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Cortical Oxygen Pressure during Acute Venous Kidney Obstruction

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Summary. Different degrees of obstruction to the renal venous drainage were produced in rabbits, and the cortical oxygen pressures in the kidney measured with the multiple-wire surface electrode of Kessler and Luebbers. Sudden complete occlusion of the inferior vena cava (IVC) above the renal vein, or above and below the renal vein simultaneously, produced a slight decrease of about 27%. Sudden complete occlusion of the renal vein itself caused a severe decrease of 67% (obstruction near the IVC) or 100% (obstruction near the kidney). One hour later different degrees of incomplete recovery were found. The PO_2 curves for the renal cortex revealed different pressure-dependent pathophysiological changes in the microcirculation. The multiple-wire surface electrode may well prove to be of use during renal surgery in which venous obstruction is a critical factor.

Key words: Renal venous obstruction, Intrarenal pressure, Microcirculation, Cortical oxygen pressure, Multiple-wire surface electrode, Kidney surgery.

Introduction

Kidney disease resulting from acute obstruction of the venous drainage is not particularly common, but when it does occur the structure and function of the organ can be rapidly destroyed. Likely causes of such a condition include thrombosis, tumour, injury, vascular disease and surgery. The site, spread and rate of spread of the lesion on the one hand, and the ability of the system to compensate on the other, together determine the clinical picture.

It is particularly during the surgical treatment of carcinoma of the kidney, following the rupture of a renal vein or the IVC, or during the resection of an aortic aneurysm, that the surgeon is compelled to ligate one of the major veins of the system draining the kidney, with possible far-reaching effects on the blood-supply of the organ itself. The further progress of the condition is usually impossible to follow, owing to the lack of suitable tests, and the construction of a vascular shunt may be attended by considerable risk. To enable rational treatment to be carried out, there is a pressing need for some method of obtaining the requisite physiological data during the operation and with minimal disturbance of the organ itself.

Quite recently several research teams have succeeded in developing direct methods for obtaining the significant parameters of cellular respiration in a highly localised region, with maximum accuracy and without causing tissue damage. One example of such a probe is the multiple-wire surface electrode (Mehrdraht-Oberflächenelektrode) of Kessler and Luebbers. The construction of this apparatus and its practical use on a number of organs has been fully described elsewhere [14–17].

Sinagowitz and his co-workers have investigated the relationship between the microcirculation and oxygen supply in cases of acute arterial ischaemia of short duration and in the presence of acute and chronic retention of urine. In the light of these results, they have been able to use these probes for the first time on the human kidney, both as a help in deciding during operation on the desirability of nephrectomy and for assessing the function of a transplanted kidney [30].

Material and Methods

Experiments were carried out under standard laboratory conditions on female rabbits between 5 and 7 months old, and with an average body weight of 3,650 g. The animals were anaesthetised for between 4 and 8 h with pentobarbitone (Nembutal®); 6.5 mg/1,000 g wt. The kidneys were mobilised through a longitudinal abdominal incision and the fatty capsule laid open, the fibrous capsule and renal parenchyma being left undisturbed. Loss of water, salt and energy was replaced by infusion, and heat loss prevented by laying the animal on non-conducting material and using an infra-red beam.

The level, wet anterior surface of the kidney supported the multiple-wire surface electrode of Kessler and Luebbers satisfactorily. It weighed only 1.2 g, extended over 1.8 cm^2 of the surface and thus exerted a pressure of 0.7 g/cm^2. It therefore caused no distortion of the tissue, which might render the readings unreliable. The probe

