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Part I.—Original Articles.

Alcoholism, Crime, and Insanity. By W. BEVAN LEWIS, M.Sc., L.R.C.P.Lond. Medical Director of the West Riding Asylum, Wakefield.

POSSIBLY the title superscribed to this short article may prove misleading; if so, I must crave your indulgence, as the subject to which perhaps such a title is more applicable could scarcely be done justice to within the short limits of time allotted us. I merely wish to skim over the surface of this question of alcoholism and indicate some special features which have appealed to my interest in this connection, prefacing my remarks on the geographical distribution of alcoholism and insanity with a cursory review of the physiological action of alcohol upon heat-formation and its effects on those centres presumably operant in muscular sense discrimination.

Alcoholism and thermogenesis.—For many years past the heat-formation and heat-discharge of the organism under the influence of alcohol has been a subject replete with interest to my mind, and the parallel results of clinical observation and of physiological experiment upon animals, in my opinion, afford a field of research which well repays further investigation. So far back as 1876 and 1880 I began my observations upon thermogenesis in animals subjected to various alkaloids and other drugs, embracing, amongst others, strychnine, ergotine, picrotoxine, hyoscyamine, atropine, solanine, chloral, caffein and alcohol.

Calorimetric observations on animals, such as the rabbit, to

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which graduated doses of alcohol have been given indicate very clearly a primary and secondary stage, the initial stage being one of stimulation of the vasomotor centres in the medulla, *contraction* of arterioles with increase of blood-pressure, the subsequent stage being one of *paralysis* of the same centres or its inhibition through the agency of the pneumogastric nerve. Now, this initial stage of stimulation in which the normal vascular tone is heightened appears to be in inverse proportion in intensity and duration to the dose administered; and it will be noted as a significant fact that in this we are not concerned with a *diminished* heat-discharge or a period of *heat-retention*, since in such a case the body temperature would necessarily be *raised*, whereas *definite lowering* of the body temperature occurs in all cases.

Here, then, we have a clear instance of decreased thermogenesis; the initial stage of alcoholic stimulation, be it short or be it prolonged, is one of lessened heat-formation (Table I). To obtain a typical and prolonged initial stage the quantity of alcohol given must be small; large quantities hurry on the second stage and obscure its effects.

In the second stage of vascular paresis and fall in blood-pressure the heat-discharge is proportional in its duration and degree to the dose administered, the *thermal crisis—i. e.*, the period of *maximum* heat-formation—occurring later on with stronger doses, whilst the prolongation of the total heat-discharge is in such cases a very notable feature. In one instance (Table II) a rabbit, to which three drachms of alcohol had been given, betrayed the onset of the paretic stage in the second quarter-hour, the thermal crisis in the fifth quarter-hour interval, and the heat-discharge had not fallen to its normal under two and a quarter hours from the administration of the drug. In this second stage we have therefore a reversal of the thermal conditions, and the large evolution of heat with vasomotor paresis is coincident with increased thermogenesis. The proof of this, of course, lies in the fact that if the large heat-discharge were not accompanied by increased heat-formation the body temperature would rapidly fall, whereas the reverse occurs, the temperature being well maintained.

In lieu of the formula of Binz and Wood, that alcohol in very large doses lowers temperature by directly checking tissue metamorphosis, we are far more in accord with Professor

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Parkes, who distinctly expressed his disbelief in the view that alcohol checks tissue change—thermogenesis is certainly checked in the *initial* stage, but the later action, the more pronounced and prolonged with the greater dose given, is certainly that of an *increased heat-formation and heat discharge*. Whatever, therefore, be the explanation of the so-called febrifuge action of alcohol, it can *not* be attributable to a *decreased tissue change* involving lowered temperature.

Now let us compare the action of another potent drug, chloral. Here you will observe the same phenomena, only more marked in degree. The highest registry of heat-formation with alcohol (Table II) was 8720 h.u. or 4·28 h.u.(1) per gramme of body weight; but with chloral we register 10,875 and 16,907 units, or the enormous evolution of 7·7 and 11·3 h.u. respectively.

I will not attempt to dwell upon the mechanism whereby this result is brought about: I merely wish to indicate the similarity of the action of these two toxic agencies and the powerful means they afford us, separately or conjointly, for modifying thermogenesis.

In Table I you will observe the effect of a lethal dose of chloral; the *stimulant stage* is absent, and the large heat-discharge due to vasomotor paresis falls immediately under observation. In the other two cases smaller non-lethal doses given betray an early stage of stimulation of the vasomotor centres, a small or inappreciable heat-formation, with loss of body temperature, to be followed by the large evolution of over fifteen or sixteen thousand heat units, or 11·3 h.u. per gramme of body weight by surface dissipation. In the initial stage of stimulation there is heightened vascular tension, as indicated by sphygmographic tracings, a fact testified to by Anstie, Andrews, and Bouchat. One cannot, then, but be struck by the similarity in action of these two narcotic drugs.

