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The Structure of the Juxtaglomerular Apparatus in Addison's Disease, Bartter's Syndrome, and in Conn's Syndrome

A Comparative, Morphometric, Light Microscopic Study on Serial Sections*

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Summary. Continuing and supplementing previous morphometric studies on the juxtaglomerular apparatus (JGA) of normal kidneys we have now investigated semi-thin serial sections of each 10 hyperplastic and hypertrophied JGAs in Addison's disease and in Bartter's syndrome, as well as 8 atrophic JGAs in Conn's syndrome. With the exception of Bartter's syndrome, where in only two out of ten JGAs the efferent arteriole, and in none of them the afferent arteriole touches immediately the macula densa, there is an almost regular direct contact between the hilar arterioles and the macula densa like in normal kidneys. The Goormaghtigh cell field invariably touches the macula densa. In Bartter's syndrome, but not in Addison's disease, a considerable enlargement of the macula densa was measured, associated with an exceptional enlargement of the Goormaghtigh cell field. In all cases examined here about 40–60% of the basal area of the macula densa do not have any direct contact with other structures forming the JGA.

Key words: Juxtaglomerular apparatus — Macula densa basal area — Juxtaglomerular contact areas — Addison's disease — Bartter's syndrome — Conn's syndrome.

From literature we know that the juxtaglomerular apparatus (JGA) of the human kidney is enlarged under various conditions as in Addison's disease (Alexander, 1968; Meyer, 1972) and in Bartter's syndrome (Bartter et al., 1962; Bryan et al., 1966; Brackett et al., 1968; Goodman et al., 1969; Wegmann, 1970; Sutherland et al., 1970; Schmidt et al., 1973; Ramanathan et al., 1973), but atrophic in Conn's syndrome (Conn et al., 1965; Cohen et al., 1965; Bohle et al., 1969; Wegmann, 1970; Meyer, 1972). We do not know, however, if all or which partial structures of the JGA take part in this enlargement or diminution, and if the size of their different contact areas with eachother will be changed in comparison with the normal JGA (Christensen et al., 1975). We think it is necessary to find an answer to these questions, for a better understanding of the function of the JGA.

Material and Methods

Renal tissue of the following cases has been studied:

1. A 59-year-old female, who a few hours after hospitalization, died in Addison's crisis. The only attainable clinical data were: RR 80/60 mmHg, serum-sodium 124 mval/l, serum-potassium 3.9 mval/l, serum-chloride 96 mval/l, and serum-calcium 4.2 mval/l. One kidney was removed one hour after death and used for this study. At autopsy we found a bilateral cortical atrophy of the adrenals.

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2. A 41-year-old female suffering from Bartter's syndrome, was known to have hypokaliaemia of 2.0 to 2.8 mval K/l and normonatriaemia of 138 to 141 mval Na/l for two years. The arterial blood pressure was subnormal (80/50 to 95/55 mmHg), the urinary aldosteron excretion slightly increased to 28 mg/day, and the renal plasma renin activity was about 20 times higher than normal in both renal veins (for further details see Schmidt et al., 1973). For histological examination we received a renal wedge biopsy.

3. A 48-year-old male with Conn's syndrome. The clinical data were: Systolic blood pressure 235 to 290 mmHg, diastolic blood pressure 120 to 135 mmHg, serum-sodium 142 meq/l, serum potassium 3.1 meq/l, serum chloride 105 meq/l, serum creatinine 1.33 mg%, endogenous creatinine clearance 93 ml/min. Ophthalmological: Fundus hypertonicus. A wedge biopsy of one kidney was made when one half of both adrenals, showing a diffuse micronodular cortical hyperplasia, was removed surgically.

The renal tissue of these three cases was prefixed in 4% formaldehyd, refixed in OsO_4, embedded in plexiglass and cut to 1.9 μ thick serial sections on a Reichert-Ultramikrotom type OmU2. Each series of 10 JGAs of Addison's disease, 10 JGAs of Bartter's syndrome and 8 JGAs of Conn's syndrome consisted of about 100 Giemsa-stained sections. Of all of the sections we took microphotographs at a final magnification of 370:1 which served for measuring the lengths of contact between the various structures of each of the serially sectioned JGAs; i.e.

a) between macula densa and Goormaghtigh cells,

b) between macula densa and afferent arteriole,

c) between macula densa and efferent arteriole,

d) between Goormaghtigh cells and afferent arteriole,

e) between Goormaghtigh cells and efferent arteriole.

