seen at 1580 cm^{-1} in the complex might arise from a unionized NH_2 group. The identification of the COOH bands at 1730 cm^{-1} etc., has been made by examining the spectra of amino-acid hydrochlorides which have the structure COOH RCO NH_3^+ Cl^-[6].

The following changes are observed for the complexed chloranil. The carbonyl band appearing in the free form at 1695 cm^{-1} is shifted to 1630 cm^{-1}, the C=C stretch band at 1575 cm^{-1} disappears. Identical behaviour has been observed in the IR-spectrum of the charge-transfer complex formed between hydroquinone and chloranil[7].

The shift of the carbonyl band to 1630 cm^{-1} and the disappearance or shift of the C=C band is attributed to charge donation into an anti-bonding orbital of the chloranil which causes a lowering of bond order and hence of frequency of vibration of these bands. It has therefore been confirmed that the amino-acid chloranil complexes show all the features of the classic charge transfer complex including a colour change which is apparent in the solid complex but not in solution[3] where the concentration is very much lower and that the amino-acid is present in the complex in the non-zwitterionic form. These observations also explain the very slow formation of these complexes in solution as over the pH range used, 4 to 9, the amount of unionized amino group present in equilibrium is very small indeed. The fact that interaction rate increases as the pH increases is also explained by the presence of more unionized amino group at the higher pH[4].

The bonding in the amino-acid complexes must arise from the donation of a lone-pair (n) electron on the nitrogen of the unionized amino group to the chloranil. Particularly interesting is the fact that the 2 aromatic amino-acids, tryptophan and tyrosin behave as n-electron donors and not as π-electron donors. This might have interesting biological consequences as lone-pair (n) electrons are fairly widely distributed in biological systems and in considering electron transport or charge transfer systems, lone pair electrons should not be excluded as possible partcipants, even in the near vicinity of π-electrons.

Zusammenfassung. Die IR-Spektren der Komplexe von Aminosäure mit Chloranil zeigen, dass zwischen Aminosäure in einer nicht zwitterionischen Form und Chloranil klassische Elektronen-Donator-Akzeptor-Komplexe gebildet werden. Die aromatischen Aminosäuren wirken als n-Elektronendonatoren.

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Renal Medullary Hypertonicity and the Pathogenesis of Acute Renal Failure

The present study was designed to evaluate a possible role of renal medullary hyperosmolarity in the development of acute renal failure as produced in rats by the injection of human methemoglobin and sodium ferrocyanide[1].

Diuresis was induced in 160–200 g male CFE rats by replacing their drinking water with 5% glucose solution. Pigment nephropathy was induced by the method of MASON et al.[1]. 48 h after methemoglobin and ferrocyanide injection the left kidney of each rat was weighed. The weights of diuresed and of control animals were compared using the student t-test to evaluate statistical significance.

In the first experiment rats were diuresed for 5 days before induction of the renal lesion. The resulting data are in Table I. In a second series, groups of rats were severally diuresed for 12, 48, 72 and 120 h before injection with methemoglobin-ferrocyanide solution. Representative data are in Table III. Histological sections of kidneys were examined.

Rats treated with sustained diuresis had significantly lighter kidneys than did control animals (Tables I and II). All rats given 5% glucose to drink showed high urine output (Table III) but kidney enlargement characterized all groups not treated with sustained diuresis. Increase in kidney weight caused by methemoglobin-ferrocyanide injection correlates closely with impairment of renal function and with the degree of histologically demonstrable renal damage[2]. Also, the heavy kidneys showed extensive cast formation, interstitial round cell infiltration and tubular hydropic changes. These data therefore show that prolonged diuresis, rather than diuresis per se, is necessary to protect rats from developing pigment nephropathy, and indicate that rate of urine was not the protecting factor.