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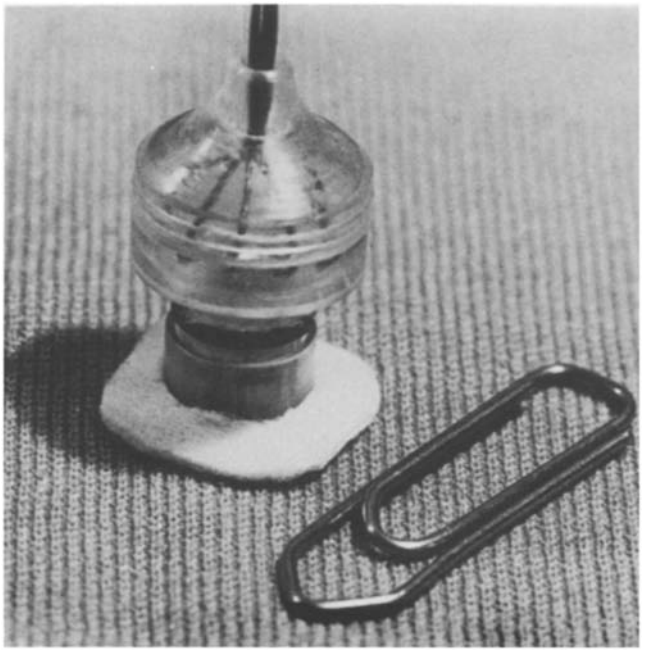

Fig. 1. The multiple-wire surface electrode

contained eight separate platinum wires, 15 μ in diameter and each surrounded by a Teflon membrane, 14 μ thick.

Oxygen molecules penetrated the adhesive capillary head-plate and produced an electric current. The hemispherical area of oxygen uptake had a diameter ranging from 20 to 25 μ; i.e. it corresponded to the size of a single cell. Data received simultaneously from different regions allowed a curve to be constructed which showed the oxygen gradient throughout the tissue (Fig. 1).

After computerised readings had been taken from the normal kidney, the junction of the renal veins with the IVC was atraumatically exposed. Sites for occlusion were chosen, both for their clinical significance, and because they were so disposed that their selective occlusion allowed varying amounts of the collateral circulation to remain open and produce an ever increasing difference in pressure together with an ever decreasing flow of blood. Occlusion was produced within 30 s with Impralen 3/0. The partial pressure of oxygen in the cortex was monitored, and a reading recorded as soon as it became stabilised.

Results

Sudden Complete Occlusion of the IVC Above the Renal Veins (Grade I)

This allowed the collateral drainage of both kidneys to remain open. The PO_2 curves showed a decrease of about 26.4%. The curve became less steep and was displaced towards the left, the values remaining normally distributed. There was no difference between the two sides. After the vessel had been kept occluded for one hour the obstruction was suddenly removed. The partial pressure of oxygen in the cortex rose to less than its original level, the difference being about 9.1% (see Fig. 2).

Sudden Complete Occlusion of the IVC Above and Below the Renal Veins (Grade II)

This occluded the remaining part of the IVC, but the partial pressure curve changed in the way described above. The values fell on average about 27.4% and normal distribution was preserved. After 1 h blood-flow was again restored, but the oxygen pressure did not return to normal and remained twice as low as in the corresponding case described above (see Fig. 3).

Sudden Complete Occlusion of the Renal Vein Near the IVC (Grade III)

The result here depended on the side chosen. On the right side, only the ureteric and capsular veins remained open;

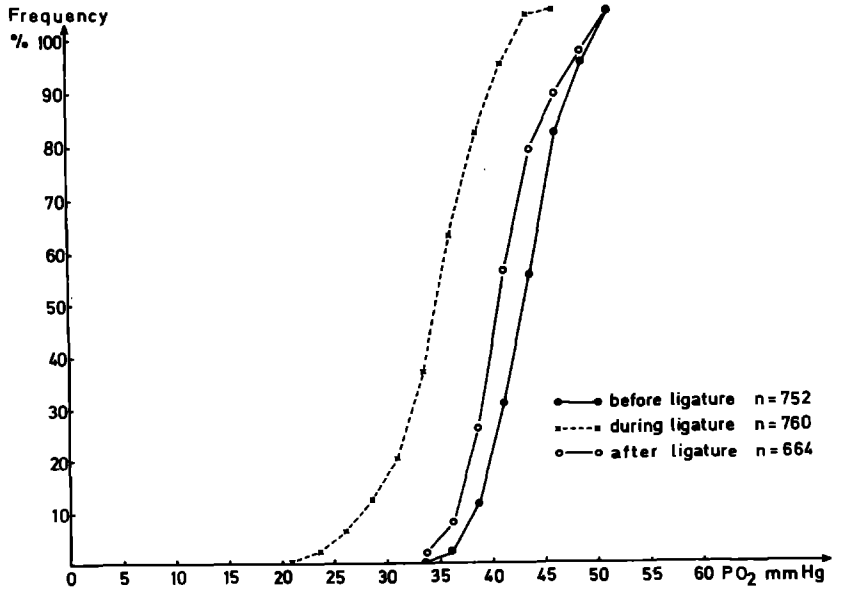

Fig. 2. Grade I. Occlusion of IVC above renal veins. PO_2 histograms of the kidney in cumulative representation

