ASSOCIATION OF CLINICAL PATHOLOGISTS:  
61st GENERAL MEETING

The 61st general meeting was held at the Royal College of Surgeons, London, on October 2, 3, and 4, 1958.

The papers delivered in honour of Virchow’s work on cellular pathology are published in full in this issue, and summaries of the other papers follow.

**Congenital Pulmonary Lymphangiectasis**

K. M. LAURENCE (Hospital for Sick Children, London) said that congenital pulmonary lymphangiectasis was characterized by a progressive, unrelieved cyanosis which generally leads to death within 24 hours of birth. At necropsy the bulky and inelastic lungs, with an exaggerated lobular pattern, prominent subpleural lymphatic vessels and cysts, are diffusely honeycombed with fluid-filled cysts measuring up to 4 mm. in diameter. Histologically these endothelium-lined cysts are situated in abundant young connective tissue under the pleura and the interlobular septi show a close relationship to blood vessels and bronchi. The lung parenchyma itself is mature. The position and structure of these cysts, which are shown by reconstructed serial sections to be part of an intricate network of irregular inter-communicating channels, indicate that they are lymphatic in origin.

A developmental disorder is suggested by its frequent association with other severe anomalies and by the early onset of symptoms. It is probably caused by a continued growth of the lung with the tissue elements maintaining their 16-week inter-uterine proportions, when the lymphatics are very much more prominent than at term. The progressive cyanosis might be due to cysts taking up some of the available thoracic space or pressing upon vessels, but more likely to the inelasticity of the lung leading to circulatory embarrassment.

The anomaly is certainly more common than the few previous reports suggest. An incidence of 1 in 100 to 150 perinatal necropsies is indicated by the 13 cases discussed. (This paper will be published in full in the *Journal*.)

**Pathology of Familial Pulmonary Hypertension**

J. TREGILLUS (Darlington) and P. N. COLEMAN (Northallerton) gave details of the morbid histology in three fatal cases of primary pulmonary hypertension in a brother and two sisters (Coleman, Edmunds, and Tregillus, 1959). There was extensive intimal proliferation in the pulmonary arteries which had caused serious occlusion in the smallest branches. It was probable that all the intimal proliferation was the result of previous thrombosis.

The hypertension was almost certainly genetic in origin, but the mode of operation of the genetic defect remained in doubt. No congenital anomaly could be found and hypertension was explained as the result of an inherited functional defect causing hypertonia in the muscular arteries. There was histological support for this in the findings of contracted arteries in the sections. Symptomless functional hypertension may have been present for many years, finally bringing about changes in the pulmonary arteries favourable to the development of thrombosis and intimal fibrosis. The obstruction then became too much for the functional reserve of the right heart and symptoms of failure appeared for the first time. In the two sisters, pregnancy may have proved an additional stress leading to an earlier development of thrombosis.

**Infantile Cardiomegaly: the Infantile Type of Endocardial Fibroelastosis**

R. I. K. ELLIOTT (Royal Sussex County Hospital, Brighton) said that the infantile form of endocardial fibroelastosis was distinguished by generalized hypertrophy of the heart with dilatation of the left ventricle and marked thickening of the mural endocardium. Affected children are, typically, healthy and normal until their terminal illness; this may take the form of progressive congestive failure, but is more commonly abrupt, catastrophic collapse and death suddenly occurring in the course of a mild febrile upset.

It is unlikely that the endocardial thickening is anything more than an incidental finding; the defect, as Brigden (1957) has suggested, is probably in the myocardium. Histological findings tend to support this; there is interfascicular and interfibrillar oedema and longitudinal banding of the sarcoplasm of the myofibrils. The condition appears to be familial, a history of unexplained sudden death in infancy among the siblings of either the child or its parents being obtained in three of the six cases in this series.

The present terminology, with its emphasis on fibroelastosis, is misleading; the old description “congenital idiopathic hypertrophy” was better; but as an alternative, more in line with modern views, the name “infantile cardiomegaly” is suggested.

**REFERENCE**

Studies on Endogenous Lipid Pneumonia and Blood Lipid Levels in Cortisone-treated Rabbits

T. J. Moran (Presbyterian Hospital, Pittsburgh, U.S.A.) described studies of the effect of cortisone on the development of bronchiolitis obliterans in rabbits given intratracheal injections of 1.0% nitric acid which showed that cortisone prevented or modified the experimental lesions of bronchiolitis obliterans but did not influence the death rate from acute pulmonary oedema or pneumonia compared with control animals. During the course of the study a peculiar form of "endogenous lipid pneumonia" was observed in cortisone-treated animals which had been given intratracheal nitric acid. The lipid material in this form of "endogenous lipid pneumonia" varied greatly in size and was chiefly extracellular as compared with the endogenous lipid pneumonia observed in animals not treated with cortisone and in human examples of this condition. The relation of this pneumonia to various causes was discussed: (1) Overloading due to high blood lipid levels caused by cortisone; (2) possible interference with macrophage ingestion of the lipid; (3) interference with the macrophage motility and activity; (4) possible physical or chemical change in the lipid itself.