Additionally, in all sections the length of the basement membrane of the macula densa was measured. From these data we computed the basal areas of the maculae densae and the contact areas between the various structures of the JGAs.

To find significant correlations, we used Spearman's rank correlation (level of significance $p = 0.05$).

Definitions

In agreement with Zimmermann (1933) and Goormaghtigh (1937) we consider the *macula densa* to be that part of the distal renal tubulus which is characterized by slender, high-prismatic epithelial cells with closely packed nuclei, and situated adjacent to the hilar region of the glomerulus. Using semi-thin serial sections one can clearly differentiate the macula densa cells from the neighbouring distal tubular cells.

We designate that hilar *arteriole as afferent* whose course we could undoubtedly trace back to its origin from an interlobular artery.

As described by Goormaghtigh himself (1932), the Goormaghtigh-cells characteristically have scanty cytoplasma, spindle-shaped nuclei with dense chromatine, and scarcely visible cell borders. They are situated between the walls of the two hilar arterioles and the macula densa, and we call the totality of these cells the Goormaghtigh cell field.

Epithelioid cells are roundish cells with abundant, hardly staining cytoplasma sometimes containing dark secretory granules, and with spherical, tenuously structured nuclei, thus imitating epithelial cells. They are frequently found in the wall of the afferent, and less often in the wall of the efferent arterioles or within the Goormaghtigh cell field. Using Giemsa-stained semi-thin serial sections, one can clearly distinguish the epitheloid cells from the darker, spindle-shaped smooth muscle cells of the arteriolar walls.

The macula densa is sharply separated from the Goormaghtigh cells by the tubular basement membrane. For an exact definition of the borderline between the cellular walls of the hilar arterioles and the Goormaghtigh cells it is necessary to have series of semi-thin sections. Finally, we considered an imaginary line across the hilar region of the glomerulus at the height of transition from the parietal to the visceral epithelial layer of Bowman's capsule to be the limit-line between the Goormaghtigh cell field and the glomerular mesangium.

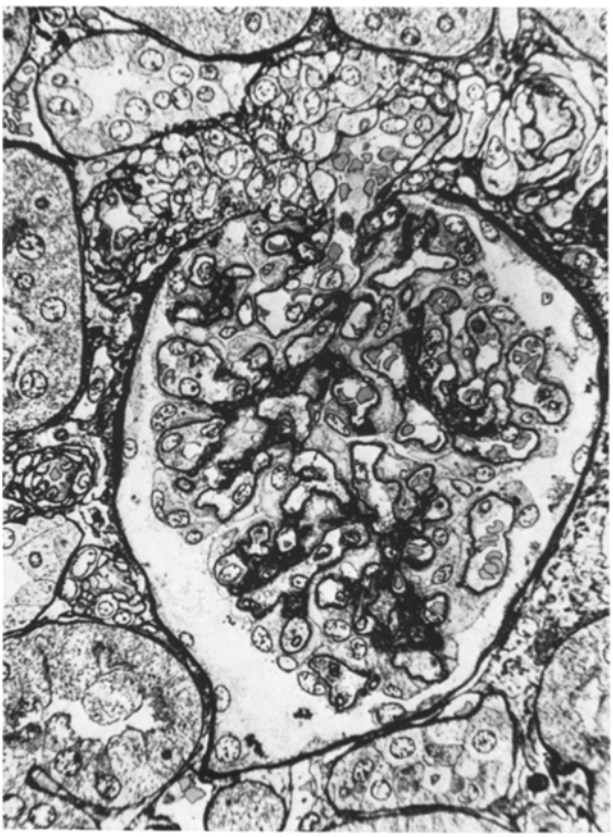

Fig. 1. Glomerulus in Morbus Addison with many epithelioid cells in a hyperplastic JGA between the afferent arteriole (right), the efferent arteriole (left) and the MD (above the efferent arteriole). 1 μ, Movat's silver impregnation, 460:1 (S 137/67)