Caffeine.—Now, if I direct your attention to Table III, you will observe that when 2½ grains of caffeine are administered to an animal weighing 1343 grammes, an augmented heat-formation and discharge begin early, and are long protracted. The second column shows the result with 4 grammes in a highly nervous and sensitive animal which struggled much when the temperature was being taken. The first interval here merely indicates loss from the body temperature from this cause whilst resting out of the calorimeter until more docile. Observations

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made at the end of the second quarter-hour interval showed immediate increased thermogenesis, the maximum attained of 4·62 h.u. at the end of the third interval, whilst 2·12 h.u. still registered at the end of the fifth observation. Now, the distinction to be noted between these two therapeutic agents is an important one. Alcohol in large doses stimulates thermogenesis and tends towards great loss of body temperature by surface dissipation; caffeine also stimulates heat-formation largely, but, on the contrary, favours retention of heat, there being a steady addition to body temperature and a re-instatement of the norma. The antagonism between alcohol and caffeine is well brought out when both are administered together, and Table III is highly instructive on this point.

Impairment of muscular sense and reaction-time.—The cortical denudations issuing in mental disease afford valuable hints as to the functional significance of the so-called motor area, and the view upheld by Charlton Bastian, that the centres herein have a kinæsthetic rather than truly motor significance, has always had a peculiar hold upon my mind, but I must confess only in a modified sense. It seemed to me that the areas represented for muscular sense discrimination had a wider range than what could be accorded to the truly motor cortex. Searching for clinical data in support of this thesis, general paralysis and alcoholism in particular appeared to me to favour this position. In 1878 in the first volume of *Brain* I wrote as follows: "No *abrupt* passage from one form of cortical lamination to that of another is ever seen in the convolutions of the vault. Changes in type are gradually assumed, and no distinct boundary exists to indicate an exact line of demarcation. The structure peculiar to one region gradually fades and blends with arrangements which foreshadow the architecture of adjacent realms, and thus we obtain *transition regions*. The ascending parietal is such a transition region, where features characteristic of the frontal lobe are blended with those exhibiting a tendency to assume the laminar arrangements of convolutions posterior to it." I have seen no reason since that date for altering this opinion, and I would indicate here that the transition region which I then proceed to describe in detail, bounds the frontal and parietal aspect of the true motor cortex. This is the fringe of cortex which in my opinion represents an extension from the truly motor

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region of a type receptive of those impressions we characterise as muscular sense. This is an area which, judging from clinical and pathological data, is *facile princeps* involved in three well-marked affections of the nervous system—alcoholism, general paralysis, and adolescent insanity, or, as some would prefer to call it, dementia præcox. In both the former the *early* implication of this cortical territory is well seen; in both the cell-mechanisms centring around the pre-central and post-central gyri suffer the more profoundly; and in many instances these tracts are almost exclusively implicated.

In all three affections there is a notable impairment of muscular sense; but the point I desire to emphasise is this: with profound loss of muscular sense the subject still retains in all cases at an early stage an exquisite sense of temperature and a keen tactile sensibility. Two deductions would naturally arise from this fact:

(1) The centres which register muscular sense discrimination are wholly distinct from, and do not overlap, those of tactile and thermal impressions.

(2) The centres receiving such muscular impressions are probably largely constituted by the pre-central, post-central, and transition realms of cortex alluded to, and thus the absolutely strict severance of a motor cortex from its guiding sensory arrangements is provided against, and is in line with the doctrines of Hughlings Jackson as regards nervous evolution in general and his views with respect to representative and re-representative centres.

The failure in muscular sense discrimination occurs quite early in general paralysis, where, as we know, the main impact of the lesion falls upon this area of the cortex, and a similar statement may be made for early stages of alcoholic insanity, revealing this decline of the muscular sense. Nay, even in the effects of alcohol upon healthy individuals transient reductions in this sense may be recognised by all who experiment on themselves or others with delicately graduated weights. First comes the stage of uncertainty as regards the finer tests, a doubt which soon extends to the grosser series, and eventually doubt gives way to absolute incapacity to determine the finer and grosser differences in weight. This early implication of muscular sense, leaving the special senses and tactile sensibility intact, is sometimes recognised in somnambulistic states. I

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will quote a personal experience: At the age of twelve I was an ardent student of osteology, and knew the human skull almost as well as I know it now. A grimy, stained specimen was always placed near my bedroom window, and whilst asleep one night I thought I awoke and saw a brand-new skull, white as ivory, on a bookcase at the foot of my bed. I clambered up by a chair, secured my prize, and holding the newly-acquired treasure in my right hand and the old friend in the other, I compared their relative merits long and critically. Then, replacing the ideal skull on the bookcase I fell asleep. On waking next morning I found, of course, that I had acted the part in a somnambulistic state—the chair was at the foot of the bookcase, the real skull was displaced from its customary site at the window. Now, the feature to which I specially draw your attention was that the sensorial centres generally were all remarkably active—full consciousness of configuration, orientation, details of structure, solidity and colour were keenly appreciated—yet I wholly failed to note the absence of weight in the ideal skull balanced against the real one. Surely this was an illustration of reductions in a special kinæsthetic area whilst the greater extent of sensorial cortex was wide awake to peripheral excitations; possibly also the vivid realisation of the ideal skull was an illustration of the dual action of the two hemispheres.

