klären, ob am primären Blastom sekundär die entzündlichen Prozesse auftraten oder ob das Blastom auf entzündlicher Grundlage entstanden ist. Ein eingehendes Studium des Materials durch einen Mitarbeiter soll sich mit diesen Fragen befassen. H. HAUSER

Veterinär-pathologisches Institut der Universität Bern, den 17. Februar 1948.

Summary

Within a period of five years a dozen cases of cutaneous nodular growths were observed in dogs of varying age and breed. They consist of agglomerations of cells with a basophil granulation in a stroma of inflammatory connective tissue.

DISPUTANDA

Some Remarks on the Aglucosuric Blood Sugar Concentration

A recent note in this journal by M. FÖLDI, G. SZABÓ, and S. ZSOLDOS[1] appears to call for some discussion.

Let us first reconsider the meaning of the aglucosuric blood sugar concentration and its mathematical derivation. Using the same notations as FÖLDI *et al.*, viz.

Ag = aglucosuric blood sugar concentration mg%
Pg = actual blood sugar concentration mg%
Rg = reabsorbed glucose mg/min
Fg = filtered glucose mg/min
P = plasma creatinine or insulin concentration mg%
U = urinary creatinine or insulin concentration mg%
Ug = urinary glucose concentration mg%

and introducing

F = rate of glomerular filtration cm^3/min
R = rate of tubular resorption cm^3/min

we get

$$\frac{F \cdot Pg}{100} = Rg\,, \qquad (1)$$

as long as $Rg < Tm_g$[2], where Tm_g = maximal quantity of glucose that can be reabsorbed per unit time. If we increase Pg—by means of glucose infusion—Rg reaches Tm_g and, by definition, the limiting value of Pg, without glucose in the urine, is Ag. Thus

$$Ag = \frac{100 \cdot Tm_g}{F}\,. \qquad (2)$$

A further increase in Pg will bring about the excretion of glucose in the urine, the reabsorption of glucose being fixed at Tm_g. Now, evidently

$$Fg = \frac{F \cdot Pg}{100} = \frac{(F - R) \cdot Ug}{100} + Tm_g\,. \qquad (3)$$

Assuming that F remains constant, if we increase Pg—which assumption is fairly well-supported by SHANNON and FISHER's data—we can calculate Ag without knowing the actual value of Tm_g and F. Expressing Tm_g from (3) and putting it into

$$Ag = Pg - \frac{P}{U} \cdot Ug, \text{ since } \frac{F - R}{F} = \frac{P}{U}\,. \qquad (4)$$

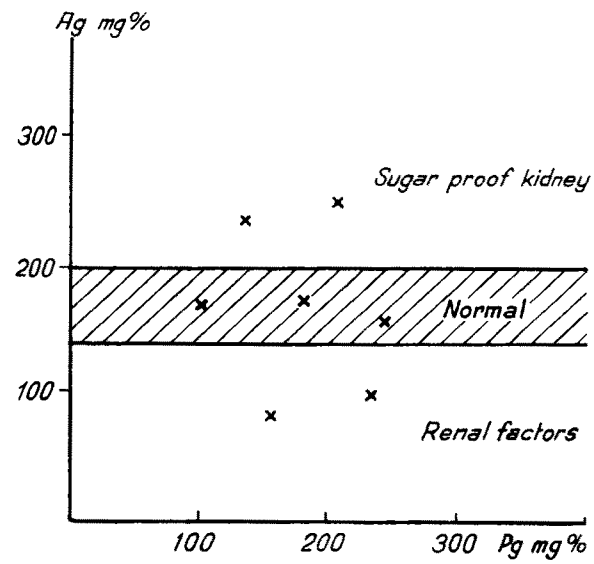
Clearly, with the assumptions made, Ag is a constant for any given individual, depending only on Tm_g and F, but can be, as we see, calculated from Pg, Ug, and P/U.

Now it will appear that Ag, a constant, is *not* a function of Pg, as stated by FÖLDI *et al.*—not at least in the same individual. It is one thing to calculate a constant from a formula, and quite another thing to say that the constant actually is a function of some other observable value. E.g. we may calculate the boiling point—a constant—of a liquid from its behaviour at two given temperatures, but we cannot say that the boiling point is a function of these actual temperatures.