Results

1. Addison's Disease

In comparison with normal human kidneys the JGA in Addison's disease was enlarged, mainly due to a transformation of nearly all Goormaghtigh cells into epithelioid cells (Fig. 1). Additionally a great number of epithelioid cells were visible within the muscular layer of afferent arterioles, and sometimes of efferent arterioles, too. The basal area of the macula densa averaged 5,700 μ^2, thus being only slightly increased as compared to the mean basal area of the macula densa in a normal kidney which we found to measure about 5,300 μ^2 (Christensen et al., 1975).

Out of the 10 JGAs of the kidney examined here, the macula densa had a direct contact with the afferent arteriole in 7 cases, and with the efferent arteriole in 6 cases, while the Goormaghtigh cell field was in direct contact with the macula densa, the afferent and the efferent arterioles in all 10 JGAs. The mean sizes of the areas of contact between the various structures of the JGA

Table 1. Size (mean ± S.E.) of the macula densa basal area and of the areas of direct contact between the structures of the JGA in two normal kidneys ([a]values according to Christensen, 1974) and one case each of Addison's disease, Bartter's syndrome and Conn's syndrome. n = number of corresponding measured areas

	Normal kidneys[a] (μ^2)	Addison's disease (μ^2)	Bartter's syndrome (μ^2)	Conn's syndrome (μ^2)
Macula densa basal area	5,298 ± 208 ($n = 43$)	5,728 ± 665 ($n = 10$)	8,154 ± 1300 ($n = 10$)	2,882 ± 394 ($n = 8$)
Area of contact between:				
a) Goormaghtigh cell field and macula densa	2,067 ± 117 ($n = 43$)	2,918 ± 357 ($n = 10$)	4,734 ± 815 ($n = 10$)	421 ± 110 ($n = 8$)
afferent arteriole	1,053 ± 70 ($n = 43$)	1,772 ± 238 ($n = 10$)	2,983 ± 475 ($n = 10$)	271 ± 82 ($n = 7$)
efferent arteriole	599 ± 42 ($n = 42$)	885 ± 147 ($n = 10$)	1,803 ± 384 ($n = 10$)	227 ± 56 ($n = 6$)
b) Macula densa and afferent arteriole	605 ± 60 ($n = 42$)	263 ± 103 ($n = 7$)	0 ($n = 0$)	418 ± 168 ($n = 7$)
efferent arteriole	266 ± 33 ($n = 41$)	378 ± 127 ($n = 6$)	155 and 565 ($n = 2$)	253 ± 79 ($n = 6$)

in Addison's disease are listed in Table 1. Contrary to the normal kidney (Christensen, 1974) no correlation between the size of the areas of contact with each other could be stated statistically, neither did the size of the basal area of the maculae densae correlate to the direct contact areas between the maculae densae and the associated Goormaghtigh cell fields (Table 2).

About 63% of the basal area of the macula densa were in direct contact with other structures of the JGA, 51% of which were in contact with the Goormaghtigh cell field (Table 3).

2. Bartter's Syndrome

As in Addison's disease the JGAs were strikingly enlarged (Fig. 2). The Goormaghtigh cell fields consisted of a great number of small Goormaghtigh cells as well as epithelioid cells occasionally containing secretory granules, the ratio of both cell types varying widely in the individual JGAs. In Bartter's syndrome the macula densa was extremely increased and revealed a mean basal area of about 8,100 μ^2, i.e. an enlargement of nearly 53% compared with a normal macula densa.

In contrast with Addison's disease no increase of epithelioid cells in the walls of the hilar arterioles was registered. None of the 10 serially sectioned JGAs showed a direct contact between the macula densa and the afferent arteriole, and only in 2 cases the efferent arteriole touched the macula densa directly (Table 1). The areas of contact between the macula densa and the Goormaghtigh cell field, as well as between the latter and both hilar arterioles were larger than in normal kidneys and occurred in all 10 JGAs (Table 1).