The total reaction-time to sight and sound stimuli is, as is well known, notably delayed under the influence of moderate alcoholic stimulation. The subject usually thinks he is reacting with his customary rapidity, or even in excess of his customary speed; in like manner the judgment pronounces *favourably at the time* as regards the glibness of speech, the contents of conversation, or the intrinsic value of writing and composition under the action of alcohol; and many an amusing story could be gleaned from individual experience of the eagerness with which the brilliant speaker next morning would wish to disclaim the remarks made by him on festive occasions. Not so, however, with the muscular sense: the judgment, it seems to me, is not so blinded to the steady advance in failure of this sense discrimination, and records only too painfully its growing impotence. Now, it appears to my mind that this failure of delicate discrimination of the action of the smaller musculatures is one of the very earliest to occur in cerebral reductions,

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physiological or pathological; and notably is this the case in alcoholic forms of brain disease.

In the magnificent monograph on *Cortical Localisation* recently given to the world by Dr. Alfred Campbell, for which all of us as psychologists should be deeply indebted, I observe that the conclusions arrived at by me as to the "transition" regions of the cortex cerebri are fully identified and confirmed, corresponding in the main to his post-central and intermediate pre- and post-central cortex; but I glean from his remarks that he assigns the site of muscular sense discrimination to the post-central area only, strictly limiting this localisation to post-central realms. In all other particulars we are in most absolute agreement.

Geographical incidence and heredity.—We have all to familiarise ourselves with the geographical limitations of disease in general: the cosmic and telluric agencies of climate, soil, temperature, moisture, vegetation, which so largely condition the prevalence of certain forms of disease, and which constitute so fascinating a study for the public health officer. But although as affecting large communities from a racial or ethnic standpoint certain valuable generalisations have been arrived at, the relationships of alcoholism, pauperism, crime, and insanity amongst smaller communities, bound together by similar political interests, the same legal enactments, and subject to intimate individual intercourse, action, and reaction, is one of perennial interest to the psychologist. In the following remarks I wish to disarm all criticism by at once admitting how fully I recognise the deceptive flexibility of statistical inquiry, more particularly when it concerns the labyrinthine intricacies of the social fabric. I venture, however, to believe that there is a kernel of truth in the results I am about to place before you, and which further experience and inquiry may enucleate and sever from all adventitious matter—facts to which I first drew attention in an article published in the *Fortnightly Journal* for 1893. Limiting our inquiry to England and Wales, it is seen by reference to the table that the convictions for drunkenness per 1000 of the county population show remarkably diverse ratios, beginning with a ratio of ·7 for Cambridge, and ending at Durham with the high ratio of 17·5 as the maximum. Now, the average ratio for all England and Wales was 5·8, and whilst thirty-four counties were below this average, thirteen counties exceeded it. Specially

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notable for this inebriate proclivity were Northumberland, Glamorgan, and Durham, with ratios respectively of 11·0, 11·3, and 17·5 per 1000. On more closely analysing our figures we find that by far the greater part of the west coast, and the whole of the east and south coast from the East Riding of Yorkshire down to Devon, give us a very moderate ratio of convictions for drunkenness; and that exactly similar conditions prevail in at least thirteen out of the fifteen inland, rural, and *agricultural* counties, in which the highest ratio attained is 3·3. In other words, the maritime as well as the inland counties populated by an *agricultural* community are by a long way the most temperate in our island home, so far as England is concerned. Now, if we compare the mining and manufacturing communities along the remaining sea-board, we find that six give us ratios above the average—*e.g.*, Chester and Cumberland at 6·8, Lancaster at 8·4, Northumberland at 11·0, Glamorgan at 11·3, and Durham at 17·5.

Again, of the nine *inland* counties distinguished by their mining and manufacturing industries, all but one give ratios above the average, whilst four greatly exceed the latter. To quote from a former statement of mine, "We may therefore accept it as a fact that of the telluric and geographical influences by which a community is conditioned the agricultural pursuits of inland counties, as also of the sea-board, with its associated maritime industries, are far more favourable to national sobriety than the employments originating in the mineral resources of the counties, with their associated manufactures, and that probably the relative local position of sea-board (apart from the seasonal exodus from the dense centres of life) or inland communities does not materially affect the question, although undoubtedly the mining and manufacturing industries with an extended sea-board afford us the highest of all ratios."

