Genetic variation in the feral horses of the Namib Desert, Namibia

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ABSTRACT

Genetic variation at 7 blood-group and 10 biochemical genetic loci was examined in 30 horses from a feral herd from the Namib Desert of Namibia, Africa. The observed genetic variability was extremely low compared with that found in domestic horse breeds. The low variation was most probably a result of recent small population size and a small founding population size. Genetic comparison of the Namib horses, which were of unknown origins, to domestic horse breeds, showed that the Namib horses had the highest genetic similarity to Arabian type horses, although they did not closely resemble this type of horse in conformation.

Key words: biochemical genetics, blood groups, feral horses genetic distance, genetic variation.

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INTRODUCTION

Feral horse populations frequently live in harsh environments, because they are forced into these areas to prevent competition with grazing livestock such as cattle and sheep. The feral horse population of the Namib Desert perhaps occupies the most inhospitable environment of any group of horses.

The horse population is concentrated around a well (the Garub water hole, the only permanent source of water in the area) about 20 km from the village of Aus in Namibia[22]. Although no written records exist, the horses are known to have been in this area for at least 80 years and appear to have adapted well to these difficult conditions. During favourable years the horses appear to be in good condition and show evidence of being well-bred, quality horses, although they show evidence of stress in dry years.

According to Greyling[10] and Rutherford and Westfall[18], the area where the horses are found varies in topology, geology and climate. The area is characterised by low mountain islands. Elevation ranges from 760 m near Garub to 1100 m in the east. Dry rivers penetrate the area and vast plains covered with sand expanses are common. The surface of the region varies from gravel slopes, desert crust, desert pavement plains and sand dunes. The region is described as a cool desert with average temperature around 18 °C. Rainfall ranges from 0 to 200 mm with an average around 65 mm per year. The area where the horses occur is an ecotone of the Desert biome and the Succulent Karroo biome[18]. The horses compete for grazing with gemsbok (*Oryx gazella*) and springbok (*Antidorcas marsupialis*).

The origins of these horses are not known. One hypothesis is that early immigrants from the Cape Colony moved through this area from the south, bringing their Cape Horses with them. Another is that a cargo steamer ran aground on the Namib coast with a load of Thoroughbreds destined for Australia and that some of these horses reached shore and survived. Another suggestion is that during a World War I campaign in this region, military troops abandoned or lost horses in the desert. Finally, there is speculation that the horses came from the farm of Baron von Wolf, who bred horses for the army up to just after the start of World War I. This farm was situated on the edge of the Namib Desert about 150 km from the Garub water hole. Von Wolf died in action in 1916, and it is possible that horses from his farm found their way to the Garub water hole during the turmoil following the German defeat in colonial South West African[22].

In this study we examine genetic variation in the Namib horses based on 7 blood-group and 10 biochemical genetic loci. Genetic variation in this population is a concern, owing to a probable small founding population size and continuous low population numbers due to the harsh conditions of the environment. Also, genetic comparisons of the Namib horses to other domestic horse breeds were made in hope of shedding some light upon the origins of these populations.

MATERIALS AND METHODS

Blood was collected by jugular venipuncture from a total of 30 Namib Desert horses. Eighteen of these horses were born in the desert, while the remainder was born in captivity as part of a breeding herd. One of the captive-born horses had desert-born parents that were not sampled. This horse was included among the desert-born horses for analysis. The captive herd was formed in 1987 and maintained at the Onderstepoort Veterinary Institute. Some of the samples were from horses captured during a drought in 1992 and now in private ownership in Potchefstroom.

Standard immunological procedures involving haemagglutination and complement-mediated haemolysis[20,21] were used to detect variation of red cell alloantigens at 7 blood-group loci. Starch and polyacrylamide gel electrophoresis and isoelectric focusing were used to detect variation at 10 serum and red blood cell lysate protein loci[4,5,11,12,19,16].