Table 2. Results of significance according to Spearman's rank correlation coefficient for the correlation of the structures of the JGA being in direct contact with each other. Level of significance $p = 0.05$. N.S. = no significance. [a] values according to Christensen, 1974

	Normal kidneys[a] (43 JGA)	Addison's disease (10 JGA)	Bartter's syndrome (10 JGA)	Conn's syndrome (8 JGA)
1. Macula densa basal area correlated to:				
a) the area of contact between Goormaghtigh cell field and macula densa	$p < 0.0005$	N.S.	$p < 0.005$	N.S.
b) the area of contact between Goormaghtigh cell field and efferent arteriole	$p < 0.025$	N.S.	$p < 0.005$	N.S.
c) the area of contact between Goormaghtigh cell field and efferent arteriole	N.S.	N.S.	N.S.	N.S.
2. Contact area between macula densa and Goormaghtigh cell field correlated to:				
a) the area of contact between Goormaghtigh cell field and afferent arteriole	$p < 0.005$	N.S.	$p < 0.005$	N.S.
b) the area of contact between Goormaghtigh cell field and efferent arteriole	N.S.	N.S.	N.S.	N.S.

Table 3. Mean percentages of the maculae densae basal areas which are in a direct contact with various structures of the JGA in two normal kidneys, and one case each of Addison's disease, Bartter's syndrome and Conn's syndrome

Mean macula densa basal area (100%) in contact with:	Goormaghtigh cell field (%)	Afferent arteriole (%)	Efferent arteriole (%)	Total percentage of direct contact (%)
a) normal kidneys	39	11	5	55
b) Addison's disease	51	5	7	63
c) Bartter's syndrome	58	0	4	62
d) Conn's syndrome	15	15	9	39

As proved by statistical analysis of the data obtained, there was a positive, linear correlation between the size of the basal areas of the maculae densae and the areas of contact of the corresponding Goormaghtigh cell fields with the adjacent maculae densae as well as with the afferent arterioles, but not with the efferent arterioles (Table 2). Thus the various structures of the JGAs in Bartter's syndrome relate to each other as in normal kidneys (Christensen et al., 1975).

Comparable to Addison's disease about 62% of the basal area of the macula densa were in direct contact with other structures of the JGA (Table 3).

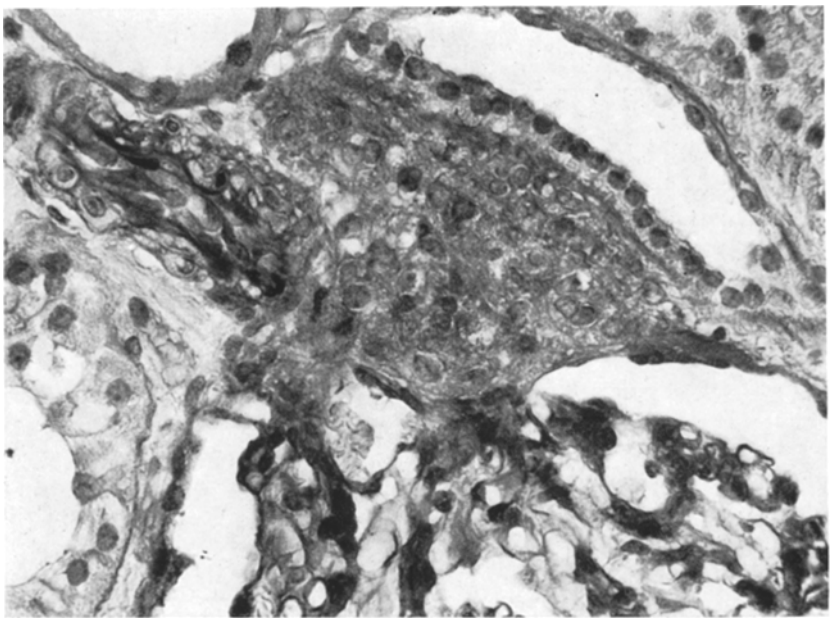

Fig. 2. Extremely hyperplastic JGA in Bartter's syndrome having pushed aside the afferent arteriole (left) from the likewise hyperplastic MD. 6 μ, PAS-staining, 440:1 (72/1/1572)