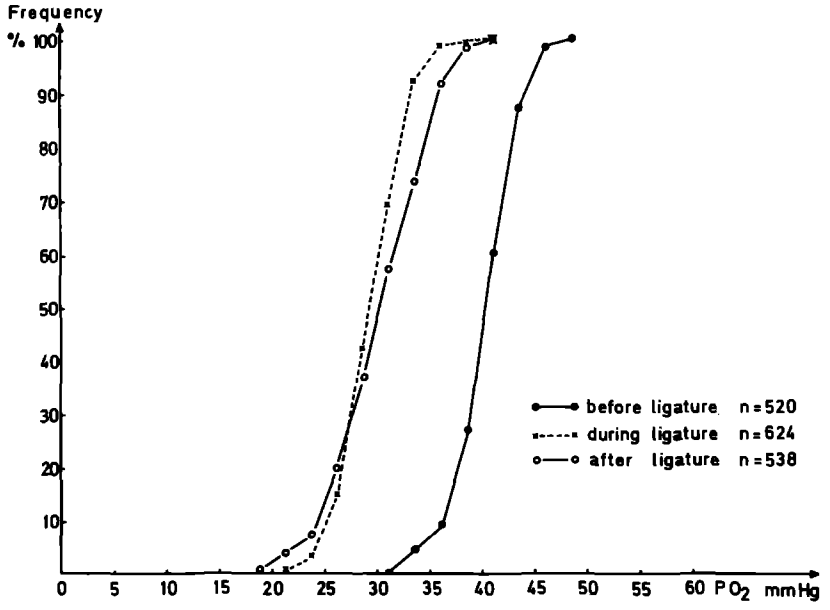

Fig. 3. Grade II. Occlusion of IVC above and below renal veins. PO_2 histograms of the kidney in cumulative representation

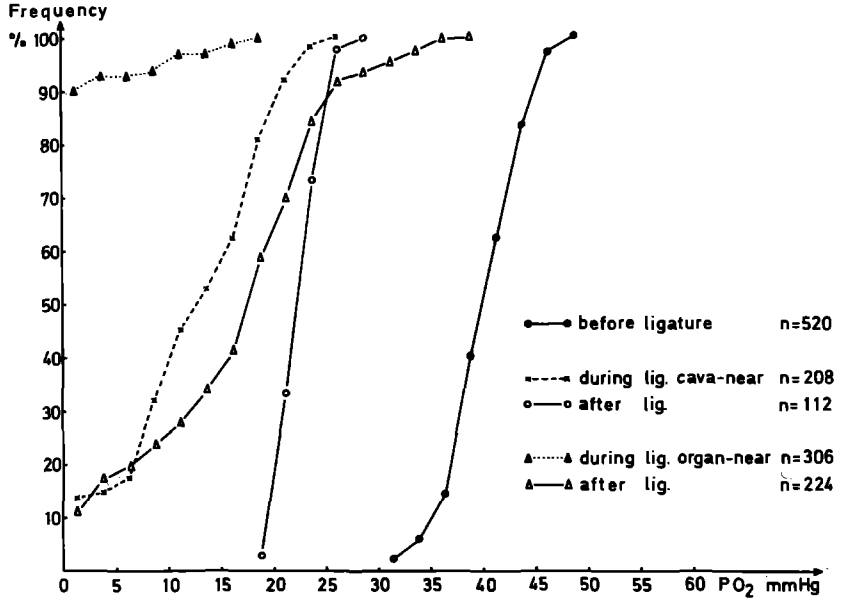

Fig. 4. Grade III and IV. Occlusion of renal vein (III) close to the IVC, (IV) close to the kidney. PO_2 histograms of the kidney in cumulative representation

but on the left side the veins from the ovary and adrenal were also available. The curve was displaced to the left and its slope reduced. The values decreased (on average) by 66.85%, when a critical point was reached. There was no difference between the two sides. When the circulation was restored after 1 h the values increased again to 25.63%, producing a smooth curve but on a very low general level (see Fig. 4).