The blood-group loci examined were the *A, C, D, K, P, Q,* and *U* horse blood-group loci and the biochemical protein loci were alpha-1 β-glycoprotein (*A1B*), albumin (*ALB),* serum esterase (*ES),* vitamin D binding protein (*GC),* glucose phosphate isomerase (*GPI),* alpha-haemoglobin (*HBA),* 6-phosphogluconate dehydrogenase (*6-PGD),* phosphoglucomutase *(PGM),* protease inhibitor *(PI)* and transferrin *(TF)* loci. Nomenclature for variants at all 17 loci is in accordance with internationally standardised usage for horses[2,3] except for variants at some loci, which have not yet received international recognition.

All analyses were calculated for the 19 desert-born horses and for the total sample. Gene frequencies for biochemical loci were calculated by direct count. Frequencies of alleles at blood-group

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loci were calculated by the allocation method[1]. Genetic variation was measured as observed heterozygosity *(Ho),* Hardy-Weinberg expected heterozygosity *(He),* unbiased expected heterozygosity *(Hu)*[14], effective number of alleles *(Ae),* and the total number of variants found in each population *(Na). Ho* was calculated for biochemical loci only because of the presence of recessive alleles and/or ambiguous genotypes at blood-group loci. Therefore, for direct comparison, *He* and *Hu* were calculated for biochemical loci only (in an ideal population, *He (Hu)* should equal *Ho*), for blood-group loci, and for all 17 loci. Tests for the genetic effects of a population bottleneck were examined using the programme BOTTLENECK[6]. In addition, population inbreeding level was estimated by Wright's *Fis* =1–*(Ho/He).* Values of genetic variation of the Namib horses were compared to those of domestic horse populations and other feral horse populations that have been tested at the University of Kentucky. Genetic relationship of the Namib herd to these other domestic breeds was investigated using Rogers' genetic similarity coefficient *(S)*[17] and Nei's modified distance *(Da)*[15]. Restricted maximum likelihood analysis (RML)[9] was used to construct the dendogram of Fig. 2.

RESULTS

Allele frequencies for the variants found at the 17 loci are shown in Table 1. One variant observed at the *Pi* locus (*P–q* in Table 1) is, in our experience, unique, as we have not seen it in any other horse breed (approximately 100 domestic horse breeds and 40 feral horse populations). No variants were found in the captive-born horses that were not seen in the desert-born horses. Estimates of genetic variability in the Namib population are shown in Table 2. Also shown in Table 2 are the same variability measures from 9 domestic horse breeds chosen to demonstrate the range of variability in the horse and mean values based upon data from 99 domestic horse breeds. There was statistically significant evidence for a recent population bottleneck in the Namib herd based upon the method of Cornut and Luikart[6], although caution must be used in this interpretation because only 13 variable loci could be analysed and 20 are recommended for this test. The distribution of allele frequencies for all 17 loci is shown in Fig. 1.

Values of *S* and *Da* for the Namib horses compared to a number of domestic breeds are shown in Table 3. The domestic breeds are grouped according to relationships among the breeds and mean *S* and *Da* values for the Namib population compared to each group also are given. The genetic associations of the Namib horses with domestic breeds are summarised in the dendogram of Fig. 2. The tree shown is a majority rule, strict consensus tree from 30 separate RML trees. The breeds that make up the branches other than those for the Oriental breeds' branch is not shown (see Cothran and Van Dyk[8] for a complete tree).

DISCUSSION

Genetic variation in the Namib horse was extremely low compared to other horse populations that have been examined. Of domestic horse populations, only the Bedouin strain of Arabian horses (also known as the Blue Star Arabians) had a lower *Ho* than the Namib horses (Table 2). The Bedouin Arabians were imported from Saudi Arabia into the USA in the middle of this century and have been maintained as a separate breeding population. They were derived from a small number of founders and are relatively inbred. Although *Ho* is lower, this strain of Arabian horses did have greater expected heterozygosity and higher allelic diversity, probably because of a greater population size.

Only 1 population out of 146 other horse populations that have been examined had lower overall heterozygosity and lower allelic diversity than the Namib horses. This was a feral population from the Marble Canyon herd management area of the Cerbat Mountains in northwestern Arizona, USA[7]. At the time of sampling in 1990, the total population size of this herd was estimated to be no more than 21 individuals (US Bureau of Land Management, pers. comm., 1990). Total *He* for this population was 0.128 compared with 0.289 for the Namib sample. This low value was primarily due to almost no variation at blood-group loci *(He* 0.023). *Ho* for the Cerbat herd was greater than that of the Namib at 0.267.