LEVITIN et al.[3] described a continuing fall in renal medullary osmolarity during a 5-day diuresis in dogs drinking 5% glucose and water. Parallel changes in rats diuresed for 5 days have been described by MORARD[4] in the acid mucopolysaccharides of the renal medulla which show increased polymerization with a decreased negative charge. This is believed to decrease the accumulation in the medullary interstitium of cations necessary for the formation of the intrarenal osmotic gradient. PARRY et al.[5] were able to prevent experimental pigment nephrosis in rats with mannitol. Mannitol diuresis decreases medullary osmolarity[6]. TESCHAN and LAWSON[7]

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Table I

Experiment	No. of rats	Treatment	Kidney weight (mg)	No. of lesions
1	5	G/W[a]	1229 ± 49.6[b]	1/5
	5	C	1914 ± 85.5	4/5
2	5	G/W	1617 ± 57.7	1/5
	6	C	1930 ± 91.8	4/6
3	6	G/W	876 ± 74.2	1/6
	6	C	1132 ± 31.1	5/6

[a] Glucose and water diuresis. [b] ± S.E.

Table II

Experiment	No. of rats	Diuresis (days)	Daily urine output/rat (ml)	Kidney weight (mg/100 g body weight)	No. of lesions
1	6	6	34.7	490.7	1/6
	6	–	5.9	390.1	5/6
2	5	3	25.6	620.3	4/5
	4	–	8.8	596.6	3/4

observed that ethacrynic acid did not induce diuresis but prevented the establishment of pigment nephrosis in rats. BEYER et al.[8] found that ethacrynic acid in the rat had no significant effect on the excretion of sodium, potassium, chloride, or water, but Dow and IRVINE[9] demonstrated that ethacrynic acid sharply reduced the concentration of sodium and urea in the rat renal medulla.

In brief, chronic diuresis, mannitol, and ethacrynic acid all decrease renal medullary osmolarity and all inhibit the experimental induction of acute renal failure. It is therefore concluded that a high solute concentration in the renal medulla facilitates the development of acute renal failure whereas a decrease or removal of the medullary osmotic gradient protects the kidney from injury.

Table III

Experiment	No. of rats	Diuresis (days)	Daily urine output/rat (ml)	Kidney weight (mg ± S.E.)	No. of lesions	*P*
E_1	5	0.5	7.5	1575 ± 51.5	4/5	0.1
	4	–	6.7	1785 ± 62.3	4/4	
S_3	6	2.0	21.5	1066 ± 71.2	5/6	0.1
	6	–	5.6	1170 ± 83.4	4/6	
S_4	6	2.0	24.1	1136 ± 61.6	6/6	0.1
	5	–	8.3	1202 ± 60.3	6/6	
S_6	6	5.0	24.5	1157 ± 70.9	5/6	0.1
	6	3.0	25.3	1160 ± 77.3	6/6	0.1
	6	–	6.7	1049 ± 92.3	4/6	
S_7	5	7.0	25.3	838 ± 31.1	1/5	0.01
	6	3.0	23.6	912 ± 22.4	5/6	0.1
	6	–	5.9	1002 ± 51.1	5/6	

Résumé. Des rats furent protégés d'une insuffisance rénal aïgue par une diurèse aqueuse chronique plutôt qu'intense. L'hypotonicité de la moelle rénale est considerée comme un facteur significatif dans le développement de la détérioration rénale.

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Influence de différents types de diurèse sur l'excrétion rénale de l'urée étudiée chez le rat, à l'aide des microponctions rénales[1]

La conception classique de l'excrétion rénale de l'urée[2], faisant intervenir une filtration glomérulaire de la substance, suivie d'une réabsorption tubulaire passive, ne s'est pas révélée exacte dans toutes les conditions expérimentales. Ainsi, plusieurs groupes de chercheurs[3,4] ont pu montrer qu'une partie de l'urée filtrée est réabsorbée activement le long des tubes collecteurs de mammifères soumis à une diète hypoprotéique.

Les résultats rapportés ici décrivent le sort intrarénal de l'urée au cours de différents types de diurèse, et suggèrent la possibilité de transports actifs d'urée dans la direction «sécrétoire». Les expériences ont été réalisées, chez le rat anesthésié au pentobarbital, par microponction des parties contournées des néphrons de surface. Des travaux récents[5,6] semblent établir que la filtration glo-

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