In the second place, a very definite relationship exists between the *occupations* of the community and the prevalence of *pauperism*, and a glance at the tabulated statistics will reveal a very high ratio of pauperism wherever we are dealing with an *agricultural* population. The average ratio of paupers to 1000 of the total population of the Union counties was for the date in question 26·9, and we find that twenty-three out of twenty-five of these counties give ratios from 30 upwards; Herts, Radnor, Hereford, Montgomery, Norfolk, Dorset, Merioneth, all range above 40,

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whilst Anglesea stands at 55·4, or more than double the average for all England and Wales.

On the other hand, should these counties be characterised by manufacturing industries, and especially should the mineral wealth of the locality foster mining operations, pauperism declines and a ratio far below the average is attained by nearly all the mining and manufacturing districts—*e.g.*, Westmoreland (22·5), Warwick (21·3), Derby (22), Nottingham (22·1), York, West Riding (18·6), Northumberland (20·3), Durham (20·6), Lancaster (18·2), Cumberland (28·8), Cheshire (20·9), Glamorgan (24·9).

In the third place, the effects of centralisation are revealed in the following columns in the densely populated manufacturing and mining centres of our boroughs and county boroughs, as modified by the free influx of a seafaring community; thus, whilst the large inland county boroughs of Leeds, Bradford, Sheffield, Halifax, Huddersfield, and Birmingham give us the low ratio for convictions of 1·7 to 6·8, the *maritime* boroughs and county boroughs fringing our sea-board rise from a ratio of 11·3 to 30·0; whilst, again, as modified by *agrarian* occupations, we may compare with these high ratios the south-coast towns of Brighton, Portsmouth, Hastings, and Southampton, which range from 1·7 to 2·7 only.

These large dense centres of activity reveal therefore still more forcibly the facts already recognised for the several counties at large as regards inebriety, that the admixture of a maritime with a mining and manufacturing class is fatal to the sobriety of the community, just as this environment fosters the criminal degenerate, who, parasite-like, preys upon his fellow-man. An all-important relationship revealed is the parallel rise and fall of insanity and pauperism, indicating the connection betwixt mental derangement and poverty, want, anxiety, and the associated moral factors; these facts are very striking if we take extreme cases:

	Convictions for Drunkenness.[1]	Ratio of Lunatics.[1]	Ratio of Paupers.[1]
Hereford	3·3	4·63	41·8
Cambridge	·7	3·01	36·9
Glamorgan	11·3	1·89	24·8
Durham	17·5	1·60	20·6

[1] Ratios per 1000 of county population.

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We may fairly conclude from such considerations that:

Inland and maritime agricultural communities are the least inebriate, have the highest ratio of pauperism, have the highest ratio of insanity.

Inland and maritime, mining, and manufacturing communities are, on the contrary, the most inebriate, have the lowest ratio of pauperism, have the lowest ratio of insanity.

Maritime, mining, and manufacturing communities are of all others by far the most intemperate, whilst revealing the lowest ratios of pauperism and insanity.

We see here, then, the *dissociation* of alcoholism from insanity. The maximum incidence of inebriety does not coincide with that of insanity, whereas it does coincide, as we all know, with crime, and as I shall endeavour to show, with epilepsies and convulsive psychoses generally. The judicial statistics reveal that of 738,061 cases of all forms of crime proceeded against summarily, 189,746, or 25·7 *per cent.*, were for drunkenness; that of 755,739 cases of all forms of crime proceeded against summarily as well as by indictment, 39,573, or 5·2 *per cent.*, were habitual drunkards; and we miss here, of course, the vast army of secret drinkers and those inured to drink who do not obtrude themselves before our law courts. In the *Fifty-ninth Report of the Lunacy Commissioners for England and Wales* we find attention called to the fact of this dissociation of drink and insanity in two maps which admirably bring out these features. On both the insane and the criminal alcohol acts upon a psychopathic basis, but when it issues in insanity it is of the degenerate and explosive type, just as it is the prime factor in evolving the criminal degenerate.

The neurotic heritage is well revealed by the following figures: At the Wakefield Asylum, of 3470 cases of all forms of insanity in whom personal intemperance was ascertained in 27·7 *per cent.*, only 6·6 *per cent.* afforded a history of parental intemperance; but of the alcoholic insane the percentage of parental alcoholism rose to 11·2 *per cent.*, whilst in the forms of adolescent insanity it was as high as 16 *per cent.*, both the latter being, of course, notably explosive forms of insanity.