The low genetic variation in the Namib horse population is most likely due to demographic characteristics of the population. There were probably a small number of founders of the population (<100) and the population has been maintained at a low number of individuals due to the harsh environmental conditions. Esti-

Table 1: **Allele frequencies for 17 genetic loci tested in the Namib Desert feral horse herd. HBG stands for Horse Blood Group. Only alleles found in this herd are shown.**

Locus	Allele	Namib Desert herd
Trf	D	0.033
	F2	0.950
	O	0.017
A1B	K	1.000
Est	I	1.000
Al	A	0.900
	B	0.100
Gc	F	0.500
	S	0.500
PGD	F	1.000
PGM	F	0.700
	S	0.300
GPI	F	0.083
	I	0.917
Hb	BI	0.183
	BII	0.817
Pi	L	0.083
	S	0.667
	T	0.050
	q	0.200
EAA	adf	0.933
	b	0.067
EAC	a	0.815
	–	0.185
EAD	dk	0.517
	deo	0.033
	bcm	0.350
	cgm	0.100
EAK	–	1.000
EAP	ac	0.034
	ad	0.428
	–	0.538
EAQ	abc	0.368
	c	0.225
	–	0.407
EAU	a	0.293
	–	0.707

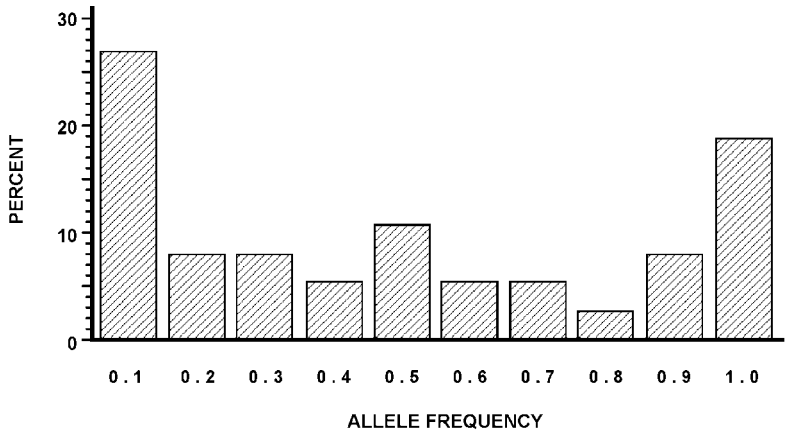

Fig. 1: **Distribution of allele frequencies within the Namib Desert feral horse herd.**