In the discussion it was pointed out that rabbits were very susceptible to cortisone, and that the picture might bear no relationship to the human disease.

There was also some discussion on "sil o fillers' disease," where the workers are exposed to NO₃, which occurs in high concentration within two days of the silo being formed, and the differentiation of this from farmer's lung, which is probably due to fungus infection, and possibly associated with "sil o emptiers' disease."

Nephritis Associated with Streptococcus Pyogenes

Type 12

D. J. H. Payne and D. A. Slade (Northallerton) gave an account of an epidemic of nephritis in north Yorkshire. Five cases occurred in a small village in 1957, during the first two months of the year. Throat and nasal swabs from the local schools showed that there was a high incidence of Streptococcus pyogenes type 12 infection in these schools. Urines of the children showed albumin, red cells, and casts in some cases, which, when compared with urines from children in schools 25 miles way, indicated kidney damage. In all, 43 children, 10 of whom had type 12 strains in their throats, had evidence of kidney damage. Sixteen months later, seven out of 43 still have abnormal findings—five with albumin, one with red cells and casts, and one with red cells only. No more cases occurred until December, 1957. From then until the present time (September, 1958), cases have occurred in the Northallerton area and York mainly during the January–March period as before. Twenty-four cases from the Northallerton area yielded 10 type 12 strains and, in York, nine type 12 strains were isolated from 25 cases and type 22 from another. In all more than 60 cases of acute nephritis have occurred in the area. The ages ranged between 4½ and 50 years, 25% being over 21 years of age. The male to female ratio was 2 to 1. Only four out of 32 did not have a history of sore throat or otitis media. Mildness of the initial symptoms was a feature, as was a shorter latent period between infection and the onset of nephritis, than has been described. Follow-up of the cases is proceeding and might, in time, throw light on the aetiology of chronic nephritis.

In the discussion which followed it was suggested that serological follow-up of, for example, ASO titre might also give some indication of the imminence of complications to this infection.

The Control of Cross-infection by Formaldehyde Disinfection of Blankets

H. Caplan (Highlands General Hospital) said that regular sterilization of blankets and bed-side curtains was an essential measure in the prevention of cross-infection. Formaldehyde possesses a direct bactericidal activity which is enhanced by moderate heat and by a vacuum (which increases the penetration of the gas).

Using a commercially available formaldehyde generator (supplied by Messrs. Manlove, Alliott and Co., Ltd., London, S.W.1) attached to the ordinary hospital autoclave, blankets are sterilized by exposure to formaldehyde vapour in a vacuum of 15 in. Hg at 55° to 60° C. for 20 minutes. Hot air is then blown through to remove the smell. Up to 50 blankets can be sterilized at a time in an autoclave chamber of 120 cu. ft. capacity. Following the introduction of this technique cross-infection in a male general surgical ward of 25 beds was eliminated. There was one wound infection in the ensuing six months compared with 17 in the preceding six months. The incidence and severity of urinary infections following prostatectomy were markedly diminished from 70 to 100% down to as low as 20% and the average stay in hospital after prostatectomy was reduced from 60 to 20 days. The consequent saving on drugs alone meant that the apparatus paid for itself in less than six months. Blankets have now been sterilized fortnightly for over a year and have not shrunk or become matted and formalin dermatitis has not been experienced.

The Antibody Content of Single Cells

M. C. Berenbaum (Greenford) described an autoradiographic technique for localizing and measuring antibody in individual cells. The paper is published in full on page 543.

Observations in Bone Marrow Transplants in Man

J. G. Humble, K. Newton, and N. H. Kemp (Westminster Hospital, London) discussed bone marrow transfusions, and said that the two features which make it difficult to apply well-known tech-
Techniques used with animals to human transfusions are lack of precise knowledge of the effects of ionizing whole body irradiation in man and the difficulty of obtaining sufficient cells for transplanting. For this reason they tried the effects of local irradiation, for example, of the ilium, and using the transplant as an infusion of cell suspension into the aorta. In two cases there was certainly improvement. In one case of leukaemia there appeared to be a definite slowing down of the process, and in another case the irradiated zone on examination had become cellular. The appearance of female clubs in the polymorphs after transfusion of female blood into aplastic marrow suggested in one case that the transplant had "taken." It was noted that the procedures were well tolerated by the patients, most of whom were extremely ill.