The above expression of Ag (4) only has a meaning if $Ug > 0$. Hence it follows that we cannot calculate the aglucosuric blood sugar level of a normal person without actually producing glucosuria. Once Pg has exceeded Ag, the calculated values of Ag should be constant for any Pg value. It may be mentioned that I have calculated Ag, using formula (4), from the data in SHANNON and FISHER's[1] paper. For values of Pg ranging 238—1,410 mg% Ag is constant and has the value $232{\cdot}2 \pm 2{\cdot}9$ mg% (dog experiment).

It is difficult to see what meaning should be ascribed to curve 1 in the note by FÖLDI *et al.* (actually the ordinate should be Ag instead of Rg, as printed).

Are we to assume that the points on the ascending part of the graph are points calculated with $Ug = 0$, and that the true value for Ag is represented by the points lying on the horizontal part of the graph? If so, we have, roughly, $Ag = 350$ mg%. However it is generally known[2] that for human normals Ag lies between 140—200 mg%, the mean value being 180 mg%. This points either to errors in the technique or to the particular case presented in the figure, being one with a very high glucose threshold.



If, on the other hand, points lying on the ascending part of the graph do correspond to values $Ug > 0$, then, it seems, the results disprove the very theory—that of SHANNON's—upon which the whole work is based. Since $Ag = \frac{100 \cdot Tm_g}{F}$, Ag could increase only if Tm_g were increasing, or F decreasing. But if either Tm_g or F were not constants, $Pg - \frac{P}{U} Ug$ in (4) would have no actual meaning and could not be considered as an expression for Ag. That is, if Ag calculated from (4) increases with increasing Pg, it is evident that the assumptions—

[1] M. FÖLDI, G. SZABÓ, and S. ZSOLDOS, Exper. *3*, 329 (1947).
[2] I. A. SHANNON and S. FISHER, Am. J. Physiol. *122*, 765 (1938).

[1] I. A. SHANNON and S. FISHER, l. c. p. 767.
[2] C. E. DUKES, Urine examination and clinical interpretation (Oxford Univ. Press, 1939), p. 131. – W. C. LOVATT-EVANS, Principles of human physiology (London, Churchill, 1945), p. 927.

constant Tm_g and F—are not fulfilled. SHANNON's conclusions and the data represented by Fig. 1 of FÖLDI *et al.* are thus seen to be incompatible.

If we accept the former it will be clear that to speak of a correlation between Pg and Ag, in the same individual, since Ag is a constant, is meaningless (although there may be a correlation between these quantities, i.e. the normal Pg and Ag, determined in a sugar-infusion experiment, considering several members of a population).

If one wishes to plot Ag against Pg (see figure), the values of Ag will be distributed about the value 180 mg% in the range 140—200 mg%[1,2]. Points lying below this zone indicate a lower tubular reabsorption (phlorizin type), points above the zone indicate a "sugar-proof kidney"[3].

In a diabetic patient with glucosuria Ag can be determined forthwith, without glucose infusion, and again, values between 140—200 mg% should be considered as normal, while values outside this range point to a case complicated by renal factors or to a sugar-proof kidney.

J. GERGELY

Department of Biochemistry, University of Budapest, and Department of Physical Chemistry, University fo Leeds, January 9, 1948.

Zusammenfassung

Zu einer kürzlich erschienenen Arbeit von FÖLDI und Mitarbeitern wird folgendes festgestellt: Ihre «aglykosurische Blutzuckerkonzentration» (Ag=die höchste Blutzuckerkonzentration, bei der eben noch kein Zucker im Harn erscheint), ist bei ein und derselben Person eine vom momentanen Blutzuckerwert unabhängige Größe, obwohl in der Formel für Ag die Blutzuckerkonzentration (Pg) vorkommt. Die Formel für Ag ist nur für $Pg > Ag$ definiert. Wenn man Ag bei verschiedenen Personen bestimmt und die Ergebnisse in ein Koordinatensystem (Abszisse Pg, Ordinate Ag) einträgt, dann fallen die Punkte, die einer normalen Nierentätigkeit entsprechen, in einen mit der Abszisse parallelen Streifen. Dieser liegt im Bereiche der physiologischen Werte der Nierenzuckerschwelle. Punkte unterhalb dieses Streifens deuten auf eine vergrößerte, Punkte oberhalb des Streifens auf eine verminderte «Zuckerdurchlässigkeit» der Nieren hin. Mit den vorliegenden kritischen Bemerkungen soll nicht bestritten werden, daß es — wie das FÖLDI und Mitarbeiter betont haben — klinisch bedeutsam ist, die durch Nierenfaktoren komplizierten Diabetesfälle zu erkennen.