Sudden Complete Occlusion of the Renal Vein Near the Kidney (Grade IV)

This procedure excluded all collaterals save a few capsular veins and lymphatics. Within a few seconds the value fell to zero. After 1 h of ischaemia the partial pressures rose again only to 41.55% and the distribution remained non-uniform (see Fig. 4).

Discussion

The physiological and pathological changes in the kidneys produced by an increase in venous pressure are more pronounced than those that follow an equivalent decrease in the arterial supply [6, 13, 29, 37]. This is probably due to the fact that relatively small changes in venous pressure occur under natural conditions, so that there has not been the same evolutionary need to develop a compensatory mechanism, and the capacity for automatically regulating the venous pressure is correspondingly less. On the arterial side, however, the capillary pressure in the glomerulus is kept

constant by preglomerular vasoconstriction, even though the systemic pressure varies between 80 and 180 mm Hg [2, 8, 19, 34]. Venous obstruction, on the other hand, produces a rise in pressure extending to the peritubular capillaries and the lymphatics. The rise of pressure in each of these regions is linearly related to the degree of obstruction [2, 4, 9, 32, 33]. Furthermore, owing to the decreased resistance resulting from preglomerular vasodilatation, the overall blood-flow and the effective filtration pressure are not affected by moderate degrees of venous obstruction [2, 18, 20, 35].

Nevertheless, significant changes can occur during this stage of moderate obstruction. There is a redistribution of blood between the outer and inner layers of the cortex, probably brought about by such hormonal agents as renin-angiotensin and prostaglandin [1, 11, 12, 18, 20]. This is possible because the length of the capillaries is not constant throughout the cortex. The length of the liver sinusoid, it may be remarked in passing, has been shown to be normally distributed [31]. In addition to this, the capillary blood-flow is not uniform. Both factors account for the different oxygen tensions which can occur in the venous blood, and which can be utilised under pathological conditions as a reserve supply for cellular respiration if the microcirculation redistributes the blood [14–17].

A further result of increased intrarenal pressure is the retention of sodium and water, with the increased absorption in turn producing extra oxygen consumption [7, 21]. The pressure gradient between tubules and capillaries will be reduced or abolished altogether, with a corresponding increase in the rate of absorption. Tubular permeability is raised and oedema (with a high sodium content) accumulates in the neighbourhood [3, 5, 10–12, 18, 20, 22, 27, 36].

With more severe obstruction, if the transmural pressure difference lies below 80 mm Hg, circulation through the entire kidney will also be reduced and the effective filteration pressure lowered or abolished. Not only sodium but also potassium – coming mainly from the damaged cells of the tubules – accumulates in the tissue [2, 5, 10, 13, 18, 22].

From the haemodynamic point of view, this phase of severe obstruction is characterised by 'low-flow' or 'no-flow' anoxia. For a short time the energy required for basal metabolic turnover can be supplied from the anaerobic breakdown of glucose, provided that acid catabolites are carried away by a minimal microcirculation [14–17]. This is seldom possible following sudden complete occlusion of the renal veins. The condition worsens as a result of intravascular haemoconcentration and interstitial oedema, both of which further impair the microcirculation [23–26, 28].

We can summarise our own experimental work on acute obstruction of the renal veins in the following way.

1. Changes in the partial oxygen pressure in the renal cortex in the presence of an occlusion of Grade I or Grade II are brought about by the centripetal redistribution of blood within the kidney and the increased consumption of energy used for the absorption of sodium. Severe disturbance of the oxygen supply and microcirculation does not occur.
2. Corresponding changes in the cortex in the presence of an occlusion of Grade III or Grade IV are produced by ischaemia of haemodynamic origin, intravascular haemoconcentration and interstitial oedema. A severe disturbance of cellular respiration is to be expected.

In addition we wish to draw attention to a sensitive method of measuring hypoxia in the kidney brought about by venous obstruction. This method can also be of great help in supporting decisions that have to be made in the theatre.

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