**JOINT MEETING WITH THE ASSOCIATION OF CLINICAL BIOCHEMISTS**

This meeting was a symposium on paper chromatography, at which Professor C. E. Dent took the chair.

**Chromatography of Androgens, Oestrogens, and Progestogens**

R. V. Short (A.R.C. Unit of Reproductive Physiology and Biochemistry, Cambridge) said that the problem of steroid determinations in the urine of domestic animals was complicated by qualitative differences in the metabolites excreted by some species and the presence of interfering substances in the urine extracts. In addition, the major excretory pathway for some steroid metabolites may be via the gastro-intestinal tract. These difficulties have emphasized the need for a simple technique that is capable of measuring the active steroids in the peripheral blood of any animal.

Such a method has already been described for the determination of progesterone in peripheral blood (Short, 1958a). With a few minor modifications, this method can be adapted for the simultaneous determination of androstenedione, testosterone, oestrone, oestradiol-17β, oestriol, and probably corticosterone and hydrocortisone (Short, 1958b).

The basic steps of the modified procedure are as follows:

1. Titration of the plasma to pH 10.0 with NaOH.
2. Extraction with ether.
3. Ether extract evaporated to dryness, partitioned between light petroleum and methanol.
4. Application of methanolic extract to a paper chromatogram.

Paper chromatography is carried out in the Bush system appropriate for the steroids to be estimated. The steroids are eluted from the paper and measured in the spectrophotometer. The Kober reaction is used for the oestrogens, and the ultra-violet absorption at 240 mλ for the αβ-unsaturated androgens and progestogens. Using a microcell attachment, this limits the sensitivity to 0.5 to 1 µg. steroid. With the application of fluorimetric techniques, it should be possible to achieve a ten- to hundred-fold increase in sensitivity. This should permit the determination of these steroids in a small sample of peripheral blood.

**REFERENCES**


**The Assessment of Disordered Indole Metabolism Using Paper Chromatography**

M. Sandler (Queen Charlotte's Maternity Hospital, London) said that assay of biological fluids for indoles by paper chromatographic techniques could only be, at best, roughly quantitative. The reasons given for this view included the lack of sensitivity of these compounds during electrolytic desalting procedures, their susceptibility to atmospheric oxidation, certain specific drawbacks of the solvent systems commonly used, and the variability of results obtained with developing reagents.

Qualitative studies, using two-dimensional techniques especially, are of great value in clinical practice, however. Diagnostic patterns may be obtained in the carcinoid syndrome, Hartnup disease, phenylketonuria, melanogenuria, and other disorders.

**The Fractionation of Serum Proteins by Ion Exchange Chromatography**

M. P. Tombs, K. G. Cooke, and N. F. MacLagan (Westminster Hospital, London) described a method which had been developed for the fractionation of serum proteins into five groups (P, A, B, G, and L in order of elution) on carboxy-methyl cellulose columns (Peterson and Sober, 1956). Protein was estimated in the eluent by the ultra-violet absorption at 280 mλ. Carboxy-methyl cellulose was chosen because it was possible to obtain higher flow rates of eluting buffer than with diethyl amino ethyl cellulose, so that a serum required about three hours for examination. Group P contained mainly α1 globulins. Albumin was fractionated between groups A and B, but it was shown by re-chromatography that the two forms of albumin were completely interconvertible. They may bear some relationship to the electrophoretically distinguishable isomeric forms of albumin reported recently. The α2 globulins were distributed throughout the elution system, and β globulins occurred mainly in group B and γ globulins in B and G. The areas of the peaks bore a strict quantitative relationship to the amount of protein eluted, and can be used for comparative purposes, provided due allowance is made for the different specific absorption of the various components at 280 mλ. It seems that resolution into electrophoretically homogeneous components is very unlikely using a single-run technique, but that re-chromatography of groups obtained in a rough elution system such as this one leads to very high resolution. There is no upper limit to the amount of serum which can be employed.

