompson and Trounce (1956) had previously shown the existence of increased cholinesterase activity in obesity, and suggested that the enzyme was in some way related to fat metabolism. Further physiological roles of the enzyme as it exists in other tissues are still obscure, but in the mammalian ileum it appears to function in the removal of non-neurogenic acetylcholine, associated with spontaneous rhythmicity, and, at least in some animals, it is associated with the destruction of nervously-released acetylcholine also (Jamieson, 1963). Complete absence or inhibition of the enzyme is compatible with life, but this may not be true under conditions of stress (Heymans and Casier, 1948).

The pharmacological role of serum cholinesterase became evident when Bovet-Nitti (1949) demonstrated its action in hydrolyzing suxamethonium. It is also of importance in the hydrolysis of local anaesthetic esters, such as procaine and amethocaine, and more recently (Nowell, Scott and Wilson, 1962) it has been shown to be intimately involved in the hydrolysis of neostigmine. Although serum cholinesterase is also active in the hydrolysis of succinylmonocholine, a separate liver enzyme has been found to be responsible for at least part of succinylmonocholine hydrolysis (Greenway and Quastel, 1955). The use of suxamethonium in anaesthetic practice increased rapidly after 1950, and abnormal reactions were initially attributed to such things as overdosage with thiopentone (Durrans, 1952) or respiratory acidosis (Davis et al., 1955). Increasing experience with this relaxant showed that in a few cases prolonged apnoea was associated with low serum cholinesterase activity (Evans et al., 1952). In such cases the administration of pseudocholinesterase concentrate (Cholase) was advocated to reduce the duration of apnoea (Evans et al., 1953). Borders et al. (1955) were unable to confirm the value of Cholase in persons with depressed cholinesterase activity, although reduction of apnoea in normal subjects was obtained. Foldes, Rendell-Baker and Birch (1956) suggested that prolonged apnoea after suxamethonium was more likely to occur in the presence of diminished cholinesterase activity if there was also poor urinary output of succinylmonocholine.

Hall and Lucas (1937) indicated that a wide range of cholinesterase activity existed in normal subjects. They stated that the values were not related to diet, exercise, fatigue, age, sex or weight, and were unaffected by pregnancy. Their study of cholinesterase activity in malignant conditions, acute and chronic infections, and other conditions failed to show any characteristic change. However, Faber (1943) showed that in liver disease, chronic anaemias, malignant states, cardiac failure and severe acute infections, serum cholinesterase activity was reduced, whilst in hypertension and diabetes it was increased. It was subsequently established (Thompson and Trounce, 1956) that, in diabetes, cholinesterase levels are unrelated to the diabetic state itself and are increased in the presence of obesity, both in diabetic and non-diabetic subjects. Other studies have shown that in malnutrition, liver and plasma cholinesterase are diminished, and recover with treatment (Waterlow, 1950; Hutchinson, McCance and Widdowson, 1951; Montgomery, 1963). Additions to the list of low-cholinesterase states include tuberculosis, uraemia, and shock (Editorial Brit. med. J., 1951). Exposure to organophosphorus compounds reduces cholinesterase activity (Barnes and Davies, 1951), and recently (Barr, 1964) it has been shown that combination occurs between organic phosphates and the esteratic site of serum cholinesterase. Low esterase activity exists in patients with blood dyscrasias (Scudamore, Vorhaus and Kark, 1951), and effective treatment produces a recovery of enzyme level. Therapeutic radiation has been found to cause diminished serum cholinesterase (Hodges and Harkness, 1954), and reduced activity may follow transfusions of albumin (Vorhaus, Scudamore and Kark, 1950). Cholinesterase deficiency has been shown to follow dialysis with the Kolff twin-coil kidney (Holmes, Nakamoto and Sawyer, 1958). The reason for this is uncertain, but the extent of the deficiency may vary according to the characteristics of particular batches of cellophane used in the coil units. Therapy with the cancer chemotherapeutic agent AB-132 causes diminished cholinesterase activity, and two cases of greatly prolonged apnoea after suxamethonium and AB-132 have been reported (Wang and Ross, 1963).

Earlier studies in cancer patients indicating low cholinesterase values have recently been confirmed (McComb, LaMotta and Wetstone, 1964), and this work suggests that the decreased enzyme activity is not merely a reflection of lowered protein synthesis, nor is it due to the production of a different enzyme.

Todrick (1954) demonstrated inhibition of pseudocholinesterase by various drugs, including promethazine and diphenhydramine, and inhibition of the enzyme by chlorpromazine has been shown (Erdös et al., 1956). Hofstee (1960) has shown that chlorpromazine binds with the enzyme at the site of negative charge, so causing competitive inhibition of activating ions such as calcium. This work has been substantiated in a study of the effects of chlorpromazine on pig serum cholinesterase (Oliver, Funnell and Oki, 1963). It is of interest in this connection that phenothiazines have been shown to exacerbate the symptoms of poisoning by anticholinesterase insecticides, and anticholinesterases potentiate behavioural changes induced by chlorpromazine (Goldberg and Johnson, 1964).

The changes in cholinesterase in liver disease, and the relationship between cholinesterase and serum albumin have been extensively studied in the past. Vorhaus, Scudamore and Kark (1950, 1951) have shown the value of serum cholinesterase estimation as a test of liver function, particularly to assess progress in chronic liver disease. This has been further evaluated by Wilson, Calvert and Geoghegan (1952) and Khan (1962), who have emphasized the value of the test in prognosis. Burnett and Cohen (1955), studying liver function after surgery, attempted to integrate serum cholinesterase values with those liver function tests concerned with protein metabolism. Their findings suggested that there was depression of liver function after surgery, most marked on the fifth day after operation, and that serum cholinesterase was a sensitive index of this. The value of serum cholinesterase estimation in the prognosis of venous shunt operations in the portal system has been assessed by Hunt and Lehmann (1960), who conclude that the esterase activity gives greater advance knowledge than any other test to show the likely clinical trend of a patient. Kekwick (1960) showed a change in the substrate specificity of serum cholinesterase in liver disease, and suggested that this was due to a change in configuration of the enzyme. The sensitivity to suxamethonium in patients with collagen diseases has been pointed out (Potts and Thornton, 1961), and the existence of diminished cholinesterase activity is presumed to reflect liver dysfunction.