3. Conn's Syndrome

In this disease one finds not only an atrophy of the Goormaghtigh cell field, the macula densa, too, was remarkably reduced in size (Fig. 3). The mean size of the basal area of a macula densa in Conn's syndrome was about 2,900 μ^2, indicating a decrease of about 45% as compared to the macula densa of a normal kidney. The area of direct contact between the atrophic Goormaghtigh cell field and the macula densa which was present in all 8 JGAs measured about 420 μ^2 on an average, which means that less than 15% of the basal area of the macula densa were in a direct relation to the adjoining Goormaghtigh cells.

A direct contact was demonstrated between macula densa and the afferent arteriole in 7, and between macula densa and the efferent arteriole in 6 out of the 8 serially sectioned JGAs (Table 1).

Statistically, there was no correlation in the size of any particular structure of the JGA to be seen (Table 2).

About 40% of the basal area of the macula densa were in direct contact with the other structures of the JGA (Table 3).

Discussion

Our examinations of semi-thin serial sections of 10 JGAs in Addison's disease and Bartter's syndrome, as well as of 8 JGAs in Conn's syndrome confirm the results of other authors and our own previous observations: In Addison's disease (Alexander, 1968; Meyer, 1972) or following experimental bilateral adrenalectomy

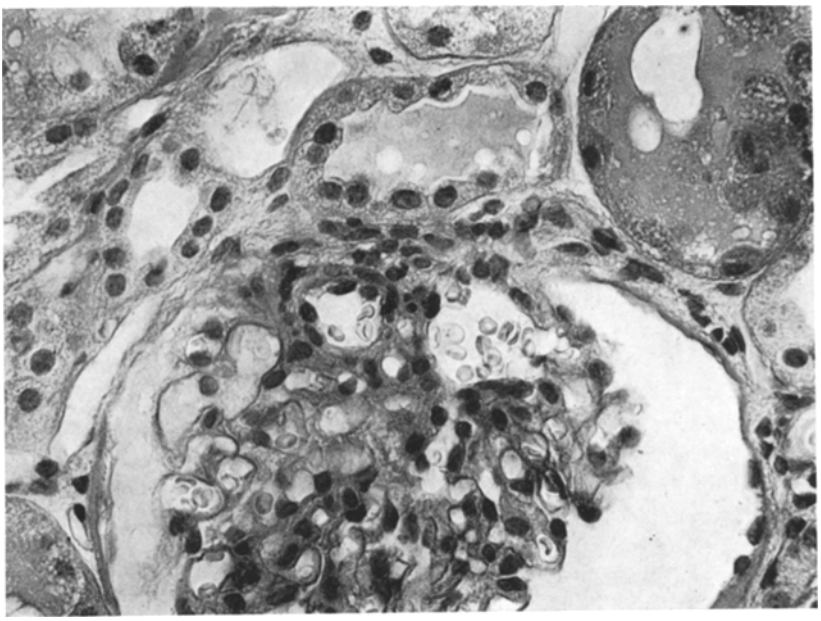

Fig. 3. Atrophic JGA and MD in Conn's syndrome. 6 μ, PAS-staining, 460:1 (67/2/1498)

(Dunihue, 1947; Bohle, 1954; Barajas and Latta, 1963) and in Bartter's syndrome (Bartter et al., 1962; Brackett et al., 1968; Goodman et al., 1969; Sutherland et al., 1970; Schmidt et al., 1973) the JGA is markedly increased, whereas it is diminished in Conn's syndrome (Conn et al., 1965; Cohen et al., 1965; Bohle et al., 1969; Meyer, 1972).

In the case of Addison's disease described here, the enlargement of the JGA is due mainly to the transformation of Goormaghtigh cells and mural cells of the hilar arterioles into epithelioid cells. Only in 2 out of the 10 scrutinized JGAs we could see a few remaining small Goormaghtigh cells. In a former planimetrical study of this kidney (Meyer, 1972) we had found the mean cut area of the juxtaglomerular cell complexes, comprising Goormaghtigh cells and epithelioid cells, to be 1,702 μ^2 in PAS-stained, 6 μ sections. This means an increase of about 85% in comparison with the mean size of the juxtaglomerular cell complexes in normal kidneys (mean $\pm$S.D. = 919 $\pm$ 54 μ^2).