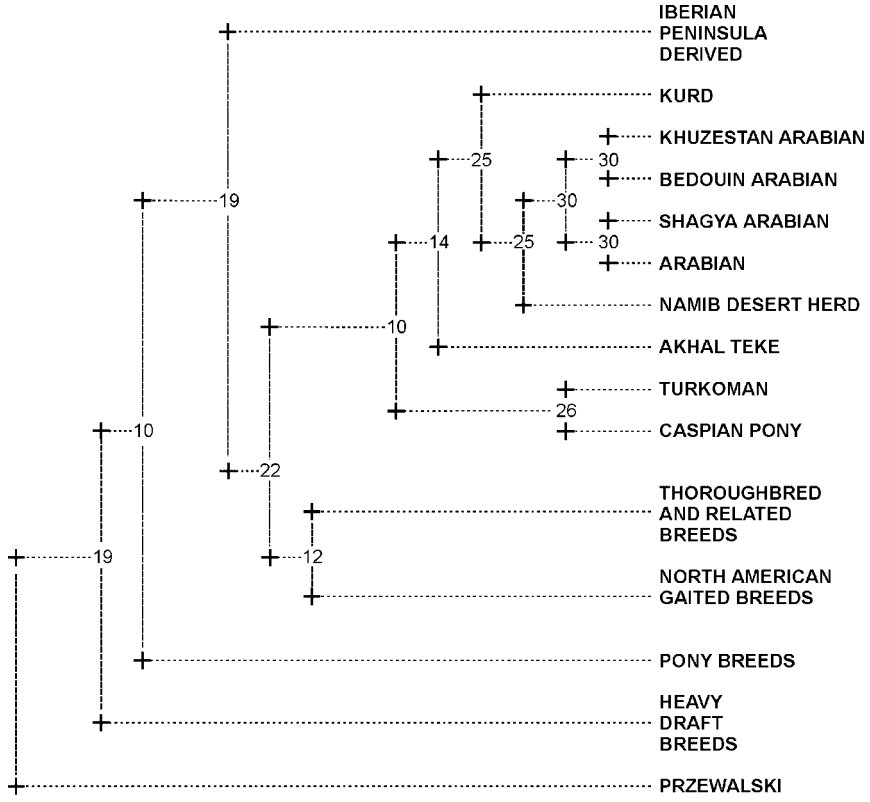

Fig. 2: **Restricted maximum likelihood consensus tree of the association of the Namib Desert feral horse population with domestic horse breeds.**

mates of population size in recent years range from 300 to 65[22]. Also, there have probably been periodic bottlenecks to the herd over the time it has been in existence due to such factors as drought. All of these conditions would be expected to reduce genetic variability through genetic drift and inbreeding. Evidence for a bottleneck in the population size of the Namib herd (the bottleneck could have been the founding population) was observed in the distribution of allele frequencies[13]. It was demonstrated that severe population bottlenecks distort the distribution of allele frequencies within a population from those expected by reducing the number of rare alleles at high frequency[13]. Figure 1 clearly shows that the Namib herd fits that pattern. This is not a surprising result as the population has always been small, but assuming that the founders came from a much larger population of a domestic breed, it shows that there has been a serious decline in variability over a relatively short period.

Genetic similarity and distance show that the Namib horses were not closely related to any domestic horse breed or group of breeds (Table 3). The greatest *S* was with the Khuzestan Arabian (Persian Arabian) from Iran and the lowest *Da* was with the Arabian (from the USA). Closest mean genetic resemblance was to the Arabian type of horse breeds. The *S* and *Da* mean values of the Namib herd to the Arabian type breeds were statistically closer than to any of the other breed groups. The Namib horses also showed no close relationship to the 3 South African horse breeds examined (the Nooitgedacht, Boerperd and Basuto Pony)[8]. The low level of resemblance of the Namib horses to the other horse breeds is to some extent due to the low level of genetic variation within the Namib population. Changes in the genetic composition of the herd due to genetic drift or selection also could have played a part.

The RML tree in Fig. 2 confirmed the results of Table 3. The Namib horses fit within the Oriental horse cluster at the base of the branch with the Arabian horse breeds. The low genetic variability of the Namib herd also corresponds with the relatively low variability found in all Arab breeds in Fig. 2 . Thus, the genetic evidence was that the Namib horses were most like Arabian type horses. This would refute the hypothesis that they were originally from the Von Wolf stud at Duwisib, because, according to written evidence of his breeding programme, Von Wolf did not use Arabians but rather favoured Thoroughbreds, Hackneys and Trakehners, believing that these breeds could bring the desired size to the remounts he was trying to breed. Although Van der Merwe[22] favoured the Von Wolf hypothesis for the origin of the Namib horses when he first observed them in 1984, his objective description of their conformation as 'of the hot blood type *i.e.* athletic, lean muscled, straight and clean limbed, with exceptional bone quality with long sloping shoulders and showing well-bred characteristics in the head, skin and coat' could fit the Arabian type as well

Table 2: **Estimates of genetic variability for the Namib Desert horse and selected other domestic horse breeds. The means of the genetic variation measures were based on data from 99 domestic horse populations. *Ho* was calculated from the 10 biochemical loci only, *He* also was for the biochemical loci only as was the unbiased estimator of *He* (*Hu*), *Heb* was from the 7 blood-group loci, while *Tt1He, NA* and *Ae* were from all 17 loci.**