In like manner the feeble-minded degenerate, the imbecile, and idiot, give us, *if we exclude epileptic forms*, the low proportion of 8·5 *per cent.*, and those in Dr. Barr's statistics come up to 4 *per cent.* at most. If, however, we take all the imbecile and

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idiot class, *including epileptics*, the proportion of those with alcoholic parentage rises to 20 *per cent.*; this, of course, is a very significant feature.

Reverting now to the criminal class, according to Dr. Henry Clarke, of H.M. Prison at Wakefield, taking all criminals together, 43·5 *per cent.* have drunken fathers, but if the epileptic criminal only be considered, 67 *per cent.* have a definite and certain history of paternal intemperance, whilst 18·2 *per cent.* further were somewhat doubtful, but almost certainly intemperate, making if included a gross total of 85·2 *per cent.* The epileptic criminal, you observe, as the epileptic idiot and imbecile, reveals the potency of paternal alcoholism.

The same facts are testified to by other observers, thus: Bourneville (quoted by Dr. Jules Morel), in the examination of 2554 idiot, imbecile, epileptic, and hysteric children, found that 41·1 *per cent.* had an alcoholic parentage, and of these conception was certainly coincident with drunkenness in 9·2 *per cent.*, and probably also in a smaller group of 3·3 *per cent.* So the idiots of St. Anne's Asylum (Paris) were found by Robinovitch to afford a proportion of 54 *per cent.* with alcoholic parentage, but when the computation was restricted to the epileptic community the proportion rose to 65 *per cent.* I would draw your attention here to the chart enlarged from that given in the charming work by Sir Lauder Brunton—*Disorders of Assimilation and Digestion*—as well illustrating some of the features I have just mentioned.

Alcoholism and heredity.— And now, as regards alcoholic inheritance, the more general conception of this disease, to which I subscribe my belief, is that of a very universal reaction of the organism to the *direct* action of the toxic agent alcohol. I say *direct* because a distinguished pathologist has lately expressed the view that the disease is the result of a secondary bacterial toxæmia induced by alcohol breaking down the first line of defence. Health, we are told by Dr. Payne, is a state of moving equilibrium, a condition of uniform motion, or, as expressed by Dr. Allbutt, "a positive conception of a perfect balance of the moving equilibrium we call systemic life; disease is a negative conception, and signifies something less than this perfect balance." Yet, as there is no absolute standard of health, the term becomes a relative one, and so the line betwixt physiological and pathological reaction can be but ill defined.

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Thus it is that inquiries into individual idiosyncrasies can alone establish when the operation of alcohol oversteps the limits of healthy systemic reaction. When, again, diseases dependent for their *cause* upon external agencies are concerned, whether traumatism, thermal, electric, or vital bacterial infections, everything depends on the impact or dose of the morbific factor, together with the individual susceptibility. The dose of alcohol which trangresses the physiological limits is probably very small, and for the specially susceptible is very minute indeed.

Parkes and Wollowicz estimated it at one and a half ounce for man, and this is probably greatly over-estimated, an opinion expressed by Dr. Sidney Martin. It has always appeared to me that the period of decreased thermogenesis, as defined in my calorimetric observations, gave the nearest approach to an accurate estimate of the dose consistent with physiological reaction, that the moment this period of stimulation of the vasomotor centres tends to be less defined and shortened in favour of the second stage of thermolysis we transgress the physiological limits strictly so to speak. Thermotaxis affords us, then, clinical indications which should ever be borne in mind.

Is alcohol inheritable? I think this question must be met by a direct negative. Alcoholism as alcoholism is not inherited. What is inherited is usually something wholly different. That alcohol, like other toxic agencies in the parent, results in certain abnormal nutritional conditions of the germ-plasm is unquestionable; it would indeed be strange if such were not the case. That the ovum nourished by the maternal blood should not be affected by its immediate environment, the soma, in metazoa, just as the protozoa are directly influenced by the cosmic environment, would be highly improbable. So far back as 1895 Féré produced monstrosities by exposing eggs to the vapour of alcohol during incubation. Ovize also in 1900, and as confirmed subsequently by Reiz, showed the toxic action of alcohol vapour on artificially hatched eggs of fowl, 36·7 *per cent.* of the progeny being structurally defective, monstrous, and of the whole number incubated 17·5 *per cent.* alone were viable. Cossar Ewart's Indian Blue-Rock pigeons, infected by the parasite halteridium, did not transmit malaria to their progeny. True, they were *weakly* offspring and *more or less sterile*, according to the potency of the parental toxæmia, but in no case did the young exhibit the parasite halteridium in their blood; in

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other words, *malaria was not transmitted.* Professor Ewart would predicate the same for gout; it is the *tendency* under certain conditions for gouty changes to arise in the offspring which is transmitted; the offspring are *never born* with gouty tissue-changes.