The obviously increased JGAs in Bartter's syndrome are composed of small Goormaghtigh cells and sometimes granulated, epithelioid cells in a varying ratio, surrounded by a considerable mass of basement membrane-like material (Brackett et al., 1968; Schmidt et al., 1973). No additional transformation of smooth muscle cells of the arteriolar walls as seen in Addison's disease were to be found in Bartter's syndrome. As we were able to show previously by planimetry, the mean size of randomly cut juxtaglomerular cell complexes in histological sections of this case was about 4,900 μ^2 (Schmidt et al., 1973).

A very remarkably difference between the morphological behaviour of the JGA in Addison's disease and in Bartter's syndrome, is the contradictory reaction

of the maculae densae: In Bartter's syndrome the mean basal area of the macula densa is about 50% larger than normal and seems to participate in the general hyperplasia and hypertrophy of the JGA, whereas it is nearly of normal size in Addison's disease. This lack of concord between the two essential structures of the JGA, i.e. the juxtaglomerular cell complex and the macula densa, in Addison's disease has already been stated in former investigations by our group, by methods of cell counting (Helber et al., 1970) and planimetrically (Meyer, 1972).

The excessive enlargement of the Goormaghtigh cell field in Bartter's syndrome may be responsible for the almost complete loss of contact between the hilar arterioles and the associated maculae densae: None of the afferent and only 2 of the efferent arterioles out of the 10 serially examined JGAs were in direct contact with their maculae densae (Table 1), whereas this contact is almost invariable in normal kidneys (Bohle et al., 1970; Christensen et al., 1975).

In the renal tissue taken from the patient suffering from Conn's syndrome all contact areas and the macula densa basal area were reduced. This is in agreement with previous planimetric observations on renal wedge biopsies of seven patients with primary aldosteronism, in which a reduction of the mean cut area both of the juxtaglomerular cell complex (mean $\pm$S.D. = 790 ± 148 (μ^2) and the macula densa could be demonstrated (Meyer, 1972).

As listed in Table 2 the various areas of contact between the structures of the JGA indicate that in analogy to a functional and morphological normal kidney (Christensen, 1974) some parts of the JGA correlate positively in size only in Bartter's syndrome, while there is no correlation at all in Addison's disease and in Conn's syndrome.

It is noteworthy that in normal kidneys as well as in kidneys of patients suffering from diseases which are accompanied by changes of the renin-angiotensin system, a rather extensive direct contact was always present between the macula densa and the Goormaghtigh cell field (Table 3) whereas a direct contact between the other structures of the JGA was not always established (Table 1, numbers in brackets). On the other hand, 40–60% of the macula densa basal area were not in direct contact with any other structure belonging to the JGA as may be read from Table 3.

Our results suggest at least two types of hyperplasia and hypertrophy of the JGA: The one—as in Addison's disease—being characterized by a transformation of many arteriolar smooth muscle cells near the glomerular vascular pole as well as Goormaghtigh cells into epithelioid cells without an equivalent increase of the macula densa, and the other—as in Bartter's syndrome—being represented by an enormous hyperplasia of Goormaghtigh cells, partly changed into epithelioid cells, but associated with a considerable enlargement of the macula densa.

We cannot say whether this incongruous reaction of the structures of the JGA is due to different ways of stimulation. Possibly, in Addison's disease as in Conn's syndrome in accordance with Tobian's hypothesis (1960, 1967) either the hypovolaemic and/or hypotensive condition, or a hypervolaemia and/or hypertension may be the decisive factor to stimulate or suppress renin release in the JGA via the hypothesized stretch-receptors within the walls of the afferent arterioles, whereas in the sodium-loosing Bartter's syndrome the stimulation of

the JGA may be induced additionally by an increased sodium chloride concentration of the distal tubular fluid at the level of the macula densa (Thurau and Schnermann, 1965) providing a renin release from, or renin activation in the juxtaglomerular cells (Thurau et al., 1972).

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