minimum iodoacetate dosage and after only one-third the usual minimal exercise (compared to model animals not given ethanol), the ethanol-fed rats developed the muscle symptomatology. In addition, both male and intact female rats developed the more severe male-pattern of injury. Histological study revealed damage exclusively to the type 2B muscle fibres.

Our preliminary findings in the ethanol-fed rats would tend to support the contention by Slavin et al.10 that selective vulnerability of type 2B muscle fibres in chronic alcoholism is the result of alcohol-induced alteration of anaerobic glycolysis. It is possible that in chronic alcoholics the syndromes of acute rhabdomyolysis and chronic muscle atrophy are opposite ends of a spectrum of type 2B muscle fibre response to varying degrees of alcohol-induced interference with energy metabolism.

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References

Elution of antibodies to Mallory's hyaline from kidneys of patients with alcoholic liver disease and mesangial IgA deposits

We were most interested to read the paper by Burns et al.1 reporting a Mallory body antigen (JMB2) in the mesangium of three patients with alcoholic liver disease. Data from our elution experiments with such kidneys help to confirm the significance of this report.

Kidneys were obtained at post-mortem from nine subjects with alcoholic liver disease and mesangial IgA deposits. Washed glomerular suspensions were eluted with a citrate buffer pH 3-2 and the concentrated eluates were tested for IgA, IgG and IgM class antibodies to Mallory bodies by indirect immunofluorescence using frozen sections of liver from a patient with alcoholic liver disease and abundant Mallory hyaline. IgA anti-Mallory body staining was seen with seven of the eluates. The same pattern of staining was seen with serum from a patient with acute alcoholic hepatitis and with two high titre smooth muscle antibody (SMA) sera. No staining was seen with an eluate from a normal kidney or with normal serum or serum from a patient with IgA myeloma. No staining was found with reagents for IgG or IgM and there was no reactivity with normal liver sections.

These findings suggest that IgA anti-Mallory body/Mallory body complexes contribute to the mesangial deposits seen in some patients with alcoholic liver disease. However, other immune mechanisms are also operative in alcoholic liver disease, notably increased gut permeability to antigens, impaired hepatic sequestration of antigens, immune complexes and IgG polymers.14 The participation of IgA in each of the above mechanisms explains the predominance of IgA in the associated hyperglobulinaemia, serum immune complexes and mesangial immune deposits.

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<table>
<thead>
<tr>
<th>Total Ig (µg IgG equiv/ml)</th>
<th>Indirect immunofluorescence staining of Mallory bodies (Graded 0-+++)</th>
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<tr>
<td></td>
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<td>9</td>
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Sera

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<td>IgA myeloma</td>
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Letters to the Editor

References


The Howie code and the price of safety

I endorse the need for caution expressed by Dr Whale about interpreting the tuberculosis figures in the last survey carried out for the ACP with help from the IMLS and others. This adds to the importance of the further data to be collected in the next survey covering 1982–83, and continued co-operation is hoped for in this important surveillance project. Attention will be paid to classification of groups other than “medical, science, MLSO” since the breakdown which served adequately for the original problem of hepatitis is less satisfactory for tuberculosis. Dr Whale’s caution about the high attack rates calculated from only two cases in “porters, assistants” should be related to the even higher rates calculated for those “technicians and attendants” in mortuary and post-mortem work in 1979–81. Hepatitis B surprised many of us when inapparent parenteral contamination emerged as an important mode of infection in laboratories, and in the light of the study by Newsom and others it will be interesting to discover whether routes other than airborne prove to be important in the spread of tuberculosis to laboratory staff.

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References


Book review


This monograph is one of the first of its kind to deal with the pathology of oxygen by an author well known in this field. It is well set out and the illustrations are clear. Overall the style is uniform and easy to read.

After two introductory chapters there is a chapter on the biochemistry of oxygen and how hyperoxia may mediate its damage through various enzyme systems. Subsequent chapters deal with the effect of oxygen on the body systems, most space rightfully being given to the lung, eye, and nervous system. I would have liked to have seen the pulmonary section expanded and balanced with some more recent references. No account is taken of the lung’s potential for repair. The fact that pulmonary fibrosis is not necessarily a permanent lesion or a definite sequel to hyperoxia receives scant attention. Pulmonary oxygen toxicity in humans is a minefield since often there has been pre-existing lung damage and in the reparative process oxygen appears often to take the blame. Thus I cannot agree with the author’s statement that “pulmonary oxygen toxicity is the principal limiting factor in the therapeutic utilisation of oxygen”. The author quotes evidence that during the proliferative phase of diffuse alveolar damage there is an increase in lamellar bodies which could represent increased surfactant production. In ARDS in humans there may well be a decrease in lamellar bodies and surfactant which is more easily deformed than normal. In the section on Idiopathic Respiratory Distress Syndrome the author confusingly refers to “honeycombed emphysematous spaces”. In fact this appears to be honeycomb lung secondary to pulmonary fibrosis and not emphysema.

However, all the above features do not detract from an important work on the effect of excess oxygen on both experimental animals and man. It will become a reference book for any worker in this field and hopefully we will see an expanded second edition in the near future.

PS HASLETON

Some new titles

The receipt of these books is acknowledged, and this listing must be regarded as sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits.


Notice

**Third International Symposium on Morphometry in Morphological Diagnosis**

The Third International Symposium on Morphometry in Morphological Diagnosis will be held in Delft, The Netherlands, September 13–15, 1984.

The purpose of this Symposium is to provide pathologists and clinicians interested in diagnostic morphometry with theoretical and practical education. In addition, diagnostic human pathologists experienced in diagnostic morphometry will find the Symposium useful as a platform to present their own applications.

A restricted number of free papers will be allowed, after approval by the scientific board. A simultaneous poster session will be organised during the Symposium. The number of participants to the Symposium will be limited to approximately 150.

For further information, please contact: “Third Diagnostic Morphometry Symposium” QLT, convention service Keizersgracht 792 1017 EC AMSTERDAM The Netherlands.