Similarly as regards tuberculosis: the diminished resistance to the growth of tubercle, whatever that may mean, is what is transmitted to the offspring of the tuberculous—*the diathesis is not the disease.* Let us for a moment take as an illustration of the loose application of the term "heredity" other cases of toxæmia. A syphilised mother with secondary symptoms begets a syphilised infant, and this instance of congenital syphilis will by some be regarded as proof of the inheritance by the offspring of a definite character acquired by the parent. It is really not a question of heredity at all; it is but an instance of simultaneous infection—either germinal, placental, or both combined—a synchronicity of morbid agency, an antenatal contamination. Similarly as regards tuberculosis, should it arise congenitally in the offspring of the tuberculous parent it is but an instance of direct infection *in utero*, yet no proof at all of the heredity of phthisis; what is handed down as a germinal variation is the hereditary predisposition to the growth of the tubercle bacillus. In fact, all the specific infective diseases, such as scarlet fever, measles, smallpox, tuberculosis, must be excluded from the category of inherited diseases, and when arising congenitally are but instances of infection through the placental circulation, or, as with syphilis, transmitted by the infected germ. There is, of course, positive evidence that pathogenic organisms do pass from the placental into the fœtal circulation. In a strict biological sense, therefore, none of these diseases are instances of hereditary transmission.

And so, passing from vital agencies such as the pathogenic bacteria to the toxic agency alcohol, we can in no sense speak of alcoholism as inheritable; the varied and elaborate tissue-changes constituting the disease chronic alcoholism are never inherited by the offspring as a definite specific character. If that disturbance of the healthy equilibrium in the parent which is indicated by the symptoms or signs of the disease we know as chronic alcoholism can be reflected upon the progeny through the parental germ-plasm, we should indeed be in the presence of a mystery explicable only by some such elaborate

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and inconceivable hypothesis as that of Darwin's theory of pangenesis. The dictum of biological science of to-day would affirm the negative—*acquired characters in this sense are not transmitted.* And yet undoubtedly this is the trend of popular belief, and would be, moreover, I presume, upheld by the Neo-Lamarckian school of to-day.

An inherent susceptibility of the tissues in certain individuals to the toxic agency of alcohol is universally admitted, and just as specific differences are recognised in susceptible animals, as when, *e.g.*, we find that the white rat is immune to the *Bacillus anthracis*, while the common rat and mouse are peculiarly susceptible, so individual resistance to alcohol is a notably varying factor of germinal transmission.

Again, it has been well shown that we can confer on animals an acquired predisposition to disease; in other words, the natural resistance can be broken down and an unnatural susceptibility established, as when hens or pigeons naturally refractory to anthrax can be readily infected if deprived of food, and thus Kanthack indicates how starvation, bad food, exposure, or fatigue may reduce the natural resistance to the pathological level. In like manner we might argue that *various agencies* might break down one's natural resistance to alcohol so that an acquired predisposition to alcoholism when indulged in would be apparent. But the point of interest to us is whether or no this acquired predisposition can be inherited by the offspring—in other words, is what is transmitted to the progeny specific or non-specific? In the first place, we have to eliminate all instances of congenital contamination through the mother's blood during gestation—as before explained, these instances are not instances of hereditary transmission at all; in the next place, specific *maternal* transmission can only be assumed where alcoholism was well established prior to conception, and is always open to some doubt as we must still eliminate a connate origin. The transmission of *paternal* qualities is not open to such fallacy; and where an acquired predisposition is clearly transmitted from the paternal side, there we have more solid ground for our contention. These cases of transmission of an acquired predisposition from the paternal side are, therefore, the simpler ones for investigation; yet even here we have to note the varied factors introduced by amphimixis. I take it that general assent will be accorded to the proposition that the

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vehicle of heredity is constituted by the chromatin threads of the germ-cell nucleus, and that the theatre for its operation is the cytoplasm of the cell itself. At first isotropous, the cell-protoplasm, step by step, through the agency of these developing forces, acquires polarity, cytoplasmic differentiation, and a stereotyped scaffolding, more or less modifiable with the age of the ovum. In early developmental stages there is betrayed a uniformity in value of the several blastomeres into which the egg has segmented, as shown in the more labile differentiation of amphioxus by Wilson; whilst in other organisms, as the sea-urchin Toxophneustes, the isolated blastomeres indicate a more fixed and stereotyped differentiation. One of the most beautiful experiments in biological investigations is that recorded by *Z*oja, who obtained perfect embryos from the two-, four-, eight-, and even sixteen-cell stage of Medusa by separating their blastomeres—each blastomere in the latter case developed into a perfect animal one sixteenth the normal size. I introduce these biological considerations to indicate that the fertilised ovum is constantly varying in its reaction to the environment; and what is predicated for one stage as the result of such environmental agencies cannot be safely asserted for a later stage of development. Cossar Ewart's remarks upon the staleness or age of the developing ovum as a potent source of variation will occur to us in this connection.