**REFERENCE**

Uro-pepsinogen Excretion in Patients with Gastric Lesions

J. Schrager (Wigan) discussed uro-pepsinogen excretion in patients with gastric lesions. The uro-pepsinogen excretion was estimated in 232 healthy adults, 188 patients with active duodenal ulcer, 66 patients with gastric ulcer, and 90 patients with gastric carcinoma. At least three 24-hour specimens of urine were examined in each individual. The available data show that the range of uro-pepsinogen excretion in healthy adults falls between 36 and 90 μg. per hour with an average mean output of 70 μg. per hour (using Armour pepsin as standard). It remained constant within 10% for each person. In many cases the tests were repeated three or four times at monthly intervals, without significant change, in spite of wide variation of urine volume. Patients with duodenal ulcer had uro-pepsinogen excretion well above normal with a mean output of 150 μg. per hour. The excretion in patients with gastric ulcer was lower than that of duodenal ulcer patients, but significantly higher than the controls, with a great variation in output, 35-270 μg. per hour. It is suggested that this is due to the pathological condition of the gastric mucosa. Histological examination of the stomachs of patients who were operated on and whose uro-pepsinogen level was previously estimated revealed widespread gastritis and metaplasia. It was possible to correlate the uro-pepsinogen excretion with the state of the gastric mucosa. The uro-pepsinogen excretion in patients with gastric carcinoma was well below normal, and a considerable number excreted no uro-pepsinogen at all. The mean uro-pepsinogen excretion in this group was 26 compared with the normal 70 μg., and 126 μg. in patients with duodenal ulcer. It is suggested that uro-pepsinogen excretion may prove helpful in differentiating between benign and malignant lesions, and may help in the discovery of early malignant changes. The disappearance of uro-pepsinogen from the urine on repeated examination suggests widespread changes in the mucosa and warrants further examination.

An Immunochemical Analysis of the Proteins of Normal Urine not Present in Blood

G. H. Grant (Royal Salop Infirmary, Shrewsbury) described the immunochemical analysis of normal urine proteins by the agar gel diffusion and immuno-electrophoretic methods. Rabbit antisera prepared against normal urine colloids, against normal semen, and against the salt-precipitated mucoprotein of Tamm and Horsfall respectively were used after absorption with normal serum.

About 11 antigenic components not present in normal blood serum were detected in normal urine. Some appear to be specific to the urinary tract; others are common to male urine, female urine, and semen, and so might be excreted by the urethral glands. In addition traces of prostatic proteins occur in the male.

The mucoprotein of Tamm and Horsfall appears to be the principal urine protein not detected in blood. It arises from the upper urinary tract and is a mixture of immunochemically similar proteins of varying electrophoretic mobility.

Published methods for the quantitative analysis of urinary mucoproteins measure the carbohydrate content of protein mixtures rather than that of a single fraction.

Comparison of Protein-bound Iodide Determinations with Radioactive Test in Thyroid Disease

K. Steinitz (Rothschild Hospital, Haifa, Israel) discussed a modification of Barker's incineration method for routine use. Incubation is at 24 ± 0.1°C for 30 minutes in an ordinary well-insulated waterbath. Colorimetry is done without the internal standard described by Barker. The two-hour and the 24-hour uptake, the eight- to 24-hour urinary excretion and the PBI111 after 48 hours are compared separately with the PBI27 values. The two-hour uptake shows a good correlation in clear cases of hyperfunction, a bad correlation in normal, and a worse one in cases with non-toxic goitre or other states of iodine avidity. About three-quarters of this group cannot be diagnosed correctly. The 24-hour uptake, to-day considered as the least valuable of all I111 tests, gave in 39 of 40 cases of non-toxic goitre or iodine avidity an abnormal increase, together with normal PBI27 values. The eight to 24-hour urinary excretion of I111 gives results comparable with the two-hour uptake, i.e., relatively good results for clear hyperfunction, and a false classification of about 70% of euthyroid cases of iodine avidity. The 48-hour PBI111, though known to be influenced by the rate of turnover of the organic iodine in the thyroid, proved to be superior to the previous three methods. Its correlation with PBI27 is much better, and even in the bad group with iodine avidity the error is less than 20%.

The Estimation of Oxalic Acid in Urine and Other Biological Material

A. Hodgkinson and P. M. Zarembski (The General Infirmary at Leeds) reviewed the existing methods for the estimation of oxalic acid in urine and described a new procedure based on that of Powers and Levatin (1944).

The principle of the method is as follows: Urine is acidified with hydrochloric acid and half-saturated with ammonium sulphate. The gelatinous precipitate formed after standing is removed by filtration and the filtrate is extracted continuously with peroxide-free ether. The extracted oxalic acid is precipitated as the calcium salt, and reduced to glycollic acid by boiling with zinc powder and sulphuric acid. Glycollic acid is estimated colorimetrically by heating with chromotropic acid.

