intervals postoperatively until the animal died. Control electrocardiograms were taken in dogs similarly anesthetized but not operated upon.

The electrocardiograms in the operated dogs showed distinct aberrations from the normal preoperative tracings.

Protocol Dog 4 operated 1/13/42. Preoperative electrocardiogram—sinus arrhythmia.

1/14/42 Electrocardiogram—inversion of the T wave in lead 1.
1/16 " inversion of the T wave in lead 1.
1/19 " inversion of the T wave in lead 1.

Changes became progressively more marked, indicative of increasing myocardial damage.

The changes were interpreted in some instances as indicative of myocardial damage and in others as showing evidence of coronary closure with infarction. There was no constancy in the electrocardiographic patterns nor did they always correspond to the definite electrocardiographic changes observed clinically in heart disease.

The exact mechanisms underlying these changes are being further studied. Observations are also being continued on the association of electrocardiographic changes with other acute intraäbdominal lesions.

Summary. Clinical observations have demonstrated the association of acute pancreatitis with transient electrocardiographic changes. We were able to obtain similar changes in dogs with experimentally induced pancreatitis.

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Relationship Between Body Size and Metabolism.

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Recently Kleiber has studied the relation in liver slices of oxygen consumption to body weight in rats, rabbits, and sheep.[1] It was shown that the Q_{O_2} was inversely proportional to a fractional power of the body weight, so that plotted on a log log grid the data gave a straight line represented by the formula

$$Q_{O_2} = 5.26\ W^{-0.24}$$

1 Kleiber, Max, PROC. SOC. EXP. BIOL. AND MED., 1941, **48**, 419.

where Q_{O_2} is in mm^3/mg dry tissue/hour and W is the body weight in kilos.

It was concluded that "the factors which determine the metabolic level *in vivo* seem still to be present in the surviving tissues cut out of the organism."

Subsequent determinations of tissue respiration and observations on the relation of total metabolism to body size justify an additional note on this subject.

A recalculation of values from the original individual records of Kleiber gives a coefficient of correlation between log Q_{O_2} and log body weight of –0.783 and a coefficient of regression of –0.228 with a standard error of ±0.017. The formula thus becomes

$$Q_{O_2} = 5.28\ W^{-0.228*}$$

Field has since obtained data on respiration in kidney and brain slices for the rat and rabbit. Although covering only two species, the regression obtained is very similar, the average being –0.199. A value as low as this might be expected to occur by random sampling in data similar to Kleiber's about once in 10 times so that the difference can hardly be considered as significant. It is thus apparent that other tissues behave in a manner very similar to liver slices. This strengthens the view previously expressed by Kleiber.

On the other hand it is interesting to compare the exponent with that required by the surface area "law", 0.333. The chances that a value as high as 0.333 could occur from sampling of Kleiber's material is less than one in 10^{+8}. There can be no doubt that the present data differ significantly from that required by the surface area "law".

We have subsequently noted that the weight-specific rate of heat production of intact animals of these species bears the same relation to the body weight as that stated above for the tissues *in vitro*. The data given in Table I were taken from Benedict[2]; the body weights

* In Article 13340, Body Size and Metabolism of Liver Slices *in vitro*, PROC. SOC. EXP. BIOL. AND MED., 1941, **48**, 419, 18 instead of 42 kg was used as mean weight of the sheep. The correct weight leads to the following relation between mean Q_{O_2} of liver slices from rats, rabbits, and sheep and their respective mean body weights:

$$\log Q_{O_2} = 0.735 - 0.21 \log W$$
$$\text{or } Q_{O_2} = 5.43\ W^{-0.21}$$

(The omission of the minus sign of the exponent in the original article is a typographical error). Fortunately the error had no significant effect on the conclusions.

2 Benedict, Francis G., *Vital Energetics*, Carnegie Institution, 1938, p. 131 and 175.

TABLE I.
Heat Production by Animals of Different Body Weights.*

Animal	Body wt, kg	Heat production Cal./kg/day
Rat	0.4	82.0
Rabbit	2.6	44.5
Sheep	45.0	25.5

*Data from Benedict Table 2, p. 131, and Table 4, p. 174. These two tables do not check. I have used figures from Table 2, except for the heat production of the sheep, which is given as 35.50, obviously a misprint for 25.50, as may be seen by comparing with Fig. 35, p. 142.

differ from those given in Kleiber's paper and presumably represent different breeds of these species. The log log plot of heat production (Cal./kilo/day) on body weight (kg) gives a straight line represented by the formula

$$H = 6.87\ W^{-0.243}$$

This power of the body weight might have occurred by random sampling in Kleiber's data once in 3 or 4 times, so that the difference cannot be considered significant. The other constant cannot be compared because of the different units employed and the fact that one is dry, and the other, wet weight. We thus see that there is complete agreement between the weight-specific heat production rate in the intact animal and the respiratory rate in the excised tissues of the same series of animals. This has not, as far as we know, been pointed out before. The relation here presented supports the opinion expressed in the previous paper.

It is clear that we must envisage a mechanism of respiratory regulation which will account for the following observed facts, a far from easy task.

a. The respiratory rate (Q_{O_2}) varies widely; the greatest range is from tissue to tissue in a particular animal (150-fold between extremes, kidney and blood, in the rat[3]), and less for a particular tissue from animal to animal with change of size (4-fold in the present data from rat liver to sheep liver, 10-fold from the mouse to the elephant, if all tissues parallel the weight-specific rate[2]). In addition there are apparently qualitative as well as quantitative differences in the enzyme systems involved (Commoner[4]).

b. The control is not immediately systemic, for example through the circulation by regulation of the supply of oxygen or of some substrate, since the relation to body weight of oxygen consumption

[3] Field, John, II, Belding, H. S., and Martin, A. W., *J. Cell. Comp. Physiol.*, 1939, **143**, 14.

[4] Commoner, B., *Biol. Revs.*, 1940, **15**, 168.

from animal to animal is the same for tissue slices in an atmosphere of oxygen as for the total metabolism of the intact organism.

c. The control is intrinsic in the sense that the relation to body weight of the respiration of the excised tissue slices parallels the metabolism of the intact animal, at least during the one or 2 hours required by the Warburg technic. If the control is ultimately central, as by some hormonal regulation of the concentration of respiratory enzymes, the peripheral "set" involved must have a large measure of inertia, as it is not readily altered by excision and the disruption of all systemic channels of control.

d. Through a striking range the intensity of respiration is regulated between tissue and tissue and from animal to animal so as to present the following picture: The relative intensity of respiration is proportional to the relative body weight of the animal, while the tissues preserve their relative relations. As a result the plot of the log oxygen consumption on the log body weight (thus placing both on a relative basis) yields a family of parallel straight lines, one for each tissue. This conclusion is necessitated not only by the relation shown above for the Q_{O_2} of kidney, liver and brain but also by the fact that the total metabolism of a series of animals parallels the Q_{O_2} of any one of the tissues for these same animals.

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Agglutination of Rabbit Leucocytes by *Staphylococcus aureus* Toxin.

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Several years ago, while testing streptococcal-extract toxin[1] for leucocidin by the Neisser-Wechsberg technic, one of us noted agglutination of the leucocytes in some of the tubes. Such agglutination appeared only on dilution of the toxins.

Recently a similar phenomenon was observed with staphylococcal toxin. This paper deals with a method developed for testing of staphylococcal toxin for these leuco-agglutinins.

Methods. Most of this work was carried out with the Wood strain

[1] Weld, Julia T., *J. Exp. Med.*, 1934, **59**, 83.