were engorged with iron-containing particles. Subsequently free particulate material disappeared from the lungs, but particle-containing macrophages were still present after 75 days. Initially there were minor inflammatory changes in lung tissue, with slight peribronchial oedema and epithelial thickening. Welding fume material was also observed in the gastrointestinal tract.

Concentrations of elements present in the welding fumes, such as iron, cobalt, chromium and antimony, were significantly increased in lung tissue. Concentrations of iron in lung tissue decreased slowly over 75 days, but cobalt, chromium and antimony concentrations decreased more rapidly, probably by elution from retained particles. Cobalt levels in liver tissue were significantly increased after 24 h. These results suggest the possibility that tissues in the vicinity of inhaled welding fume deposits may be exposed to high local concentrations of some toxic elements. This may be of interest in relation to the aetiology of respiratory distress experienced by workers exposed occupationally to welding fumes.

**Toxicological evaluation of surgical dressings**

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The widespread use of plastic materials in medical and veterinary practice has necessitated the development of some methods for evaluating their safety in use. One such use of these substances is in the adhesive coating of new types of non-woven surgical dressing products with polymeric substances. Such additives and other materials added to aid fluid absorbtion could, if transferred from fabric to patient, give rise to adverse effects. A method has been developed whereby the extractable compounds are eluted from the fabric sample under reproducible extraction conditions and subjected to chemical analysis and animal toxicological studies. The extraction method will be demonstrated and consists basically of a syringe barrel fitted into a constant temperature water bath ; the compressed air ram which operates the piston exerts a pressure on the fabric saturated with a fixed volume of solvent. The resulting extract is collected and examined using suitable test systems.

**The effect of experimental conditions on total urinary catecholamine excretion in the rat**

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Measurements of urinary catecholamine levels have frequently been used as a measure of sympathetic nervous activity in experimental animals. The metabolites of these substances are, however, excreted in much greater quantities ; the major metabolite in rats, hydroxymethoxyphenylglycol (H.M.P.G.) constitutes approximately 80% of the total excretion of catecholamines and metabolites (Ceasar, Ruthven & Sandler, 1969 ; Shum, Johnson & Flattery, 1971). In both of these studies urine was collected from male Wistar rats, in the former case maintained isolated and fasted, whilst in the latter case groups of four animals were used.

In our studies male C.S.E. rats 170–270 g were used ; analysis was made of total conjugated adrenaline and noradrenaline, using the methods of Anton & Sayre (1962) and Moelottke & Sloan (1970).

Total normetadrenaline and metadrenaline (Anton & Sayre, 1966) and H.M.P.G. and hydroxymethoxymandelic acid (Ceasar, Ruthven & Sandler, 1969) was also carried out including standards for each substance, on two 24 h samples of urine, collected using glass metabolism cages (26 cm diameter Metabowl).

Differences in the urinary excretion of catecholamines will be demonstrated in groups of rats maintained under different experimental conditions.

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## Estimation of angiotensin II blood concentrations in hypertensive rats

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Many factors are thought to be associated with the development and maintenance of experimental hypertension. Previous work in these laboratories (Finch & Leach, 1970) has been concerned with the role of the sympathetic nervous system in renal and metacorticoid hypertension in the rat, and present studies are concerned with the role of vasoactive substances in experimental hypertension. In particular, the levels of circulating angiotensin II are being investigated using the superfused isolated organ technique (Vane, 1964 ; Regoli & Vane, 1964). Angiotensin II estimation using the isolated ascending colon of the rat, is made with the preparation bathed with either blood or Krebs solution. In the case of blood superfused organs, modifications have been made to the original procedure to deal with the small blood volumes available in the rat.

Fluid is pumped by a Watson-Marlow flow-inducer, at a rate of 2 ml/min via polythene tubing (Portex Ltd.) of internal diameter 0·584 mm $\pm$ 0·076 mm ; total extracorporeal volume of the tubing, including that in the pump, is not greater than 1 ml. The whole system is cleaned and sterilized by the method described by Vane (1971).

Angiotensin II in large rats (>200 g) is assayed by superfusing the tissue directly with the rat's blood. The assay tissue is washed with Krebs solution prior to switching to blood superfusion through a three way tap.

Calibration of the tissue responses is achieved by infusion of angiotensin II solution into the superfusing blood obtained from an acutely, bilaterally nephrectomized anaesthetized rat, and therefore, free of naturally occurring angiotensin II.