Breed	*n*	*Ho*	*He*	*Hu*	*Fis*	*Heb*	*Tt1He*	*Na*	*Ae*
Namib Desert-bred	19	0.258	0.219	0.225	–0.175	0.388	0.289	37	1.577
Namib Desert horse	30	0.227	0.215	0.219	–0.053	0.412	0.298	37	1.691
American Saddlebred	259	0.404	0.409	0.410	0.013	0.470	0.435	96	2.625
Arabian	117	0.307	0.327	0.328	0.061	0.448	0.376	67	2.132
Bedouin Arabian	213	0.209	0.224	0.225	0.066	0.418	0.304	52	1.890
Chilean Criollo	173	0.375	0.383	0.384	0.022	0.564	0.457	86	2.919
Friesian	314	0.307	0.306	0.307	–0.003	0.407	0.348	54	1.901
Peruvian Paso	141	0.451	0.445	0.446	–0.014	0.503	0.469	77	2.761
Quarter Horse	168	0.396	0.393	0.394	–0.007	0.508	0.440	87	2.653
Standardbred Pacer	341	0.401	0.395	0.395	–0.016	0.441	0.414	85	2.142
Thoroughbred	265	0.294	0.288	0.289	–0.019	0.376	0.325	64	2.009
Domestic horse mean	99	0.377	0.364	0.371	–0.034	0.486	0.413	64.7	2.393
Standard deviation		0.051	0.044	0.045	0.066	0.054	0.039	11.2	0.250

Table 3: **Rogers' genetic similarity and Nei's modified distance for the Namib Desert horse herd compared to other domestic horse breeds and mean values for groups of domestic horse breeds. Standard errors of the mean in brackets.**

Saddle and harness light horses												
	TB*	BH	PI	SF	QH	HH	HN	HO	TK	SL		Mean
Rogers	0.706	0.725	0.738	0.693	0.720	0.738	0.704	0.686	0.732	0.715		0.715 (0.018)
Nei's *Da*	0.170	0.144	0.150	0.169	0.151	0.135	0.149	0.182	0.138	0.148		0.153 (0.015)
Gaited North American breeds												
	PA	TR	MH	TW	FT	MP	RM	SB				Mean
Rogers	0.712	0.716	0.727	0.701	0.732	0.714	0.710	0.713				0.716 (0.010)
Nei's *Da*	0.146	0.155	0.142	0.168	0.142	0.155	0.157	0.171				0.154 (0.011)
Arabian-type breeds												
	AR	BS	SA	YA	TU	AT	KU	CS	KA	AM	MB	Mean
Rogers	0.759	0.739	0.746	0.719	0.712	0.761	0.722	0.706	0.762	0.740	0.702	0.733 (0.024)
Nei's *Da*	0.107	0.141	0.130	0.151	0.158	0.123	0.132	0.149	0.117	0.133	0.152	0.133 (0.017)
Heavy draft horses												
	CD	BE	BR	PC	SH	SU	CL	HF	NK	PH	PV	Mean
Rogers	0.674	0.706	0.643	0.720	0.631	0.672	0.623	0.725	0.657	0.662	0.697	0.674 (0.035)
Nei's *Da*	0.183	0.171	0.195	0.150	0.211	0.203	0.227	0.165	0.226	0.188	0.161	0.189 (0.026)
Pony breeds												
	WP	HP	SP	MN	GT	DT	DL	NF	HL	IC	EX	Mean
Rogers	0.686	0.670	0.630	0.651	0.650	0.665	0.678	0.683	0.681	0.581	0.626	0.654 (0.032)
Nei's *Da*	0.146	0.199	0.238	0.200	0.204	0.209	0.188	0.192	0.177	0.258	0.219	0.203 (0.030)
Iberian Peninsula-derived breeds												
	AN	LU	PP	CP	CC	RP	PF	GR	MM	MA	BZ	Mean
Rogers	0.695	0.675	0.684	0.690	0.683	0.688	0.685	0.691	0.707	0.695	0.651	0.683 (0.015)
Nei's *Da*	0.166	0.178	0.172	0.164	0.168	0.159	0.154	0.150	0.158	0.171	0.185	0.167 (0.101)
Other breeds												
	LI	SO	PO	KR	FR	CB	NO	BP	BS			
Rogers	0.650	0.625	0.695	0.641	0.647	0.654	0.690	0.722	0.700			
Nei's *Da*	0.211	0.265	0.171	0.215	0.252	0.199	0.176	0.164	0.157			