Certain conditions, therefore, morphological and developmental, are imposed upon the germ by virtue of its specific organisation; but we must equally bear in mind, in speaking of hereditary transmission, that we are dealing with agencies infinitely diversified, subject to elaborate combination and modification quite unpredicable, and that unforeseen forces, not easily suspected and far less readily explained, step in to disturb the mathematical precision we at times hopelessly attempt to impose upon our formulæ of vital processes.

What is it, then, that is transmitted by alcoholic ancestry? I presume it is a defective organisation of the neuron, or a molecular *degradation of nerve-tissue* revealing itself in a loss or weakening of that primary attribute so characteristic of nerve-cell mechanisms—inhibition. Functional instability is, of course, pre-eminently the stamp of the neurotic heritage, but the instability resulting from an alcoholic stock appears above all other forms of instability to be indicated by (*a*) its convulsive

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nature, (*b*) its tendency to limitation (as in so-called systematised forms of insanity), (*c*) its rhythmic periodicity and paroxysmal nature. The latter time-relationships are features especially worthy of note. Epilepsy, chorea, hysteria, the convulsive psychoses (moral and impulsive forms of insanity), and certain systematised delusional states are the first-fruits of an alcoholic heritage: the *motor element of mind*, if I may so express myself, is peculiarly liable to this derangement in *paternal* forms of transmission. On the other hand, arrests of development, as indicated by congenital mental weakness, imbecility, idiocy, appear to me especially the results of *maternal* toxæmia, and largely, if not wholly, due to direct poisoning of the germ or ovum.

Instability is a relative term, for with advancing structural differentiations functional instability *must inevitably* be evolved; whilst with the parallel structural integrations that normal control becomes established over such unstable elements, which is characteristic of healthy life. Ontogeny as well as phylogeny tell us the same tale. But the instability we are concerned with is not the labile equilibrium of healthy functionating nerve-centres, but such morbid divergences which indicate unwonted explosiveness conjoined with integrative failure; the steed is frisky, but the whip-hand is also powerless to control. Functional instability may be preternaturally enfeebled or excessive as the result of molecular constitution, and issue in the one case, as so well indicated by Dr. Mercier, in mental feebleness, imbecility, or idiocy, from "failure of the developmental forces, originating in a primeval weakness of sperm- or germ-cell," and in the other case in an undue explosiveness (Hughlings Jackson's nutrition increased and degraded in quality) which, however, is so frequently associated with defective integration, emphasising, therefore, the morbidly labile state of nervous centres. It is this *latter factor* which, it appears to me, betokens the unstable nature of the sperm-cell, and explains the frequency of convulsive neuroses, epilepsy, chorea, hysteria, and the like affections in paternal inheritance.

To the minute sperm nucleus, therefore, as the bearer of paternal heredity, must we attribute this vicious development, unless we assume the still more minute sperm centrosome, which displaces the egg centrosome and becomes the dynamic

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centre of the germ in its resulting mitosis and cleavage, as having any share in effecting this germinal transmission. This opinion, however, would not be in concert with the usual belief, which could not be better expressed than in the eloquent words of Prof. Wilson: "From the mother comes in the main the cytoplasm of the embryonic body, which is the principal substratum of growth and differentiation. From both parents comes the hereditary basis or chromatin by which these processes are controlled and from which they receive the specific stamp of the race. From the father comes the centrosome to organise the machinery of mitotic division by which the egg splits up into the elements of the tissues, and by which each of these elements receives its quota of the common heritage of chromatin. Huxley hit the mark two score years ago when, in the words that head this chapter, he compared the organism to a web of which the warp is derived from the female and woof from the male. What has since been gained is the knowledge that this web is to be sought in the chromatin substance of the nuclei, and that the centrosome is the weaver at the loom."

"Neurotic heritage" is a big term and embraces complex factors; the instability more likely to result in insanity, or in epilepsy, or in crime, inebriety, chorea, hysteria, etc., may be all more or less existent in the same individual and in varying degrees, as congenital or inborn varieties; but it is reasonable to assume that environmental agencies such as alcohol, by directly acting upon either germ- or sperm-cell, may intensify or in other cases eliminate one or other of these inherited factors. For the germ-cell lives a life like the somatic elements, nourished from the same source, and acts and reacts in response to the same environmental agencies; but this, of course, is not expressing the view that *acquired characters are transmitted*.

(1) H.U. = Heat units.

Discussion,

At the quarterly meeting held on February 23rd, 1906, at the West Riding Asylum, Wakefield.