Transfusion of albumin causes a temporary decrease in cholinesterase, suggesting an autoregulative mechanism for the control of serum albumin and cholinesterase together, although in the nephrotic syndrome, albumin transfusion causes a striking rise in cholinesterase (Kunkel and Ward, 1947). The nephrotic syndrome is the only condition in which hypo-albuminaemia and a high serum cholinesterase regularly co-exist (Maier, 1956; Pietschmann, 1960; Stefenelli, 1961).

The low albumin and cholinesterase values in severely burned patients may be of greater importance than has been generally realized (Bush et al., 1962; Bush, 1964). The pathophysiological factors involved in the hepatic lesion in burns have been clarified experimentally by Arturson (1961), but Stenberg (1964) makes less definite conclusions about the qualitative and quantitative changes in liver function in burns, possibly because the burns which he produced in mice were only up to 15 per cent of body surface. Gürtner, Kreutzberg and Doenicke (1963) performed serological, pathological and enzyme histochemical investigations on sera and liver biopsy specimens from eighty surgical patients, and found a marked depletion of liver cell cholinesterase after surgical shock, the changes being reflected in the serum.

In healthy individuals, prolonged apnoea after suxamethonium is likely to be due to the existence of "atypical" esterase. Following on the work of Kalow and Staron (1957) using dibucaine (cinchocaine) numbers, more recent reviews have indicated the existence of a number of serum cholinesterase phenotypes (Lehmann et al., 1963; Harris, 1964; Bamford and Harris, 1964). Abnormal cholinesterase differs from the normal enzyme in being less active in hydrolyzing suxamethonium and more resistant to a variety of inhibitors, notably dibucaine and sodium fluoride, although organophosphorus compounds appear to inhibit both enzymes equally (Kalow and Davies,

1958). The importance of atypical cholinesterase in relation to prolonged apnoea after suxamethonium, and the management of prolonged apnoea, have recently been reviewed (Vickers, 1963; Nixon and Thiel, 1964). In subjects with atypical enzyme, an unusual urinary compound has been demonstrated by thin-layer chromatography on Silica Gel G (Rubinstein, Dietz and Czebotar, 1964), and it is of interest that the same compound has been identified in the urine of myasthenic patients treated with pyridostigmine and in a normal subject after administration of pyridostigmine. The complete absence of serum cholinesterase, with markedly prolonged apnoea after suxamethonium, has been described in a few subjects (Hart and Mitchell, 1962; Doenicke et al., 1963). Genetic studies have led to the conclusion that this rare anenzymia exists in persons who are homozygous for a "silent" gene which is an allele of the "usual" and "atypical" genes (Liddell, Lehmann and Silk, 1962; Simpson and Kalow, 1964).

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LA DEFICIENCE EN CHOLINESTERASE SERIQUE
I: MALADIE ET HEREDITE

SOMMAIRE

On présente une revue de la littérature concernant la nature, le lieu de production, les fonctions physiologique et pharmacologique de la cholinestérase sérique. Une étude de la littérature indique que les affections suivantes peuvent être associées à la déficience en cholinestérase sérique: affection hépatique, anémies chroniques, tumeurs malignes, défaillance cardiaque, infections aiguës graves, malnutrition, tuberculose, urémie, maladies du collagène, choc chirurgical, brûlures graves, dyscrasies sanguines, exposition aux composés organophosphorés, hémodialyse, radiothérapie, traitement par l'albumine, l'AB-132 et certaines phénothiazines. On passe en revue la littérature se rapportant à la cholinestérase sérique en tant que test fonctionnel hépatique, avec référence aux changements de la fonction hépatique après les interventions chirurgicales, et à l'étude des hépatites chroniques et des opérations de shunt veineux. On donne un bref aperçu des aspects génétiques de la déficience en cholinestérase sérique.

CHOLINESTERASEMANGEL IM SERUM
I: KRANKHEIT UND VERERBUNG

ZUSAMMENFASSUNG

Es wird ein Literaturüberblick über die Chemie, den Entstehungsort, die physiologische Funktion und die pharmakologische Wirkung der Serumcholinesterase gegeben. Bei der Literaturdurchsicht wurde festgestellt, daß folgende Zustandsbilder mit einem Nagel an Serumcholinesterase einhergehen können: Lebererkrankung, chronische Anämien, maligne Erkrankungen, Herzversagen, schwere akute Infektionen, Unterernährung, Tuberkulose, Urämie, Kollagenkrankheiten, chirurgischer Schockzustand, schwere Verbrennungen, Blutkrankheiten, Einwirkung von organischen Phosphorverbindungen, Hämodialyse, Bestrahlungstherapie, Behandlung mit Albumin, AB-132 und bestimmten Phenothiazinabkömmlingen. Die Literatur in bezug auf die Serumcholinesterase als Leberfunktionsprobe wurde durchgesehen, besonders mit Hinblick auf die Veränderungen der Leberfunktion nach chirurgischen Eingriffen und auf die Bewertung der chronischen Lebererkrankung und der venösen Shunt-Operationen. Auf die genetischen Aspekte des Serumcholinesterasemangels wird kurz eingegangen.