*Key to abbreviations.
TB, Thoroughbred; BH, Belgian Halfblood; PI, Wielkopolska; SF, Sella Français; QH, Quarter Horse; HH, Hackney Horse; HN, Hanovarian; HO, Holstein; TK, Trakehner; SL, Silesian; PA, Standardbred Pacer; TR, Standardbred Trotter; MH, Morgan horse; TW, Tennessee Walking Horse; FT, Missouri Fox Trotter; MP, Mountain Pleasure; RM, Rocky Mountain Horse; SB, American Saddlebred; AR, Arabian; BS, Bedouin Arabian; SA, Shagya Arabian; YA, Yabou; TU, Turkoman; AT, Akhal Teke; KU, Kurd; CS, Caspian Pony; KA, Khuzestan Arabian; AM, Moroccan Arab; MB, Moroccan Barb; CD, American Cream Draft; BE, Belgian Draft; BR, Breton; PC, Percheron; SH, Shire; SU, Suffolk Punch; CL, Clydesale; HF, Haflinger; NK, Noriker; PH, Polish Heavy Horse; PV, Posavina; WP, Welsh Pony; HP, Hackney Pony; SP, Shetland Pony; MN, Miniature Horse; GT, Gotland Horse; DT, Dartmoor Pony; DL, Dales Pony; NF, Norwegian Fjord; HL, Highland Pony; IC, Icelandic Horse; EX, Exmoor Pony; AN, Andalusian; LU, Lusitano; PP, Peruvian Paso; CP, Campolina; CC, Chilean Criollo; RP, Puerto Rican Paso Fino; PF, American Paso Fino; GR, Garrano; MM, Mangalarga Marchador; MA, Mangalarga; BZ, Brazilian Criollo; LI, Lipizzaner; SO, Sorraia; PO, Polish Primitive Horse; KR, Kladruby; FR, Friesian; CB, Cleveland Bay; NO, Nooitgedacht; BP, Boerperd; BS, Basuto Pony.

as other phenotypes like the abovementioned breeds, which are at least partially derived from oriental stock. A subsequent search in the Windhoek archives revealed that quite a few other private breeders in pre-World War I German South Africa imported and bred not only Arabian, but also Shagya Arabians (from Hungary) and that offspring of these were used by the army and police. It therefore is possible for the Namib horses to be descendents of Arabian or Shagya Arabians. However, in the absence of direct historical evidence, it must be conceded that the closest resemblance to the Arabians could have been, at least partially, a statistical artifact of the low genetic variability. Although the Namib herd showed the closest affinity to the Arabian type horses, the actual *S* and *Da* values were not high. Regardless of its origins, the current Namib desert feral horse population has extremely low genetic variation, most likely due to stochastic effects associated with small population size. It remains to be seen whether the low variability will have a negative effect on the viability of the population.

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Book review — Boekresensie

A guide to canine and feline orthopaedic surgery (4th edition)

H R Denny and S J Butterworth

2000. Blackwell Science, Oxford, 634 pp., hardcover. £55.00. ISBN 0 632 05103 5.

The first edition of this book was published in 1980 and comprised 184 pages. Subsequent editions have been expanded over the years, and the 4th edition now comprises 634 pages. The consistently stated aim of all 4 editions was to provide students and practitioners with a 'rapid reference guide' to small animal orthopaedic surgery, including the recent advances in the field. In this edition, the authors also suggest that the book 'should allow veterinary practitioners to diagnose and treat most orthopaedic and spinal problems encountered in general practice', and provide a 'sound basis' for postgraduate students. It is with these aims in mind that this book was reviewed.

The first, general section comprises chapters on topics germane to the later chapters (for example, fracture healing, bone grafts and osteochondrosis). The following 2 sections cover joint disease and fracture management, and provide the background information and principles required to diagnose and treat specific conditions. The 3 sections that follow deal with these conditions by region, namely, the skull and spine, the fore- and the hind limbs. The last section covers a variety of miscellaneous conditions, including bone neoplasia, myopathies and non-nutritional bone diseases.