Recovery of oxalic acid from aqueous solution and from urine was found to be 98 ± 2%. The procedure can be applied, with suitable modification, to the
The Inheritance of an Idiopathic Low Plasma Pseudocholinesterase Level

H. Lehmann, V. Patston, and E. Ryan (St. Bartholomew's Hospital, London) discussed the inheritance of an idiopathic low plasma pseudocholinesterase level. The short-term action of the muscle relaxant suxamethonium is due to its rapid destruction by pseudocholinesterase. If the plasma level of this enzyme is low, the response to the drug is prolonged. The usual causes of this are liver disease and to a lesser extent general debility. Very occasionally patients were observed with an abnormally long response to suxamethonium where the low enzyme level was without obvious aetiology. Investigation of the families of such people led to the discovery of healthy persons with abnormally low pseudocholinesterase levels. It was suggested that the likelihood of finding these people by chance would be well below 1 in 1,000. In an attempt to get information on the mode of inheritance it was assumed that a very low pseudocholinesterase level up to 35 units (Warburg) represented the homozygous condition for an abnormal gene, and that the high normal level above 90 units indicated the normal homozygote. It remained to be seen whether within the intermediate range 36–90 units the heterozygote could be differentiated from the two homozygotes. Kalow and Staron have described an atypical pseudocholinesterase and have examined the mode of its inheritance, but it is not yet certain whether the homozygotes for the gene determining the abnormal enzyme correspond with the abnormal homozygotes of the present study. Earlier findings seemed to be consistent with the assumption that the inheritance of a recessive gene might be responsible for an idiopathic enzyme level less than 36 units. Allott and Thompson, however, described a family where the enzyme levels in three children and one sister of a male propositus were roughly halfway between those seen in the propositus and in his wife, who had normal levels. They suggested that a single gene might find expression in the phenotype. The normal plasma pseudocholinesterase level has a range of 61 to 120 units. A level within the 36 to 60 range in otherwise normal persons should therefore indicate the heterozygous state. Random findings of such people is unusual, but amongst 72 relatives of 14 propositi 16 of them were discovered (22%). The 72 relatives were composed of the propositi's children and grandchildren, their siblings with their children and grandchildren, and their parents with their siblings. Forty-three of the remaining 56 relatives showed values within the normal 61–120 range. This range might well be subdivided into one greater than 90 units definitely indicating the normal homozygous state, and another 61–90 units within which both normal homozygotes and heterozygotes could be found. The following states could then be presumed:

Less than 36 units = abnormal homozygotes
36–60 units = heterozygotes
61–90 units = heterozygotes and normal homozygotes
Greater than 90 units = normal homozygotes

If this assumption is correct no parents of children of persons with an enzyme level less than 36 units should show a level greater than 90 units, but some should be found amongst their siblings, the number depending on whether their parents were all heterozygotes or included some abnormal homozygotes. By studying the families of 14 propositi data were obtained which appeared to fulfil these conditions and which might allow the conclusion that the inheritance of the plasma pseudocholinesterase level is determined by two allelic genes, neither of which shows dominance.

A Blood Serum Test for Leukaemia and Neoplasia

D. Starke Murray and J. M. Daly (Group Laboratory, Kingston-on-Thames) reported a theory of the mechanism involved in leukaemia which postulates a flaw in D.N.A. production in the bone marrow through a histidine or imidazole compound. This would have the double action of removing zinc ions which normally inhibit D.N.A. synthesis and of acting directly as an accelerator of D.N.A. synthesis activity. A search for this substance revealed a compound, probably a conjugate since it can be detected only after hydrolysis in a hot alkaline solution (0.5 ml. serum boiled for 30 seconds in 1 ml. 20% NaCO₃), and which appears to be present in excess in neoplastic cases. It is measured by adding 2.5 ml. of diazo
reagent and reading with an Ilford filter 404 in the colorimeter against a blank prepared at the same time. No false negatives have been obtained and only two proven false positives out of 346 tests. The test appears very reliable as a diagnostic aid. The normal range is very narrow and distinct from abnormal. Highest “neoplastic” readings are found in myelogenous leukaemia; in lymphatic leukaemia these are not so high. In carcinoma the figure is always above normal but very variable although all carcinomas give a positive reading. It is possible that some other forms of malignancy do not give positive results, but sufficient cases have not yet been tested. The test is upset by raised urea figures, and false positives are probably related to liver damage. The test may prove to have a value in assessing the effect of different forms of treatment of the neoplastic diseases.

Association of Clinical Pathologists Broadsheets

The following broadsheets (new series) are published by the Association of Clinical Pathologists. They may be obtained from Mr. G. S. Corden, Swan Press Ltd., 20 Bakers Row, Farrington Road, London, E.C.1, price Is. each.

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