Dr. Mercier, who was first called upon, confessed that he had had no time to digest the paper in the way it was entitled to be treated, a paper to which so much thought had been given, and which ranged over such a wide variety of subjects. One of the points in it was the question of the sense of muscular discrimination brought about by alcoholic liquor and analogous conditions, especially in general paralysis. Of course, they were very much impressed with the extraordinary

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similarity of the symptoms of general paralysis and the symptoms of alcoholism. "And it seems to me, if it is a fact, as I take it to be from Dr. Bevan Lewis's paper, that the sense of muscular discrimination is much impaired in these conditions, that that will account for a good deal. It will account for the exaggerated estimate that an individual places upon his own capacity. It is clear that if a man has his sense of muscular discrimination so impaired that he cannot discriminate between one weight and a much greater weight, neither will he be able to discriminate between the effort necessary to do one thing and the effort necessary to do a much greater thing—the effort, for instance, necessary to jump over a stool and the effort necessary to jump over a table." They only needed, said Dr. Mercier, to exaggerate that incapacity to find a person who would be unable to discriminate the effort necessary to jump over a house; and, as they knew, a general paralytic would announce his ability to do any one of these things. And not only in crude matters, but in things more elaborate. He thought that the sense of muscular discrimination probably lay at the root of their ability to estimate their powers in every direction. Hence a person whose muscular discrimination was wanting might eventually so exaggerate his capacity as to become possessed of the opinion that he was emperor, or lord, or king, or monarch of the universe. Into the lessons to be learned from the variations caused by alcohol and thermogenesis he was not competent to enter. Dr. Bevan Lewis had waved before them the flag of the non-inheritance of acquired qualities. To him that was like waving a red flag before a bull. It was impossible for him (Dr. Mercier) to decline the challenge, and he wished to declare his unalterable belief that acquired qualities were inherited. It was a most extraordinary thing that they could look upon the whole universe and see organisms varying to a most incredible extent, and in the most varied directions—one adapted, say, for an aquatic medium, one for aerial medium, and another for terrestrial locomotion —and to say that these varied surroundings had nothing to do with the form or shape or function of those organisms. To say that the influence of the environment in organisms could not be transmitted to the offspring seemed to him to be one of the most preposterous doctrines ever advanced, and rested, not on evidence, but on assumption. He could only say that if acquired qualities were not inherited the doctrine was one of pessimism and blank despair. By no means could we hope to leave the world better for our own exertions if our successors were to start from the point, not where we left off but from where we began. He did not believe it.

Dr. Robert Jones could not see why environment was so much left out of consideration in questions of heredity. They had only to look at Alpine plants, grown at high altitudes, which had characteristics of their own. When grown in the plains these special features changed. In just the same way plants of the desert had characteristics decided for them by their environment, and these acquired characteristics came true by the seed, which demonstrated that acquired characteristics can be transmitted. Of course, it is understood that it takes a long time before characters thus acquired through the environment can become fixed, but the ultimate tendency is for them to become fixed, and then they are transmitted. Relating an experience of his own for the purpose of illustration, Dr. Jones showed that some time ago he went to a school in Essex and took the weight, height, and other details of children of various ages attending there, and classified them. Then he examined some children from Dr. Barnardo's Homes, children who came from the worst slums, who suffered from various ailments due to faulty environment, *i.e.*, chilblains, adenoids, rickets, discharges from the ears, and many other complaints. Yet these same children, when medically supervised and treated with a well-considered dietary, life out of doors, physical drill, and school work, after a residence of several years compared most favourably with the best country children. Again, he thought the craving for alcohol was a very definite physical condition, and that such physical conditions, which were inflicted upon people by their environment, were of necessity transmitted, for we found the ancestry of over 40 *per cent.* of dipsomaniacs to give a history of alcoholic excess, showing as definitely as could be ascertained in any circumstances that acquired characters may be transmitted. As to the question of the muscular sense, it was difficult to analyse quite the full effects of alcohol in regard to this particular system, knowing the different effects of alcohol upon

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different structures of the body in different persons. The whole question was one of alcohol attacking the *locus resistentiæ minoris*. In asylums they hardly ever saw a case of cirrhosis of the liver, showing that drunkards, when insane, usually gave way on their nerves and brain side rather than on the side of other organs, and that the nervous system in the insane was the *locus resistentiæ minoris*.

Dr. BEVAN LEWIS, in replying, thanked those who had spoken and those who had listened so attentively to the discussion of a subject which he felt must be a trifle obscure.

Amentia and Dementia: a Clinico-Pathological Study.

By JOSEPH SHAW BOLTON, M.D., M.R.C.P., Fellow of University College, London; Senior Assistant Medical Officer, Lancaster County Asylum, Rainhill.

PART III.—DEMENTIA.

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INTRODUCTION.

THE present and final division of this paper contains a description of those types of mental disease which are classed by the writer under the heading "Dementia." Under this term

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