In general, the text is clear and concise, well-indexed and cross-referenced. The content, although often appropriate to the book's stated aims, often lacks balance. For example, the section on total hip replacement (a technically difficult procedure, generally not the province of an average (private) practitioner) is 6 pages long and quite detailed. On the other hand, there are important topics that are dealt with rather superficially. Examples include the pathogenesis of osteochondrosis and osteoarthritis, the initial evaluation/assessment of the fracture patient, the anatomy and assessment of the peripheral nervous system pertaining to the limbs, the biomechanics of spinal fractures, the description of sacral fractures, and the technique and limitations of cerclage wire. There is also scant information regarding the post-operative management and support of patients, especially regarding the important issues of analgesia, antimicrobial therapy and physical therapy.

In the chapter on osteochondrosis, the authors intimate that ununited medial epicondyle of the humerus and fragmentation of the caudal glenoid rim are 'common or well-recognised' manifestations of osteochondrosis. The former is certainly not common in South Africa, and attributing the development of both of these conditions to osteochondrosis is rather controversial. The authors offer no information or recommendations regarding screening tests and breeding practices to reduce the incidence of this condition, for example those provided by the International Elbow Working Group, and now well established in Europe.

The 3 regional sections are generally well written, succinct and accurate, and provide the reader with the necessary theoretical information to understand the conditions mentioned. The authors show clear preference for certain procedures, which, given their accumulated experience, will be of great value to practitioners. The authors also provide useful hints, often omitted from standard surgical texts, which may significantly simplify a procedure. Many of the chapters start with a list of differential diagnoses or conditions that provide a reference system for easy access to the rest of the chapter. I believe that these should have been page-referenced as an additional aid for the reader to negotiate the text.

The chapter on the skull is particularly disappointing. There is no information on the biomechanics of mandibular fractures, especially related to their relative stability. These principles, as well as the relevant anatomy of the region, could easily have been illustrated. There is also no information on the differences in the nature of fractures in the dog and cat. There is little advice offered on decision-making, for example, the role of teeth within the fracture line or the most appropriate treatment for various fracture patterns. Some of the most commonly-performed techniques, for example interfragmentary wiring and tape muzzling, are poorly or inadequately described and illustrated. Some of the described techniques are generally recognised as poor, or at the least controversial. These include transramal (transmandibular) pinning and lag screw techniques, and the extraction of teeth to correct malocclusion. There is no mention of specific post-operative complications associated with fracture repair in this region and scant information on the often-difficult after-care of these patients.

There are a number errors in the terminology that detract from the scientific accuracy of an otherwise well-written text. These include 'Vaulkmann' canal (Volkmann's canal), 'Howship' lacuna (Howship's lacuna), 'Mitchel' trephine (Michelle trephine), 'conjugal' ligament (intercapital ligament) and 'horizontal and vertical ramus' (body/corpus and ramus) of the mandible. There are also a few factually incorrect statements, for example that dermoid 'cyst' (sinus) is an 'infolding of skin'.

My greatest reservation regarding this book is, however, related to the quality of the illustrations and photographs and the often inadequate or confusing annotations. Unfortunately the majority of these are poor; some are so poor that they cannot be interpreted at all (Figs 13.2, 13.21, 15.5, 19.4, 22.3, 24.8), while others are confusing/misleading (Figs 1.3, 9.1, 26.6, 28.1). Some illustrations are drawn so amateurishly that they are almost cartoon-like in character (Figs 16.7, 19.2). With the availability of first-class illustrators and computer-generated graphics, this deficiency is unacceptable. The success of surgical textbooks hinges on the quality of their illustrations, as the surgery 'message' is usually a visual one. If one considers the number of surgical textbooks with truly excellent illustrations, this book measures up very poorly.

The book is well referenced, although there are a number of references from the 1960's and 1970's that could probably have been replaced by more up to date work. It is unfortunate that the authors used few review articles as references in the suggested reading sections, as these are particularly useful to the busy practitioner.

In conclusion, *A guide to canine and feline orthopaedic surgery* sets out to cover a massive field in veterinary practice and I believe that it is partially successful. I do not, however, believe that it has convincingly accomplished its aim of providing practitioners with the information to 'treat most orthopaedic and spinal problems', as the illustrations and descriptions of surgical approaches are often inadequate. Most practitioners would require additional texts on anatomy and surgical approaches. If I were a practitioner in the market for a surgical guide, this would not be my first choice.

N E Lambrechts
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