[1] C. E. DUKES, l. c. - W. C. LOVATT-EVANS, l. c.

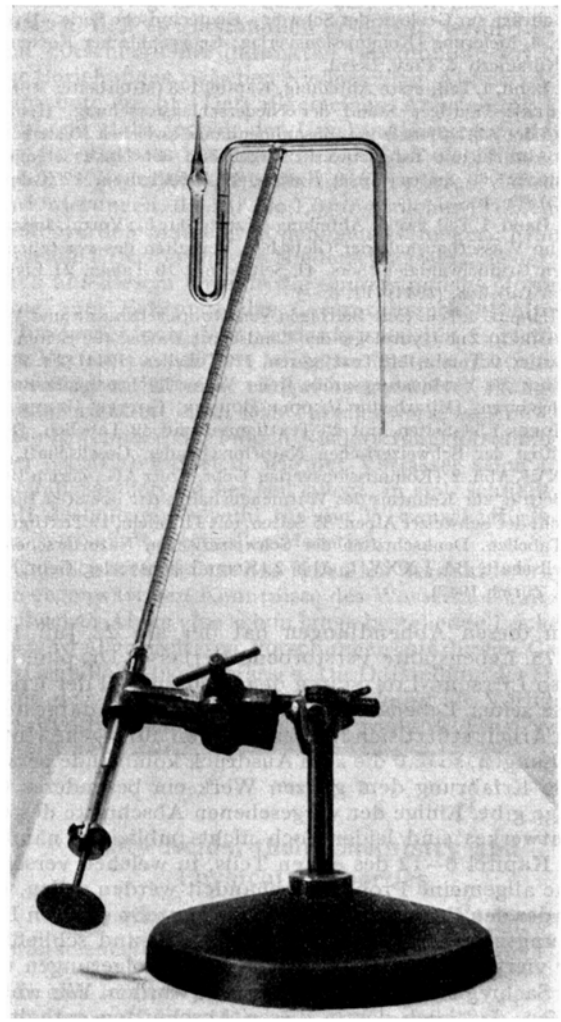
[2] According to SMITH (H. W. SMITH, Lectures on the Kidney [Kansas, 1943], p. 36) Ag = 256 mg%. Using his data (pp. 89–91) one obtains by our formula (2) values of Ag ranging from 189–330 mg% with a mean of 258 mg%. It is somewhat puzzling that these values are definitely higher than the standard text-book value of the renal glucose threshold. The essence of our argument is of course unaffected by the choice of the range of normal Ag values; this determines only the position and width of the marked zone in the figure.

[3] M. FÖLDI, G. SZABÓ, and S. ZSOLDOS, l. c.

PRO LABORATORIO

Microburetta di precisione senza rubinetti

Le comuni microburette graduate in mm³ sono tutte praticamente inservibili per un lavoro accurato e spedito in quanto il lubrificante dei loro rubinetti ne sporca inesorabilmente il capillare graduato. Per questo motivo la microburetta qui raffigurata è stata ideata senza rubinetti. Essa funziona a mercurio ed è graduata di 0,5 in 0,5 fino a 110 mm³. Un capillare ad U con un po' di mercurio, una bolla ed una graduazione ausiliaria sostituisce il rubinetto e consente il riempimento. La graduazione ausiliaria permette la correzione quantitativa di even-



tuali spostamenti della chiusura a manometro durante la titolazione; la bolla serve a trattenerne il mercurio durante il riempimento. Uno stantuffo facilmente amovibile imprime con un movimento a vite spostamenti finemente graduabili alla colonna di mercurio. È possibile misurare soluzioni *indifferenti di fronte al mercurio* fino al decimo di mm³ approssimato. Le interpolazioni si fanno coll'aiuto di una lente.

La buretta viene costruita dalla ditta Ing. G. Terzano, Milano.

E. SARTORI

Clinica Pediatrica dell'Università di Pavia, il 10 novembre 1947.

Zusammenfassung

Es wird eine neue Präzisionsmikrobürette beschrieben, die es gestattet, Lösungen, die gegenüber Quecksilber indifferent sind, bis auf ungefähr $^1/_{10}$ mm³ zu messen. Im Gegensatz zu den bisher üblichen Mikrobüretten hat sie keine Hahnen und verfettet daher nicht.